The Evolution of Personality and Individual Differences

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Introduction

He who does not understand the uniqueness of individuals is unable to understand the working of natural selection.
(Mayr, 1982, p. 46)

Individual differences are indispensable for natural selection. Without heritable variants, natural selection—the only known process capable of creating and maintaining functional adaptations—could not occur. This truism is so central to evolutionary biological thinking that life scientists take it for granted. Selection is typically seen as a homogenizing force. Favored variants tend to spread throughout populations, leading to species-level characteristics that we all share—human universals in the case of our species.

The present volume crystallizes a counterpoint to this species-typical view, and captures a scientific change in thinking about individual differences that has been building over the past 15 years. Rather than viewing variability as merely the raw material upon which selection operates, the contributing authors provide theories suggesting and empirical evidence supporting the view that personality and individual differences are central to evolved psychological mechanisms and behavioral functioning.

This claim may be controversial among some. Some scientists historically have viewed individual differences as noise or error variance to be controlled for or eliminated by careful experimental design. Among non-scientists, some believe that something as intimate as ‘personality’ is uniquely human, inherently mysterious, unexplainable, or even Godly. Indeed, the *Oxford English Dictionary* defines personality as “the quality which makes a human being.” Nonetheless, there are compelling reasons to believe that personality and individual differences are both created and maintained by selective forces, not simply eliminated in the selective drive toward species-typical characteristics. First, individual differences, such as those captured by the five-factor model of personality and the six-factor HEXACO model, show stability over time, situations, and cultures (e.g., Ashton & Lee, 2001; McCrae & Costa, 2008; Saucier, 2009). Second, careful cross-species comparisons have revealed important continuities among humans and non-animals in the architecture of personality (Gosling, 2001). Third, measures of these personality traits show impressive predictive power in forecasting objectively-measured manifest behavior (Fleeson & Gallagher, 2009). Fourth, dozens of behavioral genetic studies converge on the finding that these traits show moderate heritability, which opens the door for an important role for evolutionary genetics (Plomin, DeFries, McClearn, & McGuffin, 2008; Penke et al., 2007). Fifth, and perhaps most important, these stable individual differences have been shown to have weighty consequences for evolutionarily-relevant components of fitness, such as survival, mating success, status ascension, offspring production, and parenting (Buss & Greiling, 1999; Nettle, 2006; Ozer & Benet-Martinez, 2006). For all these reasons, understanding personality and individual differences within an evolutionary framework becomes a necessary, not an optional, component for a mature science of evolutionary psychology.

Social relationships and more transient interactions are important features of the adaptive landscape within which humans evolved, and within which evolved mechanisms must operate. Thus, whatever their origins, the personality characteristics of others in each person’s social
milieu also play a key role in our solving adaptive problems. When selecting a mate, friend, or an alliance partner, for example, species typical characteristics (e.g., bipedalism, upright gait) recede to the background. Instead, humans hone in on how people differ in qualities such as intelligence, dependability, ambitiousness, and aggressiveness. Because individual differences in others with whom we associate carry significant consequences for outcomes historically linked with reproductive success, it is reasonable to hypothesize that humans have evolved adaptations dedicated to tracking and acting upon these individual differences, that is, “difference-detecting adaptations” (Buss, 1991, 1996, this volume).

At the same time, stable individual differences also create adaptive problems. If personality traits afford an increment in predicting who will defer in competition, cheat in social exchange, free-ride in coalitions, or employ cost-inflicting strategies to get ahead in the hierarchy, then adaptations designed to assess these traits can offer an advantage in anticipatory problem solving. Personality traits of others can also interfere with the solution to existing adaptive problems, as when an impulsive or emotionally unstable man interferes with the success of a carefully planned coalitional raid.

Taken together, all these considerations—the stability of traits over time and cultures, their continuity with non-human animals, their predictive power in forecasting behavior, their moderate heritability, their implications for the components of fitness, and their role in creating and solving adaptive problems—render personality traits and other stable individual differences prime candidates for evolutionary psychological analysis.

Despite their apparent importance, personality and other stable individual differences surprisingly have been neglected by the field of evolutionary psychology, with some notable exceptions (e.g., Buss, 1984, 1991; Buss & Greiling, 1999; Figueredo et al., 2005; Hawley, 1999; MacDonald, 1995; Nettle, 2006; Wilson, 1994; Wilson, Near, & Miller, 1996). Instead, evolutionary psychology has been most successful in providing theories about, and evidence for, human universals such as adaptations for survival, sexual strategies, parenting, cooperation, kinship, and aggression (e.g., Buss, 2005; Crawford & Krebs, 2008). These theoretical and empirical successes, however, have been achieved at the level of illuminating species-typical (characteristic of most or all humans) or sex-differentiated adaptations. Individual differences within each sex—profound and integral to all human functioning—have been almost entirely ignored.

There are important reasons for this relative neglect, starting with a paucity of powerful theoretical frameworks that can account for the evolution of personality. Cogent theories exist for predicting and explaining sex differences (e.g., the theory of sexual selection; Buss, 1995; Geary, 1998) and species-typical adaptations (e.g., kin selection theory, the theory of reciprocal altruism). Evolutionary psychologists have successfully synthesized these models with principles of modern psychology to create unique theories such as social contract theory (Cosmides & Tooby, 2005), theories of morality (e.g., Krebs, 2005), theories of human mating strategies (Buss & Schmitt, 1993), theories of social conflict (e.g., Kenrick, Sundie, & Kurzban, 2008), and theories of error management (Haselton & Buss, 2000), which, in turn, have led to important empirical discoveries (e.g., Haselton, Nettle, & Andrews, 2005). In sharp contrast, comparably powerful theories that predict and explain personality and individual differences have largely eluded evolutionary psychologists.

Why have these theories proven so elusive? One reason can be traced at least in part to a foundational assumption in evolutionary biology—that natural selection tends to reduce or
eliminate heritable individual differences. Traits that are advantageous tend to spread over time to fixation in a population and become species-typical. Consequently, some of the key founders of evolutionary psychology have tended to focus on human universals. Accordingly, they have argued that heritable individual differences in personality are best viewed either as random genetic differences that are part of a defense against parasites, or as superficial “noise” that is irrelevant to the basic functioning of the psychological machinery, much like differences in the colors of the wires of a car engine do not affect its basic functioning (Tooby & Cosmides, 1990). This assumption seemed reasonable 20 years ago, but has been seriously challenged by key theoretical developments in evolutionary biology with input from developmental psychology (see, e.g., Houle, 1998; Penke, et al., 2007).

Some of our contributors have roots in developmental psychology, a domain which has long been infused with evolutionary biological thinking (e.g., Baldwin, 1902; Bowlby, 1969; Piaget, 1978) and committed to understanding the development of individual differences. These authors have invoked well substantiated developmental processes in their theoretical treatments of the individual differences (Harris, 1995; Sulloway, 1996; Hawley, 1999). All contend, for example, that competition in various species-typical contexts (peer groups, family) are primary shapers of personality as individuals strive to occupy niches where benefits can be secured or costs minimized.

Another theoretical perspective for explaining the evolution of personality characteristics has been adopted from evolutionary developmental biology—life-history theory (e.g., Charnov, 1993; Roff, 1992). Life history theory is a broad formulation that construes development as a sequence of resource allocation decisions to various components of fitness (e.g., growth, reproduction, parenting) that can shift in response to environmental conditions (Kaplan & Gangestad, 2005; Wolf et al., 2007).

A third source of theoretical inspiration is derived from advances in evolutionary genetics. Evolutionary genetics deals with mathematical models of the impacts of the major classes of evolutionary change—mutation, selection, drift, and migration—on the genetic structure underlying traits within populations (Maynard Smith, 1998; Roff, 1997; Penke et al., 2007). One of the most important evolutionary genetic models is frequency-dependent selection, where the fitness of one phenotype (morphological or behavioral) depends on its frequency in the population relative to other phenotypes (Maynard Smith, 1982). For example, as the frequency of cheaters relative to cooperators increases, the average success of a cheating strategy declines because there are fewer cooperators to cheat on. Behavioral strategies characterized by psychopathy (marked by cheating, exploitation, and deception), for example, have been proposed to be maintained via frequency-dependent selection; they are effective as long as they are rare (Mealey, 1995). Frequency-dependent selection is a form of balancing selection, which is one class of evolutionary forces that some authors of this volume believe can explain many forms of personality and individual differences (e.g., Penke, this volume). Other authors argue that mutation-selection is a major contender for explaining individual differences ranging from
psychological disorders to variation in overall intelligence (Keller & Miller, 2006; Keller et al., this volume).

Only recently have scientists from disparate disciplines—animal biologists, developmentalists, behavioral ecologists, evolutionary psychologists, evolutionary geneticists, personality psychologists, and molecular geneticists—converged in realizing the need to understand and explain individual differences from an evolutionary perspective. Important articles have recently been published in leading journals such as Behavioral and Brain Sciences, Quarterly Review of Biology, and Nature, introducing these new developments (e.g., Keller & Miller, 2006; Miller, 2007; Penke et al., 2007; Wolf et al., 2007).

The goal of the current volume is to showcase the most important scientific breakthroughs in understanding the evolution of individual differences, and to herald a sea-change in the interdisciplinary science of human nature. The first section, Personality and the Adaptive Landscape, introduces two complementary perspectives from which stable individual differences can be examined. Daniel Nettle provides a brief overview of some of the key evolutionary forces responsible for the creation of stable individual differences. He then provides his theory of the origins and functions of the five major factors of personality that many believe represent the most well-established global personality traits. David Buss flips perspectives, and examines the evolutionary importance of personality traits of the self and of other people residing in each person’s social landscape, regardless of their evolutionary origins. He explores the hypothesis that humans have evolved difference-detecting adaptations to attend to, and act upon, those individual differences because they are central to creating, interfering with, and solving social adaptive problems.

The second section, Developmental and Life History Perspectives on Personality, focuses on personality through the lens of ontogeny and life history theory. Patricia Hawley introduces the key theoretical constructs of phenotypic plasticity and life history theory, and then provides an illustration of these concepts as they relate to social dominance and Machiavellianism, with a special focus on competition and strategic differentiation in the peer group. Frank Sulloway explores the developmental process of strategic niche specialization further, but within the context of family dynamics, specifically inter-sibling competition. Judith Rich Harris advances a modular theory of personality and social development, with a focus on the relationship system, the socialization system, and the status system. Marco del Giudice and Jay Belsky provide a multi-stage theory of developmental switch points within a life history theoretical orientation, each with a phase linked to specific evolutionary functions. Bruce Ellis also adopts a modular perspective, and advances a theory of the development of individual differences in reproductive strategies by drawing on West-Eberhard’s model of switch-controlled systems. The final chapter in this section, by Aurelio José Figueredo, Pedro Wolf, Paul Gladden, Sally Olderbak, Dok Andrzejczak, and W. Jake Jacobs, proposes that quantitative theoretical ecology provides the best explanation of individual differences. These authors argue that no single evolutionary force can explain individual differences. Their theory integrates frequency-dependent niche-splitting, developmental plasticity, genetic diversification, directional social selection, and behavioral flexibility. Taken together, the chapters in this section provide a rich array of theories about developmental and life-history perspectives on personality, and outline a cogent empirical agenda for the next decade.
The third section of the book focuses on the *Evolutionary Genetics of Personality*. Lars Penke provides an overview of the major evolutionary forces that can produce and maintain heritable individual differences. These include recent selective sweeps, balancing selection, and mutation-selection balance. He provides an integrated model using life history theory that focuses on variation in environmental stability in selecting for adaptations ranging from adaptive conditional adjustments at one end (high environmental instability) through universal and sexually dimorphic adaptations at the other end (low environmental instability). Matt Keller, Daniel Howrigan, and Matthew Simonson review the major proximate and ultimate sources of genetic variation. They then outline the currently-available methods that can be used to examine these major sources, with an emphasis on molecular genetic methods, such as assays of mutation load and whole-genome association studies. They conclude by arguing for the necessity of “consilience” between evolutionary psychology and molecular genetics. Nancy Segal picks up on the theme of consilience and highlights the importance of the more traditional methods of behavioral genetics such as the classic twin method and adoption studies. She then illustrates how these methods can illuminate important issues in the evolution of individual differences, highlighting substantive topics such as individual differences in social closeness, age at menarche, and fluctuating asymmetry.

Steven Gangestad explores two major scenarios of the rich and developing conceptual ideas about the evolutionary forces that are potentially responsible for explaining genetic variation in personality—mutation as an opposing force and variability in selection. Gangestad maintains that there is currently no compelling empirical evidence supporting the causal primacy of one evolutionary force over another—alone or in combination—responsible for genetic variation in personality. Accordingly, he urges scientists to consider the importance of novel empirical tests, and provides an illustration using molecular genetic methods. Geoffrey Miller proposes that the field must take seriously the notion that evolution has accelerated during the Holocene. This rapid recent evolution, he suggests, might call into question three foundational assumptions of evolutionary psychology—(1) that the most important environment of evolutionary adaptedness (EEA) for explaining individual differences resides in the Pleistocene; (2) that evolutionary equilibrium models such as balancing selection are the most relevant theories for explaining heritable variation in personality, cognitive, and mental health traits; and (3) the degree to which psychic unity or species-typicality characterizes human psychology.

Denis Réale and Niels Dingemanse argue for the application of the ‘phenotypic selection approach’ for understanding the evolution of personality traits in humans and non-human animals alike. In addition to heterogeneous selection and life-history trade-offs, forces emphasized by many authors of this volume, Réale and Dingemanse emphasize sexually antagonistic selection (within-species arms races between males and females due to conflicting fitness interests) and correlational selection (when individuals with specific combinations of traits have an advantage) as plausible candidates. They then proceed to provide empirical evidence for selection of personality traits in the wild in non-human animal species, and finally focus on evidence within human populations. They conclude by highlighting the complementarity of traditional adaptationist approaches that emphasize historical selection and current selection for genetic variation.

The section concludes with a chapter by Andrea Camperio Ciani, who provides empirical evidence bearing on three competing evolutionary genetic scenarios—neutral selection, mutation
selection, and balancing selection. Using an innovative research design involving personality assays of inhabitants of three mainland villages and a host of nearby islands, he concludes that the findings support only the balancing selection explanation, while refuting (or finding no support for) the neutral and mutation selection scenarios.

The volume concludes with a chapter on evolutionary psychopathology by Leif Edward Ottesen Kennair. He argues for an expansion of the field of evolutionary psychology to include not just personality traits within the normal range, but also the evolutionary analysis of psychological disorders. Adopting the evolution-based harmful dysfunction definition of psychological disorders advanced by Jerome Wakefield (2005), Kennair provides a meta-theoretical framework that includes an individual’s genetic potential, developmental adaptations, and environmental influences. He highlights classes of markers for psychopathology, such as the failure of an evolved psychological mechanism to operate as it was designed to operate, and ancestral adaptations that are mismatched with the modern environment.

It is clear that many authors within this volume share some common conceptual tools, such as life history theory, various theories of balancing selection and mutation-selection balance, and differing forms of developmental or phenotypic plasticity. At the same time, the authors also disagree about which of the conceptual tools, or which combination of tools, they view as most important for explaining the evolution of personality and individual differences. All authors stress the need for novel empirical tests of the competing models.

We see the current volume as heralding a sea-change in thinking within evolutionary psychology, and indeed within the broader field of psychology as a whole, notably developmental, personality, social, cognitive, and clinical psychology. No longer can the field of psychology remain focused primarily on species-typical and sex-differentiated mechanisms, despite the successes it has achieved in these domains. The cumulative evidence for the importance of the evolution of individual differences is now too overwhelming to ignore. Given the key theoretical developments and the dramatically increasing profusion of scientists focusing on this topic, we envision that the next decade will witness rapid progress in understanding the evolution of personality and individual differences. We hope that this volume, by taking stock of the current status of the field—its conceptual tools, the current empirical evidence, and areas of future theoretical and empirical growth—will hasten this scientific paradigm shift.

References


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The Evolution of Personality and Individual Differences
Part I Personality and the Social Adaptive Landscape
1 Evolutionary Perspectives on the Five-Factor Model of Personality

Daniel Nettle

Readers of this volume will hardly need reminding that the evolutionary perspective is a powerful and growing theoretical influence in psychology, nor of the fact that individual differences is one of the areas of psychology where the incorporation of Darwinian thinking has moved relatively slowly. This chapter discusses how we might affect the marriage—or at least the courtship—of Darwinian theory with an existing empirical framework, namely the five-factor model (FFM: Costa & McCrae, 1992; Digman, 1990; Goldberg, 1990), in personality research.

Since the FFM is a trait model of how individuals differ, in Section 2 I discuss how inter-individual variation is explained within evolutionary biology in general. I conclude that the existence of intra-population phenotypic variation is often (though not always) evidence of a fluctuating selective regime, where certain phenotypic values are sometimes favored and sometimes disfavored. I also argue the pattern of covariation of individual traits gives us clues about how behaviors are synergistically related to each other or relevant to common situations. In Section 3, I use these principles to consider how variation in each of the big five might be understood from the evolutionary perspective. Section 4 briefly discusses what Darwinian thinking suggests in terms of new avenues for empirical work in personality psychology. Personality research is an empirical, often inductive and applied, endeavor. Thus many of its practitioners will only engage with Darwinian thinking to the extent that the latter provides new empirical insights. A task for a keen Darwinizer such as myself is thus making a convincing case that the evolutionary perspective adds value where it matters.

Inter-Individual Variation in Evolutionary Biology

In this section, I briefly review the way that inter-individual variation in behavior is conceptualized within evolutionary theory. The reason for brevity is that these issues are discussed elsewhere in this volume, and in the literature (Buss & Greiling, 1999; Nettle, 2006a; Penke, Denissen, & Miller, 2007), and my objective is to move promptly from general considerations to the particular case of the FFM.

A brief history of evolutionary psychology would attest to a strong initial focus on situation-typical behavior and species-typical mechanisms, with attention to inter-individual variation entering the scene somewhat later, led by pioneers such as Buss (1991), Buss & Greiling (1999), and MacDonald (1995). This pattern is not unique to evolutionary psychology, but in fact characterizes behavioral biology more generally. The classical period of behavioral ecology was mainly concerned with situation-specific optima for solving problems such as foraging, with inter-individual differences appearing as little more than noise in the data (see for example Krebs & Davies, 1993). Only more recently has much attention been paid to the reasons why stable inter-individual variation might be found (Dall, Houston, & McNamara, 2004; Dingemanse, Both, Drent, & Tinbergen, 2004; Sih, Bell, & Johnson, 2004; Wolff, van Doorn, Leimar, & Weissing, 2007).

Inter-individual variation has forced itself onto the attention of theoreticians for several reasons. First, the extent of phenotypic polymorphism in wild populations has turned out to be rather large. Much of this polymorphism is developmentally induced. That is, early-life environmental
inputs cue life-long, stable changes in the individual’s phenotype (Bateson et al., 2004). For example, grasshoppers raised on dark backgrounds become black instead of green (Burtt, 1951); freshwater snails grow into different shapes depending on whether the chemical signature of predatory fish or of crustaceans is present in the water in which they develop (DeWitt, Robinson, & Wilson, 2000); and rats calibrate their hormonal function and adult reproductive behavior according to the maternal licking and grooming they receive (Cameron et al., 2008). In all these diverse systems, the early-life cue functions as a ‘weather forecast’ of the type of ecological context that the individual is most likely to experience (Bateson et al., 2004). Such systems can only have evolved if it is quite often the case (a) that there is not one optimal phenotype for all contexts, but rather a range of contexts experienced over evolutionary time, each of which selects for a slightly different phenotypic value; and (b) that there are costs or constraints on being infinitely plastic in adulthood, leading to individuals committing stably and irreversibly during development to one phenotype or reaction norm.

A second reason that inter-individual variation has become more salient for behavioral biologists is that the extent of heritable variation in important characteristics is greater than would have been predicted by classical population genetic theory such as that of Fisher (1930). In fact, heritabilities are often high in wild populations, even for traits directly relevant to fitness (Houle, 1992). On a related note, heritability estimates for key human behavioral and cognitive traits are substantial (Bouchard, 2004), and the extent of molecular polymorphism in the human genome, including in coding regions, is much higher than would have been predicted some years ago (Levy et al., 2007), not least because of the existence of sizeable chunks of genetic material which exist in different numbers of copies in different individuals (Redon et al., 2006).

Given the ubiquity of variation, then, theoreticians have been naturally concerned to provide ultimate explanations of its existence. In fact, there are two rather different sets of questions that such variation raises. I shall call these the issue of variation, and the issue of correlation.

Why Variation?

The more obvious of the two sets of evolutionary questions concerns why phenotypic variation itself should persist. In cases of developmentally-induced variation, the answer must be that it is not possible to build a phenotype optimally adapted for all the different local contexts recurrently encountered over evolutionary time, and it is impossible or excessively costly to maintain complete plasticity in adulthood. As long as there are cues available of the local state of the environment during ontogeny, and as long as that environment forecasts the future one moderately well, this can lead to the evolutionary emergence of responsiveness to specific early environmental inputs.

Where variation is genetically inherited, there are, also as mentioned above, a number of possible reasons why selection has not been able to drive variation down to zero. The first is neutrality: variants that have no impact on fitness are invisible to natural selection and can change by mutation and drift. For traits consequential to fitness, another possible reason for variation is that the number of genes, and hence the number of targets for mutation in every generation, is sufficiently large that selection cannot outpace the input of new mutations (Barton & Keightly, 2002; Keller & Miller, 2006). This argument has been applied to the maintenance of heritability in human physical symmetry, intelligence, and mental disorders (Keller & Miller, 2006;
Prokosch, Yeo, & Miller, 2005). The winnowing power of selection is further attenuated if it fluctuates; that is, if the phenotypic value with the highest fitness varies from place to place, time to time, or individual to individual. Fluctuating selection arises through a number of mechanisms including spatial or temporal heterogeneity in the habitat, sexually antagonistic selection (where alleles are favored in one sex but disfavored in the other), and negative frequency dependent selection (where alleles are favored when they are rare but disfavored when common). Thus, regardless of whether the proximate mechanism producing phenotypic variation is environmental cues or genetic polymorphism, the same idea about different levels of a trait being adaptive in different contexts turns out to be potentially potent in retaining phenotypic variation.

Given the variety of possible reasons genetic variation persists, the reader might be forgiven for thinking that hypotheses are untestable and the inquiry speculative in this area. This is not the case, since clear predictions can be made about the relationship between the trait under study and consequential outcomes such as survival and reproduction. If variation is maintained by mutational load alone, then selection on the trait should be consistently directional; high scorers on physical symmetry, which is a phenotypic indicator of low mutational load, should always have better outcomes than less symmetrical individuals, for example. If some kind of fluctuating selection is at work, then life outcomes should sometimes be positively associated with the trait, and sometimes negatively so. For example, Dingemanse et al (2004) showed that male great tits that were high on a heritable dimension of exploratory behavior were more likely to survive and reproduce than low scorers in years when resources were abundant, but exactly the opposite was true in years when resources were scarce. This is clear evidence of temporally fluctuating selection, as is the finding that bold guppies are advantaged in habitats lacking predators but disadvantaged where predators are present (Dugatkin, 1992; O’Steen, Cullum, & Bennett, 2002). Similarly, Foerster et al. (2007) showed, in red deer, that parents whose sons had higher than average reproductive success had daughters with lower than average reproductive success, direct evidence that sexually antagonistic selection is maintaining variation in phenotypic characteristics in this case. Thus, hypotheses about why variation is maintained can be tested in other species. The challenge, to which we return in Sections 3 and 4, is testing them in humans.

**Why Correlation?**

The second set of evolutionary questions arises from the fact that phenotypic characteristics often covary across individuals. In the freshwater snail *Physella vargata*, some individuals have long shells and some short, some narrow shells and some rotund, some grow fast and some grow slowly, and some crawl out of the water for periods and some do not (DeWitt et al., 2000). Strikingly, these distinct traits all covary, so that long shells go with narrow shells, which go with fast growth, and not crawling out of the water, whereas crawling out of the water goes with short, rotund shells and slower growth. Personality psychologists will immediately recognize that what we are dealing with is an underlying dimension or factor here, of ‘growth strategy,’ which has a number of different phenotypic manifestations.

The question is why selection would have caused these particular attributes to covary in the way that they do. This question becomes tractable once the adaptive problem faced by these snails is understood. The snails are victim to several different types of predator. Predatory fish crush them
whole in their jaws. The optimal avoidance strategy is thus to grow as rotund as possible, giving greater crush-resistance, and to keep out of the water when it is not necessary. Predatory crayfish, by contrast, cannot crush the shells, but enter the shell aperture of small snails only. The optimal defense against crayfish is to remain narrow (small aperture), and grow as fast as possible in order to leave the window of vulnerability. Clearly, it is not possible to optimize one’s defenses against both fish and crayfish simultaneously, since by leaving the water, one cannot feed, and therefore cannot grow maximally fast, and shell rotundity is traded off against aperture narrowness. On the other hand, since both fish and crayfish are recurrent adaptive problems, each continues to select for the relevant anti-predator traits. Once the adaptive context is understood, the reasons for the covariation of traits become clearer, because the whole manifold can be considered as a strategic package. There would be little benefit to remaining narrow without growing fast, or crawling out without growing rotund, and so on, and so selection has linked them into an associated suite. In behavioral ecology, such suites are often referred to as behavioral syndromes (Sih et al., 2004), but the concept is very similar to that of a broad personality dimension with a number of correlated facets.

In this particular case variation along the dimension is developmentally induced by chemical cues of either fish or crayfish received during early development, but even if the variation were genetic, covariation of the parts of the package could evolve since selection can act not just on traits themselves but also on the genetic correlations between traits. For example, Dingemanse et al. (2007) showed that, across 12 populations of sticklebacks, the presence of predators not only changes the mean level of several behavioral traits, but also changes the pattern of association between them. Thus, whether they are heritable or not, we are able to ask why selection has caused patterns of differential traits to covary in the way that they do.

Explanations for the covariation between traits, in both human personality psychology, and in biology more generally, have tended to focus on the proximate level, invoking common neural or developmental mechanisms. For example, all the various aspects of extraversion have something to do with reward and depend on reward circuits in the brain (Depue & Collins, 1999), or all the aspects of neuroticism reflect activity of neural mechanisms for detecting threat of punishment (Gray & McNaughton, 2000). These kinds of explanations, though not wrong, are incomplete from an evolutionary perspective (Dingemanse et al., 2007). Evolution generally favors designs in which mechanisms solve only one class of problem, and can develop and change somewhat independently of one another (Buss, 1995; Tooby & Cosmides, 1992). Given that this is the case, why should there be mechanisms dealing with ‘threats in general,’ or ‘rewards in general,’ rather than, separately, food rewards, status rewards, sexual rewards and so on? Of course, selection has to build mental mechanisms up from systems already in place, and so it is quite plausible that systems for feeding, say, would become exapted for approaching other kinds of benefits, but selection is perfectly capable of decoupling different domains if there are fitness disadvantages to having one’s libido yoked to one’s appetite for food, for example. Thus, as well as these proximate hypotheses, we need ultimate explanations of why selection would allow mechanistic correlations across suites of behaviors in different domains to persist.

The freshwater snail example gives us a possible insight into this problem. Perhaps the apparently disparate components of a behavioral syndrome or personality dimension represent aspects of a coherent behavioral package, so that it makes adaptive sense when showing any one of them to a
high degree, to show the others, too. There are two ways which this could be brought about. First, there could be correlations in the environment, such that the same environmental conditions that select for a high level of behavior A also select for a high level of behavior B, whilst environmental conditions that select for a low level of B also select for a low level of A. This could produce associations of A and B via selective advantage of individuals high in A and B, or low in A and B, but not those high in one whilst low in the other. Let us call this the correlated environment hypothesis.

Alternatively, there could be synergistic tradeoffs between the component behaviors. For example, spiders which are bold in foraging are also high in aggression in intra-specific encounters (Reichert & Hedrick, 1993). Bold foragers may, as a consequence of their foraging actions, have larger food packages to defend from rivals, and thus the benefit of an increment of aggression will be larger for an individual high in boldness than one low in boldness, selecting for a covariation of foraging boldness and aggression. I will term this the synergistic behaviors hypothesis.

Both correlated environments and synergistic behaviors result in a selection pressure to couple the development of several separate traits together.

At the proximate level, this might be achieved in a number of ways, for example by using partly shared neural control mechanisms, so that a polymorphism or developmental influence upstream impacts downstream on all of the components. Alternatively, there might be specific evolved mechanisms which ‘read off’ one aspect of the developing phenotype and use this to calibrate the appropriate level of other aspects. This is related to what Tooby and Cosmides (1990) called ‘reactive heritability,’ where psychological mechanisms in one domain calibrate themselves according to heritable variation in other aspects of the phenotype. We return to the proximate mechanisms leading to behavioral associations in Section 5.

**Darwinizing the Big Five**

In this section (summarized in Table 1.1), I review the application of the evolutionary ideas discussed in Section 2 to the big five personality dimensions in humans. Taking each dimension in turn, I discuss possible evolutionary answers to the variation question (Why do individuals differ on this dimension?) and the correlation question (Why do the different facets covary?). The question of variation has been raised before, and I am drawing heavily on my earlier accounts (Nettle, 2006a, 2007). In these, I dismissed neutrality as a possibility, given the abundant evidence of personality effects on fitness-relevant outcomes such as life expectancy, mating, relationships, and health (Friedman, 1995; Kelly & Conley, 1987; Ozer & Benet-Martinez, 2006; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007), and instead suggested possible fitness costs and benefits of being high or low on each dimension.

The question of correlation has been less widely discussed and may turn out to be of broad interest to personality psychologists. This is because each of the big five consists of a number of component motivational or behavioral tendencies, sometimes known as facets or aspects (DeYoung, Quilty, & Peterson, 2007), which often belong to quite different domains of life. At first blush it is not obvious why these aspects should bundle together as they do. The discovery that they do has been entirely inductive, coming from factor analysis of large lexical or rating data sets. Whilst this is a valid approach, it has left some with the feeling that the FFM is unsatisfactorily atheoretical, offering little deep insight into why the different aspects should go together (Block, 1995; Denissen & Penke, 2008a). Although there have been attempts to address
this issue in terms of proximal and ontogenetic factors (McCrae & Costa 2001), adopting an evolutionary perspective may provide deeper explanatory insights.

11

**Extraversion**

Table 1.1. The big five personality dimensions, with suggestions of fitness benefits and fitness costs for an increasing level of each dimension, and a list of some of the key components of the dimension. Note that the components are an informal inventory culled from the literature (especially Nettle 2007, DeYoung et al. 2007, Denissen & Penke 2008a), not a formal claim about the factor analytic sub-structure of the big five.

<table>
<thead>
<tr>
<th>Dimension</th>
<th>Benefits</th>
<th>Costs</th>
<th>Components</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extraversion</td>
<td>Increased status</td>
<td>Sexual partners</td>
<td>• Ambition</td>
</tr>
<tr>
<td></td>
<td>Sexual partners</td>
<td>Resource access</td>
<td>• Competitiveness</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>• Assertiveness</td>
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<td></td>
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<td>• Sociability</td>
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<td></td>
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<td></td>
<td>• Exploratory tendency</td>
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<td></td>
<td></td>
<td></td>
<td>Sexual motivation</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Vigilance to threats and dangers</td>
<td>Stress-related illness, consequences for</td>
<td>• Vigilance to physical threat</td>
</tr>
<tr>
<td></td>
<td></td>
<td>relationships</td>
<td>• Vigilance to social threat</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Perceived vulnerability to disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Angry hostility</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>Planfulness and care in premeditated tasks</td>
<td>Rigidity, difficulty adapting to changing</td>
<td>• Industriousness</td>
</tr>
<tr>
<td>Agreeableness</td>
<td>Cooperative ventures and harmonious alliances</td>
<td>Failure to maximise personal returns; victim of cheaters</td>
<td>• Orderliness</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Cooperativeness</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Theory of mind processing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Norm-following</td>
</tr>
<tr>
<td>Openness</td>
<td>Creativity</td>
<td>Disorganised or psychotic thinking</td>
<td>• Intellect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Imagination</td>
</tr>
</tbody>
</table>

**Why Do Individuals Differ?**

High levels of extraversion are associated with increased numbers of sexual partners (Nettle, 2005), increased social status and social attention (Ashton, Lee, & Paunonen, 2002; Nettle, 2007), and increased levels of physical activity (Kircaldy, 1982). These are all plausibly fitness-enhancing under appropriate circumstances. However, all carry some degree of risk, of accident, disease, social conflict, or resource depletion. Consistent with this view, high extraversion scorers have been found variously to have
greater risk of accidents (Nettle, 2005), of arrest (Samuels et al., 2004), and shorter life expectancy than low scorers (Friedman, 1995).

Thus, whether increasing extraversion will bring about a net increase or decrease in reproductive success will vary in two ways. First, there may be characteristics of individuals that determine how much extraversion is optimal. Physically stronger, more attractive individuals with good immune function may be better placed to bear the risks of an extraverted strategy going wrong, and thus their optimal level will be higher. On the other hand, there may be ecological contexts that generally favor this kind of risk-raking. When social structures are fluid or the habitat novel, there may be big payoffs to taking risks, attempting to influence the local group, and seeking multiple matings. When the habitat is already saturated and hierarchies stable, it may become optimal to be more cautious. Interestingly, Chen, Burton, Greenberger, & Dmitrieva (1999) showed that ‘long’ alleles of the D4DR gene in humans are more common in populations that are nomadic or have historically completed long migrations than in populations that have been sedentary for a long time. The D4DR long alleles are also associated with personality traits and behaviors related to extraversion (Ebstein, 2006). This suggests that nomadism and social fluidity select for increasing extraversion. (Chen et al. [1999] consider the opposite possibility, that extraverts are more likely to embark on migrations, but conclude that this is less likely as an explanation of the pattern observed; see also section on consequential outcomes and person-environment fit.)

Given that the optimal level of extraversion will thus be varying across individuals within a habitat (due to their different phenotypes), across habitats, and within a habitat over time, it is not surprising that a full range of phenotypic variation is maintained.

Why Do the Different Facets Covary?

The extraversion dimension has a number of distinct components. High scorers are ambitious, assertive, and competitive, but they are also sociable, physically active, and sexually motivated. There is no necessary reason that these different components should go together; it would be logically possible, for example, for ambition and sociability to be inversely correlated. These different components could have come to covary through synergistic effects. Ambitious and competitive individuals are more likely than average to achieve high status, and for individuals with high status, a motivation to achieve multiple mates is more likely to lead to an actual increase in reproductive success than if such a motivation were found in individuals with low status. Thus, the payoff to higher mating motivation will be greater if ambition is also higher. Similarly, sociability and ambition could increase the payoff for each other; in small, tight-knit groups, the individual who seeks high status without also being motivated to expend energy on creating and maintaining alliances is liable to run into trouble. The relationships also make sense in the negative direction. An individual who is not disposed to spend energy vying for status need perhaps not allocate so much energy to creating social alliances, and an individual whose mating strategy is not based on seeking multiple mates may derive less benefit from an increment of status.

It is also possible that there are certain types of ecology that advantage high (or low) levels of all of the extraversion components. As mentioned above, it may be that nomadism and novel environments favor all of sociability, assertiveness, sexual motivation, and high physical activity, whilst sedentism and established social structures favor none of them.
Neuroticism

Why Do Individuals Differ?

High neuroticism scorers face the serious drawback of increased stress-related physical and mental illness (Neeleman, Sytema, & Wadsworth, 2002), and difficulties in relationships as a consequence of their negative affect and anxiety (Kelly & Conley, 1987). This would seem like a formidable selection pressure constantly reducing the level of neuroticism.

However, negative emotions exist for a reason, namely to detect and deal with threats. Theory suggests that the more prevalent threats are in an environment, the more sensitive threat-detection mechanisms should be, even at the expense of producing numerous false positives, where threats that do not in fact exist are detected (Haselton & Buss, 2000; Haselton & Nettle, 2006; Nesse, 2005). Empirically, in fish, environments where predation pressure is high cause the rapid evolution of increased vigilance and caution, despite the negative impact this has on feeding (Dingemanse et al., 2007; O’Steen et al., 2002). Thus, it would be quite possible to be too low in neuroticism for a particular context, and thus fail to detect threats that are in fact present.

Evidence for such effects in humans is admittedly scarce. However, Oishi, Diener, and Lucas (2007) have shown that the individuals most successful in terms of income, education, and political engagement are not those with the highest levels of subjective well-being, but those who are closer to the mean well-being level. Since subjective well-being is closely associated with neuroticism (Costa & McCrae, 1980), this squares with direct evidence that students high in neuroticism enjoy increased academic performance, perhaps through the motivating effects of detecting the bad outcomes if they fail (McKenzie, Taghavi-Knosary, & Tindell, 2000).

Thus, it is plausible that in environments where the level of actual threat is high, or in individuals poorly able to deal with undetected threats, selection favors increased neuroticism, whilst in more benign situations, selection tends to reduce it. Relatedly, high neuroticism and associated affective disorders are more common amongst women than amongst men (Costa, Terraciano & McCrae, 2001). Though environments experienced may not differ much between the sexes, the impacts of undetected threats may do so. Succumbing to either a physical hazard or social ostracism in adulthood will tend to have more serious effects on reproductive success for a woman than a man because of the greater knock-on effects on offspring survival (Campbell, 1999).

Why Do the Different Facets Covary?

High neuroticism scorers show increased sensitivity to physical dangers, increased perceived vulnerability to disease, and increased angry hostility in response to social challenges (Denissen & Penke 2008b; DeYoung et al., 2007; Nettle, 2007). Again, we might have expected all these rather different types of negative outcomes to be appraised using entirely separate mechanisms, and so there is no logical reason that the responsiveness to one type would predict responsiveness to the others. However, we may once again be dealing with some combination of synergy and correlated environments.

One of the most ancestrally salient threats—predation—is a physical one (and predator sensitivity is the closest analogue to neuroticism in the animal personality literature). However, the presence of predators has many consequences for behavior above and beyond predator avoidance itself (Lima & Dill, 1990). For example, if there are predators around, then the cost of becoming...
diseased becomes much higher, since disease impairs predator escape abilities. Similarly, the costs of not being in a social group are greatly increased by the presence of predators, since a major function of mammalian grouping is predator avoidance (Silk, 2007). Thus, it makes sense that where sensitivity to physical threat is high, perceived vulnerability to disease should also be high, as should concern and anxiety about social exclusion and social position. Why angry hostility should be part of the syndrome is less obvious.

Conscientiousness

Why Do Individuals Differ?

High conscientiousness is associated with making plans and sticking to them, which has the advantage in the modern environment of leading to high attainment in office and school (Barrick & Mount, 1991). Being able to stick to internally-generated plans and goals in the face of distraction may be advantageous in some ancestrally-relevant contexts too, particularly when facing repeated harvesting tasks where the outcomes and optimal schedules are predictable. However, some situations—sudden attack or opportunistic hunting, for example—cannot be planned for, and instead an ability to respond spontaneously and without the need for extensive reflection is useful. High conscientiousness-scorers can be rigid and inflexible, characteristics that reach a damaging extreme in obsessive-compulsive personality disorder. It is thus likely that the optimal balance between planfulness and spontaneity depends on the precise details of the local ecology and personal role, and in particular the nature of the resource-extraction tasks one specializes in. This variability would suffice to sustain variation in conscientiousness.

Why Do the Different Facets Covary?

The two main distinguishable aspects of conscientiousness are termed industriousness and orderliness by (DeYoung et al., 2007). Industriousness describes the planfulness discussed above, whilst the latter consists of personal neatness, tidiness in dealing with objects, and attention to detail. Quite why these two aspects should be associated is not immediately clear, though it is possible that both are useful where the same resource is to be exploited recurrently in a predictable manner, and thus has to be maintained and managed. Neither would be particularly advantageous where resources are exploited opportunistically or unpredictably on a one-time basis.

Agreeableness

Why Do Individuals Differ?

High agreeableness is associated with high investment in cooperative joint ventures (Ben-Ner, Kramer, & Levy, 2008; Koole, Jager, van den Berg, Vlek, & Hofstee, 2001), and harmony in inter-personal relationships (Asendorpf & Wilpers, 1998). Humans are of course a highly social species, and coordinating with others in joint ventures can often be favored by selection. Thus, the advantages of higher agreeableness are not difficult to identify. However, there are contexts when joint economic ventures are unprofitable, and individuals would do better fending for themselves. This would depend very much on the type of resources being extracted and the local social structure. Highly agreeable individuals may over-invest in social relationships, at the expense of their own material and status interests, at such times (Nettle, 2007). More specifically, once high agreeableness and cooperation are widespread in a population, there can be large advantages to less agreeable ‘cheaters’ who exploit the joint
ventures surrounding them. Such frequency-dependent advantages to low agreeableness would maintain a mixture of phenotypes at equilibrium. In addition, variation in agreeableness might be maintained by sexually antagonistic selection. Studies across many countries have found higher agreeableness scores in women than in men (Costa, Terraciano & McCrae 2001; Schmitt, Realo, Voracek & Allik 2008). I have argued that men have on average gained more over evolutionary time from a unit of personal status relative to a unit of social support than women have. This has produced quite marked sexual dimorphism in agreeableness (Nettle & Liddle, 2008), but may also be an engine for the maintenance of inter-individual variation more generally.

**Why Do the Different Facets Covary?**

Central components of agreeableness are prosocial behavior itself (Ben-Ner et al., in press; Koole et al., 2001), the differential allocation of attention to the mental states of other people (Nettle & Liddle, 2008), and a tendency to follow social norms (DeYoung et al. 2008 call this component politeness, but it may be broader than this word suggests). These are plausibly synergistic. Given that other humans are highly discriminating conditional cooperators with the propensity to punish, exclude those who default, and occasionally cheat, then if one is to enter into complex cooperative joint ventures, there would be high payoffs to carefully monitoring the mental states of other participants, and being attentive to the social norms created by the interactions. On the other hand, if one is disposed to engage in more solitary tasks and eschew joint ventures, then some energy can also be saved in terms of mental state processing and being bound by norms. This would be sufficient to ensure the covariation of the agreeableness package.

**Openness**

**Why Do Individuals Differ?**

One of the striking things about the fifth of the big five, openness, is that it is linked in the literature about equally often with positive and with negative outcomes. On the negative side, high openness is associated with paranormal beliefs (McCrae & Costa, 1997), contact with psychiatrists (Soldz & Vaillant, 1999), schizotypal personality disorder (Gurrera et al., 2005), and perhaps even psychosis (Burch, Hemsley, Pavelis, & Corr, 2006). On the positive side, it is correlated closely with artistic creativity (McCrae, 1987) and more weakly with intelligence (DeYoung, Peterson, & Higgins, 2005).

I have suggested elsewhere (Nettle, 2001) that the creativity, with consequent increase in social and sexual attractiveness (Miller, 2000; Nettle & Clegg, 2006) represents a key benefit of increased openness, whereas the unusual beliefs and potentially disorganizing psychotic experiences represent a cost. Here it may not be so much different ecologies selecting for different levels of openness, as different levels of openness being optimal depending on other characteristics of the individual.

**Why Do the Different Facets Covary?**

Several authors agree that there are two main components of openness: an ‘intellect’ aspect related to intelligence, quickness of mind and verbal abilities; and an ‘imagination’ aspect which is linked to loose semantic associations, fantasy, daydreaming, paranormal belief and psychosis-like experiences (DeYoung et al., 2007; Nettle, 2007). The fact that these are so different from one another has led to a fair amount of disagreement about the appropriate name for the fifth factor, and even whether it represents a coherent entity at all. The current perspective suggests a novel way of looking at this problem: it could be that the intellect aspect and the imagination
aspect are distinct, but have synergistic fitness relationships between them. An individual high in intellect may benefit from being high in imagination, as this will allow them to exhibit their intellect in arresting ways that will garner them social esteem and attention. Conversely, an individual low in intellect will find the loose mental associations of high imagination difficult to manage and they may become disorganizing fantasies or even delusions and hallucinations. Thus, selection is for high imagination in individuals with high intellect, and low imagination in individuals with lower intellect, with intellect itself being perhaps determined by developmental stability and general mutational load (Keller & Miller, 2006; Prokosch et al., 2005). This would be enough to maintain intra-population diversity in imagination levels.

To my knowledge, there is no direct evidence for this position, although research has shown that successful poets and artists score as highly on scales of psychosis-proneness as schizophrenia patients do, but differ from the patients in other ways (Nettle, 2006b), which would plausibly include premorbid intelligence (Gilvarry et al., 2000). Thus, the effects of high imagination would be highly condition-dependent. This may explain why artistic creativity is found so attractive; it signals that the person is of high enough intellect to develop high imagination in a successful way (Nettle & Clegg, 2006; Shaner, Miller, & Mintz, 2004). It also suggests that serious psychological disorders including psychosis might be thought of as the interaction of a particular cognitive style with adverse general developmental conditions, a position which is consistent with a number of lines of evidence.

Conclusions and Prospects

Having reviewed the possible evolutionary reasons for variation and for correlation in each of the five-factor dimensions, it is time to conclude, and to assess whether the ideas discussed above have any usefulness for personality psychologists, whose focus is understandably trained on specific empirical predictions. I believe that they do. Although many of the ideas discussed in Section 3 are little more than speculations, they do lead to both interpretations of existing unsolved problems and novel empirical predictions. In the remainder of this chapter I review some areas where the evolutionary approach adds value in this way.

The Broad Versus Narrow Issue

An ongoing debate for both evolutionary and personality psychologists is that of the appropriate breadth of construct. Personality psychologists have long noted the superior predictive power, especially in single instances, of constructs that are much narrower in terms of their situational scope than umbrella dimensions like the big five (Mischel & Shoda, 1995). Likewise, evolutionary psychologists, with their argument from the adaptive reasons to expect domain-specificity of psychological mechanisms, would tend to predict that coherent traits will refer to only one class of adaptive situation and be uncorrelated to those referring to other behavioral domains. Yet, set against this, the statistical evidence for both the behavioral coherence and the long-term predictive power of broad dimensions is rather strong (Fleeson, 2001; Ozer & Benet-Martinez, 2006; Roberts et al., 2007).

The considerations of this chapter shed some light on this paradox. Mental mechanisms are domain-specific, but because there are either synergistic combinations of traits, or types of environment that select for a whole suite of different responses, sets of domain-specific traits

...
have become partially yoked together during development. This might explain the observed hierarchical structure of behavioral variation in humans, with narrow-domain traits governed by a single mechanism and very coherent amongst themselves, clustering more loosely into umbrellas such as the big five, and possibly the big five showing even broader and looser clustering into one or two very general superfactors (Digman, 1997; Musek, 2007). These superfactors could reflect the response of many behavioral domains to extremely general life-history parameters such as the rate of mortality or expectation of life (Figueroedo, Vasquez, Brumbach, & Schneider, 2004).

**Consequential Outcomes and Person–Environment Fit**

The evolutionary perspective focuses attention on consequential life outcomes of personality variation, such as life expectancy, health, relationships, and status. This has of course been of long-standing interest to personality psychologists, though they have seldom conceptualized the outcomes they measure as components of biological fitness. However, the evolutionary perspective more specifically predicts that being high on a personality dimension will bring benefits to some components of fitness and costs to others, and this can lead to novel ways of framing empirical results (Nettle, 2005). It also predicts that the net effect of a particular personality profile on fitness will vary with ecological context, and this is where there are novel empirical predictions to test.

The first study I know of that has satisfyingly shown such personality by context interactions is by Eisenberg, Campbell, Gray and Sorenson (2008). This study examined the effects of DRD4 genotype on body mass amongst the Ariaal of Kenya, a population which is chronically nutritionally stressed. Long alleles of DRD4 are associated with high extraversion, and are found at greater frequency in nomadic than settled populations. Some of the Ariaal continue to live their traditional nomadic existence, whilst others have settled. The study found that long DRD4 alleles were associated with significantly increased body mass in the nomadic group, but significantly decreased body mass in the settled group. This is exactly the interaction that the Darwinian costs-and-benefits framework—but no previous framework for thinking about the big five—would predict.

**Coherence: Successful and Unsuccessful Trait Combinations**

If the arguments of this chapter concerning synergy amongst different behavioral domains are correct, then we can frame some novel predictions about the fitness consequences of different combinations of traits. For example, I argued that being high on the imagination aspect of openness was beneficial to the extent that the person also scored highly on the intellect dimension. This makes the specific prediction that people high on imagination and low on intellect, or low on imagination but high on intellect, should have less favorable outcomes than those who are either high on both or low on both. This is a testable prediction.

Similarly, people with a high motivation to enter into cooperative joint ventures, but a low propensity to theory of mind processing, should fare worse in social life than individuals where both cooperation and theory of mind are high, or both are low. It will be adaptive to track mental states if one is involved in cooperative ventures, or not track them and not be involved, but what will be harmful will be to enter into them and then fail to follow the social chess of what is happening. In general, there should be fitness payoffs for coherence amongst the components of any of the big five. Since the correlations between the components are much less than 1, it will be
possible to find individuals with relatively high and relatively low levels of coherence for any particular dimension and test this directly.

Developmental Pathways

The evolutionary perspective may also suggest avenues for research on developmental determinants of personality. The consensus in the literature is that personality variation is due to a combination of heritable and non-shared environmental effects (Bouchard & Loehlin, 2001), but relatively little progress has been made on establishing what the environmental factors are, or why they should have the influence they have (Plomin & Daniels, 1987). The evolutionary perspective suggests that the emphasis should be on:

(1) Finding early-life cues that might have, over evolutionary time, predicted the state of the adult environment, and establishing whether these calibrate personality. These cues could be subtle, age-critical, and specific in their actions, such as the effect of father absence on a girl’s age at menarche (Ellis 2004), or the developmental induction effects observed in the animal literature. Does early-life geographical mobility make for greater adult extraversion? Does maternal stress during pregnancy specifically impact neuroticism? Does giving children joint tasks produce more agreeable adults?

(2) Finding pathways through which different aspects of the developing phenotype influence each other, leading to adult personality coherence. For example, people with more attractive faces become more extraverted (Pound, Penton-Voak, & Brown, 2007), and physically large men become less agreeable (Ishikawa, Raine, Lenez, Bihrlke, & Lacasse, 2001). Presumably here the non-behavioral characteristics influence the degree to which particular behavioral suites will be effective for that individual, and thus ‘reactive personality formation’ makes adaptive sense.

These are all fruitful areas for further research, and areas where the evolutionary perspective helps frame hypothesis. As is generally the case, the greatest progress will come not by forming a sub-discipline of evolutionary personality psychology separate from the rest of personality psychology, but rather suffusing the way we think about personality in general with a warp of ultimate, Darwinian logic, to weave into the weft of careful empirical research.

References


2 Personality and the Adaptive Landscape: The Role of Individual Differences in Creating and Solving Social Adaptive Problems

David M. Buss

Dora’s social world came crashing down when she discovered that her husband had cheated on her with her best friend. The double-betrayal meant that she lost what she thought was her lifelong mate as well as her closest female confidant and ally. Although Dora took revenge by having a retaliatory affair with her best friend’s husband, it failed to stanch her losses (Meston & Buss, 2009). It turned out that her husband was high on the personality trait of narcissism—a trait that predicts infidelity (Buss & Shackelford, 1997). And her best friend was low on agreeableness, a trait that predicts betrayal in social relationships (Nettle, 2006). Had she chosen a mate and friend with a different constellation of personality traits, Dora would not have been forced to deal with the adaptive problems of her double-barreled loss.

The central hypotheses of this chapter can be stated succinctly. Stable personality traits in oneself and in inhabitants of one’s social environment are linked to the creation of adaptive problems as well as to the solution to adaptive problems. The personality-problem linkages occur for each of the major classes of social relationships—coalitions, friendships, enemies, hierarchical relationships, kinships, and mateships. As a consequence of these recurrent linkages, humans have evolved difference-detecting mechanisms, or personality assessment adaptations, that function to avoid some adaptive problems and facilitate the solution to other adaptive problems. The recurrent personality-problem linkages, in short, are the raison d’être for the evolution of personality assessment adaptations. The personality characteristics of others in each person’s milieu, therefore, define a social adaptive landscape that difference-detecting adaptations help to navigate. This chapter elaborates on these hypotheses, summarizes available empirical evidence in support of them, and outlines a research agenda and conceptual directions for their further exploration.

Social adaptive problems require social psychological solutions. Individuals inhabiting each person’s social world differ from each other in myriad ways. Some differences reflect temporary states. Individuals differ in transient health status, for example—variation in who happens to be infected by a local virus at any particular point in time. Solving the problem of avoiding infection requires identifying and selectively avoiding close contact with those who happen to be infected (Navarette & Fessler, 2006). A second example of a state difference centers on transient emotions. Humans show exceptional ability to rapidly and accurately detect an angry face from among a sea of faces (Öhman et al., 2001). This difference-detecting ability alerts individuals to sources of potential danger from other humans and enables taking action to avoid the threat or to defend against the threat. Humans clearly have adaptations designed to perceive and act upon transient state-differences in other humans.

Other individual differences are more stable and reflect relatively enduring personality traits such as those captured by the five-factor model, the HEXACO model, as well as individual traits of well-documented importance such as narcissism, intelligence, and some psychological disorders (Ashton et al., 2004; Buss & Chiodo, 1991; Goldberg, 1990). These traits are now known to provide at least moderate predictive power in forecasting the future behavior of others (Fleeson &
Gallagher, 2009). If that behavior has consequences for the creation of, or solution to, adaptive problems involved in interacting with those others, selection should fashion adaptations designed to detect, evaluate, and act on those stable differences (Buss, 1991a, 1996). Personality assessment adaptations are hypothesized to represent one class of mechanisms for solving social adaptive problems.

From this perspective, the most important theoretical and empirical issues become: (1) identifying each of the key social adaptive problems humans have recurrently confronted; (2) identifying which individual differences in others are most central to creating these social adaptive problems; (3) identifying which individual differences in others interfere with solving adaptive problems; (4) identifying which individual differences in others are most central to solving these social adaptive problems; and (5) identifying difference-detecting adaptations, including (a) the cues people use to assess the key individual differences, (b) psychological mechanisms, including motivational and emotional circuits, used to process information about key individual differences, and (c) the behavioral output directed as solving problems based on these individual differences.

Consider as an example an adaptive problem that women almost certainly have faced over deep time—the problem of sexual exploitation at the hands of some men. There is good empirical evidence for stable individual differences among men in which some, but not others, pursue a sexually exploitative strategy—the roughly 4% of men who have the cluster of traits captured by the term psychopathy (Lalumiere et al., 2005). These men score low on the personality traits of agreeableness and conscientiousness, and lack some common human emotions such as guilt or remorse. They frequently pursue a social strategy of deception, manipulation, and conning, so avoiding becoming a victim of psychopaths is an adaptive problem that many individuals face, both men and women. For women specifically, men high on psychopathy tend to pursue short-term mating strategies marked by deception, manipulation, the threat of force, and the use of force (Lalumiere et al., 2005).

Sexually exploitative strategies usually inflict heavy fitness costs on victims. These include bypassing female choice, impregnation by a man who is unwilling to invest parentally, damage to the victim’s social reputation, damage to her perceived mate value, and social stigma attached to resulting children. Given these heavy fitness costs, women who had the ability to identify which men were more prone to using a sexually exploitative strategy, even if this detection ability gave them a modest increment in predictive power, would have a selective advantage over women who were oblivious to this individual difference.

The advantage would come from adaptive action taken subsequent to detecting cues to this male strategy, such as selectively avoiding sexually exploitative men, selectively choosing mates and friends who have the physical formidability and psychological fortitude to act as “body guards,” and enlisting the aid of kin or other social allies to deter sexually exploitative men. In short, a social adaptive problem created by some men more than by other men can, in principle, be solved or ameliorated by difference-detecting adaptations that lead to adaptive action. Of course, difference-detecting adaptations undoubtedly play a role in detecting all sorts of social exploiters, and there is evidence that psychopaths are not just sexually exploitative, but also tend to cheat in social exchange and other social relationships (Mealey, 1995).

This example raises an interesting issue that involves sexual conflict and sexually antagonistic co-evolutionary arms races (Arnqvist & Rowe, 2005). Men who pursue a sexually exploitative
strategy also face a problem whose solution would be aided by a difference-detecting adaptation. The ability to

identify more vulnerable victims—perhaps those who are more gullible, naïve, or cognitively challenged—would afford these men the ability to neutralize or circumvent whatever evolved defenses exist in potential victims and more successfully carry out their sexually exploitative strategy (Buss & Duntley, 2008). Just as cheetahs who selectively avoid gazelles who exhibit ‘stotting’ behavior (an evolved anti-predator defense that signals to the cheetah the athletic condition of the gazelle—FitzGibbon & Fanshawe, 1988) are more successful at predation, psychopaths who selectively avoid non-exploitable women will be more successful at sexual exploitation. Sexual exploitation, of course, is not limited to psychopaths, but is perpetrated, albeit at lower frequencies, by men not so characterized (Haselton et al., 2005).

In this sphere of sexual conflict, difference-detecting adaptations are central to the social problems faced by both victims and perpetrators. They are critical for women in avoiding men dispositionally inclined to sexual exploitation. They are critical for exploiters for selectively targeting some women as victims and selectively avoiding other women who are less vulnerable to exploitation. They are critical for women in implementing direct or ancillary adaptive solutions, such as choosing men dispositionally inclined to serve as effective body guards. And they are critical for men in avoiding satellite adaptive problems of a sexually exploitative strategy, such as incurring physical damage due to the choice of a non-exploitable victim. The ability to detect and act on stable personality traits of others, in short, is important for all players in this sexually antagonistic arena for solving their respective adaptive problems.

Identifying Social Adaptive Problems

Ultimately, it would be desirable to formulate a comprehensive taxonomy of all key social adaptive problems. This would permit examining each problem through the theoretical lens of difference-detecting adaptations and formulating a program of research to test hypotheses about their role in evolved solutions. The field of psychology is a long way from this goal, although a few preliminary attempts have been made (e.g., Buss, 1986; Kenrick, Griskevicius, Neuberg, & Shaller, 2010). Nonetheless, some excellent candidates provide a starting point—the key social relationships that appear to occur in all social groups. Any comprehensive taxonomy must include, at a minimum, adaptive problems associated with mating relationships, coalitions, friendships (dyadic alliances), enemies, hierarchical relationships, and kinships (Buss, 1986). This chapter illustrates the importance of personality assessment adaptations for solving social problems entailed by each of these classes of relationships.

Interestingly, the field of evolutionary psychology has invested a large amount of effort and produced striking success in identifying the many design features of one difference-detecting adaptation—the psychology of assessing individual differences in physical attractiveness (Sugiyama, 2005). In contrast, evolutionary psychologists have almost entirely neglected difference-detecting adaptations for psychological and behavior traits, although some personality and social psychologists have explored difference-detecting mechanisms outside of an evolutionary framework (e.g., Borkenau, Mauer, Riemann, Spinath, & Angleitner 2004; Naumann, Vazire, Rentfrow, & Gosling, 2009). This paper provides a conceptual framework for the evolutionary psychological analysis of personality assessment adaptations.
Personality Traits Linked With Habitual Welfare Tradeoff Ratios

We begin with hypothesized links between personality traits and a variable hypothesized here to be linked to a host of social adaptive problems—welfare tradeoff ratios. A welfare tradeoff ratio (WTR) refers to how much weight an individual places on his or her own interests relative to those of another person (Tooby, Cosmides, Sell, Lieberman, & Sznycer, 2008; Sell, Tooby, & Cosmides 2009). WTR has been hypothesized to be an internal regulatory mechanism that affects, and is affected by, variables such as genetic relatedness and physical formidability. The recalibration theory of anger, for example, proposes that anger is an adaptation designed to alter the WTR of the target of the anger in favor of the person displaying the anger (Sell et al., 2009).

Two empirical studies have confirmed specific predictions from this theory (Sell et al., 2009). Physical formidability is a stable trait linked with the ability to inflict costs on others, giving formidable individuals a better bargaining position and ability to prevail in social conflicts. Formidable men are more prone to anger and more strongly endorse attitudes toward using force to resolve interpersonal and even international conflicts (Sell et al., 2009). Women high on physical attractiveness—an attribute linked with the ability to confer benefits because of its link with fertility—are also more prone to display anger and have a greater sense of entitlement. Both findings support the notion that the stable traits of physical formidability in men and physical attractiveness in women influence WTRs and the tactics used to alter them in others, such as the display of anger.

If stable personality traits also influence how an individual habitually perceives WTRs, as well as the tactics used to alter WTR in his or her favor, then adaptations to assess those traits would help to solve a suite of problems associated with conflicts of interest in social interactions and relationships. A prime personality trait that should influence WTRs is that of narcissism. Two cardinal features of narcissism are a strong sense of personal entitlement and being self-centered. Narcissistic acts that reflect these elements include: He asked for a large favor without offering repayment; he took the last piece of dessert without asking if anyone else wanted it; she assumed that someone else should pay for dinner when she was low on cash; he refused to share his food with others; she cut into a long line ahead of her turn (Buss & Chiodo, 1991).

Narcissists also display a lack of empathy, which is reflected in acts such as: He did not show much feeling when his friend was upset; she did not listen to other people’s problems; and he ignored a friend who was sad. Another hallmark of narcissism is being interpersonally exploitative. Exploitative acts include: He insisted that his friend drop everything to see him; she did the favor only when twice as much was promised in return; she asked someone else to do her work for her (Buss & Chiodo, 1991). Narcissism may be accurately gauged from observing these acts over time or through social reputation (Buss & Chiodo, 1991) or through physical appearance cues such as expensive or stylish clothes, and for females, the amount of cleavage that they show (Vazire, Nauman, Rentfrow, & Gosling, 2008).

In short, those high on narcissism display a wide range of acts that indicate that they habitually place a higher weight on their own welfare relative to the welfare of others than do individuals lower on narcissism. One explanatory possibility is that narcissists possess a selfishly-skewed WTR because they are more physically attractive, and hence affiliating with them provides benefits not provided by those who are less attractive—an effect that should be especially pronounced for women, given the centrality of attractiveness to a woman’s fertility and hence
mate value (Buss, 1989; Symons, 1979). The results of this are equivocal. One study found that narcissists think that they are hotter than average, but they are not (Bleske-Rechek et al., 2008). Another found a small positive correlation of +.23 between narcissism and observer-evaluated physical attractiveness (Vazire et al., 2008). Perhaps future research will reveal some other positive externality emitted by those high on narcissism that might justify their selfishly-skewed WTR; or perhaps not.

Personality assessment adaptations that gauge the levels of narcissism in others, as well as the subcomponents of sense of entitlement, lack of empathy, selfishness, and interpersonal exploitativeness, would aid in solutions to adaptive problems associated with conflicts of interest in social relationships. These include mate selection, friend selection, selection of coalition partners, decisions about which kin to invest in, and decisions about strategies for interacting with individuals who vary in their position within the local status hierarchy. A spouse with a selfishly-skewed WTR, for example, could inflict a wide array of costs on their partner—using joint resources excessively for personal gain and withholding resources that might be allocated to the needs of their partner. Those who score high on narcissism are more likely to be sexually unfaithful in marriages than those low on this trait (Buss & Shackelford, 1997), perhaps because their selfishly-skewed WTR leads them to believe that they are entitled to extramarital sex. Infidelity, however, diverts valuable reproductive resources away from their spouses.

The ability to identify individual differences in narcissism may even help to solve problems associated with kinship. The theory of parent–offspring conflict predicts differences between the fitness interests of parents and their children, as well as between siblings (Trivers, 1974). A kin member high in narcissism, by holding a selfishly-skewed WTR, would have an increased likelihood of exploiting their kin, attempting to resolve conflicts of interest more in their favor than kin not high on narcissism. Consequently, knowing the level of narcissism of kin members would provide valuable information for resource allocation decisions. There should be a higher return on resources allocated toward kin low on narcissism than toward kin high on narcissism. The same logic should apply to friendships and coalition partners.

A second personality trait likely to play a key role in WTRs is agreeableness, one of the global factors of the five factor model of personality. Those high on trait agreeableness tend to exhibit a relatively high frequency of the following acts: He volunteered to make dinner for his friends; she offered to help her friends move something; he helped a friend with a difficult assignment; she volunteered to take responsibility when no one else did; he showed sympathy for his friend’s problems; she volunteered to get the drinks for the party; she loaned her friend something when he/she needed it (Botwin & Buss, 1989). The qualities captured by the agreeableness factor have been hypothesized to reflect a cooperative strategy (Buss, 1991a) or an altruistic proclivity (Denissen & Penke, 2008).

Based on these proclivities, it is reasonable to advance the hypothesis that dispositionally agreeable individuals tend to set their WTRs in a way that places the welfare of their social partners at a high value relative to their own. This provides a powerful selective rationale for why agreeableness is so highly valued across a variety of social relationships and the ability of observers to assess this personality trait in others, even based on physical appearance cues (Naumann, Vazire, Rentfrow, & Gosling, 2009; Yamagishi, Tanida, Mashima, Shimoma, & Kanazawa, 2003).
Just as stable physical traits such as strength and physical attractiveness influence WTRs, stable personality traits also reflect habitual WTRs. If this hypothesis is supported by future empirical tests, it provides at least one powerful selective rationale for the evolution of personality assessment adaptations. Evolution should select for difference-detecting adaptations to the degree that they aid in the solution to adaptive problems associated with the major classes of social relationships.

**Coalitions**

Just as wolves form packs for the purpose of hunting, humans historically formed coalitions for hunting, especially when the capturing and killing large animals requires the coordinated efforts of several or many (Tooby & DeVore, 1987). Humans are an unusually coalitionary species, however, in that they form coalitions for multiple functions. Historically, and in some cultures currently, females formed coalitions in order to render mutual assistance in child-tending (Tooby & DeVore, 1987). Male coalitions historically have had different functions—to attack other groups to capture fertile women or other reproductive resources; to defend their group against the attacks of other groups in order to protect their own reproductive resources; to hunt large game; and within-groups, to ascend status hierarchies (Buss, 2008).

Because the functions of male, but not female, coalitions involve tasks that require substantial physical strength and psychological bravery, it is reasonable to hypothesize that selection for inclusion in men’s coalitions should center on dimensions of individual differences likely to aid in the solutions to the adaptive problems for which coalitions are formed. Since female coalitions for goals such as mutual assistance in tending children require somewhat different traits for their success, women should attend to a somewhat different set of coalitionary criteria, such as social perceptiveness and conscientiousness.

DeKay, Buss, and Stone (unpublished ms.) explored these questions by asking men and women to evaluate how desirable 148 potential characteristics were in a coalition member. A coalition was defined as “a group of people with whom you identify because you pursue a common goal.” Each characteristic was rated on a scale ranging from –4 (extremely undesirable in a coalition member) to +4 (extremely desirable in a coalition member). The list of characteristics included markers of the major personality variables of Dominance, Agreeableness, Conscientiousness, Emotional Stability, and Openness, as well as characteristics derived from evolutionary hypotheses about the functions of mateships, friendships, and coalitions. For example, the item “Being brave in the face of danger” was included to test the hypothesis that two functions of male coalitions, and hence the basis on which men select coalition partners, are aggression against other coalitions for the goal of capturing reproductive resources and defense of one’s group from attack for the goal of protecting one’s reproductive resources.

Both men and women rated the following characteristics as highly desirable in a coalition member: Being hardworking, being intelligent, being kind, being open-minded, being able to motivate people, having a wide range of knowledge, having a good sense of humor, and being dependable (see also Cottrell, Neuberg, & Li, 2007). There were notable sex differences, however, that point to the distinct functions of men’s and women’s coalitions. Men, more than women, found the following characteristics to be significantly more desirable in coalition members: Being brave in the face of danger (2.40 for men v. 1.66 for women), being physically
strong (1.07 v. 0.43), being a good fighter (1.30 v. 0.42), being able to protect others from physical harm (1.37 v. 0.89), being able to tolerate physical pain (0.75 v. 0.36), being able to defend oneself against physical attack (1.90 v. 1.43), and being physically able to dominate others (0.35 v. – 0.42). Similarly, men evaluated the following qualities as more undesirable than did women in a coalition member: Being poor at athletic activities (– 0.68 v. – 0.23) and being physically weak (– 1.08 v. – 0.55).

Women, on the other hand, place an especially high premium on the trait of conscientiousness (2.63 for men, 3.36 for women) and being socially perceptive (2.23 v. 2.87) in coalition partners (DeKay et al., unpublished). These traits were hypothesized to be especially valuable for females that form coalitions for the purpose of mutual aid in child-rearing. Furthermore, women more than men especially dislike in coalition members the trait of sexual promiscuity, such as being sexually active with multiple partners (– 0.65 v. – 1.45). Sexually promiscuous women are likely to be especially undesirable for female coalitions because they interfere with other women’s long-term mating strategies (Buss, 2003). Sexually promiscuous women siphon off other women’s mate’s resources and also decrease men’s willingness to commit to long-term partners (Buss, 2003). In short, although women and men are alike in valuing some traits in coalition members such as dependability and intelligence, women and men differ in the traits they seek in coalition members, corresponding to the respective adaptive functions of coalitions and the problems faced within those coalitions.

This is merely one study using a limited sample of American undergraduates, so no grand conclusions can be drawn. Certainly, this study should be replicated in different cultures. Nonetheless, it is interesting to note that even in the modern context of an American university, seemingly so distant from the small-group warfare that characterized the human ancestral past, men still apparently select coalition members in part based on stable traits that will help the coalition to succeed in group-on-group aggression and physical defense.

Coalition members face other adaptive problems that can undermine the emergence and success of their coalition—defection and free-riding.

Defection sometimes occurs during war raids among the Yanomamö of Venezuela (Chagnon, 1983). While a coalition of Yanomamö approaches a rival coalition in preparation for an attack, a man will sometimes claim that he has a sharp thorn in his foot or a stomach ache, and so must turn back to the home base prior to the risky attack. These defections jeopardize the success of the coalition, since it is weakened by the loss of potential combatants, and relative group size often determines the outcomes of wars (Wrangham, 1999).

Another adaptive problem that confronts individuals who form coalitions consists of the potential costs inflicted by free-riders—those who obtain benefits from the successes of the coalition but fail to contribute their fair share of work to its successes (e.g., Price, 2005). In a hunting coalition, for example, when encountering a dangerous but desirable animal, an individual might surreptitiously recede from danger, allowing the others in the coalition to assume a greater risk, but then fully partake of the bounty of meat that results. The premium placed on the personality traits dependability and being hardworking in selecting coalition partners (DeKay et al., unpublished) may represent partial solutions to the problems of coalitional defection and free-riding. Other partial solutions may include punitive sentiments toward free-riders (Price, 2005) and ostracism of those who violate the coalition (Williams, Forgas, & von Hippel, 2005).
In summary, those who use stable personality traits such as dependability, hardworking, and bravery to select coalition members will accrue coalitional advantages over those oblivious to these traits. Iterated over time, selection favors the evolution of difference-detecting adaptations to identify coalition members with the right constellation of traits to solve problems involved in the formation and the implementation of coalitional strategies.

**Dyadic Alliances—Friendships**

There is good evidence that humans have adaptations to form friendships, including true friends characterized by deep engagement, as well as more casual reciprocity-based alliances (e.g., Tooby & Cosmides, 1996; Bleske & Buss, 2000). Just as with coalitions, friends can provide a bounty of resources that aid in the solutions to core adaptive problems—food, protection, deterrence of aggressors, assistance during times of sickness, help in finding and guarding mates, help with child-rearing, and many others. Friends can also inflict costs, creating new adaptive problems. And they can interfere with successful solutions to existing problems. Friends can become rivals for desirable mates, poach one’s existing mates, and betray the friendship in ways ranging from revealing damaging personal information to others to spreading false rumors (Bleske & Shackelford, 2001). If personality dispositions provide predictive information about the likelihood of receiving these benefits or incurring these costs, they should play a key role in friend selection. A friend low on dependability, for example, might become a “frienemy” by defecting or failing to show up during a time of need or by disclosing personal information to others that is damaging to social reputation.

Dekay et al. (unpublished ms.) found that both sexes placed a premium in friend selection on the traits of helpfulness, kindness, dependability, being hardworking, being open-minded, being intelligent, and having a wide range of knowledge—the high ends of the big five traits of agreeableness, conscientiousness, and intellect-openness. Bleske-Rechek and Buss (2001) found that revelations about a friend’s dishonesty, lack of trustworthiness, and disagreeableness figured heavily in reasons for the dissolution of friendships. Personality traits, in short, play a key role in the selection and dissolution of friendships. The recurrent statistical links between traits and resource accrual and cost infliction undoubtedly intensified selection pressure for the evolution of personality assessment adaptations.

It is important to note that personality traits carry with them costs as well as benefits (Nettle, 2006). High conscientiousness, for example, affords dependability, hard work, and delay of immediate gratification. At the same time, the conscientious ally, by delaying gratification, may show rigidity and miss immediate fitness gains (Nettle, 2006) that could benefit the friendship alliance. The conscientious ally also may also fail to be sufficiently flexible to adjust the steepness of the future discounting function as adaptive circumstances vary (see Wilson & Daly, 2004, for an example of men’s adjustment of future discounting as a function of exposure to attractive women). In short, although traits such as agreeableness and conscientiousness are highly valued in friends, these and other socially desirable traits carry costs, providing a selective rationale for the maintenance of individual differences on these traits (see other chapters in this volume for overviews of evolutionary forces that create and maintain individual differences).

**Rivals and Enemies**

Assessing the formidability of enemies is critical to making decisions about whether to confront them in an aggressive social interaction or placate them with submissive signals. Sell and his
colleagues (2009) provide compelling evidence that humans have cognitive mechanisms for assessing the physical formidability of others. People showed accuracy in assessing the physical strength of male targets when viewing photos of them. Even when viewing photos of faces from the neck up, people were accurate at inferring objectively-measured upper body strength. These findings provide evidence for a difference-detecting adaptation—the ability to accurately assess individual differences in physical formidability, which aids in the solution to problems related to aggressive social interactions.

A similar logic applies to the assessment of a rival’s personality traits. A rival high on trait aggressiveness and low on trait fearfulness poses greater physical risk than an enemy low on aggressiveness and high on fearfulness. An enemy’s stable level of intelligence may provide information about the likely sophistication and cunning of his or her future strategies of attack.

The traits relevant to evaluating an enemy’s formidability are likely to differ according to gender and adaptive circumstance. Traits such as bravery and fearlessness are more relevant to males when physical contests are involved in the rivalry. Traits such as emotional intelligence, low agreeableness, high physical attractiveness, bitchiness, and ability to gossip effectively may be more relevant assessing the formidability of a woman’s enemy. Traits such as Machiavellianism and psychopathy are probably relevant to assessing the formidability and malevolence of enemies of either sex. Personality assessment adaptations, in short, can help to better solve problems posed by social rivals and enemies.

Hierarchical Relationships

Humans live in groups, and all known groups contain status hierarchies— formal, informal, or both. Position in status hierarchies is linked to access to reproductively relevant resources, such as food, territory, health care, and mates (Buss, 2008). Occupying positions of subordinate status, equal status, and superordinate status clearly has been associated with different suites of adaptive problems over human evolutionary history (Buss, 2008). Among chimpanzee males, for example, those low in dominance get few copulations, and perforce pursue “sneaky” mating strategies (de Waal, 1982). Alpha chimp males gain most of the copulations, but must contend with sneaky males, females who sometimes accede to these sneaky males, and up-and-coming males who seek to challenge and displace the alpha.

In humans, men are highly responsive to relative status in their decision making about resources (Ermer, Cosmides, & Tooby, 2008). Men take more risks to recoup resource losses when they thought they were being viewed by other men of equal status, but not when they believed they were being viewed by men of higher status. This provides evidence that men are sensitive to relative status states, and vary their risk-taking behavior accordingly. The fact that these effects were found only for men, and not for women, adds support to the notion that status hierarchies historically were more central to men’s fitness than to women’s fitness (Buss, 2008).

Assessment of the personality traits of others aids solving problems associated with status hierarchies. Some individuals are dispositionally dominant, others dispositionally submissive—traits that can be inferred from the frequencies of observable dominant and submissive acts (Buss & Craik, 1980, 1981). Dispositionally dominant individuals, for example, tend to set goals for the group, take charge after accidents, demand that others run errands for them, take the lead in
organizing projects, and settle disputes among members of the group. Dispositionally submissive individuals tend to agree to the plans of others, even if they lack confidence in those plans; accept verbal abuse without defending themselves; apologize repeatedly for a minor mistake; say nothing when they get short-changed at the store; and say nothing when others cut in front of them in long lines (Buss & Craik, 1980, 1981).

Accurate assessment of these hierarchically-relevant personality traits provides information about solving problems within status hierarchies. When attempting to extract resources from someone within the status hierarchy, for example, greater success will be achieved by targeting dispositionally submissive rather than dominant individuals. Indeed, empirical evidence has documented precisely this among capuchin monkeys—they target those in subordinate positions for stealing food (de Waal, 1992). Knowledge about whether a new entrant to the group will pose a threat to one’s position in the status hierarchy is afforded by accurate assessment of dominant and submissive dispositions. Assessment of personality dispositions, in short, can provide valuable information about who is likely to ascend status hierarchies, as well as about who is likely to remain low in status or fall in status.

Finally, personality traits provide information about with whom to ally oneself for the goal of status ascension. The personality trait of ‘capacity for status’ (which is positively correlated with dominance) predicts who will rise in status in the future and conversely who is unlikely to rise (Gough, 1996). People can make better decisions about alliance formation for the purpose of elevating their status by accurate assessment of personality dispositions.

**Kin Relationships**

Genetic relatives can help an individual solve a host of adaptive problems. Parents, for example, can provide their children nurturance during times of illness, food during times of scarcity, and valuable information about strategies of hunting and gathering, tactics of hierarchy negotiation, or even effective methods of mating. Siblings can offer coalitional support against attack, protection from exploitative parents, stepparents, or other siblings, and more generally, important information about the social adaptive landscape.

Kin can also inflict costs, creating problems for their genetic relatives. Parent–offspring conflict theory (Trivers, 1974) predicts that children often have evolved adaptations designed to extract more resources from parents than is in the best interests of the parents to cede. Conversely, parents have adaptations to manipulate and exploit their children in ways that are not necessarily in the best fitness interests of those children. Fathers may divert resources away from one set of children in order to pursue new mating opportunities or to invest in children who are only half-siblings of the original set. Parents often discriminate, favoring one child over another in resource allocation, creating a problem of resource scarcity for the non-favored child. Parents may differ in their baseline welfare trade-off ratios for their different children, varying how much they prioritize their own interests relative to those of each child.

Children too can create adaptive problems for parents or help to solve problems for parents. Children who are sickly or needy can absorb a disproportionate share of parental resources. Children who inflict costs on their siblings damage their parent’s reproductive ‘vehicles.’ Conversely, children can be used or exploited by parents for solving adaptive problems. They can be forced to work on the family farm, literally or metaphorically. Daughters can be traded by polygynously inclined fathers to acquire additional wives, resulting in non-optimal mateships.
from the perspective of the daughters. Parents can manipulate their children to invest in their siblings to a degree that sacrifices the child’s interests to those of the parents.

Siblings sometimes attempt to out-maneuver one another in competition for parental resources. They can manipulate parents through information or disinformation into favoring them and disfavoring their sibling. Siblings even compete for available mates when their mating pools overlap. Sibling rivalry, in short, can extend over the entire array of reproductively relevant resources, given that, ceteris paribus, each sib is expected to value its own welfare twice as much as it values the welfare of its full siblings and four times as much as it values its half siblings.

If personality characteristics are statistically linked with problem creation and problem solution by kin, then accurate assays of those traits in kin can lead to superior decision making. Parents who are unreliable or emotionally unstable will be erratic in their provisioning of resources, protection, and health care. Children who accurately assess each parent’s level of reliability and emotional stability can make wiser decisions whether they can count on them during food shortages, conspecific threats, or bouts of illness, or whether alternative social allies need to be cultivated as backups for parents who are unreliable or emotionally unstable. Parents low on cognitive ability or social intelligence may give inaccurate or misleading advice about navigating the social landscape. Knowledge of the personality traits of parents, since they predict life-history strategies such as divorce, family abandonment, and infidelity, can help children to anticipate adaptive problems they will face in the future, and deploy anticipatory problem-solving strategies, such as cultivating back-ups. Accurate evaluation of these traits helps children to make better decisions about whose advice to trust and whose advice to ignore or discount.

Personality traits of children provide parents with valuable information about which of their offspring are likely to rise in the social hierarchy, which sons have good prospects for polygyny, and which daughters are likely to marry up. Given limited parental resources to invest in offspring, it should pay fitness dividends for parents to invest more resources in sons who are high on the personality traits of dominance, conscientiousness, intellectance, and capacity for status—traits linked with successful hierarchy negotiation (Lund et al., 2007) and hence traits likely to propel a son over the polygyny threshold.

The personality traits of siblings too are statistically linked to the creation and solution of adaptive problems. Siblings high on extraversion, for example, exude positive externalities by virtue of their success in commanding a wider social network and attracting a larger pool of possible mates. Allying with these extraverted siblings leads to access to these social resources. Siblings high on agreeableness, because of their greater empathy, trustworthiness, and other-oriented welfare trade-off ratio, can be relied upon as confidants who will not disclose vital personal information to others in damaging ways and will help rear or protect one’s own children. Conversely, siblings low on agreeableness, conscientiousness, and emotional stability inflict costs—the disagreeable through a selfish WTR, the unconscientious through unreliability and erratic follow-through, and the emotionally unstable through inability to handle stress in times of family peril. Distancing oneself from sibs low on these traits may reduce the costs associated with them.

Personality assessment adaptations, in short, contribute to successfully solving an array of problems posed by interacting with kin.
Mating Relationships
It is difficult to point to a more reproductively-consequential decision than selecting a long-term mate. Mates can potentially offer a bounty of direct and indirect reproductive benefits. They provide resources, offer protection in times of danger, invest in mutually-produced offspring, provide social connections that improve hierarchical position and coalitional strength, and afford help during fitness-threatening times of sickness or resource scarcity.

Conversely, mates can inflict an astonishing array of costs on their partners (Buss, 1989a, 1991b). They can act possessive or overly dependent, restricting freedom and absorbing resources that could be otherwise allocated. They can be unreliable, failing to provide resources promised or erratic in aid during times of need. They can be sexually unfaithful, diverting valuable reproductive resources to others. They can be moody or “high maintenance,” absorbing attention, effort, and resources that could be used to solve other adaptive problems. And they can be emotionally, physically, or sexually abusive, inflicting psychological, bodily, or reputational damage. Selection will favor the evolution of difference-detecting adaptations to the degree that personality traits in potential mates are recurrently linked with reproductively-relevant benefits and reproductively-damaging costs.

Empirically, personality characteristics do figure prominently in expressed mate preferences. Buss and Barnes (1986) found that nearly all of the dozen most highly rated qualities in a potential spouse consisted of personality traits—honest, considerate, affectionate, dependable, intelligent, kind, understanding, loyal, and faithful. Another study found that the upper ends of the major factors that comprise the five-factor model were preferred in a mate—extraversion, agreeableness, conscientiousness, emotional stability, and intellect-openness (Botwin et al., 1997). A study of 37 cultures found that personality traits figured prominently in expressed preferences for qualities desired in a spouse—dependable character, emotional stability and maturity, and pleasing disposition (Buss et al., 1990). Although mate preferences cannot always be translated into actual mating decisions due to constraints of one’s own mate value, operational sex ratio, and arranged marriages, a considerable body of evidence suggests that mate preferences often do affect actual mating decisions (Asendorph, Penke, & Back, in press; Buss, 2008; Buss & Schmitt, 1993; Luo & Zhang, 2009).

Some of these mate preferences are relationship-specific in their desirability (Łukaszewski & Roney, 2010). People prefer mates who are kind and trustworthy when these traits are directed toward themselves, friends, and family, but prefer somewhat lower levels of these traits when they are directed toward same-sex others. From a fitness standpoint, to the degree that kindness reflects a generous, benefit-bestowing disposition, individuals should want from a mate benefits bestowed on themselves, their family, and their friends rather than indiscriminately. In short, extensive findings from studies of expressed mate preferences reveal that personality traits are extremely important in selecting a long-term mate and that mate selectors want trait-representative behavior to be differentially directed toward themselves, family, and friends.
From the current theoretical perspective, we can ask: Which adaptive problems are solved by selecting mates with these traits? Conversely, which costs are avoided by selecting mates with these traits?

The trait agreeableness reflects a cooperative strategy (Buss, 1991a) and an altruistic proclivity (Denissen & Penke, 2008). Those who score high on agreeableness display more altruistic behavior in public goods games (Koole, Jager, van den Berg, & Hofstee, 2001). Interestingly, stable altruistic dispositions can be identified with some level of accuracy merely from viewing 20 second silent video clips of individuals (Fetchenhauer, Groothuis, & Pradel, in press). There is even evidence that cooperators look different from cheaters (Yamagishi et al., 2003). A cooperative strategy and altruistic proclivity are likely good indicators that a mate who can be trusted is unlikely to defect, will make personal sacrifices, and will invest time and resources in partners and offspring. Agreeableness, in short, is an excellent indicator of good long-term partner traits as well as good parenting proclivities (Miller, 2007).

Low agreeable individuals, because of their selfishly-skewed welfare trade-off ratios, tend to impose multiple costs on their long-term mates, creating adaptive problems for them (Buss, 1991b). They tend to be verbally and physically abusive. They tend to be selfish and self-absorbed. They tend to be sexually unfaithful (Schmitt, 2004). Unfaithful males divert resources to other women. Unfaithful females inflict the problem of paternity uncertainty on their partners. In short, those low on agreeableness inflict a host of costs on their mates, while those high on agreeableness confer a bounty of benefits.

The trait of conscientiousness reflects the capacity for reliable work and enduring commitment (Buss, 1991a, 1996), dependability in adhering to social contracts such as marriage, and tenacity in goal pursuit (Denissen & Penke, 2008). Conscientiousness is also an excellent empirical predictor of educational attainment, job security, higher occupational status, greater financial security, and higher levels of commitment in social relationships (Larsen & Buss, 2010). Those high in conscientiousness put in long hours of hard work needed to get ahead in work and status hierarchies (Lund et al., 2007). Conscientious individuals, in short, have the capacity to acquire economic resources reliably over time and commit those resources to a mate and children. Those low on conscientiousness are particularly susceptible to engaging in sexual infidelity, including one-night stands, brief affairs, and serious affairs (Buss & Shackelford, 1997; Schmitt, 2004). Finally, impulsivity, the low end of conscientiousness, predicts divorce, particularly among men (Kelly & Conley, 1987).

The third major personality trait highly valued in long-term mates is emotional stability. Emotional stability has been conceptualized as the ability to handle stress while remaining on an even keel (Buss, 1991a). Those high on emotional stability suffer fewer mood swings, experience less fatigue over the course of the day, and report better physical health and fewer physical symptoms than their more emotionally unstable counterparts (Larsen & Buss, 2010). Those low on emotional stability inflict many costs on their long-term mates and drain reproductively-relevant resources from them. They tend to get depressed and suffer from anxiety, especially social anxiety. Spouses of emotionally unstable individuals report that their mates are possessive, jealous, dependent, and abusive—qualities that absorb valuable psychological, temporal, and physical resources from their partners, as well as inflicting opportunity costs on them (Buss, 1991b). Time and energy devoted to calming and placating an anxious, clingy, needy, jealous, and abusive partner is time and energy that cannot be devoted to solving other adaptive problems.
On the other hand, emotionally unstable individuals often invest heavily in their close relationships (Denissen & Penke, 2008b) and may provide benefits in the form of greater vigilance to dangers (Nettle, 2006).

In summary, there is compelling empirical evidence that personality traits, particularly agreeableness, conscientiousness, and emotional stability, are reliably associated with a host of social adaptive problems involved in mating. Those high on these traits provide a bounty of reproductively-relevant benefits, and are reliable indicators of being a good parent and a good partner (Miller, 2007). Those low on these traits inflict reproductively-relevant costs, drain resources, and create adaptive problems that could be avoided by choosing mates who score higher on these traits. The recurrent statistical links between personality traits and the provision of benefits and the infliction of costs provides a compelling theoretical rationale for why these traits are so highly valued in mate selection. Personality assessment adaptations, in short, have evolved in part to solve adaptive problems associated with the reproductively-consequential decisions of mating.

Adaptations for Self-Assessment

Just as social adaptive problems can be better solved by detecting differences in others, many of these problems also can be more effectively solved by adaptations that assess personal qualities of the self. Some of these personal qualities may reflect temporary states: Am I healthy enough to confront a rival today? Am I angry enough to sever a friendship with someone who has failed to reciprocate a favor or let me down at a time of need? Do I currently have sufficient status and respect among members of my coalition to attempt to oust its current leader? Other adaptations involve assays of one’s own personality traits and enduring physical attributes such as formidability and physical attractiveness.

Tooby and Cosmides (1990) provide one candidate self-assessment adaptation with their concept of reactive heritability. Individuals show heritable differences in their size, muscularity, and athleticism. Self-assessment of these traits provides input into decision rules about whether to adopt an aggressive or cooperative social strategy. Those who are more physically formidable can better succeed in adopting an aggressive strategy, such as using implied threats to get others to cede resources to them. Thus, accurate self-assessment of stable physical characteristics can aid in the solution to social adaptive problems. Fox (1997) found evidence for spontaneous self-assessment of fighting ability. Men engage in more frequent spontaneous self-assessment of fighting ability relative to other men than do women relative to other women, suggesting a sex-differentiated design feature inherent in this self-assessment adaptation.

A similar logic applies to self-assessment of stable psychological qualities. The success of an aggressive strategy depends not merely on physical formidability, but also on stable personality traits such as bravery or courageousness, or conversely cowardice or fearfulness. If accurate self-assessment of these personality traits leads to better decisions about whether an aggressive social strategy will succeed or fail, selection will favor the evolution of adaptations to self-assess on these personality traits.

Analogous logic applies to self-assessment on other personality traits. As noted earlier, the trait of conscientiousness indicates the capacity for reliable work and enduring commitment (Buss, 1991a) and tenacity of goal pursuit (Denissen & Penke, 2008). Accurate self-assessment about one’s own level of conscientiousness could aid in the solution to a suite of adaptive problems: Decisions about the type and magnitude of work tasks to tackle, decisions about the quality of
mates to seek, and decisions about the amount of coalitional responsibility one can afford to take on. If conscientiousness reflects tenacity in goal pursuit and this trait translates into resolve in mate pursuit, then individuals high on conscientiousness may choose to go after mates at the top end of their mate-value range, knowing that their persistence often pays off (Buss, 2003). Self-knowledge will lead the high conscientious individual to take on a heavier load of coalitional responsibility, safe in the knowledge that he or she will not let the group down. Accurate self-evaluation of one’s level of conscientiousness should also lead to better decision-making about level of aspiration within some work hierarchies. Circumstantial evidence for this hypothesis comes from studies that show that the trait of conscientiousness is linked with effective tactics of hierarchy negotiation in the workplace (Lund et al., 2007).

A cardinal benefit of self-assessment is social self-assessment—a realistic appraisal of one’s social reputation (Craik, 2008). Consider the adaptive problem of reputational damage repair. Knowledge that others view you as undependable or uncooperative highlights the domains in which reputation damage repair is needed. Knowledge that others assess you as intelligent or socially savvy highlights social niches in which you are likely to reap social dividends, such as solving complex social problems for the group. Knowledge that others fear you because of your hair-trigger aggressiveness will facilitate the success of a strategy of getting others to cede resources in order to avoid a damaging fight.

These hypotheses do not imply that self-assessment will be designed to be perfectly accurate. Indeed, just as it sometimes pays to exaggerate one’s level of desirable traits in the early stages of mate attraction, especially for short-term mating (Buss, 2003), the effectiveness of these exaggerations may be facilitated by a certain amount of self-deception (Trivers, 2000).

In summary, just as assessing the personality dispositions of others affords better solutions to a suite of social adaptive problems, self-assessment mechanisms can also yield better solutions to adaptive problems. They provide information about which social strategies will yield the largest payoffs, which social niches can be effectively exploited, the areas in which effort to reputational repair must be allocated, and the attainable levels to which one should aspire in domains such as mating and hierarchy negotiation.

**Discussion**

Most work in evolutionary personality psychology has been dedicated to discovering the evolutionary origins of stable individual differences (e.g., Camperio, Ciani et al., 2007; Hawley, 1999; Keller & Miller, 2006; Nettle, 2006; Penke, Denissen, & Miller, 2007; Wilson, 1994; Wolf et al., 2007). This chapter, in contrast, flips the perspective and looks at evolutionary personality psychology through the lens of hypothesized difference-detecting adaptations.

Abundant evidence supports the hypothesis that humans have evolved adaptations for detecting and acting on individual differences in physical traits such as attractiveness (Sugiyama, 2005) and formidability (Fox, 1997; Sell et al., 2009). This paper elaborates on the hypothesis that humans also have evolved adaptations for assessing and acting on individual differences in personality traits (see Buss, 1991, 1996). The selective rationale for the evolution of personality assessment adaptations is based on the notion that, over evolutionary history, there have been recurrent statistical links between personality traits and the creation of, and solution to, social adaptive problems.
This chapter summarizes available evidence that supports the hypothesis that personality traits indeed are reliably linked with the conferral of specific classes of benefits and the infliction of specific classes of costs. Consequently, forming relationships with individuals possessing relevant personality traits can aid greatly solving some adaptive problems and avoiding being forced to allocate effort to others. The costs and benefits reliably linked with personality traits have profound fitness-relevant consequences for individuals in forming mating relationships, coalitions, friendships, hierarchical relationships, and kinships, as well as for evaluating enemies. Many of the hypotheses advanced in this paper about the statistical links between personality traits and the bestowal of benefits and infliction of costs remain to be tested empirically. Future work will undoubtedly reveal other costs and benefits associated with personality traits. For example, which personality traits are differentially linked with value as a short-term mate versus value as a long-term mate? Is conscientiousness, because of its connection with tenacity of goal pursuit, linked with formidability in social conflicts involving wars of attrition? Which personality traits afford more value in friendships characterized by deep engagement versus those characterized by more superficial reciprocal exchange? Will an analysis of the facets of the global personality traits provide a more incisive level for examining the costs and benefits associated with traits in different types of relationships? Do humans have adaptations for tracking other units of personality, such as personal projects (Little, 1983). In this sense, the current chapter provides just the start of a foundation for a future research agenda on personality assessment adaptations. This discussion highlights some cornerstones of this foundation.

Welfare trade-off ratios. A key component of this foundation will involve documenting the links between personality traits and habitual welfare trade-off ratios—the value people place on their own interests relative to the value that they place on the interests of those with whom they form relationships. Do those high on narcissism, for example, truly have selfishly-skewed baseline WTRs, or do they emit positive externalities as yet undocumented that offset their seemingly selfish WTRs? Do those high on agreeableness truly have a relatively selfless WTR, or is their altruism so easily afforded that it is an honest signal of their greater resource holding potential? Or do agreeable individuals inflict negative externalities in other domains such that their selflessly-skewed WTR is needed to compensate for those negative externalities? Habitual WTRs are likely to have profound consequences for all social relationships, from kinships through mateships.

Accuracy, predictable inaccuracy, and co-evolutionary arms races in adaptive personality assessment. All else being equal, accurate personality appraisals should be favored over inaccurate or biased appraisals. But there are several principled reasons for expecting personality assessment adaptations to be predictably inaccurate. One is based on error management theory (Haselton & Buss, 2000). When assessing the personality traits of strange males, for example, it may pay to be adaptively biased—erring on the side of attributing higher levels of aggressiveness in order to avoid the more costly error of assuming agreeableness when the individual is pursuing a cost-inflicting strategy. Another source of inaccuracy comes from self-presentation. There are sometimes fitness benefits to concealing one’s undesirable traits and exaggerating one’s possession of desirable traits. A man might inflate his bravery and fearlessness when attempting to gain entry to a coalition. A woman might suppress her emotional instability and a man might exaggerate his ambitiousness in the early stages of mate attraction. Furthermore, there are predictable individual differences—
those high on narcissism, for example, habitually self-enhance in their presentation to others (e.g., Buss & Chiodo, 1991; John & Robbins, 1994).

As a consequence of these predictable forms of inaccuracy, co-evolutionary arms races may develop between difference-detecting adaptations and trait-relevant behavioral displays. Difference-detectors should be selected to discount unreliable information, much as women are ‘commitment skeptical’ about men’s declarations of the depth of their feelings in mating contexts (Haselton & Buss, 2000). Personality assessment adaptations should be selected to correct for predictable forms of biased self-presentation. And strange males should exaggerate their level of agreeableness in order to compensate for the error management biases that difference-detectors have about them, resulting in the co-evolution of a more stringent error management bias on the part of detectors.

Co-evolutionary arms races will also emerge in presentation and interpretation over the personality components of social reputation. Just as people might show a positivity bias about the personality information they convey to others about their close friends, mates, and valued kin, receivers should be selected to discount personality information, or gauge its accuracy according to the source of the reputational information. Analogously, mating rivals can be expected to bias the personality information they convey about their competitors in a derogatory fashion, while receivers can be expected to correct for these source biases (Buss & Dedden, 1990).

Empirical research is needed to uncover error-management biases, source biases, and other causes of predictable biases, as well as the footprints of co-evolutionary arms races pertaining to personality presentation and difference-detecting adaptations.

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Design features of difference-detecting adaptations. Future research also will have to explore other design features of personality assessment adaptations. It will have to uncover the sources of information people use to assess these individual differences, such as manifest act frequencies (Buss & Craik, 1983), non-verbal cues (Naumann et al., 2009), habitat (Gosling, Mannarelli, & Morris, 2002), and social reputation (Craik, 2008). Research on personality assessment adaptations will explore not only problem-specific shifts in which traits are relevant, but also whether there are problem-specific shifts in which cues are given primacy and which are discounted. And it will have to explore how individuals integrate conflicting cues, as when a hostile and aggressive social reputation conflicts with an observed high frequency of agreeable acts.

Future research also will have to identify the generality or specificity of the traits in their importance across different social relationships and relevant adaptive problems. Dependability, for example, may be important for coalitional allies, friendships, mateships, kinships, and even hierarchical relationships. Dispositions for sexual fidelity, in contrast, may be most central to long-term mating relationships and less important for friendships (Shackelford & Buss, 1996). The finding that personality traits represented by the five factor model emerge as important in so many different relationships, from mate selection to hierarchy negotiation, suggests some degree of domain-genericity. On the other hand, even these global traits show some level of differential desirability by sex and by relationship type. Furthermore, the facets subsumed by the global traits may show greater domain-specificity with respect to solving social adaptive problems. The anxiety facet of emotional stability, for example, may be most relevant to solving adaptive problems requiring vigilance to threat. The angry hostility facet, in contrast, may be a useful facet in coalitional aggression.
A further complexity centers on the concept of *multiple social personalities* (Craik, 2008). Although this chapter has focused primarily on global stable personality traits, it is likely that individuals display different personality traits to different individuals, different social groups, and in different contexts. Students may view a male teacher as conscientious at school while his spouse sees him as an untidy slob in the home. A person may assess a sea captain as trustworthy with respect to his life, but not trustworthy with respect to his wife.

Finally, the research agenda on adaptive personality assessment will require identifying the behavioral output—the adaptive action—deployed toward solving each of the key problems deeply enmeshed with these stable individual differences. One form of behavioral output will be the selection or rejection of individuals as mates, friends, coalition partners, and kin based on their personality traits. Within these social relationships, after

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selection, adaptive problems associated with traits must be solved. A person might go to a socially intelligent friend for sage advice about navigating a social dilemma, beseech an agreeable friend to defend his reputation when it is attacked, and enlist the brave and physically formidable friend to deter enemies from aggressing. A man might expect an agreeable mate to comply with a sexual request, an extraverted mate to help build his hierarchical alliances, and a conscientious mate to keep the home fires burning while he is exploring new territory for resources. All of these complexities point to a rich conceptual and empirical agenda in the study of difference-detecting adaptations.

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**References**


DeKay, W.T., Buss, D.M., & Stone, V. (unpublished ms.). Coalitions, mates, and friends: Toward an evolutionary psychology of relationship preferences. Department of Psychology, University of Texas, Austin, Texas.


Fetchenhauer, D., Groothuis, T., & Pradel, J. (in press). Not only states but traits—Humans can identify permanent altruistic dispositions in 20 s. Evolution and Human Behavior.


Part II Developmental and Life History Perspectives on Personality
3 The Role of Competition and Cooperation in Shaping Personality: An Evolutionary Perspective on Social Dominance, Machiavellianism, and Children’s Social Development

Patricia H. Hawley

Everyone says you should be nice all the time but no one really is, so if you do what you should and be nice all the time, you’re probably gonna get screwed.

The above quote was drawn from Claudia Hart’s *A Child’s Machiavelli: A Primer in Power* (1998). Part of the appeal of this tiny and charmingly illustrated book is how Ms. Hart brilliantly captures Machiavellian philosophy in childlike terms and in ways that ring true. What is more, the quote conveys what every evolutionist knows; unrestrained altruism is a strategy that generally does not pay.

Can children really be so diabolical, as the Machiavelli reference suggests? His name, after all, evokes the scoundrel concerned only with personal power and glory. The psychological literature on Machiavellianism surely reflects this interpretation (e.g., Paulhus & Williams, 2002). Or shall we instead heed Isaiah Berlin’s alternative analysis, that Machiavelli’s treatise is an astute commentary on human nature, one that simply reflects a pagan (power, competition, social and material success) rather than a Christian morality (divinity, sin, redemption; Berlin, 1979/2001)? If we accept Berlin’s unemotional view, we can see a striking resemblance to the theory of evolution by natural selection: Success is not defined by forgiveness and renunciation of worldly goods, but rather by social and material reward, the delicate balance of self and other, and ultimately, of course, reproductive success.

The present chapter attempts to describe and explain children’s social and personality development through evolutionary lenses. I will ultimately frame Machiavellianism in evolutionary terms, but the first step to doing so is reintroducing a construct that has been of interest to behavioral ecologists and evolutionists for decades; namely, social dominance. Even though I have maintained the construct’s agonistic, competitive flavor found in the literature of origin, I have deliberately shifted the focus of the measurement model from the form of behavior (agonistic contest) to its function (resource control). By doing so, I have moved gently away from its zoological roots to firmly integrate prosociality and its central role for achieving power in social groups (Hawley, 1999a). Now the way is paved for an evolutionary understanding not only of children’s personality development (including Machiavellianism), but also of their interpersonal relationships and the nature of human social competence itself.

I will begin by briefly outlining the modern history of evolution and individual differences, including key concepts (e.g., phenotypic plasticity) and a very promising meta-theoretical perspective (life history theory). I will then reintroduce the construct of ‘social dominance’ and present arguments to support the critical roles of both prosocial and antisocial behavior in inter-individual competition. Third, I will sketch out the theoretical (and methodological) implications of social dominance relations for human personality development, and then exemplify with a theory (Resource Control Theory; Hawley, 1999a) and body of empirical work exploring children’s social dominance relations and the strategies they employ, including a mixed strategy I
have referred to as Machiavellian behavior (Hawley, 2003). I will close by suggesting that human personality cannot be fully understood without evolutionary theory.

**Evolution and Individual Differences**

Until fairly recently (e.g., after the mid-1990s), “the evolution of personality” was considered nearly an oxymoron. “Adaptations are universal” and individual differences are thought of as mostly error variance (but see Tooby & Cosmides, 1990). At the same time, however, animal behavior researchers (Figueredo, Ross, & Petrinovich, 1992; Hawley, 1994) and practitioners (Forkman, Furuhaug, & Jensen, 1995) were busily discovering the utility of measuring animal personality and drawing both cross- and within-species comparisons (the theoretical foundations of which have since been well explicated: Wolf, van Doorn, Leimar, Weissing, 2007; Gosling, 2001; McElreath & Strimling, 2006).

The logic in dismissing individual differences is fairly simple: If natural selection (i.e. stabilizing or directional, v. disruptive) bears ‘universal adaptations,’ then how can it be used to explain human individual differences? Natural selection of course acts on naturally occurring phenotypic and genotypic variability as the raw material for evolution. Any leftover individual differences is considered by most as ‘noise.’ Accordingly, most evolutionary psychologists (with some notable exceptions) have traditionally set their sites on human universals. As such, individual phenotypic differences have tended to be minimized in the evolutionary psychology enterprise except for broad classifications presumed to be central to sexual selection such as gender and stage in the lifespan.

**Polymorphic Diversity**

In contrast to the (implausible) ¹

¹ Implausible because a) there is good evidence in other taxa that individual differences are anything but ‘noise’ (see polymorphic diversity below), b) selective neutrality may be difficult to achieve in terms of genetic probabilities, (e.g., Ford, 2002), and c) human individual differences have been shown to predict very important outcomes for over a century (Buss & Greiling, 1999; Ozer & Benet-Martinez, 2005).

idea that individual differences are merely error variance, game theoretic models and field research have demonstrated unequivocally that multiple morphs can co-exist in a population, each taking advantage of a slightly different environmental niche (West-Eberhard, 2003). Long common to most genetic polymorphism models is the assumption that variegated phenotypes will evolve to some equilibrium, each enjoying comparable average fitness payoffs (e.g., Haldane, 1930; West-Eberhard, 2003). Accordingly, the evolution of this theoretical state of equilibrium has been handled well by game theoreticians who evoke the terms ‘players,’ ‘strategy,’ ‘mixed strategy,’ ‘evolutionarily stable strategies (ESS),’ and conditional strategies (exempt from the equal payoff assumption), etc (e.g., Lewontin, 1961; Colman, 1999; Maynard Smith, 1974). Noted by Darwin himself (1871), reproductive polymorphisms are the most common (e.g., male, female). Trophic polymorphisms are perhaps the most striking; conspecifics appear so different that they are mistaken for different species (many species of fish and insects, for example: Gadgil, 1972; Gross, 1991; Van Duzee, 1917).

Several interrelated mechanisms can give rise to variegated intraspecific phenotypes; genetic/allelic differences (e.g., sex; maintained by frequency dependent advantage), genetically and environmentally regulated phenotypic plasticity (see below), and, less relevant to the present
discussion, developmental stages (think caterpillars to butterflies). From a behavioral genetic position, intraspecific polymorphisms associated with distinct genotypes are the most remarkable. Sex determination in humans may be considered a relatively simple case; the presence or absence of the SRY gene determines sex.

The most common selective mechanisms that have been proposed in psychological circles for genetic polymorphisms associated with behavior are related to frequency or density dependence \(^2\) (see also balancing selection); that is, the relative advantage of a given genotype depends on the relative frequency of other genotypes/phenotypes in the population (Tooby & Cosmides, 1990; Wilson, 1998). Mealey (1995), for example, entertained the relative advantages of a psychopathic phenotype when it is rare. Strategies characterized by defection are effective so long as there is a population of susceptible targets (i.e., non-psychopaths). Other traits discussed as plausible in humans include female sexual strategies (Gangestad & Simpson, 1990), the shyness-boldness continuum (Wilson, 1994), and the Big 5 personality traits (Figueredo et al., 2005; Nettle, 2005; MacDonald, 1995).

Though genetic polymorphisms have been documented in many taxa and are essentially non-controversial, genetic polymorphism in human personality traits have proven far more elusive. We now have more than enough evidence to conclude that human personality traits are heritable (see Bouchard & Loehlin, 2001 for review). From this basic reality, however, one cannot conclude that the underlying genetic mechanisms are simple (i.e., based on few loci), a prerequisite for balancing selection models (Penke, Denissen, & Miller, 2007). In fact, to date there is little compelling evidence to suggest that measurable human personality traits are associated with single loci, or even an identifiable few (Burton et al., 2007). I will leave the complexities of genetic models to colleagues much more knowledgeable on the matter (e.g., Penke et al., 2007; Keller & Miller, 2006; Gangestad, Penke, and Miller, all in this volume). Suffice it to say here; frequency-dependent theoretical models have tremendously appealing heuristic value for personality traits in general, and even Machiavellianism in particular (see, e.g., Wilson, Near, & Miller, 1996; Hawley, 2006; Jones & Paulhus, 2009). That being said, cautious readers should be aware that such models may not be supportable with genetic realities (Miller, this volume), but stirring discussions are presently emerging in the literature.

**Phenotypic Plasticity**

Perhaps the most promising model for evolutionary discussion of individual differences with the widest applicability (especially from developmental perspectives like the one represented in this chapter) is one of developmental plasticity and its overarching meta-theoretical framework, life history theory (LHT) (Charnov, 1993; Roff, 1992: a.k.a. developmental evolutionary ecology; Chisholm, 1999). The appeal lies in LHT’s explicit recognition that organisms are flexible in their developmental course and responsive (though not infinitely) to complex inputs from their physical (e.g., temperature, resource scarcity, predation pressures) and social environments (e.g., intra-sexual competition, quality of parental investment, population density). LHT approaches can be applied to both cross-species and within-species questions in that each species has an evolved modal range of responses around which individuals vary in response to local socioecological conditions (West-Eberhard, 2003). This ‘variation’ includes not only behavioral traits (e.g., ‘aggressiveness’ in humans; Tooby & Cosmides, 1990), but also physical
characteristics (e.g., environmental sex determination in some turtles, snakes, and reptiles; Bull, 1983).

Growth (somatic), development, and reproduction (timing of sexual maturation, parental investment, quantity of offspring, etc) involve a lifelong stream of adaptive tradeoffs for allocating resources towards competing life functions. Life history orientations attempt to understand the coherency across the life span of the stream of resource allocation ‘decisions’ across species or individuals, not as consciously executed choices, but rather as genetically coded, environmentally-conditioned switches (West-Eberhard, 2003). Accordingly, life historian biologists and psychologists have aggressively sought to understand gene by environment by phenotype interactions and, in doing so, have documented a remarkable degree of condition sensitivity in even the ‘simplest’ organisms (West-Eberhard, 2003). These models focus on species—typical, environmentally-sensitive regulatory mechanisms (Reznick, 1982; Figueredo, Vásquez, Brumbach, & Schneider, 2004). Key environmental inputs (e.g., harshness, unpredictability; Brumbach, Figueredo, & Ellis, 2009) trigger a cascade of genetically-regulated hormonal and neurological events, calibrating the system towards what is presumably a favorably competitive strategy (strategic differentiation) given the environmental conditions that the organism finds itself in at various points of development.

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Work in rodents, for example, has shown that systematic variation in physical environments influences parent–offspring interactions which then calibrate the stress-response system. In turn, the organism is biased towards either a fast (fearful, early mating) or slow (explorative, delayed mating) reproductive pattern (see Ellis, this volume, for details). Indeed, life history work has typically focused on the timing of reproductive maturation and parental investment strategies (including offspring quality) and antecedent growth patterns (i.e., current vs. future reproduction tradeoff; Chisholm, 1999), the tradeoff of which positions the life history strategy on a fast-slow continuum (early reproduction, briefer gestation vs. protracted development, later reproduction, longer gestation). “The core assumption of LH theory is that over evolutionary and developmental time, individuals and lineages systematically adjust LH strategies in response to specific risks and opportunities afforded by the environment...” Ellis, Figueredo, Brumbach, & Schlomer, 2009, p. 211). (For interesting applications in human reproductive strategies, see Draper & Harpending, 1982; Figueredo et al., 2006; del Giudice, 2009; Ellis, 2004).

From behavioral ecological perspectives, this latter notion of risk refers to the variance around a mean of expected outcomes, spanning from gains to losses (Figueredo & Jacobs, in press). “Loss” might include a low payoff for investment of time that could have been spent on more beneficial activities, or at the extreme, death. Thus, two strategy trajectories may have equal mean level rewards, but one may be ‘riskier’ in terms of the potential costs that may be incurred. Social scientists tend to view behaviors designed for long term gains more favorably than those that produce short term gains and long term losses (Figueredo & Jacobs, in press); accordingly, developmentalists label behaviors that increase the probability of negative developmental outcomes (teen pregnancy, alcohol related accidents) as ‘risk’ behaviors (sex, alcohol use). This notion of risk will be readdressed below.

Three primary points from LHT are important for the discussion that follows. First, is the notion of organismic flexibility; namely, many organisms—including humans—have evolved a degree of intrinsic flexibility to extrinsic forces, including structured responsivity to key social and material cues. This first point is non-controversial. Second, early exposure to factors such as poor
quality parenting, unpredictable and/or harsh environments, and resource scarcity can calibrate behavioral systems in ways that may create enduring individual differences (traits) that, in LHT parlance, can be considered ‘strategies’ (West-Eberhard, 2003; Ellis et al., 2009). Finally, frequency-dependent models of selection and LHT both suggest that a) variegated behavioral phenotypes coexist in populations, b) individual differences in behavior (including sociality) can be seen as inherently competitive and strategic (individuals attempt to maximize gains in the presence of others who are also attempting to do so), and c) environmental cues (including characteristics of one’s own phenotype) carry information about important aspects of the competitive landscape.

The present perspective focuses on inter-individual competition and claims that said competition is at the core of human sociality and interpersonal relationships. In order to understand human personality development, one must first visit differences in relative competitive ability known as social dominance.

**Competition and Social Dominance**

The competitive essence of natural selection suggests that individualism and ‘selfishness’ are inherent to organisms and this individualism is well captured within a good many of phenomena studied in evolutionary psychology circles, including the life history and game theoretic approaches outlined above. Evolutionists well know, however, that this competition need not take overt, agonistic forms. In fact, extreme altruism could out-compete alternate phenotypic variants via multilevel selection forces (e.g., Wilson & Sober, 1994).

Yet, at the same time, these highly visible agonistic contests (in general predicted by size, pugnacity, and sex; Pusey & Packer, 1997) seem to have cornered the market in the behavioral ecology literature on social dominance and dominance hierarchies (a “sexually selected manifestation of conflict”; Alcock, p. 332: see also Krebs & Davies, 1997; Hofer & East, 2003; Pelletier & Festa-Bianchet, 2006). The conspicuous peck-order of chickens gave rise to this aggression-based view of social dominance (Schjelderup-Ebbe, 1922), and was promptly similarly applied to social groups of children (e.g. Bühler, 1927). The legacy of this work has been the continued focus on the form of behavior (the measurement model defined by agonistic contests, fighting ability, submissive gestures), even though the function of such contests is always understood to be resource control (the theoretical model; see Hawley, 1999a, b for extended discussion).

Post dominance affiliative interactions (reconciliation) have been proposed to maintain social standing and group cohesion (de Waal, 2000; Aureli, Cords, & van Schaik, 2002).

As a consequence of this focus on form over function, socially dominant individuals (typically males) are generally thought to hold the central positions in their social groups (e.g., attention, ally selection, grooming, priority access to mating; Dunbar, 1988; de Waal, 1982).

The above described mismatch between the measurement and theoretical models inherent in the social dominance literature has led to a number of quandaries and theoretical oversights. First, the social patterns associated with social dominance defined by agonism found in the animal
behavior literature only seem to apply to humans up to a certain point in development; namely, even successful agonistic competition in isolation will not lead to social centrality beyond the age of five (Hawley, 1999a). This discontinuity (more apparent than real) between humans and animals dealt a blow to the research (e.g., ethology) that was seeking to extend itself to the study of human behavior (Vaughn, 1999). Second, the thrill and allure of agonistic contests led (and continues to lead) to an unnecessary focus on masculinity and sexual dimorphism, and a prejudicial oversight of the more subtle yet significant (e.g., long-lasting) manifestations of competition among females (Hrdy, 1981/1999; Hawley, Little, & Card, 2008). Third, and most important for the present chapter, by shifting focus and attending to function first (and form secondarily), the social dominance construct not only can be brought firmly in line with other psychological constructs of profound theoretical significance (e.g., social competence in humans; Hawley, 2002), but also be shown to play a central organizing role in human social groups, as biological approaches have long suggested it should.

To grasp the implication of this shift for personality development in general, and Machiavellianism in particular, the evolution of prosociality must be briefly considered.

Cooperation as Competition

Darwin’s ‘struggle for existence’ (Darwin, 1859) unnecessarily creates powerful imagery of aggressive competition both within and between species. This alignment is especially unambiguous in the sexual selection literature (Darwin, 1871; Trivers, 1972; Pellegrini & Archer, 2005). Yet, early lesser known writings (e.g., Kropotkin, 1902) contended that under harsh conditions and low population density, cooperation would evolve over aggressive competition. According to this minority view, the “struggle for existence” also demands ‘individual against environment’ and that this struggle could best be won via mutual aid (see also Wilson, 2006).

Trivers’s seminal work on reciprocal altruism (1971) united individual level selection with a viable theory of other-oriented behavior (see also Hamilton, 1964). Here, “altruism” bears limited cost when one considers delayed social and material benefits; that is, social individuals perform other-oriented acts with the implicit expectation that favors will be reciprocated (see also social exchange theory: Byrne, 1964). Thus, in a very real sense, material goals can be achieved via sociality as well as aggression.

A Dualism in Human Nature

This dualism—that competitive forces give rise to both antagonistic and other-oriented behavioral strategies—underlies the present theoretical perspective, resource control theory (RCT; Hawley, 1999a). 6

6 The bulk of psychology considers antisociality and prosociality opposite ends of a single continuum and, as such, assumes that they serve opposing functions. In contrast, here they are considered to serve the same function or “two sides of the same coin” (Hawley, 2002; Charlesworth, 1996). As a consequence, they may be assumed to be either independent or positively related (for extended discussion see Hawley, 1999a).

According to the outline above, successful competition can be achieved directly or indirectly. Direct means—without consideration for the goals and motivations of others—are relatively straightforward (agonistic contests, aggression). Indirect means of resource competition derive from evolutionary models of cooperation (e.g., Charlesworth, 1996). Here, competition takes on a more non-zero-sum quality; interaction partners both stand to gain in this cooperative or reciprocal context (but not necessarily equally). Instead of bypassing the social group as direct means do, indirect strategies exploit the mediating effect of the social group to access to
resources *prosocially*. Thereby are the social groups’ norms followed, social relationships spared, and material resources won. There is no reason to assume that this ‘cooperation’ assumes any altruistic flavor either at the level of the individual or gene.

Resource control theory attempts to capture this dualism concretely and directly in its translation of these theoretically-based strategies into its measurement model. Competition comes in at least two broad forms: *Coercive strategies of resource control* are direct, aversive, and immediate (e.g., taking, threatening) and as such are derived from traditional conceptions of agonistic social dominance of behavioral ecology. *Prosocial strategies of resource control* include reciprocity, cooperation, and positive alliance formation (i.e., friendships). In contrast to coercive strategies, prosocial strategies are indirect, prolonged, and stand to win positive group regard. Neither strategy is entirely risk free; coercion stands to attract punishment from the group and prosocial strategies can be exploited by others. Nonetheless, because the variance in potential outcomes is greater for coercion (material rewards at one extreme and death on the other) than the variance associated with prosociality (material reward versus net loss from exploitation), coercion is considered ‘riskier’ (Figueroedo & Jacobs, in press).

### Theoretical and Methodological Implications for Human Social Dominance and Personality Development

The relatively simple shift of RCT from form to function has three implications for human social and personality development that contest-based models of social dominance failed to realize. The first implication is methodological: The two-strategy approach implies that prosocial and coercive strategies should either be positively correlated (as suggested by their common function) or independent (i.e., neither opposites nor negatively correlated). 7

7 There is no reason to believe these broad classes of strategy are unique to humans, but are probably more characteristic of species with reasonable cortical and social complexity.

This assumption implies that a) the strategies can be measured on independent scales, and b) once they are measured, *types* of individuals can be identified based on their employment of the two strategies relative to other social group members (i.e., high-high, low-low, high-low, low-high, etc). This *person-centered approach* is known to uncover aspects of functioning unavailable to more commonly employed variable-centered approaches characterized by regressions and correlations (see Hawley, Johnson, Mize, & McNamara, 2007 for extended discussion).

Second, this methodological twist leads us directly to a novel approach to Machiavellianism in children (and adults) *if* we interpret Machiavellianism to mean the tendency to use both prosocial and coercive strategies of resource control to a high degree (cf. Christie & Geis, 1970; Paulhus & Williams, 2002; Byrne & Whiten, 1988).

Third, we can now discuss children’s resource-directed behavior in ways that will lead us back to life history theory and alternate approaches to phenotypic diversity; namely, we have reason to believe that prosocial and coercive strategies are ‘calibrated’ by the parent-child relationship and honed among peers. Each of these points will be elaborated below.

### Strategy Differentiation and a Person-Centered Approach: Identifying the Machiavellian Bistategic Controller

The first step to the above research program is to establish whether the two classes of strategy indeed function to attain social dominance (i.e., successful resource control *relative to others*) as proposed. Observational studies with preschool children centered on highly attractive play
material supported our novel view (Hawley & Little, 1999; Hawley, 2002). Here, prosocial strategies were operationally defined as making suggestions, issuing polite requests, initiating (often unequal) trades, and offering ‘help’ (which effectively commandeered the play material). Coercive strategies were operationally defined as taking, aggression, and insults. First, and somewhat surprising to us given the age of the children, prosocial strategies were employed at twice the frequency of coercive strategies (which suggests that socialization works). Second, and as we expected, both prosocial and coercive strategies were highly associated with possession of the play material (rs = .53 and .46 respectively), and positively correlated to each other (r = .67). Together, the strategies effectively accessed the play material 71% of the time (Hawley, 2002).

Older children, adolescents, and young adults can self-report their own behavior and intentions, as well as report those of peers. Questionnaire items include “I get what I want by promising friendship,” “...by being nice,” or “...reciprocating” (prosocial strategies) and “I get what I want by bullying,” “...threatening,” or “...taking” (coercive strategies). Teacher questionnaires are also highly informative (though outcomes differ in important ways; Hawley, Little, & Pasupathi, 2002). On the basis of the relative degree of strategy employment, subgroups or types can be defined depending on individuals’ placements in distributions divided into tertiles; bistrategic controllers by definition are in the top tertiles of both prosocial and coercive strategies, coercive controllers are in the top tertile of coercive strategies only, prosocial controllers are in the top tertile of prosocial strategies only, and noncontrollers are in the lowest tertile of both strategies. Typical controllers comprise the remaining largest group. Regardless of whether the types are formed via teacher report (Hawley, 2003a), self-report (e.g., Hawley, 2003b), or peer nomination (Hawley, Card, & Little, 2007), bistrategic controllers prove themselves to be the most successful at resource control by far, followed by prosocial and coercive controllers, with the noncontrollers being the least successful. Thus, bistrategic controllers are considered to be of the highest social dominance status and noncontrollers the lowest from this view by definition. This body of work can be seen to address the validity of our approach: Prosocial and coercive strategies seem to work, especially if they are used in combination. Moreover, the groups form by arbitrary cut-offs have been replicated using a data-driven classification technique (Geldhof, Stump, Howard, & Hawley, 2010).

**Characteristics of the Resource Control Subtypes**

Over the course of a decade, prosocial controllers have shown themselves to be warmly drawn to others, agreeable, and socially skilled. Consequently, they are well-liked by peers and enjoy intimate, high-quality friendships (Hawley, Little, Card, 2007). We see this as evidence that prosocial strategies are doing what they were ‘designed’ to do—procure resources effectively while at the same time maintaining group cohesion and long-term, resource-yielding bonds. In contrast, coercive controllers are aggressive, hostile, impulsive, unskilled (Hawley, 2003b), and motivated by power and popularity. They are rejected by peers, and what friendships they have are low-quality and conflictual (Hawley et al., 2007). Instrumental aggression is known to be an effective goal attainment strategy, but one that risks breaking bonds and ultimately ostracism (Boehm, 2000).

Neither of the above patterns associated with pure prosociality and pure coercion (i.e., pure strategies) are particularly surprising or noteworthy as they coincide with what we have long
known about children’s behavior. Bistrategic controllers, however, make the greatest novel contribution to our understanding of human behavior in developmental circles. First, the dual strategy approach to resource control leads to very high material success relative to peers. Like coercive controllers, bistrategics are aggressive (cf. Jones & Paulhus, 2009), manipulative, have a high need for recognition, and value material attainments over relationships (Hawley, Shorey, & Alderman, 2009; see also Stewart & Stewart, 2006). At the same time, they have a rather sophisticated understanding of others (unlike coercive controllers) and a certain moral attunement (Hawley, V a, b). Despite their highly visible ‘dark sides’ (i.e., the characteristics that are in general believed to garner public censure), they enjoy a good deal of social success with their peers; they are popular, liked by others, and have many reciprocated friendships (and may even enjoy differential reproductive success; Jokela & Keltigangas-Järvinen, 2009).

Interestingly, despite the high levels of aggression employed by bistrategics, they tend to be equally male and female across all ages. Moreover, female bistrategic controllers tend to engage in high levels of behavior considered ‘masculine’ (e.g., overt aggression) while male bistrategic controllers have skills associated with females (e.g., attunement to others). For this reason, we have tended to think of bistrategics as a whole as a rather androgynous group who have mastered all humans skills and strategies and can employ these strategies flexibly and in ways that garner some degree of group approval. Thus, not only are they meeting their material needs, they appear to be meeting their social needs. Accordingly, we have argued elsewhere (Hawley, 2002; Stump et al., 2009) that this behavior pattern may well represent a morally neutral view of human social competence.

Bistrategics clearly demonstrate that visibly high levels of aggression need not lead to ostracism, but instead can lead to material and social success when balanced with positive acts and attitudes. Thus, in many ways,

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bistrategies are the successful embodiment of Machiavelli’s philosophy: be “... merciful, faithful, humane, frank, religious” but “preserve a disposition which will make a reversal of conduct possible in case the need arises” (Machiavelli, 1513/1966, p. 63). Do this and you stand to be supported by your people.

Biologizing Machiavellianism

Evolutionary game theory. David Sloan Wilson and colleagues (1996) readdressed and reformulated the Machiavellianism construct into evolutionary terms by applying game theoretic models and aligning Machiavellianism with the willingness to ‘defect’ in multi-strategic games. In terms of game theoretic models, these authors noted, high-Machs may use cooperative and defection strategies more flexibly than others, or may be more willing to defect as a first strike option. Wilson and colleagues suggested that this quickness to defect would undermine long-term interactions (though our research program shows this to be less true than predicted). In contrast, low-Machs would be cooperators, and as such would outperform high-Machs in contexts calling for coordinated interaction. Populations composed of individuals using various strategies would theoretically stabilize after multiple ‘generations’ in terms of the resultant numbers of cooperators and defectors.

Non-controllers: A losing strategy. From an RCT perspective, the bistrategic controllers map on well to Wilson’s construal of Machiavellians. The two perspectives, despite their similarities, have a number of key points of departure. First, low-Machs in our view may use cooperative
strategies (prosocial controllers), but also very importantly, may use no strategies at all (non-controllers).

We do not view ‘non-controllingness’ to be an evolved alternate strategy that enjoys similar average payoff as the other types. In fact, a quick comparison of the social and personality profiles of non-controllers and bistrategics show they are quite opposite; every characteristic on which bistrategies are high, non-controllers are low (resource control, extraversion, agency, social skills, aggression, social status, and social acceptance). Non-controllers are not akin to the well-documented ‘sneaker strategy’ in the animal world; they are more like the anxious and withdrawn individuals who don’t try and consequently fail to thrive. ²

² In school children, they are the gravest clinical significance (Stump, Ratliff, Wu, & Hawley, 2009). See also Gilbert’s approach to powerlessness (Gilbert, 1992).

They are—by all measures and views—socially incompetent.

First Steps to Applying a Life History Framework to Social Dominance and Machiavellianism

Humans have a modal tendency toward a combined capability for sophisticated prosociality and aggression. Indeed, an average use of prosocial strategies and coercion defines most of us as ‘typical controllers.’Aggression is everywhere from minute fishes to mega-fauna, and thus needs no further discussion. Prosociality of the variety that serves long-term, resource-yielding relationships, however, is likely reserved for those species with a reasonably complex social system, a protracted juvenile period with concomitant parental care, and long-term affiliations with known group members; in other words, a species with an evolved tradeoff favoring an extended juvenile growth period over early reproductive maturation (i.e., slow life history strategy; Ellis et al., 2009).

³ We also remain open to the possibility that prosociality can take very simple forms, and as such exist across many taxa. Though, in general, we would not expect sophisticated prosocial strategies to emerge in ‘r’ selected species (i.e., fast).

At the same time, individual differences are distributed around this modal pattern. To developmentalists especially, the source of these individual differences in prosociality and aggression—predominantly though not exclusively those attributable to environmental influence—are of keen and enduring interest. Consistent with LHT (though not derivative from it), developmentalists have looked to parents (parental investment), peers (conspecifics), and the material environment (SES, urbanity, school quality, media influences) as the usual suspects. For example, it is well known that aggression is first learned in the family circle (e.g., Patterson, Dishion, & Bank, 1984) and is further shaped by peers (e.g., Dishion, Spracklen, Andrews, & Patterson, 1996) and other socioecological conditions (e.g., urban stress; Sampson & Laub, 1994).

Parental Calibration: Attachment

In the life history literature, the quality of parental investment is argued to vary according to ecological contextual variables and to ‘calibrate’ an accordingly favorable life history strategy in the developing organism. In harsh and/or unpredictable environments, for example, when there is relatively high risk of morbidity and mortality (i.e., short average life expectancy), selection tends to favor fast strategies that bias offspring quantity over quality (Ellis et al., 2009). Parenting behavior (itself sensitive to environmental conditions)
may be a potent ‘cue’ that signals environmental conditions to the developing organism (Chisholm, 1996; Quinlan, 2007; Belsky, Steinberg, & Draper, 1991). Low investment from parents (low responsivity, low warmth) may signal environmental harshness (because high investment yields no differential benefit under harsh conditions) and the developing organism would ‘do well’ to get on with development and reproduction. In such environments, investment in intimate relationships that are a) unlikely to last, and b) unlikely to yield material benefit, would be a waste of finite energy and organismic resources that could better be applied elsewhere.

In developmental circles, parental investment has been addressed in the attachment literature. Bowlby (1969; 1988) proposed that young humans have an evolved predisposition (a species-typical LH pattern) to maintain attachments to caregivers to secure protection and resources. Yet, individuals vary in the features of these attachments. Secure attachments (won by warm, responsive parenting) free the developing child to confidently explore the interpersonal and material worlds (Ainsworth, Blehar, Waters, & Wall, 1978). Securely attached children become increasingly efficacious because they believe that a) others are available and responsive to their needs, and b) the world is a safe and predictable place where material resources can be readily attained (Ainsworth et al., 1978; Bowlby, 1969).

In RCT terms, securely attached individuals should value the role of the social group in resource allocation and, importantly, view others positively as avenues for successful goal attainment. Secure schemas thusly should underlie ‘slow’ prosocial strategies of resource control. Indeed, and as expected, prosocially controlling college students enjoy the attachment profile most consistent with attachment security (Hawley et al., 2009). Furthermore, even though prosocial controllers value material pursuits to a high degree (as attachment theory would predict of the securely attached), they value their social relationships even more. Cast in this light, it should come as no surprise that prosocial controllers are cherished group member across all age groups (i.e., they are very well liked). In LHT parlance, prosocial controllers embrace a ‘slow’ strategy on the slow-fast continuum.

In contrast, attachment avoidance results when infant bids are met with caregiver reproach (Main, 1990). To adjust, the developing individual may turn toward material goal pursuits and away from non-rewarding interpersonal contact (Mayseless, 1996). Accordingly, the avoidantly attached undervalue personal relationships (nothing useful comes of them) while overvaluing material goals (Feeney, Noller, & Hanrahan, 1994). Attachment avoidance would thus seem to underlie coercive strategies of resource control (Hawley et al., 2009), a ‘fast’ strategy on the slow-fast continuum where long-term bonds are eschewed (Ellis et al., 2009).

Attachment anxiety is characterized by hypervigilance for rejection cues and a “preoccupation” with relationships that can manifest as an eagerness to please others to avoid social rejection (Bartholomew & Horowitz, 1991; Mikulincer & Shaver, 2003). Thus, in some sense, anxiety should underlie prosocial strategies (“be nice or you will be ostracized”; see also the self-monitoring and anxiety of Machiavellians: Snyder, 1974; Jones & Paulhus, 2009). This more strategic flavor of prosociality in fact underlies the behavior of bistategic controllers (Machiavellians) who are as avoidant as they are anxious (Hawley et al., 2009). Bistategic controllers tend to be consciously aware of the role of others for the realization of their goal pursuits. Accordingly they ‘value’ interpersonal relationships (albeit instrumentally). At the same
time, bistrategics value material goals even more than personal relationships, and their avoidant attachment schemas facilitate their direct goal pursuit via coercive strategies and aggression. The complex attachment pattern can account for their relatively high levels of prosociality and aggression.

Summary. The roots of social dominance and its concomitant strategies may well lie in the attachment relationship, a relationship, that in the view of attachment researchers, lays the foundations for understanding (schemas) the value ascribed to the material world and the role that other people play in meeting material needs (internal working models), and thus, personality. Are others understood to be valued sources of material need fulfillment? If so, then establishing long-lasting, intimate relationships with them will insure long-term pay off (a slow strategy). Prosocial strategies agreeably employed will achieve this end. Are others of little use and only stand in the way? If so, then short-term relationships, extrinsically motivated, will facilitate short-term pay off (a fast strategy). In this case, coercion and deceit maximize benefit and minimize cost. In LHT parlance, this biologically and psychologically significant relationship signals important information about the environment into which one is born that biases the developing organism toward an adaptive trajectory.

Conclusions
In developmental circles I am often asked, “Are bistrategic Machiavellians born or made?” The answer is, of course, they are both. As argued here, humans possess a modal life history pattern that embraces both prosociality and coercion for individual level (and group level) goal attainment. This pattern (which is not entirely unique to humans) is a product of our evolutionary history. Presumably, the phenotypic variability we enjoy is genetically regulated and environmentally sensitive.

We additionally know, however, that the picture is even more psychologically complicated. At the proximal level, inborn, stable temperament or personality characteristics influence whether or not one orients to the material world (e.g., surgency, shy-bold continuum; Wilson, Clark, Coleman, & Dearstyne, 1994) as well as the ‘flavor’ of this orientation (e.g., agreeable or disagreeable; Paulhus & Williams, 2002). Aggressiveness is known to be heritable (Dodge, Coie, & Lynam, 2006). At the same time, we know these intrinsic qualities are open to outside influence (Casi, McClay, Moffitt, Mill, Martin, Craig, Taylor, & Poulin, 2002) and are further shaped by parental behaviors (Dodge, Petit, & Bates, 1994), family climate (Patterson, DeBaryshe, & Ramsey, 1989), the peer group (Berndt, 1979), and peers’ social norms (Steinberg & Monahan, 2007), all of which have been spoken to by evolutionary thinkers (e.g., Ellis, 2004; Hawley & Little, 1999; Harris, 1995; Sulloway, 2007; Boyd & Richerson, 1992; Figueredo & Jacobs, 2009).

Life history theory, however, additionally shines a new light of a different hue on the parent-child relationship and child personality development than does traditional attachment theory; it explicitly adds an ecological dimension to the calibration process in a way that highlights the adaptive nature of pure versus mixed strategies as well as completely integrates the genetic endowment of the developing organism.

In the end, can children’s (human’s) personality development be completely understood without evolutionary lenses? No. So long as we bias our inquiry with the assumption that prosocial behavior (“good”) opposes antisocial behavior (“evil”) as developmental and social psychologists generally are wont to do, then we will continue to fail to frame these classes of behavior as viable
alternate paths to the same end, that in combination, may exquisitely portray the facultative nature of human strategic behavior and the importance of evolutionary theory for understanding personality development and individual differences.

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References


4 Why Siblings Are Like Darwin’s Finches: Birth Order, Sibling Competition, and Adaptive Divergence within the Family
Frank J. Sulloway

Parents often express surprise at the range of differences in their children’s personalities. For example, one child may be unusually fun-loving and sociable, whereas another child may be shy and introverted. Or one child might be a highly organized neat-freak, but another may revel in clutter. A Darwinian perspective on family dynamics sheds considerable light on these kinds of sibling differences, which are driven by principles that are well known to evolutionary biologists.

Given their impressive disparities, human siblings are a lot like Darwin’s finches, that famous group of birds from the Galápagos Islands that has played such an important role in evolutionary theory. The 14 species of Darwin’s finches are all derived from a single ancestor that colonized these volcanic islands more than two million years ago (Grant & Grant, 2008). From an ancestral species that is thought to have resembled the present-day warbler finch, the original colonists have evolved into 7 species of insectivorous tree finches, 2 species that consume the flowers and fruits of cactus, 1 species that eats fruits and leaves, and 4 species of ground finches that have their beaks graduated according to the size of the seeds they consume (Figure 4.1). So extensive is the diversity among the 14 species in this remarkable avian subfamily that Charles Darwin, during his five-week visit to the Galápagos Islands in 1835, mistook some of these Finch species for members of distinctly different bird families. Only after returning to England

Figure 4.1. Four of the 14 species of Darwin’s Galápagos finches (Geospizinae), illustrated in the second edition of Darwin’s Journal of Researches (1845:379). Top left, the large ground finch; top right, the medium ground finch; bottom left, the small tree finch; bottom right, the warbler finch, which is closest to the ancestral form of this avian group.
was Darwin convinced by John Gould (1837), a distinguished British ornithologist, that these species were all closely related, prompting Darwin to surmise their evolutionary origins (Sulloway, 1982). Commenting in the second edition of his Journal of Researches about the extraordinary range of morphology found within this closely related avian group, Darwin hinted at the evolutionary explanation he revealed to the world fourteen years later in the Origin of Species (1859): “Seeing this gradation and diversity of structure in one small, intimately related group of birds, one might really fancy that from an original paucity of birds in this archipelago, one species had been taken and modified for different ends” (1845:380).

Like Darwin’s Galápagos finches, human siblings tend to diversify in adaptive ways. Whereas Darwin’s finches have diverged phylogenetically, through the gradual evolution of genetic differences, human siblings become increasingly dissimilar during ontogeny, through learned differences in family roles, strategies, and other behaviors. Such behavioral differences eventually become encapsulated in personality as well as in familial sentiments, which include attitudes toward parental authority and feelings of closeness toward other family members. The fact that humans accomplish through learning what Darwin’s finches and other species have achieved through organic evolution does not mean that sibling behavior is no longer subject to the pressures of evolution by natural selection. Rather, the playing field on which natural selection expresses itself includes not only phylogenetic changes but also various ontogenetic adaptations that help individual offspring to survive childhood and to reproduce. Strategies for dealing with sibling competition, and for evoking sibling cooperation, are among the principal functional mechanisms that govern successful adaptation within family life.

Darwin’s Principle Of Divergence

The main reason why Darwin’s finches and human siblings have so much in common goes back to what Darwin (1859) termed his “principle of divergence.” To use one of Darwin’s own examples from the Origin, if a plot of ground is sown with several different species of grasses, rather than with a single species, a larger number of plants and a greater weight of dry herbage can be reaped from this plot because the different species do not compete for the same limited resources. Natural selection, Darwin argued, tends to favor species that face the least competition from other organisms. Darwin’s principle of divergence explains why species become increasingly disparate over time. Along with the theory of natural selection, Darwin considered his principle of divergence to be the “keystone” of his revolutionary arguments about the evolutionary process (Darwin, 1991; 8 June 1858 letter to Joseph Hooker).

One of the most compelling demonstrations of Darwin’s principle of divergence is a phenomenon known as character displacement, or the process by which morphological differences arise in two or more closely related species when they overlap geographically. Darwin’s finches provided one of the earliest documented examples of this evolutionary process (Brown & Wilson, 1956; Lack, 1947). Upwards of 10 different species of Darwin’s finches coexist on the largest islands within the Galápagos group. Nevertheless, some islands have a much smaller number of species. On islands where only one or two species of ground finches are resident, the birds have developed a generalist beak size to take advantage of a wider range of available resources, thus venturing into niches normally occupied by other closely related species (Figure 4.2). Over millions of years, the cumulative outcome of repeated
Figure 4.2. Character displacement in Darwin’s Galápagos finches. On Floreana and San Cristóbal islands, where the small ground finch (*Geospiza fuliginosa*) and the medium ground finch (*G. fortis*) are both resident, the two species have distinctly different dimensions for beak depth, which dictates the size of the seeds these species can crack open. On Daphne and Los Hermanos islands, where only one of the two species is present, beak depth is similar, indicating character release. After Lack (1947:82).

Speciation and character displacement is adaptive radiation, the evolutionary process that has produced the 14 species of Darwin’s finches, the 15 species of Galápagos *Scalesia* (ancestral daisies that have evolved into bushes and 10-meter-high trees), and more than 80 species of Galápagos land snails (Parent, Caccone, & Petren, 2008).
The Biology Of Sibling Competition

Competition is what drives the closely related processes of character displacement and adaptive radiation. Like species, siblings compete over valued resources. On average, among sexually reproducing organisms, siblings share half their genes (unless they are identical twins). In setting forth the concept of kin selection—one of the most important evolutionary insights since Darwin’s theory of natural selection—William Hamilton (1964a, b) hypothesized that full siblings will tend to compete for scarce resources whenever the benefits of doing so are more than twice the costs, because it takes two sibs to equal the genetic material that is shared with the self.

From the perspective of Hamilton’s theory of kin selection, which involves the tendency for organisms to behave altruistically toward close relatives in proportion to their shared genes, sibling competition and parent-offspring competition are closely linked. Parents are equally related to all of their offspring, and thus have good reason to invest equally in their offspring. By contrast, offspring are twice as related to themselves as they are to their siblings, so ideally they want parents to provide themselves with twice as much investment as the parents give to another sibling. Hence, children are generally in conflict not only with their siblings over the allocation of parental investment, but also with their parents. Children are also in conflict with their parents and siblings over the timing of parental investment (Trivers, 1974). Weaning conflicts exemplify such timing disputes. The unweaned offspring attempts to secure additional parental investment from breast-feeding, which not only gives the offspring greater food resources but also generally delays the conception and birth of a sibling competitor (Figure 4.3).

Sibling competition has been widely documented among animals, birds, fish, and insects (Mock & Parker, 1997; Mock, 2004; Simmons, 2002). Such conflicts are especially prevalent among seabirds and predatory birds and sometimes end in siblicide. Two types of siblicidal competition are observed in nature: obligate (when siblicide almost always occurs) and facultative (when siblicide occurs only under specific individual and ecological conditions). Among Verreaux’s eagles (Aquila verreauxii), which breed throughout Africa, siblicide is obligate. The elder chick pecks the younger chick to death within the first three days of hatching (Figure 4.4). In species where siblicide is obligate, parents are rarely capable of successfully rearing more than one offspring owing to large food requirements of a single offspring. The second egg, which constitutes a minimal physiological investment by the mother, insures that valuable time is not lost during the breeding cycle if the first egg is infertile or if the older chick dies soon after hatching (Mock, Drummond, & Stinson, 1990).

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Figure 4.3. A mother fur seal attacking her older, previously nursing offspring (on the right) while a newborn pup rests on the left. After the birth of a younger sibling, the likelihood that a yearling pup will die increases by about 60 percent. Older pups exhibit aggression toward younger siblings, by biting them and chasing them away from the mother (Trillmich & Wolf, 2008).

Among seabirds, such as blue-footed boobies (*Sula nebouxii*), parents sometimes successfully rear two or even three chicks, depending on the available food supply. Older chicks are dominant over their younger chicks, limiting their access to food. Siblicidal pecking by the older chick begins only when its body weight drops to about 80 percent of normal. Parents do not intervene in these lethal conflicts, and it is not in their genetic interests to do so (Drummond & García-Chavelas, 1989).

In some species natural selection has engineered specialized traits that help offspring to compete with their siblings. During the tadpole stage, spadefoot toads develop formidable teeth, which they use to cannibalize their broodmates (Bragg, 1954). Similarly, piglets are born with eight “eye teeth” that are later shed. The piglets use these teeth to defend access to the mother’s anterior-most teats, which have the richest supply of milk. By the third week following their birth, mortality among piglets forced to nurse from their mother’s posterior teats is twice the rate for their anterior-nursing littermates.

Photograph courtesy of Peter Steyn.
Figure 4.4. A six-day-old Verreaux’s eagle chick (top) has opened up a large wound in its one-day-old sibling. In one documented instance, an older Verreaux’s eagle chick inflicted 1,569 pecks to the head and body of its younger sibling during the latter’s three-day lifespan (Gargett, 1978).

(Trivers, 1985). Even plants engage in sibling competition and have evolved specialized weapons for this task. The India black plum (Syzygium cumini) develops seeds with up to 30 ovules. The first ovule to be fertilized secretes a “death chemical” that kills off all the other ovules by preventing them from metabolizing sucrose (Krishnamurthy, Uma Shaanker, & Ganeshaiah, 1997). The widespread nature of sibling competition, including its occasional resolution in siblicide, exemplifies a gene’s eye view of evolution, as epitomized by Dawkins’ (1976) well-known metaphor of the “selfish gene.”

Sources Of Sibling Differences

Findings From Twin Studies

Just as the study of Darwin’s finches has been enriched by recent genetic discoveries (Petren, Grant, & Grant, 1999), behavioral scientists have increasingly drawn on genetic investigations to understand the sources of individual differences among siblings raised in the same family. Results of these studies, which typically involve comparing identical twins raised together and apart, have turned out to be almost as surprising as Darwin’s Galápagos finches were to the zoologists who first examined the birds nearly two centuries ago. By comparing the personalities and intellectual abilities of twins reared under differing conditions, behavioral geneticists have obtained keen insights into the relative contributions of genes and environment in human development.
Typically, behavioral geneticists subdivide the sources of human characteristics into three classes, namely, those stemming from (1) genes, (2) the shared environment (for example, growing up in the same home and living in the same neighborhood), and (3) the nonshared environment (experiences that are unique to each individual—both inside and outside the home). Based on results from numerous twin studies, genetic differences appear to explain about 40 percent of the variance in individual personality. The nonshared environment explains another 35 percent of the variance. By contrast, the shared environment explains only about 5 percent of the variance in personality, leaving the remaining 20 percent of the variance to be explained by errors in measurement (Dunn & Plomin, 1990; Loehlin, 1992; Plomin, DeFries, McClearn, & McGuffin, 2001). The gist of these findings is that siblings are little more alike than people plucked randomly from the general population (Dunn & Plomin, 1990; Plomin & Daniels, 1987).

Why Family Environments Are Rarely Shared

These surprising results from research in behavioral genetics have led some commentators to claim that parents and the family exert almost no influence on personality (Harris, 1998; Pinker, 2002; Rowe, 1994). Although this assertion has received considerable coverage in the popular press—in large part because it goes counter to most people’s intuitive assumptions about the family and its influence on offspring—the claim is misleading. The real insight from this behavioral genetic research is not that the family has little influence on personality. Rather, the correct conclusion is that the bulk of the family’s influence, including that of parents, is not shared by siblings (Sulloway, 1996; Turkheimer, 2000; Turkheimer & Waldron, 2000).

There are nearly endless ways in which seemingly shared family experiences are not truly shared and prompt siblings to become different. For example, offspring react differently to the same parental behaviors, since every parental action is filtered through a distinctive sibling brain. Parents, in turn, react differently to each of their offspring, because offspring—unless they are identical twins—are themselves genetically different, and are different as well owing to previous environmental influences.

In addition, siblings typically occupy differing niches within the family system. Through differences in these family niches, siblings develop specialized roles based on such factors as age, sex, personality, interests, and ability—all of which cause their daily interactions with parents and other family members to differ. Communal family experiences are never truly shared for another important reason: differences in age cause siblings to experience the same events at different developmental stages (Dunn & Plomin, 1990). In addition, unless offspring happen to be twins, parents are at different ages when their children are born. With each successive child, parents bring differing skills, experiences, marital relationships, and life-stage concerns to their child-rearing efforts.

Much of the support and encouragement that parents give to their children tends to augment differences among their offspring rather than making them more alike. As children are growing up, parents seek to identify useful talents among their offspring and to fine-tune these abilities through instruction and encouragement. Owing to differences in the genetic make-up of each child, some abilities are expressed more strongly in one child than another. For example, parents may help a bold and athletically inclined child to become a figure skater, enhancing this child’s prior disposition to take risks. The same parents may encourage a more pensive sibling to pursue an interest in competitive chess, accentuating this child’s intellectual orientation. The net
consequence of parental shaping is that children become increasingly different as they grow up, a phenomenon that is analogous to Darwin’s principle of divergence.

Parents also create differing environments for their children by coercing behavior toward the biosocial norm, as when they seek to inhibit the behavior of a hyperactive child and try to stimulate that of a shy and overly quiescent child (Buss, 1987). Such differing parental responses to offspring reflect what are known as “genotype-environment correlations” (Scarr & McCartney, 1983). These correlations help to explain the fact that, in behavioral genetic studies, seemingly “environmental” influences on behavior frequently contain a genetic component. Three types of genotype-environment correlations have been documented: (1) passive, as when an offspring receives from parents a genetic predisposition for higher intelligence, and is also supplied with more books in the home to read; (2) reactive, as when parents respond more affectionately toward particularly amiable and affectionate offspring; and (3) active, as when offspring with differing genotypes seek out environments that best suit their genetic predispositions. Because all nontwin siblings differ genetically, genotype-environment interactions are continually causing siblings to experience their world—including the family environment—differently.

Five Mechanisms Causing Sibling Differences

Research on birth order and family dynamics helps to illuminate the role of the nonshared environment. This research highlights at least five key mechanisms—psychological as well as biological—that cause sibling diversification. These mechanisms include (1) differences in parental investment; (2) dominance hierarchies among siblings; (3) deidentification, or the tendency for siblings to actively seek to differentiate themselves from one another; (4) the closely related principle of niche picking within the family, by which siblings seek different specializations and roles; and (5) birth-order and gender stereotypes, which tend to cause others to react differently to siblings and to assign them specific roles, thereby accentuating disparities (Table 4.1).

Differences In Parental Investment

Differences in parental investment have been documented prenatally in the amount of nutrition received by the fetus as well as the kinds of hormones in which the fetus is bathed (Sulloway, 2007a). All fraternal twins, and approximately one-third of identical twins, have different chorions, the outermost membrane that envelops the growing embryo and its placenta. As adults, identical twins who have previously shared the same chorion are more similar in their personalities and in some physical attributes (Sokol et al., 1995). Differences in fetal environments also arise as a result of maternal autoimmune reactions to previous male fetuses, which cause sons of higher birth rank to exhibit elevated rates of homosexuality (Blanchard, 2004). Evidence from multiple twin studies suggests that differences in fetal environments explain upwards of 20 percent of the variance in IQ scores (Devlin, Daniels, & Roeder, 1997).

Although parents generally seek to invest equally in offspring after children are born, they nevertheless adjust their investments based on differences in physical and personality characteristics, special needs of offspring, and other factors. Differences in real and perceived prospects for survival and reproduction also affect parental investment. Birth order and gender turn out to be important considerations in this Darwinian calculus. Only two centuries ago, half of all children did not survive past the age of five, and infant mortality rates are still almost this high today in poorer parts of the world. Even small differences in quantity and type of parental investment during childhood can determine which offspring survive and which ones do not.
### Gender Biases in Parental Investment

Table 4.1. A family dynamics model encompassing five developmental mechanisms affecting sibling differences in health, personality, social attitudes, intellectual ability, and strategies to increase Darwinian fitness.

<table>
<thead>
<tr>
<th>Developmental mechanism</th>
<th>Associated trends in health, personality, and other attributes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Disparities in parental investment</td>
<td>Differences in parental investment among offspring occur prenatally as well as postnatally. Prenatally, such differences involve hormones, nutrition, and the health of the mother. Postnatally, differences include the duration of breast-feeding, vaccination rates, verbal stimulation, affection, education, and inheritance. Differences in parental investment influence mortality rates, overall health, closeness to parents, intelligence, personality, and social attitudes. Older siblings are generally dominant over younger ones, and they often use their power to garner more resources within the family. Likewise males and larger children may be more dominant, which affects the nature of sibling interactions and the development of personality.</td>
</tr>
<tr>
<td>2. Sibling dominance hierarchy effects</td>
<td>Siblings are often most different from those brothers and sisters who are adjacent in age and birth order. Siblings sometimes vary in the parent to which they are closest, reflecting competitive specialization in the quest for parental affection.</td>
</tr>
<tr>
<td>3. Sibling deidentification</td>
<td>Siblings specialize in their family roles. Firstborns are often pressured more by parents to uphold family values and traditions. Surrogate parenting (primarily by firstborns and oldest females) can lead to differences in conscientiousness and other aspects of personality. Laterborn offspring tend to excel in sports, to take greater risks, and to be more unconventional and open to experience.</td>
</tr>
<tr>
<td>4. Niche partitioning within the family system</td>
<td>Birth-order, birth-order stereotypes can reinforce birth-order differences. Gender stereotypes and other stereotypes, as well as stereotypes based on age and physical size, also influence family roles. Childhood mortality rates vary by sex in ways that promote the reproductive interests of their parents. As Trivers and Willard (1973) realized, natural selection should favor a tendency for adult females to produce male offspring when females are in good physical condition because the healthiest and most robust male offspring can potentially produce numerous progeny after reaching adulthood. This is especially true when there is intense competition among males for mates, and some males do not mate at all. By contrast, when adult females are in poorer health, they are better off having female offspring, because the physical condition of female offspring is less critical for reproductive success, inasmuch as almost any female can be impregnated by a willing male.</td>
</tr>
</tbody>
</table>
Animals and insects accomplish these adaptive shifts in sex ratios of offspring through physiological mechanisms. Humans achieve similar manipulations of sex ratios through postnatal differences in parental investment (Hrdy, 1999). As Boone (1986) and Voland (1990, 2007) have shown by drawing on large historical samples from Portugal and Germany, the critical factor in the adjustment of sex ratios among humans is not the physical condition of the mother, but rather the social class and wealth of the parents. Among wealthier Portuguese families, for example, sons were more likely to outreproduce daughters, whereas in poorer families, daughters were more likely to outreproduce sons. These and analogous findings implicate differences in parental investment, including the duration of breast-feeding, hygienic care, the provisioning of nutrition and medical care, inheritance practices, and parental resources devoted to dowries (Beise & Voland, 2002; Gaulin & Robbins, 1991; Voland & Dunbar, 1995).

**Birth-order Biases in Parental Investment**

Relative to younger siblings, older siblings have already experienced more of the childhood diseases that can cause early death. For this reason, older siblings generally represent better Darwinian bets for survival and reproduction and hence should garner even more parental investment (Daly & Wilson, 1988; Rohde et al., 2003; Salmon & Daly, 1998; Sulloway, 1996). Not surprisingly, in societies where infanticide is practiced, no society condones the killing of an older sibling in place of a newborn (Daly & Wilson, 1988).

Accordingly, mortality statistics and other measures of health indicate that parents often invest less in laterborn offspring. Repeated studies have shown that laterborns are less likely to be vaccinated than are their older siblings, with rates of vaccination declining 20-30 percent with each successive birth rank in the family (Hertwig, Davis, & Sulloway, 2002). Lower vaccination rates in turn contribute to the higher childhood mortality that has been documented among laterborn children. Drawing on demographic data from various Latin American countries, Puffer and Serrano (1973) found that compared with firstborns, children of fifth and higher birth ranks experienced two-to-three times the usual rates of infant mortality. In an investigation of 1,903 Philippine households, Horton (1988) determined that younger siblings, on an age-adjusted basis, were shorter and weighed less than older siblings, indicating poorer nutrition among the younger siblings, who must increasingly subdivide limited parental resources. Similarly, younger daughters tend to reach menarche at a later age than do their older sisters, probably because of poorer nutrition (Surbey, 1998).

Even when they survive childhood, younger siblings often face discrimination by parents in favor of their older siblings. In a survey of 39 non-Western societies, Rosenblatt and Skoogberg (1974) found systematic differences in parental favor by birth order. In these non-Western societies, first children of either sex were generally privileged over their younger siblings through a wide variety of social customs, including birth ceremonies, leadership recognition, and inheritance practices.

In Western and other societies, biases in parental investment have long been manifested in inheritance practices, especially those related to sex and birth order. Going back to the Middle Ages and earlier, the policy of primogeniture—leaving all or most of the property to the eldest son or child—was widely practiced in Europe and other parts of the world to insure the perpetuation of family property and the family name, especially in countries where land was a limited resource (Hrdy & Judge, 1993). In his investigation of the upper nobility in medieval Portugal, Boone (1986) found that younger sons—typically finding themselves landless as a
result of primogeniture—were less likely to marry and to leave offspring than were their elder brothers. Among men as well as women, Boone observes, “birth order had a catastrophic effect on the probability of marriage” (1986:869). Landless younger sons were also more likely than eldest sons to take part in the expansionist military campaigns in distant parts of the world, such as Africa and India, where they died in battle or from disease, or where their military service paid off in honors and a share of the spoils of war. This then provided younger sons with an alternative, but highly risk-laden, route to acquiring the resources need for increased Darwinian fitness.

Underinvestment in Middleborns

In their efforts to garner parental investment, middle children are often disadvantaged relative to eldest and youngest siblings. When parents—especially mothers—have passed the age of reproduction, youngest children are the last offspring the parents will ever have. It makes Darwinian sense for parents to invest extra resources in their last, most vulnerable, youngest child. Whereas firstborns tend to receive privileged parental investment based on their greater prospects for survival, and lastborns are favored as well, middle children are left holding the short end of the parental investment stick (Salmon & Daly, 1998; Suitor & Pillemer, 2007; Sulloway, 1996, 2001). For example, Lindert (1977) documented total child-care hours in 1,296 American families and found that middleborns received about 10 percent less care than either firstborns or lastborns.

Compared with other siblings, middleborns tend to experience less parental investment for another reason. If parents, using an equity heuristic, allocate resources by dividing their resources equally between all existing children, middleborns will end up with fewer resources than other siblings because at all times during their development they must share these resources with other children (Hertwig et al., 2002). By contrast, firstborns receive 100 percent of parental investment as long as they remain only children. After the birth of a second child, both offspring receive 50 percent of total parental investment. With the arrival of a third child, the average amount of parental resources is divided three ways, and investment per child declines to about 33 percent. Eventually, only the lastborn is left at home. Because there usually is never a time when middle children are not sharing parental investment with one or more siblings in the home, middle children seldom reap the advantage that oldest and youngest siblings do when no other children are present. The counterintuitive consequences of parents allocating their resources equally is that middleborns generally receive less cumulative investment than do eldest and lastborn offspring.

Such differences in parental investment may help to explain why middle children are reported to have lower self-esteem than other siblings (Kidwell, 1982) and to be more self-conscious (Chao, 2001; Sulloway, 2001). Perhaps because middleborns typically receive less parental investment, they are not as close to their parents as are firstborns and lastborns. For example, studies have shown that middleborns are generally less likely to turn to parents for emotional support in response to traumatic events (Rohde et al., 2003; Salmon & Daly, 1998). Middleborns also have more positive attitudes toward their friends than do children of other birth ranks; and when they are in monogamous sexual relationships, middleborns are the least likely birth-order group to cheat on a partner (Salmon, 1998, 1999, 2002).

Parental Investment and Intellectual Performance

Considerable evidence indicates that intelligence is positively correlated with parental investment, which is in turn related to the birth order of offspring. Firstborns tend to have higher
IQs than their younger siblings, and IQ scores also tend to decline with each increase in birth rank (Belmont & Marolla, 1973; Bjerkedal, Kristensen, Skjeret, & Brevik, 2007; Kristensen & Bjerkedal, 2007; Zajonc & Sulloway, 2007). These differences in intellectual performance are consistent with the equity heuristic of parental investment as well as with resource-dilution theories (Sulloway, 2007b), including Zajonc’s (1976) confluence model, which offers explanations of birth-order differences in IQ in terms of an impoverishment of the family’s intellectual environment. The birth of the first child reduces the family’s average intellectual environment, and additional children increasingly impoverish this overall environment. According to the confluence model, earlierborn children end up with higher IQs because they spend more time growing up within an environment characterized by frequent interactions with parents. Zajonc’s model also includes a role for a “teaching function” by which older siblings tutor younger siblings in their role as surrogate parents, and thus they benefit by developing their intellectual abilities from such teaching efforts.

Parental Investment and Personality

Differences in parental investment, and conflicts with parents over such differences, affect personality. In a multinational study of 6,053 adults who rated themselves and a sibling on personality scales representing all 30 facets of the Five Factor Model, participants who asserted that their parents had favored another sibling tended to score lower on conscientiousness and higher on openness to experience (Sulloway, 2001). Such effects are consistent with a tendency for children who are victims of lower parental investment to identify less with parents’ values and to question parental authority (Sulloway, 1996), making it easier for such offspring to break away from parental influence and to explore independent life-course options that may better serve their own interests (Belsky, Steinberg, & Draper; Ellis, 2004; Ellis & Essex, 2007). For offspring who receive lower parental investment, there are often potential benefits associated with the rejection of parental values. Such offspring are less constrained by these values, as well as by possible parental manipulation, and hence they are freer to reject the status quo and to become early adopters of new ideas (Sulloway, 1996). Among immigrant families, offspring who do not share their parents’ values have been found to be quicker in their adoption of new cultures and are also more successful, socioeconomically, in such cultures (Manaster, Marcus, & Chan, 1998).

Sibling Dominance Hierarchies

Because siblings differ in age, they also differ in size, power, verbal mastery, and overall maturity. Birth order is correlated with these factors and is a proxy for them. Close age-spacing, however, diminishes the utility of this proxy, as the closer siblings are in age, the smaller the differences between them. With this qualification in mind, older siblings of both sexes are likely in childhood to be larger, stronger, and more verbally proficient than their younger siblings, and hence to be the “top dogs” of their sibling group. Older siblings are able to employ strategies of dominance that are generally unavailable to younger siblings, who instead must resort to low-power strategies including whining, begging, bargaining, and appealing to parents for protection (Sutton-Smith & Rosenberg, 1970), as well as the positive, low dominance strategies of trying to be supportive, affectionate, cooperative, and cute in order to elicit greater parental investment. Domineering, bossy behavior is typically associated
with the family role of firstborns (Beck, Burnet, & Vosper, 2006; Paulhus, Trapnell, & Chen, 1999; Sulloway, 2001).

**Deidentification**

One of the ways by which siblings differentiate themselves is through a process called “deidentification.” This process refers to the tendency for siblings who are adjacent in the family constellation to exhibit opposing personality traits. In a study of siblings’ interests and personalities in three-child families, Schachter, Gilutz, Shore, and Adler (1978) found that thirdborns were more similar to firstborns than they were to their next older sibling. Among same-sex pairs of siblings, deidentification was greater than among opposite-sex siblings, for whom rivalry is often reduced by sex-role differentiation.  

1 Using previously unpublished data from a multinational sample (Sulloway, 2001), I have analyzed the relationship between sibling rivalry (the dependent variable) and birth order, sex, and sex of sibling. None of the main effects were significant, but the interaction effect between sex and sibling’s sex was significant ($F_{1, 2105} = 28.36, r_{pb} = .12, p < .0001$).

Because of sibling deidentification, birth order trends sometimes exhibit zigzag trends, with each sibling being maximally differentiated in personality from adjacent sibs compared with more distant sibs (Skinner, 1992). Schachter (1982) has also pointed out the phenomenon of split-parent identifications. When one offspring develops a close relationship with one parent, another offspring is likely to develop a close relationship with the other parent. In this manner, siblings avoid intense competition for the affections of the same parent.

Additional evidence for sibling deidentification is provided by longitudinal studies of sibling personalities. Over a ten-year period, Loehlin, Horn, and Willerman (1990) documented negative correlations for personality traits among 83 biological siblings. As one child became more introverted over time, for instance, the comparison child tended to become more extraverted. In this same study, negative correlations among the same personality traits were also observed in a larger sample of 312 unrelated children who were reared together as siblings, suggesting that the family environment is pushing children raised together to differentiate themselves.

**Family Niches**

An ecological niche is defined in terms of an organism’s adaptive fit with those aspects of its environment that allow it to survive and reproduce more successfully than its competitors. Species living in ecological communities can increase their fitness by evolving so as to minimize competition over the same resources. For instance, the ability to crack open particularly large and hard seeds is part of the adaptive repertoire associated with the ecological niche filled by the large ground finch in the Galápagos Islands. One of Darwin’s finches—nicknamed the “vampire finch”—has evolved the unusual behavior of drinking blood from small wounds it creates at the base of the tail feathers of nesting boobies; and another remarkable member of this avian group—the “woodpecker finch”—has evolved the ability to use small twigs and cactus spines as tools to pry insects from crevices in trees (Lack, 1945; Grant & Grant, 2008).

In an analogous fashion, siblings living within the same family tend to develop disparate and complementary roles and adaptations within the family system, thereby creating different “family niches” for themselves in an effort to obtain desired resources. These differing roles and specializations are affected by age and gender, as well as by many other individual differences,
including aptitude and life experience. Birth order plays an important part in the development of family niches because it is a proxy for various age-related roles and attributes that are involved in such specializations. Age-spacing between siblings is important in the establishment of family niches because it influences functional birth order, which may not be identical to biological birth order. For example, a secondborn who is separated by a gap of many years from a next older sibling may function like a firstborn or only child (depending on whether additional younger siblings are present in the family). Close spacing also alters the dynamics of birth order, particularly when a younger sibling is bigger and stronger than an older sibling and is able to compete favorably in physical encounters, despite the age difference.

Firstborns often occupy the role of a surrogate parent, assisting parents with child care, and therefore become junior parents. Surrogate parental care by firstborns often continues in adulthood and may involve economic and other forms of assistance that enhance the fitness of younger siblings (Draper & Hames, 2000). In a study of 1,558 adults living in the Netherlands, Pollet and Nettles (2007) found that firstborns in sibships of three children were twice as likely as were their younger siblings to keep in touch on a weekly basis. Sometimes the role of a surrogate parent falls to a younger sibling rather than the oldest one. Owing to sex-role stereotypes, for example, the surrogate parent role may be thrust upon the eldest female if older children are male, and if there are younger siblings needing child care. Because birth order is only a proxy for differing family niches (together with their associated roles), data about actual family roles is a better predictor of personality than is birth order (Sulloway, 2001). Lacking such specific and possibly overriding information, birth order is nevertheless a useful predictor of the roles likely to have been adopted, and of the individual characteristics likely to have been promoted, because of the particular niches occupied as a consequence of birth order.

Firstborns are usually the first child from among their siblings to attend school. Once they begin school, they seek to maintain parental favor by fulfilling parental standards of responsibility and achievement. Firstborns tend to excel scholastically by becoming the "studious" sibling (Paulhus et al., 1999). Partly as a consequence, firstborns are overrepresented among people listed in *Who’s Who*; among American presidents and other world political leaders; and among eminent scientists, including those who have won the Nobel Prize (Altus, 1966; Clark & Rice, 1982; Sulloway, 1996). Seeking a different niche to fill than their older siblings, younger siblings sometimes differentiate themselves by developing abilities in sports. In a large national sample of college freshman that included numerous demographic controls \( N=193,422 \), Theroux (1993) found that laterborns were more likely than firstborns to win a varsity letter in high school. Compared with firstborns, younger siblings were also more likely to spend time discussing sports with their friends.

One instructive example of sibling specialization comes from the family of Ralph Nader, the consumer advocate and several-time candidate for president of the United States. When Nader and his three older siblings were adolescents, they divided the world into four equal parts. Each sibling took one quarter of the world, and they subsequently specialized in the history, culture, and languages of his or her own particular portion of the globe. As part of this sibling bargain, Ralph Nader learned three different languages (Chinese, Russian, and Arabic) that were associated with his chosen geographic domain. In accordance with Darwin’s principle of
divergence, the Nader siblings intuitively understood that they were better off by specializing in different areas of study and then pooling their collective resources (Sulloway, 1996). This example underscores the fact that within-family divergence and niche picking not only reduce competition, but they also facilitate potentially beneficial cooperation, with siblings profiting from each other’s specialties and talents, and with inclusive fitness potentially being enhanced by such cooperation.

**Birth-Order Stereotypes**

Several different studies have documented the existence of birth-order stereotypes (Baskett, 1985; Herrera, Zajonc, Wieczorkowska, & Cichomski, 2003; Musun-Miller, 1993; Nyman, 1995). Not surprisingly, these stereotypes are similar to differences that are found when siblings rate one another on measures of personality, as many stereotypes are based on commonly observed differences, such as those associated with gender. Firstborns, for example, are generally thought to be more intellectually oriented than laterborns, and they are expected to achieve higher social status. Females are expected to be more nurturing and to invest more in their younger siblings than are their male counterparts. Stronger children are asked to do more physically difficult tasks than are weaker children, and so on. Such stereotypes sometimes influence patterns of parental and sibling investment, thereby transforming cultural expectations into self-fulfilling prophecies that have psychological and even biological consequences, through their influence on fitness.

**Birth Order And Personality**

More than five hundred studies have been conducted on birth order and its effects on personality. Many of these studies have reached conflicting conclusions, leading some researchers to conclude, wrongly, that the results cancel themselves out and that birth order exerts little influence on personality (Ernst & Angst, 1983; Harris, 1998; Schooler, 1972). One major source of confusion in this extensive published literature involves the large number of studies that contain major confounds because they did not control for social class or sibship size. Large sibships are more prevalent among lower-class families. Hence poorer families are biased for an overrepresentation of laterborns. If a birth-order effect is found in a sample that has not been controlled for social class or sibship size, one cannot be certain that the observed effect is free of the most crucial confounding influences (Ernst & Angst, 1983).

**Within-family Studies of Birth Order and Personality**

The clearest evidence for birth-order differences in personality comes from studies in which brothers and sisters have made ratings of themselves and the siblings with whom they were raised. This within-family design eliminates any confounding due to between-family differences. Ten such studies using self and sibling ratings, and involving more than 7,000 participants, have assessed personality according to the Five Factor Model. To provide an estimate of mean-weighted effect sizes from these ten studies, I have assessed these findings meta-analytically in Table 4.2. The results reveal that firstborns are rated by themselves and their siblings as being more conscientious than laterborns; and laterborns, compared with firstborns, are rated as being more extraverted, agreeable, and open to experience. The results from these ten studies show significant differences in the magnitude of birth-order effects among the principal dimensions of
personality. For example, conscientiousness is more highly correlated with birth order than are the other four dimensions.  

2 Assessments of differences in the magnitude of effect sizes for birth order and personality are based on the procedure of Meng, Rosenthal, and Rubin (1992) for comparing correlated correlation coefficients. For conscientiousness versus extraversion in Table 1, $Z=2.19$, $p=.03$. For conscientiousness versus agreeableness, $Z=3.87$, $p<.001$; for conscientiousness versus openness to experience, $Z=3.44$, $p<.001$; and for openness to experience versus two self-consciousness scales on the dimension of neuroticism, $Z=2.36$, $p=.02$.

These disparities in the magnitude of birth-order effects suggest that niches shaped by birth order exert more influence on some personality characteristics than others.

Birth-order effects in personality exhibit both linear and quadratic trends, with linear trends generally being larger than the quadratic trends for most dimensions of personality (Sulloway, 2001). For this reason, middleborns and lastborns are usually more similar to one another in overall personality characteristics than they are to firstborns. Linear trends are expected based on the dominance-hierarchy hypothesis, as well as from influences that derive from differences in sibling age and size. By contrast, quadratic trends correspond with the hypothesis that middleborns differ from firstborns and lastborns owing to differences in parental investment (Hertwig et al., 2002). In addition, quadratic trends are consistent with the hypothesis that siblings who are closer together in birth rank will seek to differentiate themselves, making those furthest apart in birth rank somewhat more similar in personality.

Table 4.2. Meta-analytic findings for birth order and the Big Five personality dimensions, based on direct sibling comparisons.  

<table>
<thead>
<tr>
<th>Personality dimension</th>
<th>Mean-weighted correlation with birth order</th>
<th>N</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSCIENTIOUSNESS</td>
<td>−0.18</td>
<td>6,208</td>
<td>.0001</td>
</tr>
<tr>
<td>Firstborns are achievement oriented, conscientious, hard-working, organized, reliable, responsible, scholastically successful, and self-disciplined.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EXTRAVERSION</td>
<td>0.13</td>
<td>5,346</td>
<td>.0001</td>
</tr>
<tr>
<td>Laterborns are affectionate, excitement-seeking, extraverted, fun-loving, and sociable.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OPENNESS TO EXPERIENCE</td>
<td>0.11</td>
<td>7,218</td>
<td>.0001</td>
</tr>
<tr>
<td>Laterborn are attracted by novelty, liberal, prone to fantasy, nonconforming, rebellious, and unconventional.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGREEABLENESS</td>
<td>0.10</td>
<td>5,458</td>
<td>.0001</td>
</tr>
<tr>
<td>Laterborns are agreeable, easy-going, modest, submissive (unassertive), tender-minded, and trusting.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEUROTICISM</td>
<td>0.00</td>
<td>4,704</td>
<td>.93</td>
</tr>
<tr>
<td>There are no overall birth-order differences for being anxious, depressed, emotionally stable, self-conscious, and vulnerable.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(But firstborns are more anxious; and laterborns are more self-conscious.) (−.06) (.06) 3,346 4,198 .001.0001
Sample sizes for the ten studies included in this meta-analysis are as follows: Beck et al. (2006), $N=96$; Chao (2001), $N=412$ to 426 ($N$ varies by personality dimension); Healey & Ellis (2007), $N=161$ and 174 pairs; Paulhus et al. (1999), $N=148$, 194, 240, and 369; Rohde et al. (2003), $N=1,036$; and Sulloway (1999, 2001), $N=3,548$ to 4,510. The findings of Beck et al. (2006) and Healey and Ellis (2007) are controlled for age-spacing between siblings. The findings of Paulhus et al. (1999) are controlled for sibship size, as are those of Rohde et al. (2003), who also controlled for age-spacing. The findings of Chao (2001) and Sulloway (2001) are controlled for age, age-spacing, sex, sibship size, and social class.

A positive point-biserial correlation indicates that laterborns scored higher than firstborns.

Firstborns and laterborns are not expected to differ in being “creative,” since creativity can be expressed in different ways that exemplify “intellect” (a firstborn trait) as well as “unconventionality” (a laterborn trait). Consistent with this expectation, Paulhus et al. (1999) found a correlation of only .01 between birth order and being “creative.” These researchers did not make a prediction regarding the direction of this effect, which, for the same reason, is not included in this meta-analytic review.

Beck et al. (2006) classify “dominance” under extraversion. In factor analysis of 30 bipolar adjective pairs chosen to represent the 30 facets of the NEO PI-R Five Factor Model (Costa & McCrae, 1992), Sulloway (2001) found that “assertive (dominant)/unassertive (submissive)” had its highest loading on agreeableness (−.55) rather than on extraversion (.32). The results for this trait are therefore included with agreeableness in this meta-analysis (see also Sulloway, 1996:74).

**Between-family Studies of Birth Order and Personality**

Results based on between-family studies are generally consistent with those of within-family studies. In a meta-analysis of 188 study outcomes published between 1940 and 1999, significant birth-order trends emerged for all five dimensions of the Five Factor Model of personality, after controlling for differences in sibship size and social class (Sulloway, 1995, 1996, 2002a). Firstborns scored higher than laterborns in conscientiousness, and to a modest degree in neuroticism; and laterborns scored higher than firstborns in agreeableness, extraversion, and openness to experience. Overall, meta-analysis revealed more than five times as many confirming outcomes as opposing outcomes for the Big Five dimensions as a whole. The most consistent differences were those documented for conscientiousness, extraversion, and openness to experience.

Like the findings from within-family studies, between-family outcomes suggest that personality entails an adaptation to the family environment. For example, laterborns seek to discover unique family niches that have not already been taken by older siblings, and hence to obtain greater parental investment through experimentation and risk taking, which together reflect aspects of extraversion and openness to experience (Sulloway, 2001). Risk taking tends to be adaptive whenever organisms, including humans, seek to increase their status in social groups (Ermer, Cosmides, & Tooby, 2008)—including the family group. In addition, the lower a child’s likelihood of surviving or reproducing, the more it pays to take risks (Daly & Wilson, 1988). Consistent with these theoretical expectations, studies have shown that laterborns are more likely to have unconventional interests and to take physical risks (Sulloway, 1996, 2001), and, in the domain of mating activities, to have multiple partners and to pursue “short-term” mating strategies (Michalski & Schackelford, 2002; Theroux, 1993).
Perhaps the most studied aspect of birth order and risk taking involves participation in dangerous sports. In a meta-analysis of 8,340 participants in 24 different studies of athletic participation, laterborns were found to be 1.5 times more likely than firstborns to engage in dangerous sports such as rugby, football, and soccer, whereas firstborns and only children preferred safer sports such as swimming, tennis, and track (Sulloway & Zweigenhaft, 2010). This same study analyzed 700 brothers who played Major League baseball since 1876. Younger brothers were 10.6 times more likely than their older brothers to attempt to steal more bases (the odds ratio), and they were also more adept at doing so without being thrown out.

In general, birth-order differences documented in between-family studies are smaller than those found in within-family studies, although experimental manipulations involving between-family samples have sometimes yielded impressive effects (Courtiol, Raymond, & Faurie, 2009; Salmon, 1998; Sulloway, 2002a). The extent to which birth-order effects transcend the family environment, and the degree to which family-related primes and experimental manipulations can influence such effects in nonfamilial settings, are important topics that deserves further research. What seems clear is that some birth-order differences are predominantly situational, reflecting ongoing rivalry and differing roles within the family system rather than permanent features of personality.

**Birth Order And Social Attitudes**

Birth order is related to social attitudes. In by far the largest study on this subject, which included 193,422 participants and was controlled for sex, sibship size, and social class, Theroux (1993) found that laterborn college freshmen in the United States were more likely than firstborns to endorse what might be characterized as liberal views. Laterborns, for example, were more likely to support legalization of abortion, to oppose laws prohibiting homosexual relationships, and to endorse casual sex. Compared with firstborns, laterborn college freshmen were also less likely to attend church on a regular basis (see also Saroglou & Fiasse, 2003).

Differences in social attitudes by birth order appear to be closely linked with parental identification, which is in turn mediated by parental investment. For example, higher levels of parent–offspring conflict are associated with more liberal social attitudes among offspring, as well as with greater openness to experience (Sulloway, 1996, 2001). In a study of 649 Chinese Tokok families living in Indonesia, Skinner (1992) found that parents systematically favored older siblings by providing them with more of the family’s limited resources for education and favorable marriages. At the same time, parents expected older offspring—especially the eldest son—to accept an arranged marriage that benefited the family socially and financially, to provide support for the parents in their old age, to be responsible for commemorative rituals of ancestor worship, and to comply with Confucian ideals about filial obedience and familial responsibility. In turn, older Tokok siblings were found to be more conservative, socially and politically, and more obedient to their parents’ wishes, than were their younger siblings.

Such trends linking birth order with social attitudes have also been observed among immigrant families. The process of being absorbed into a new culture can lead parents to pressure offspring—particularly older offspring—to preserve the family’s cultural values. In a study of the transmission of social attitudes from parents to 1,042 second-generation Japanese-Americans, Manaster et al. (1998) found that firstborns were more likely
than laterborns to hold onto their Japanese culture. Firstborns, for example, were more likely than their siblings to live in Japanese neighborhoods, espouse Japanese values, place greater importance on religion, and adhere to the Buddhist or Shinto faith of their parents. In the realm of politics, Japanese-American firstborns were 1.4 times more likely than laterborns to vote for conservative political candidates.

In a meta-analysis of 20 previous studies of birth order and social attitudes (Sulloway, 2001), the mean-weighted correlation between birth order and endorsing a liberal viewpoint was 0.09 (N=11,240; controlling sibship size and social class). An effect size of this magnitude is equivalent to laterborns being 20 percent more likely than firstborns to endorse a liberal political position or candidate, which, in the United States, is roughly equivalent to the gender gap in conservative/liberal voting behavior (Clark & Clark, 2008).

**Radical Historical Revolutions**

In Western history, laterborns have been more likely than their eldest siblings to support radical revolutions. In a survey of 121 historical events, including 28 revolutions in science and more than 90 political revolutions and reform movements, laterborns were generally twice as likely as firstborns to support the radical alternative (Sulloway, 1996, 2002b, 2009). During the Copernican and Darwinian revolutions, for example, younger siblings initiated and supported novel scientific ideas that challenged the literal truth of the Bible. Nicholas Copernicus, who displaced the earth from the center of the solar system and demoted it to just one planet among many, was the youngest of four children. Charles Darwin and Alfred Russel Wallace, who codiscovered the theory of natural selection, were both the fifth of six children (Figure 4.5). Typically, the most vehement opponents of these radical doctrines—such as antievolutionists Louis Agassiz, Georges Cuvier, and William Paley—were firstborns.

Just as laterborns have been more supportive of radical change than firstborns during major innovations in science, they have also tended to endorse radical political upheavals. In a survey of 31 political rebellions taking place over the last four centuries, laterborns proved to be twice as likely as firstborns to back the radical alternative (Sulloway, 1996, 2002b). These findings about laterborn participation in political rebellions are consistent with the results of six within-family studies (N=2,427), which asked participants to identify the “rebel” of their sibling group (Chao, 2001; Paulhus et al. 1999; Rohde et al., 2003). Compared with firstborns, laterborns were 1.8 times more likely to be designated as the family rebel, controlling sibship size.
Figure 4.5. *Left:* Darwin (age seven) and his younger sister Catherine, who was the youngest of Darwin’s five siblings (from Darwin, 1903, 1: Frontispiece). *Right:* Darwin (ca. 1857), as he was writing the *Origin of Species* (from Seward, 1909: Frontispiece). Darwin’s family experience influenced his scientific career in several important ways. Based on a 10-variable logistic regression model, including five variables coding for within-family differences, Darwin’s predicted likelihood of endorsing the theory of evolution was 94 percent (compared with more than three hundred other scientists who spoke out on this theory—see Sulloway, 1996). Alfred Russel Wallace, who was politically more liberal than Darwin, was predicted to have a 96 percent likelihood of endorsing this same theory. In this multivariate model, the most significant predictors of support for Darwinian theory were having liberal social attitudes, being laterborn, being young, having experienced extensive conflict with a parent, and having traveled widely, which Darwin and Wallace both did. World travel exposed Darwin and Wallace to potentially fatal accidents and diseases, but it also provided them with compelling evidence of speciation occurring within oceanic archipelagos. This important biological evidence was a major factor in each of their decisions to accept the theory of evolution (Sulloway, 1979).

Historically, the benefits accruing from laterborn risk taking and rebelliousness have been manifested in changing laws that have created greater opportunities for people without inherited wealth. It is not surprising that radical social and political revolutions in Western history have repeatedly targeted the practice of primogeniture and have taken steps to abolish it, as was done by Martin Luther and other leaders of the Protestant Reformation and was also part of the sweeping legal reforms brought about by the French revolution. Similarly, advocates for the poor, including political activists supporting labor protests, have tended to be laterborns (Sulloway, 1996; Zweigenhaft & Von Ammon, 2000).

Even the Darwinian revolution was not without an implicit ideological message that favored younger siblings. Darwin, for example, wrote about “the evil consequences” of primogeniture in *The Descent of Man* (1871, 1:170); and he once avowed to a colleague: “But oh, what a scheme
is primogeniture for destroying natural selection” (1903, 2:34). Similarly, Patrick Matthew, who anticipated the theory of natural selection in 1831, declared that primogeniture was “an outrage on this law of nature [natural selection] which she will not pass unavenged” (Sulloway, 1996:242). Although primogeniture is no longer a common policy in modern societies, this and associated practices that discriminate parental investment by birth order are still observed in less developed societies, especially where wealth is based on land ownership.

A Darwinian Perspective On Individual Differences

Considering that most children in premodern times did not survive childhood and that many children still die in the poorest countries, the strongest selection pressures in human history have fallen on infants and young children. These pressures involve within-family dynamics, such as differential parental investment, parental manipulation to induce surrogate parental investment by older siblings in younger ones, and various sibling strategies (including role-taking and other aspects of niche-picking) aimed at altering the type, and increasing the amount, of parental investment. These kinds of within-family dynamics are closely linked with the development of sibling differences and are reflective of Darwin’s principle of divergence.

In a Darwinian world, sibling competition over parental investment represents a powerful engine of phenotypic novelty, causing siblings to behave differently from one another in their efforts to garner the resources needed for survival and reproductive success. Such differences in personality are analogues of the disparities in morphological traits, such as bill size and shape among birds, that relentlessly drive the evolution of species in nature. Although they are typically modest, the magnitude of the various individual personality differences that are associated with disparities in parental investment, as well as with differences in such factors as birth order and gender, is about the same size as morphological selection differentials found in the rest of nature. 

3 The mean point-biserial correlation observed for gender differences in personality is approximately .15 (Feingold, 1994; Hyde, 2005; for other within-family differences, see Turkheimer & Waldron, 2000). For typical effect sizes associated with rates of natural selection, see Hoekstra et al. (2001) and Kingsolver et al. (2001).

Such individual personality differences provide a more-than-sufficient basis for natural selection to operate on sibling behavior and strategies within the family.

In the course of human development, seemingly small differences early in life can have impressive cumulative effects over the lifespan, including differential reproductive success. For example, based on his or her SAT scores, a firstborn is about 13 percent more likely than secondborn sibling to be admitted to a top college (Sulloway, 2007b). Once admitted to a top college, a firstborn is more likely to be admitted to an elite graduate school, to win a post-doctoral fellowship, and, down the line, to end up being listed in Who’s Who and other biographical dictionaries devoted to famous achievers (Altus, 1966; Sulloway, 2009). With each crucial step in life, life paths diverge more and more. This is especially obvious where life presents dichotomous outcomes such as being admitted to college or not, or finding a mate or not. Over siblings’ lifetimes, the collective consequences of initially modest differences between brothers and sisters can be substantial. Charles Darwin’s remarkable scientific career is a case in point. Like many other contemporary laterborns, Darwin displayed a strong desire to travel to exotic places; and, based on his birth order, his odds of actually doing so were three times greater than for a nineteenth-century firstborn (Sulloway, 1996). Had Darwin not volunteered to go as naturalist on the Beagle voyage—an opportunity that arose after his teacher, a firstborn, declined this offer—he would never have visited the Galápagos Islands or encountered the many unusual
species that prompted him to develop his theory of evolution. Had he not been more flexible in
his religious views than his older sisters, and more intellectually daring and open to experience
than his older brother, he might never have generated that controversial theory of evolution by
natural selection, which undermined traditional theological beliefs. Like all of us, Darwin’s life
and career progressed one step at a time in ways that were shaped by earlier experiences within
his own family. For Darwin, these developmental experiences played a critical role in his
instigation of one of the greatest scientific revolutions in Western history. To that momentous
revolution we owe a whole new way of looking at the natural world, including considerably
greater insight into the question of why siblings are so different, and how these differences relate
to evolutionary fitness.

References

445.
1096–1101.
birth order among young Norwegian men (conscripts) analyzed within and between families. *Intelligence, 35*, 503–514.
Blanchard, R. (2004). Quantitative and theoretical analyses of the relation between older brothers
Psychology, 53*, 1214–1221.
Clark, C., & Clark, J. (2008). *Women at the polls: The gender gap, cultural politics, and


Chapter 5: Explaining Individual Differences in Personality: Why We Need a Modular Theory

Judith Rich Harris

People vary in personality and social behavior. It is generally accepted that some of this variation is due to differences in genes and some to “environment”—that is, to differences in people’s experiences. This chapter is about the latter source of individual differences, the variation that is not due to genes. More precisely, it is about theories designed to account for environmental influences on personality and social behavior by specifying some of the ways these outcomes are affected by people’s experiences.

A theory designed to explain how experience affects behavior is a theory of how the human mind works. The first question to be asked of such a theory is whether it makes sense in evolutionary terms. The human mind was shaped by natural selection, which means that during the evolutionary history of our species the modern version of the mind must have been tested against many alternative versions. It won out against the competition because it was more successful in enabling its owners to survive and reproduce. Thus, a theory of how experience affects behavior has to pass the test of evolutionary plausibility: Would a mind that works that way increase its owner’s fitness?

The second question is: How well does the theory account for the data? There are a great many findings on individual differences, reported by researchers from many areas of psychology using a wide variety of methodologies. Though it is unreasonable to expect any theory to account for all these data, there are some findings that are too important and too reliable to be ignored. If a theory cannot account for these findings, or generates predictions at odds with them, it has failed the second test.

In the next section of this chapter, I summarize some of the findings that a theory of environmental influences on human behavior should be called upon to explain. Then, in the following section, I will describe a new theory designed to account for these findings. In the final section I will examine some alternative theories.

Important Findings on Individual Differences in Personality and Social Behavior

The goal of researchers who study individual differences is to figure out what makes people vary. Why are some people fearful and others bold, some conscientious and others careless? One way of investigating human variation is by means of the methods of behavioral genetics (BG).

The Behavioral Genetic Evidence

The distinctive feature of BG methodology is that subjects participate in pairs. This gives the researchers a tool, admittedly imperfect but better than nothing, for distinguishing among various sources of individual differences. BG methods were formulated on the basis of two reasonable assumptions: that one of the reasons people differ is that they have different genes, and that another reason is that they grew up in different homes and were reared by different parents.

To everyone’s surprise, the second assumption was not supported by the data. For most of the outcome variables that have been measured, two people who grew up in different homes (but
within the same culture) are not noticeably more different from each other than a pair who grew up in the same home. To put it another way, sharing a childhood environment does not make people more alike. Once the effects of shared genes have been taken into account, the effects of the “shared environment”—which includes the neighborhood and school as well as the home itself—

1 By definition, shared environment includes all environmental factors that make reared-together siblings more alike. In BG studies, any measured similarities between reared-together siblings that cannot be attributed to shared genes are attributed to shared environment. Thus, if shared environment effects are found, they are not necessarily due to the shared home environment: They could be due to the school both siblings attend, the neighborhood they live in, the peer group they both belong to, or anything else they have in common.

—are negligible. This result holds for identical and fraternal twins, for ordinary siblings and half-siblings, and for biologically unrelated adoptees reared in the same family. On average, identical twins reared together are no more (or less) alike in personality than those reared apart, and adoptive siblings reared together are no more (or less) alike than two adoptees chosen at random (Bouchard & McGue, 2003; Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Plomin, Asbury, & Dunn, 2001).

The bottom line is that roughly half the variance in personality and social behavior remains unaccounted for in BG studies. Something other than genes must be producing variation among the participants in these studies—and producing it with uncanny consistency. The amount of nongenetic variance is approximately the same, whether the pairs of subjects grew up together or separately, and whether they are twins, ordinary siblings, or adoptive siblings. Identical twins are more alike in personality than other kinds of siblings, but once you allow for the similarities due to genes, you end up with about the same amount of unexplained variance. This means that the nongenetic differences between identical twins are as wide as the nongenetic differences between other sibling pairs. Whatever makes identical twins (reared together or apart) differ from each other in personality also, and to the same degree, makes ordinary siblings differ.

These counterintuitive findings present a real challenge to theorists. A successful theory will have to account not only for the nongenetic differences between ordinary siblings, but also for the equally large nongenetic differences between identical twins. This is a stumbling block for some otherwise promising contenders. For example, a current trend in psychology (e.g., Barry, Kochanska, & Philibert, 2008; Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Moffitt, Caspi, & Rutter, 2006) is to attribute unexplained variation to interactions between genes and environment (GxE interactions). The idea is that a particular environmental factor, such as the parents’ child-rearing style, might have different effects on different children, depending on their genotype. For example, if one sibling reacted to parental harshness by becoming more aggressive, while the other became fearful and meek, that could explain the failure of BG studies to find “main effects” of shared environment.

However, GxE interactions cannot explain the differences between identical twins. An interaction between genes and environment cannot produce differences between two individuals with the same genes. It would take a difference in environment—a main effect, not an interaction. But even for ordinary siblings, the GxE approach runs into trouble. To account for the BG data, we would need interactions in which some individuals respond one way to a given environmental input, while others respond in the opposite way, so that the results average out to zero. But such interactions are rarely found.
Most reported interactions involve differences in sensitivity, in which some individuals respond to a particular environmental input and others do not, or some respond more strongly than others.\(^2\)

\(^2\) Although GxE interactions of the sensitivity type no doubt exist, they have proved to be surprisingly difficult to demonstrate reliably. Studies that have been called into question due to failures to replicate include Caspi et al., 2002 (see Prichard, Mackinnon, Jorm, & Easteal, 2008) and Caspi et al., 2003 (see Risch et al., 2009).

Interactions of this type do not cancel out main effects—on the contrary, they are usually accompanied by main effects (Harris, 2006; Rowe, 2001).

**The Value of Experience**

Perhaps the unexplained variation is due to random processes in biological development, called *developmental noise*. Boomsma (2006) has listed some of the “endogenous factors” that might cause identical twins to differ: somatic mutation and recombination, differences in tissue-specific methylation patterns (methylation is an epigenetic process), and differences in X inactivation patterns in female twins. Another candidate is copy-number variation, the number of times a particular segment of DNA is repeated. Researchers have recently found copy-number differences between identical twins (Bruder et al., 2008). Other prenatal and postnatal factors, including exposure to viruses and injuries, might also play a role.

Some of the personality differences between identical twins are undoubtedly due to random biological processes of this sort. Since the same processes are at work in the chromosomes and brains of individuals who are not twins, they must contribute to the population variance. Thus, at least some of the unexplained variance measured in BG studies—variance usually attributed to the nonshared environment—must be of biological origin. Some of it may even be genetic.

But surely there must be differences that are truly environmental in origin! Although random biological differences may account for some of the unexplained variance in personality, it is implausible that they can account for all of it. If all the variance (aside from the negligible portion attributed to shared environment) were due either to conventional genetic effects or to random biological processes, it would mean that personality is entirely built in. Biology would indeed be destiny. What human children learned while they were growing up—growing up quite slowly, compared to other mammals—would have no long-term effects on their behavior. Despite their voluminous brains, the young of our species would be incapable of profiting from their social experiences by making long-term modifications in their social behavior.

Fortunately, there is enough solid evidence for long-term environmental effects on personality to rule out the possibility that all personality differences are biological. Consider, for example, cultural influences. A cross-cultural study of personality (McCrae, Yik, Trapnell, Bond, & Paulhus, 1998) found that, on average, the personalities of Chinese people in Hong Kong differed somewhat from those of Canadians. But individuals of Hong Kong-Chinese origin who grew up in Canada had personality profiles similar to other Canadians. Those who immigrated to Canada as adults retained the personality profile of their homeland, but those who were raised there developed Canadian personalities, even though the parents who raised them were Hong Kong-Chinese.

So environment matters. But that opens the door to another kind of randomness: environmental randomness. People’s life stories are full of chance events that set them on one path rather than another, sometimes with dramatically changed outcomes. You fell off your bike and broke a leg,
and there went your chance to be an athlete. Lucky guesses on an important exam increased your score just enough to get you into the class with the smart kids.

That kind of randomness must also play a role in development. But the plasticity seen in the young of our species, illustrated by the Canadian children of the immigrants from Hong Kong, must be there for a reason: Evolution does not provide expensive gifts unless they have a payoff. Why provide children with the ability to be modified, if the modifications are random? Random modifications would not, on average, increase their owner’s fitness. As Buss pointed out, “Status-striving mechanisms, for example, could not evolve unless they reliably produced classes of acts that actually led to the increase or maintenance of positions within social hierarchies” (1991, p. 464). Mechanisms that allowed themselves to be buffeted here and there by the vagaries of chance would not reliably produce classes of acts that had fitness-enhancing effects. Therefore, we are looking for evolved mechanisms that are capable of sifting the wheat from the chaff—mechanisms that respond preferentially to signals that, over evolutionary time, have proved to be useful guides for making long-term adjustments in behavior.

**How Do Children Learn?**

The process by which children profit from their experiences is called learning. A theory that posits environmental influences on long-term patterns of social behavior is, implicitly or explicitly, a theory of learning. What do we know about the way children learn?

The conventional view is that learning works this way: Through experience, instruction, or imitation, children acquire a response to a stimulus.

The stimulus may be a social partner, setting, or situation; the response is a pattern of behavior, an expectation, or an emotion. Once it is acquired, the response is automatically generalized to other similar stimuli. The baby learns that his mother is dependable; he subsequently expects other people to be dependable. The child learns to say “thank you” at home; she subsequently says “thank you” in school.

To illustrate generalization, many psychology textbooks still cite the experiment (Watson & Rayner, 1920) in which an infant called Little Albert was “conditioned” to fear a white rat—or was it a rabbit?—and supposedly generalized this fear to other furry animals and objects. It is true that Watson and Rayner stated their conclusion forcefully, but their methodology was haphazard and their results unconvincing. According to the original report, conditioning trials were given with three different animals: rat, rabbit, and dog. When the child was tested in a different room, he showed little or no fear of the rat, so he was given additional conditioning in that room, to “freshen the reaction” (p. 9). Albert was a stoic child and a thumbsucker; the researchers often had to “remove the thumb from his mouth before the conditioned response could be obtained” (p. 13).

More recent research, using better methodology, leads to a different conclusion: that true generalization is rare in our species (Detterman, 1993). Human infants appear to be born with a bias against generalizing; they are alert to any signs that this is a different situation, or a different person, and therefore might require different behavior. A baby taught to kick her foot in order to make a mobile jiggle will fail to kick if the crib is moved to a different room (Rovee-Collier 1993; see also Adolph, 2000). A toddler who has experienced nothing but kindness from everyone he knows will draw back in fear from a stranger (Eibl-Eibesfeldt, 1989). Infants of depressed mothers are subdued with their mothers but behave normally with other familiar caregivers (Pelaez-Nogueras, Field, Cigales, Gonzalez, & Clasky, 1994). Children who are
bossed around by older siblings at home are not more likely to behave in a submissive manner with peers (Abramovitch, Corter, Pepler, & Stanhope, 1986); a child who behaves like a subordinate with one peer can take a dominant role with another (Hawley & Little, 1999). And the child who says “thank you” at home may say “gracias” or “danke” at school (see Harris, 1998). The human mind is capacious enough to enable children to discriminate among their various social partners and form separate expectations for each of them. Likewise, children are capable of discriminating among their various social settings and acquiring patterns of behavior specifically tailored to each of them.

True, correlations between behavior in different settings are often found. Some children are fearful both in the laboratory and on the playground, or conscientious both at home and in school. But unless the research method includes a control for genetic influences on behavior, such correlations cannot be interpreted as evidence of learning and generalization. BG studies using a technique called multivariate genetic analysis have shown, for example, that correlations between fearful (or bold) behavior in two different contexts are due almost entirely to genetic influences on these behaviors. In contrast, environmental influences on fearfulness are context-specific (Saudino, 1997). Learned behaviors are tied to the context in which children learned them, but their genes go with them wherever they go.

For the same reason, genetic influences on behavior tend to persist over time. Although genes can produce developmental change as well as consistency, the long-term stability of personality traits is due primarily to the heritable component of such traits (Caspi & Roberts, 2001). Early-appearing traits that persist into adulthood are often interpreted as evidence that early experiences can mark a child’s psyche forever, but early-appearing, persistent traits are more likely to be a sign of inborn predispositions.

Thus, a theory that explains how children profit from their experiences does not have to give special weight to what happens in the first few years. The prolonged period of immaturity in humans is not a waste of time. Japanese children whose parents were temporarily stationed in California acquired American patterns of social behavior if they arrived in the U.S. by the age of 9 and remained for at least four years (Minoura, 1992). These behaviors led to social difficulties when the children went back to Japan, but they were able to re-adapt to Japanese norms if they returned by the age of 14 or 15.

### Anthropological Evidence

Current theories of child development mesh with current styles of child-rearing. They are part of our culture. Other cultures have (or had) different theories and different child-rearing practices. In the hunter-gatherer, tribal, and small-village societies studied by anthropologists and ethologists, child-rearing practices follow a traditional pattern quite different from our own. Most of these cultures are polygynous, and men in polygynous cultures generally play little role in rearing their children (Lancy, 2008). Mothers pay close attention to their babies’ physical needs, but seldom talk to them or try to teach them anything. Once they have been weaned, children spend most of the day in the local play group. According to ethologist Eibl-Eibesfeldt, “It is in such play groups that children are truly raised.... the child’s socialization occurs mainly within the play group” (1989, p. 600). Neither mothers nor fathers in these societies feel it is their job to entertain or play with their children (Lancy, 2008).
When considering theories of environmental influences on child development, it should be remembered that many of the parental behaviors endorsed by our culture would have been seen as aberrant in earlier times.

**Socialization versus Personality Development**

The acquired patterns of social behavior observed in the Japanese children moved temporarily to California, and in the children of the Hong Kong-Chinese immigrants to Canada, are the results of socialization. Socialization is the process by which children acquire the behaviors, attitudes, language, and skills appropriate for their culture. The result of socialization is that children become more similar to their same-sex peers in behaviors, attitudes, and so forth. The Japanese children in California learned to behave like the children they went to school with, despite the efforts of their parents to keep them from becoming Americanized (Minoura, 1992). A longitudinal study of Swedish children found that boys who behaved in a shy or timid manner at the age of 6 were no longer distinguishable in behavior from other boys by the age of 16 (Kerr, Lambert, Stattin, & Klackenberg-Larsson, 1994).

Thus, socialization (as defined here) leads to a reduction in behavioral variation, which makes it a poor candidate to account for the unexplained variance. In spite of becoming socialized, children continue to differ in personality and social behavior. Most of this variation is found within cultures, not between them.

A theory of social development should be able to explain why, during development, children become more alike in some ways (in language, customs, attitudes, etc.), while in other ways they maintain or increase their individuality. This observation makes more sense when we consider the various adaptive problems that must be solved in childhood if the child is to become a successful member of his or her society. One of the guiding principles of evolutionary psychology is that different adaptive problems require different psychological mechanisms to solve them (Buss, 1991; Cosmides & Tooby, 1994; Pinker, 1997). There is no all-purpose learning device but instead a suite of specialized devices, each with its own job to do. The answer to the question about how children learn is that they learn different things in different ways.

**A Modular Theory of Social Development**

In my earlier work, I followed generations of developmental, social, and clinical psychologists in attributing all environmental influences on social behavior to socialization; I proposed a “group socialization theory of development” (Harris, 1995, 1998). Individual differences were attributed vaguely to “differentiation within the group,” with no clear explanation of how or why that happens.

It now has become clear to me (Harris, 2006, 2009) that more than one adaptive process must be involved in social development. While they are growing up, children become more alike in some ways, less alike in others. They are motivated to conform (to be just like their peers), but they are also motivated to compete (to be better than their peers). The fact that these incompatible motives coexist, and sometimes lead to dilemmas in which an individual has to decide which internal voice to heed, is an important clue.

The expanded version of my theory (Harris, 2006) distinguishes between socialization, which adapts children to their culture, and personality development, which preserves or widens individual differences in behavior. I propose that three specialized psychological mechanisms are responsible for socialization and personality development in young humans. These mechanisms...
evolved to solve three distinct developmental problems: forming and maintaining beneficial relationships, becoming and remaining an accepted member of a group, and competing successfully with one’s rivals.

These three mechanisms respond to different environmental signals, collect different kinds of data, process the data in different ways, provide different motivations, generate different emotions, and have different developmental trajectories. In order of appearance, they are the relationship system, the socialization system, and the status system. I call them systems, rather than modules, by analogy with the visual system. Like the visual system, each makes use of a number of lower-level devices, for which the term modules is more appropriate.

The systems described here sometimes cooperate in producing a behavioral output (just as, in the visual system, several modules contribute to depth perception). Sometimes one system takes precedence over another, and occasionally they find themselves at loggerheads.

The Relationship System

Newborn human infants look avidly at human faces. It takes them only two or three days to prefer their mother’s face to that of a stranger (Pascalis & Kelly, 2009). In order to perform this feat, the baby needs not only a face-recognition module but also a module that collects and stores information about the individuals attached to these faces (“Mommy is the one who feeds me”). Pinker (1994, p. 420) proposed that the human mind is equipped with a “mental Rolodex: a database of individuals, with blanks for kinship, status, or rank, history of exchange of favors,” and so on. I call this module the people-information acquisition device (PIAD).

Like many mental mechanisms, the PIAD provides its own motivation. Babies look avidly at faces; older children and adults listen avidly to gossip. We eagerly collect information even about people we’ve never met—even fictional characters in novels—which shows that the PIAD makes use of face-recognition information but does not require it. The capacity of the PIAD is awe-inspiring. Modern humans store (and can retrieve) information about thousands of individuals: friends and neighbors; people they met in school or workplace; strangers they sat next to on the plane; and famous politicians, movie stars, and sports figures. The motivation to collect this information lasts a lifetime. Gossiping continues to be an enjoyable activity in the nursing home. The adaptive function and evolutionary history of the PIAD are easy to surmise. Like other primates, we humans are better off if we know something about the individuals with whom we might interact, so that we will know how to behave with them. Are they trustworthy? Do we owe them a favor? Can they beat us up? Are they fun to play with? Might they be a good trading partner? Might they be a desirable mate?

Finding and winning a desirable mate is a problem for which Buss (1991) has proposed a specialized psychological mechanism. In my model, this mechanism is part of the relationship system—a relatively late-developing part. Whether or not we are in the market for a mate, when we meet new people we immediately begin to store information about them, even though we don’t know at that point whether they will become friends, enemies, lovers, or business partners. If a friend becomes a lover, we might learn new things about him or her, but the information is recorded in the same mental Rolodex. We don’t have to start all over from scratch finding out what this person is like.
But finding out what people are like means occasionally having to change our minds about them, because sometimes the relationship system makes what social psychologists call the *fundamental attribution error* (Ross, 1977). When we meet someone for the first time and observe a small slice of her behavior (e.g., she acts angry), we tend to attribute her behavior to an enduring characteristic (she has a hair-trigger temper), rather than viewing it as a temporary state caused by her current situation. Although this attribution may prove to be incorrect, it is a reasonable one for the relationship system to generate. In the absence of other information, the best indication of how a person will behave in the future is how she behaved in the past. The relationship system is sometimes called upon to make a quick decision about how to behave toward a particular individual, so it swiftly collects whatever information it can get.

The relationship system goes to pains to distinguish one individual from another, even if the two individuals appear at first to be quite similar. If you get to know a pair of identical twins, your PIAD will collect information about each of them separately. People who are married to an identical twin sometimes dislike, and are usually not attracted to, their spouse’s twin. Only 13 percent of the husbands of identical twins said they could have fallen for their wife’s twin, even though she looks almost exactly like their wife (Lykken & Tellegen, 1993). The mating module might find her attractive, but its opinion has been overruled by other, more discriminating components of the relationship system. The same thing happens with biological relatives. You might have a gorgeous sister or brother, but on their page in your PIAD there is a checkmark next to “kin.” Other social interactions are also affected by that checkmark. You are more likely to help someone or share with them if they are your kin. On the other hand, kinship gives you greater leeway to behave badly, because people are less likely to harm or reject you if you are their kin (Daly & Wilson, 1988).

The relationship system generates powerful emotions, but these emotions are linked to specific individuals. Loving or hating your father does not make you feel the same way toward your boss (Mischel, 1968). True, some individuals have good (or bad) relationships with a variety of social partners, but genetic influences on personality, affecting traits such as agreeableness and neuroticism, can account for these persistent patterns (Caspi et al., 1997; Jockin, McGue, & Lykken, 1996; Nettle, 2007).

Though children’s experiences with parents and siblings have no long-term effects on the way they behave with people outside the family, such experiences leave very long-lasting memories. These memories are of the type called *episodic* or *declarative*—consciously retrievable memories of consciously experienced events. Because much of the information collected and stored by the relationship system is available to the conscious mind, this system takes up an inordinate share of our conscious thoughts. Humans talk, think, brood, and dream about their relationships.

**The Socialization System**

The relationship and socialization systems both collect information about people, but they handle the data in different ways. Whereas the relationship system specializes in finding out about individual people, the socialization system collects data on categories of people. Within every society, people in different social categories—male or female, adult or child, landowner or serf—behave differently. Thus, in order to learn how to behave appropriately, a child first has to figure out what sort of person he or she is. Once a child has
categorized herself as a “girl” (a female child) she will tailor her behavior to that of other girls. She will not take one particular girl as her model, because that girl might be atypical. Instead, she will form a concept of “girl,” much the way she forms concepts such as “bird” and “chair.”

This kind of processing is carried out below the level of consciousness—even people with amnesia can do it (Knowlton & Squire, 1993)—and results in the formation of a prototype (Rubenstein, Kalakanis, & Langlois, 1999). A prototype is the central tendency of a category, the mathematical average of the attributes of its members. Humans form prototypes of all sorts of categories, including patterns of dots shown to them by researchers (Smith, 2002). Babies start categorizing people, and forming prototypes for the various categories, before they are 6 months old (Pascalis & Kelly, 2009; Rubenstein et al., 1999).

The child will use the prototype of her social category as her standard for proper behavior, again largely without the aid of her conscious mind. Because these processes leave little trace in conscious awareness or episodic memory, people do not know how they were socialized. They attribute it to their parents because the relationship system devotes a good deal of space to parents and its data are readily accessible to the conscious mind. When people do not know why they behaved in a particular way, they use whatever information they have and make up a plausible explanation (Nisbett & Wilson, 1977).

Children are not socialized by parents or other adults; they socialize themselves. The motivation to identify with their own social category (which in childhood is often an actual group of children, such as a kindergarten class or play group), and the motivation to conform to the norms of that group, are provided by the socialization system. This explains why the children of immigrants adopt the language and social behavior of their peers, rather than those of their parents (Harris, 1998; Minoura, 1992; McCrae et al., 1998). Children whose parents are not immigrants can continue to use the language they learned at home, but the same is not true of social behavior. Developed societies demand very different behaviors at home and outside the home (Dencik, 1989). For example, displays of emotion that are acceptable at home would be seen as quite inappropriate in public. In addition, the relevant social categories may differ in different social contexts (Turner, 1987). In school a child may categorize herself as a girl, but at home or in a neighborhood play group, she may categorize herself simply as a child.

Though the human socialization system performs some cognitively sophisticated jobs, it has deep evolutionary roots. No doubt distinguishing males from females was one of its original jobs, but another ancient and evolutionarily important function was to distinguish “us” from “them.” Many species of nonhuman animals, from ants to chimpanzees, make this distinction and act upon it. Goodall (1986) reported that when a troop of chimpanzees split up and formed two smaller groups, members of one group attacked members of the other, even though they had played together as juveniles. Familiarity was not enough to override the distinction between “us” and “them.”

The socialization system is responsible both for the willingness to defend one’s group and for the merciless behavior, seen in humans especially during periods of intergroup warfare, towards members of other groups. As in the case of Goodall’s chimpanzees, “groupness” can override the opinions of the relationship system. But sometimes, when these two systems issue conflicting orders, the relationship system wins. Romeo loved Juliet even though she was a Capulet.
The Status System

The first two systems collect information about other people. The status system, in contrast, collects information about the self. In order to be successful children, and thus increase their chances of becoming successful adults, individuals need to acquire self-knowledge. They do this by comparing themselves with others in their own social category (Festinger, 1954). Answers to questions such as “Am I pretty or plain?” “Am I strong or weak?” depend on the prettiness or strength of the competition—others of one’s sex and approximate age. The fitness-enhancing result of collecting this self-knowledge is that one learns to avoid contests one is unlikely to win and to focus on those that offer better odds. A boy who discovers that he cannot compete with others his age in strength has time to develop another strategy. Perhaps he can specialize in being funny or smart, or in drawing pictures.

The status system develops more slowly than the other two. Ask a 4-year-old, “Who is the toughest boy in your nursery-school class?” and he will probably say “Me!” And yet he will defer to a larger or stronger member of his class if they both want to play with the same toy (Omark & Edelman, 1976). Dominance and submission are aspects of personal relationships and are monitored by the relationship system. Knowledge of one’s standing in the group requires more cognitive power, and it takes longer to gather and process the relevant data.

A boy can assess his strength in the course of rough-and-tumble play with other boys (Pellegrini & Smith, 1998). But how do children assess their own smartness, funniness, prettiness, or artistic ability? One useful clue is how often they are looked at; high-status members of primate groups are looked at more often (Chance, 1967/1976). But we humans are capable of obtaining more nuanced kinds of self-information from our interactive partners because, at least to some extent, we can read their minds (Baron-Cohen 1995).

In other words, we can guess what they are thinking and how they are reacting to us. The subtle, multidimensional information that young humans obtain from the feedback they receive from others enables them to make subtle, multidimensional modifications in their behavior.

Part of the status system’s job is to assess the characteristics that individuals are born with—their strength, prettiness, smartness, and so on—and adjust their social behavior to those attributes. Thus, the pretty girl and the big, strong boy are more likely to develop assertive, self-assured personalities (Jackson & Huston, 1975; Jones, 1957; Quinn & Wilson, 1989). Such adjustments, however, cannot account for the unexplained variance. To the degree that personality is a function of inherited traits, the results would show up in BG studies as genetic variance. But the link between genetic endowment and personality is not that tight. Many things, including random biological processes, can intervene between genetic plans and developmental outcomes. Malnutrition, illness, or injury could cause a boy to be weak and puny, even if he carries genes for size and strength. One who achieved his full genetic potential might find himself in a cohort that happened to contain a bigger, stronger boy. If the link between genes and optimum behavior were tight, a status system would be unnecessary; optimum behavior could simply be built in. The point of the status system is that it issues its advice on the basis of its owner’s phenotype, rather than genotype, and takes into account the actual environmental conditions at that time and place. Because the relevant factors are largely unpredictable, evolution provided the young of our species with the ability to make long-term adjustments in behavior on the basis of experience. Some of that experience comes from children’s own comparisons of themselves with their peers,
some from figuring out how they are viewed by other people in their community (adults as well as children), and some from assessing the opportunities afforded by the local environment.

Though personality can change, to some extent, even in adulthood (Caspi & Roberts, 2001), the status system does most of its work during childhood and adolescence. Consider, for example, the effects of being tall. Why do tall men receive higher salaries? The evidence suggests that the extra pay is dealt out, not for tallness per se, but for personality traits associated with tallness. Jones (1957) followed two groups of boys—slow and fast maturers—from early adolescence to adulthood. She found that boys who had been taller and physically more mature than their peers in adolescence had more dominant and self-assured personalities in adulthood, even though the slow maturers eventually caught up to them in size. More recently, researchers found that height in adolescence was a better predictor of adult salary than height in adulthood (Persico, Postlewaite, & Silverman, 2004). Taller, more mature boys tend to have higher status in the peer group (Weisfeld & Billings, 1988), and having higher status in the peer group in adolescence has lasting effects because personality is still plastic at that age but will become less so over the next few years. For the immigrants who arrived in Canada in adulthood, it was too late to develop Canadian personalities (McCrae et al., 1998). A boy whose physical maturation is slow may reach his full size too late to develop a self-confident personality. ³

³ A recent study (Case & Paxson, 2008) attributed the higher salaries earned by tall men to the fact that, on average, taller individuals have slightly higher IQs. But IQ cannot be the whole story. Persico et al. (2004) reported that the relationship between adult salary and height in adolescence was reduced only slightly when measures of childhood intelligence were included in their regression analysis. And Jones (1957) found persistent differences in personality as a function of height in adolescence.

The situation is more complicated for girls. Greater physical maturity does not necessarily enhance a girl’s status in her peer group. Prettiness does, but there is a dearth of relevant data on the long-term effects of having been pretty in adolescence.

The motives provided by the status system—to compete, to win, to outdo one’s rivals—sometimes conflict with those provided by the other two systems. While the socialization system is urging its owner to cooperate with the group and conform to its norms, the status system is striving for personal success. The resulting dilemmas are familiar ones. Should I advance my own interests by cheating or should I play by the rules? Which matters more to me, having high status or being accepted and liked?

**Sex Differences**

All intact humans have arms and legs, but some have longer legs or stronger arms than others. Likewise, all neurologically normal humans have the same mental mechanisms, but there are individual differences in these mechanisms. The motivation to form relationships or affiliate with a group or outdo their rivals seems to be stronger in some individuals than others. Some may be better at guessing what other people think of them, or at adapting their behavior to group norms.

To some extent, such differences may be a function of sex. A recent study (Benenson et al., 2009) designed to test the hypothesis that females are more sociable than males led to a different conclusion: that both sexes are sociable, but in different ways. Females place more importance on close personal relationships. Males are less concerned about personal relationships; they are more inclined to engage in group activities and to cooperate with other members of the group. A modular theory can easily accommodate these findings: In males the socialization system tends to take priority over the relationship system; in females it’s the other way round.
In males, the status system, too, seems to take priority over the relationship system. When boys and girls divide up into same-sex groups in childhood, there are noticeable differences in the way the members of these groups behave. According to Maccoby (1995), boys tend to be more competitive than girls and more concerned about their status, and their groups are more hierarchical. From an evolutionary viewpoint, status is more important to males than to females, because it affects a man’s reproductive success more than it does a woman’s (Buss, 1994).

**Testing the Theory**

As mentioned earlier, adolescent boys who are taller than their peers tend to develop more dominant, self-assured personalities and on average earn higher salaries in adulthood (Jones, 1957; Persico et al., 2004). I interpreted these results as evidence that experiences with peers in adolescence have long-term effects on personality, but an alternative interpretation is possible: Perhaps whatever causes a boy to reach his full height quickly (e.g., higher levels of androgens) also causes him to have a dominant personality.

Fortunately, there is a way around this problem. A boy might be a little taller and more mature than his peers, and consequently have higher status in the peer group, not because of genes or hormones but simply because he is a little older. Most schools in North America and Europe divide children into grades according to their birthdates, and the oldest students in a given grade—usually those born in fall and winter—have as much as a 12-month advantage in age over the youngest ones. My prediction is that if we compare males who were older than average for their grade in school with those who were younger than average, we would find differences, called relative-age effects, in personality. In terms of the five-factor model, the relatively older ones should score higher in extraversion, the relatively younger ones higher in neuroticism and perhaps agreeableness.

Relative-age effects became a topic of research interest when Barnsley and Thompson (1988) reported that top-ranked young hockey players in Canada are far more likely than chance to be born in the months from January to June, and linked this finding to the fact that the cutoff date for boys’ hockey teams is January 1. Subsequently, the discovery that major league baseball players are more likely to have birthdates between August and October was attributed to the fact that the cutoff date for Little League baseball teams is August 1 (Abel & Kruger, 2005). Similar results have been found for men’s soccer (Glamser & Vincent, 2004). The relative-age effect appears to be weaker or nonexistent in women’s sports (Vincent & Glamser, 2006).

Boys who are physically more mature are picked for better teams and get more practice playing against skilled competitors. Hence, the relative-age effect in sports might be due to differences in practice-based skills, rather than to differences in personality. But a review of relative-age effects (Musch & Grondin, 2001) concluded that psychological factors such as “perceived competence” play a role. The reviewers hypothesized that children who are older than their classmates or teammates develop higher self-esteem. This hypothesis is consistent with the evidence (Jones, 1957) that boys who are ahead of their peers in physical maturation develop more self-assured personalities.

Two more findings support the prediction that relative age influences personality. First, Nettle (2007) reported that Northern Europeans born in fall and winter score higher in traits related to extraversion, such as sensation-seeking. Second, researchers found that Canadians who had been younger than their classmates in school were more likely to commit suicide (Thompson,
Barnsley, & Dyck, 1999). The researchers attributed the heightened risk of suicide to “lowered confidence and self-esteem” (p. 82).

I have used the relative-age effect as supporting evidence because it is difficult to find systematic differences in social environment that are independent of the effects of genes. The relative-age effect cannot account for the personality differences between twins, however, because twins have the same birthdate. But relative age is only one of the many things that affect children’s status in their peer group and how they are perceived by their peers and by other members of their community. The mind of each of these perceivers is furnished with a relationship system, and each relationship system is noticing and recording any differences between the twins. Consequently, the twins will get different signals, different feedback, from the members of their community. The feedback will be used by the status system, along with other relevant information, to map out a behavioral strategy for each twin. This will happen whether they grow up together or apart, but it is easier to see it happening when they grow up together. For example, one twin might happen to be a little more talkative than the other on a particular occasion, and pretty soon people are addressing their questions and comments to that twin. She becomes the spokesperson for the pair and develops a more outgoing personality than her sister. Her status system is responding to self-information she obtained from other people’s relationship systems.  

4 Note that this theory does not attribute personality differences between twins to the twins’ having different friends or belonging to different peer groups. Reared-together twins generally belong to the same peer group.

The result is an increase in within-group diversity. In human groups—and in ants’ nests and bee hives as well—diversity leads to division of labor

within the group, and vice versa. Fewell (2003) has explained how division of labor can occur in insect colonies as a result of positive and negative feedback loops. The performance of a task by one individual reduces the chances that another individual will attempt to perform it (a negative feedback loop). The performance of a task by one individual increases the chances that the same individual will perform it again in the future (a positive loop). Putting this in human terms, if your group already has an artist, you might be better off finding something else to do. But once you start drawing pictures, practice causes your skill to increase, which makes it more likely you will continue to draw pictures.

The evidence cited so far in this section supports my explanation of how the status system produces variation in personality and social behavior. There is also evidence to support my proposition that the status system and socialization system work independently. Research has shown that feedback about group acceptance or rejection, and feedback about status within the group, have independent effects on subjects’ self-esteem (Leary, Cottrell, & Phillips, 2001). One can have positive feelings about one’s acceptance by the group and, at the same time, negative feelings about one’s status in it. Another study showed that college students who had high self-esteem regarding their status tended to be more aggressive than average, whereas those who felt good about being socially accepted were less aggressive than average.  

5 Kirkpatrick et al. (2002) assessed aggressiveness in their subjects by giving them an opportunity to administer punishment to a (nonexistent) person who had supposedly given them a bad grade on an essay. The punishment involved eating food spiked with hot sauce in a fictitious taste test. Subjects were asked to decide how much hot sauce the grader of their essay would have to consume. Those who chose to administer high amounts of hot sauce were rated more aggressive.
The researchers pointed out that aggressiveness is a risky tactic for individuals who pride themselves on their social acceptance; it could tarnish their reputation for being reliable, cooperative members of the group (Kirkpatrick, Waugh, Valencia, & Webster, 2002).

The proposition that socialization is context-specific has been tested in two studies designed to investigate how children acquire the self-discipline necessary to meet the behavioral requirements of the school environment. The researchers controlled for genetic influences on the relevant behaviors and found that parents’ child-rearing style (e.g., their enforcement of family rules) was unrelated to teachers’ judgments of the children’s behavior in the classroom (Wright & Beaver, 2005). The second study showed that the strongest influence on a child’s behavior in the classroom was the behavior of the other children in the classroom (Beaver, Wright, & Maume, 2008).

Wright and Beaver (2005) kept teachers’ judgments and parents’ judgments separate and reported both sets of judgments. Studies in which parents’ reports are used to assess how children behave outside the home (e.g., Straus, Sugarman, & Giles-Sims, 1997), or parents’ and teachers’ reports are combined into a single measure (e.g., Jaffee, Caspi, Moffitt, & Taylor, 2004) yield ambiguous or misleading results and cannot be used to test the predictions of my theory.

Other Theories of Individual Differences in Personality and Social Behavior

In this section of the chapter, I briefly examine some currently popular theories of personality development and individual differences.

Attachment Theory

The basic assumption of attachment theory is that relationships with caregivers (particularly the mother) in the first few years of life have lasting and pervasive effects. As a result of what happens in these early relationships, babies form either secure or insecure relationships with their caregivers. Those who form secure relationships are predicted to be more successful in their dealings with other people throughout their lives; those whose early relationships are insecure run a greater risk of a variety of social and emotional problems, including troubled friendships in childhood and troubled romantic relationships in adulthood (Schneider, Atkinson, & Tardif, 2001; Simpson, Collins, Tran, & Haydon, 2007; Weinfield, Sroufe, Egeland, & Carlson, 1999).

Does this theory pass the test of evolutionary plausibility? Imagine an ancestral population in which some babies were equipped with a mind that worked according to the precepts of attachment theory and others were equipped with a different kind of mind. Babies who inherited the second kind of mind would not be permanently disadvantaged if their relationship with their mother happened to go poorly but they nonetheless managed to survive infancy. They would not form their expectations of what the world was like, and whether it was safe to rely on people, on the basis of their experiences with their mother. Instead, they would go out and see for themselves what the world was like, and learn which people could be relied upon. Wouldn’t children with this second kind of mind have a fitness advantage over those with the attachment-theory mind? Wouldn’t the second kind of mind be favored by natural selection?

Evidence summarized earlier in this chapter supports my contention that in fact human babies are provided with the second kind of mind. Despite
the fact that heritable personality characteristics must play a role in all the child’s relationships, these relationships show a remarkable degree of independence. Nonetheless, heritable personality characteristics do exist; they tend to show up early and to persist over time. As noted previously, such characteristics are often misinterpreted as evidence of the long-term effects of early experiences. The type of research cited to support this interpretation almost never includes adequate controls for genetic influences on the measured outcomes.

**The Life History Approach**

An influential paper by Belsky, Steinberg, and Draper (1991) blended attachment theory with evolutionary psychology in an effort to account for individual differences in females’ sexual behavior and rate of sexual maturation. Belsky et al. proposed that a girl’s early experiences with her parents determine whether her reproductive strategy, 10 or 12 years later, will be short-term or long-term. If these early experiences indicate that the world is a friendly place and that people—in particular, men—can be relied upon, her strategy will be long-term: Physical maturation will be relatively slow and she will delay reproduction until she establishes a stable relationship with a dependable mate. If her early experiences are unfavorable, she will adopt a short-term strategy: Reproduce as quickly and as often as you can, and maybe some of the offspring will survive.

Again I ask, Would a mind that works the way the theory proposes increase its owner’s fitness? Would it benefit a girl to base her reproductive strategy on her experiences with her parents in the first few years of life? Are these experiences, now 10 or 12 years out of date, the best information she can obtain? Because many things might have changed in the interim, it would make more sense to wait as long as possible to settle on a strategy. Because her parents might be atypical, she would be better off collecting as much data as she can, by observing other people in her community. Birds are capable of making a decision about next year’s nesting site on the basis of information they collect by observing other nests in the area (Doligez, Danchin, & Clobert, 2002). If a bird has the mental capacity to do that, should we expect less of a child?

The research cited as support for the life history approach generally employed a standard methodology. The girls who participated in these studies each came from a different household, so there was no way to tell if a troubled household had a similar effect on all the children who grew up in it. In most cases there were no controls for genetic influences on the behavioral outcomes, even though genes shared by parents and offspring influence the

parents’ reproductive behavior, as well as the offspring’s (Bailey, 1997; Rowe, 2000). Some studies did control for the mother’s age at menarche, but personality traits are heritable too, and a girl inherits half her genes from her biological father. Another tactic was to follow the subjects longitudinally, but as the authors of one study admitted in a footnote, “Of course not even this longitudinal design can provide a truly adequate test of the theory because it cannot rule out gene effects” (Ellis & Essex, 2007, p. 1800).

The best way to rule out gene effects is to use BG methods. This has been done, in several large twin studies, for rate of physical maturation in girls. The consensus was that heritability accounted for 50 to 80 percent of the variance in age at menarche; the remainder of the variance was attributed to nonshared environment and measurement error (Ellis, 2004). So the environment shared by identical or fraternal twins accounted for none of the variance in timing of menarche. Twins who grew up in fatherless or stepfather families did not begin to menstruate earlier, on average, than those who grew up with a biological father. This conclusion was
supported by a later study of the daughters of twins. On average there was no difference in age of menarche between the daughters reared in stepfather families and their cousins reared in families with a biological father (Mendle et al., 2006).

Environmental factors are likely to play a larger role in the behaviors with which early physical maturation is correlated. Not only are early-maturing girls more likely to engage in early sexual activity—they are also more likely to engage in other teenage problem behaviors, such as delinquency and drug use (Flannery, Rowe, & Gulley, 1993; Magnusson, Stattin, & Allen, 1985). There is a simple explanation for this finding: Adolescents are influenced by their friends (Berndt, 1992), and early maturers tend to have older friends (Magnusson, 1988). As a consequence, they are introduced to the temptations of adolescence at an earlier age, when they are cognitively unprepared to make wise decisions. Teenage problem behaviors are also more prevalent in adolescents who have older brothers or sisters (Ernst & Angst, 1983; Rodgers & Rowe, 1988; Zweigenhaft, 2002). As a rule, birth order has no reliable effects on behavior or personality outside the family context, but this is one of the exceptions. Laterborns, too, receive an earlier introduction to the temptations of adolescence, by way of their older siblings and their older siblings’ friends.

Birth Order and Other Environmental Differences Within the Family

In 1987, Plomin and Daniels asked, “Why are children in the same family so different from one another?” (p. 1). Ever since, researchers have been trying to answer that question. The goal was to account for the unexplained variance—the variation in personality and social behavior that could be attributed neither to genes nor to the environment shared by children reared in the same family. Two promising hypotheses received the most attention. The first was that siblings differ because their parents treat them differently; the second, that siblings differ as a result of their interactions with each other—for example, a firstborn becomes accustomed to dominating his younger siblings, a laterborn learns to deal with being dominated.

Both hypotheses were tested in a longitudinal study of 720 sibling pairs—twins, siblings, half-siblings, and stepsiblings—living in the same home (Reiss, Neiderhiser, Hetherington, & Plomin, 2000). The results were negative. Neither differential behavior by parents (e.g., favoring one child over another) nor asymmetrical relationships between the siblings themselves (e.g., domination of one by the other) could account for the nongenetic differences between the siblings. The parents did treat their children differently, but apparently they were responding to, rather than causing, the behavioral differences between the children.

Another major effort also yielded disappointing results. Turkheimer and Waldron (2000) found 43 studies that addressed the question of the unexplained variance and combined them in a meta-analysis. They concluded that the nonshared environmental factors measured in these studies “do not account for a substantial portion of the nonshared variability” (p. 78). Differential treatment by parents accounted for only 2 percent of the variance. Family constellation variables, including birth order and age differences between siblings, accounted for only 1 percent.

Everyone who has a sibling can remember many incidents from childhood in which that sibling was bossy, annoying, hurtful, or helpful. But these memories are the property of the relationship system. The evidence indicates that learning to get along with their siblings neither helps nor hinders children in their other relationships. Those who fight with their siblings are not at greater risk of getting along poorly with their peers (Stocker & Dunn, 1990). Firstborns and laterborns do
behave differently at home—when they play together, the firstborn tends to be more aggressive—but outside the home, as judged by researchers or teachers, they do not differ in aggressiveness (Abramovitch et al., 1986; Deater-Deckard & Plomin, 1999). Birth order effects show up reliably when subjects are judged by their parents or siblings, or when they judge themselves in comparison with a sibling. But with standard personality questionnaires filled out in a laboratory or classroom, significant effects are seldom found (Sulloway, 1999, 2007) and rarely replicated. If firstborns and laterborns do not differ reliably in their responses to standard personality questionnaires, then birth order cannot be an answer to Plomin and Daniel’s (1987) question, because most of the personality data reviewed by Plomin and Daniels came from standard personality questionnaires.

Moreover, much of that data came from studies of twins. As mentioned earlier, a theory of environmental influences on personality should be called upon to account for the nongenetic differences between identical twins, as well as the nongenetic differences between ordinary siblings. This is a problem for theorists who believe that an important source of nongenetic variation in personality is competition between siblings (as proposed by Sulloway, 1996). According to kin selection theory (Hamilton, 1964), the closer the relatedness of two individuals (i.e., the more genes they share), the more likely they are to behave altruistically toward each other. Consistent with kin selection theory, Segal (2002) has found that identical twins compete with each other less and cooperate more than do fraternal twins. Combining Segal’s finding with the theory that competition between siblings causes them to differ generates the following prediction: that nongenetic differences should be narrower between identical twins than between fraternal twins. This prediction is disconfirmed by the BG findings.

Nor are the differences narrower for twins or siblings who grow up in separate households. If siblings become different due to their interactions with each other or to their efforts to fill different niches in the family, then we would expect pairs reared together to differ more than those reared apart. That would mean negative correlations between the personalities of adoptive siblings, and lower correlations for twins reared together than for those reared apart. What BG studies actually find is that the correlation between reared-together adoptive siblings is approximately zero, not negative (Plomin & Daniels, 1987), and the correlation between identical twins is very close to .50, whether they were reared together or apart (Bouchard et al., 1990).

From an evolutionary viewpoint, the questions asked of attachment theories can also be asked of birth order theories. Would it increase a firstborn’s fitness if he went out in the world expecting to dominate others the same way he dominated his little brother? Would a laterborn be better off, from a fitness standpoint, if he never tried to assert himself because it hadn’t worked at home? It is true that siblings fill different niches within the family, but the world outside the family offers a whole different set of niches. You might be the best artist in your family, only to discover that the artist niche in your community has already been filled.

Birth order theories and attachment theories are both based on the idea that learned patterns of behavior generalize readily and automatically, from the home to the world outside the home, even in cases where generalization is counterproductive. The persistence of this idea can be traced to two commonly made mistakes. First, many theorists fail to take adequate account of genetic influences on behavior, and consequently misinterpret early-appearing predispositions as evidence of the lasting effects of early experiences. Second, many researchers fail to provide objective measures of
children’s behavior outside the home. The researchers either assume that the way children behave at home is a good indication of how they behave elsewhere, or they accept parents’ judgments as valid indicators of how their children behave in social contexts that don’t include a parent.

Why Are Siblings So Different?

Much of the evidence that led Plomin and Daniels (1987) to ask this question came from studies of identical and fraternal twins. Their evidence on personality came mostly from responses on standard personality questionnaires (though subsequent studies have replicated the results using a variety of other measures). In order to answer Plomin and Daniel’s question, it is necessary to explain not only what parents say about their children or what siblings say about each other, but also what people say about themselves on standard personality questionnaires. It is necessary to explain not only how children behave at home, but also how they behave outside the home and why they behave differently in the two settings. It is necessary to explain not only why ordinary siblings differ, but also why identical twins differ. GxE interactions can explain why children with different genomes might react differently to parental behaviors or family events, but if identical twins react differently, it cannot be due to a GxE interaction.

Plomin and Daniels asked about the differences between siblings because the evidence they reviewed came from twins and siblings. What they really wanted to know was not why siblings are so different but why people are so different. Answering the first question is a good first step towards answering the second.

Conclusions

“The mind has to be built out of specialized parts,” explained Pinker, “because it has to solve specialized problems” (1997, p. 30). This chapter has focused on three of the problems that young humans have to solve while growing up: forming and maintaining beneficial relationships, becoming and remaining acceptable members of their group, and competing successfully with others of their age and sex. I have proposed that the human mind contains three specialized mental systems for dealing with these problems. These systems operate to a large extent independently, which explains why individuals who do well (or poorly) in one of these social arenas do not necessarily do well (or poorly) in the other two. The three systems respond to different aspects of the environment, process the input in different ways, make use of different kinds of memory, produce different motivations and emotions, and follow different developmental trajectories.

An important feature of this theory is the way it handles learning and generalization. In particular, the relationship system does not generalize at all: It is a splitter, not a lumper (the socialization system is the lumper). Babies are not born expecting that everyone will treat them alike—the fact that Mommy takes good care of them does not lead them to expect equally benign treatment from other people they encounter. That is why children can be securely attached to one caregiver but insecurely attached to another, and why those who fight like cats and dogs with their siblings often have successful relationships with peers. The attachments are real and the sibling battles may be very bitter, but the behaviors and emotions they generate are tied to the specific relationships in which they occurred. In contrast, the child’s genetically influenced characteristics (including physical appearance) are likely to play a role in a variety of relationships.
Because people outside the immediate family are less tolerant than parents, children have to learn to behave in a way that is acceptable in their society. What makes this task challenging is that people in their society do not all behave alike: Members of different social categories are expected to behave differently. A child can pretend to be an adult in fantasy play, but one who does so at other times would be considered peculiar or perverse. Thus, children cannot learn how to behave appropriately by imitating their parents. My explanation of how they solve this problem is based on what is known about the cognitive processes of categorization, prototype formation, and implicit (or procedural) memory. Because these processes leave little or no trace in consciousness, introspection cannot provide accurate information about how we were socialized.

Testing this theory will require some changes in methodology (see Harris, 2009, Appendix 2). Current research methods often fail to make some crucial distinctions. Friendships, group acceptance, and group status are the products of three different systems and should be carefully distinguished. Context effects are too often ignored; children’s behavior in the presence of a parent is not a valid indicator of how they behave in other social contexts. Ideally, a research method should provide a way of controlling for genetic influences on behavior, not because they are of greater importance but because they make it more difficult to discern true environmental effects. At present, traditional BG methods are still the best tools we have for skimming off the effects of genes. Genotype studies that investigate the effects of variations in a single gene are promising, but they are not a substitute for methods that take into account the effects of the entire genome.

Finally, brain imaging studies offer another way to test this theory. Perceived rejection by a friend or lover, perceived loss of acceptance by a group, and perceived loss of status within the group should make the brain light up in somewhat different ways.

*Homo sapiens* is a group-adapted species with a highly complex social life. Acceptable social behavior differs between cultures and among the various social categories within a culture. Status in human groups does not depend solely on size and strength; a status-seeking strategy that works for one individual might be counterproductive or even hazardous for another. Dealing with these challenges requires an impressive amount of brainpower. Traditional theories of social development underestimate the complexity, capacity, and discriminative ability of the child’s mind.

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**References**


Jones, M. C. (1957). The later careers of boys who were early or late maturing. *Child Development, 28*, 113–128.


The development of individual differences has always been a primary focus of psychological research, and it continues to be an intensely debated topic to this day. Three issues in particular stand out in contemporary debate. The first pertains to the sources of individual variation, with the pressing task of understanding the interplay between genetic and environmental factors. Second, there is the issue of early experience (especially within the family) and its role in shaping later development, a role which some question (e.g., Breur, 1999; Harris, 2005) and for which there exists no comprehensive theory capable of accounting for many conflicting findings. Finally comes the issue of continuity versus discontinuity in individual differences across the life span; this subject is rendered difficult by the compartmentalized way in which development is often studied and by the lack of organizing principles for linking diverse behavioral phenomena, manifested at different points in time, into meaningful clusters. In this chapter, we illustrate how an evolutionary approach can advance understanding of all three of these issues, and how a developmental perspective can provide fascinating insights to the study of individual differences.

**An Integrative Perspective: Adaptive Variation in Life History Strategies**

A powerful way of understanding the evolutionary meaning of individual differences is to analyze them in the framework of strategic variation (Hagen & Hammerstein, 2005). Different phenotypes can be conceptualized as the manifestation of different adaptive strategies, that is, ways for an organism to balance costs and benefits in order to maximize its expected fitness (though of course this does not imply that all individual variation is adaptive). Here we focus on a specific kind of strategies, namely, life history (LH) strategies.

**Life History Strategies**

Life history theory is a branch of evolutionary biology dealing with the strategies that organisms use to allocate their limited time and energy to the various activities that comprise their life cycle (see Hill, 1993; Kaplan & Gangestad, 2005; Chapter 9 of this title). LH theory is essentially concerned with identifying optimal solutions—in fitness terms—to various trade-offs, the most important of which are that between somatic effort (i.e., growth, maintenance, and learning) and reproductive effort; and, within reproductive effort, that between mating (i.e., finding and attracting mates) and parenting (i.e., investing resources in already-born offspring). From another perspective, the crucial decisions involved in a LH strategy can be summarized by the trade-offs between current and future reproduction, and between quality and quantity of offspring (see Ellis, Figueredo, Brumbach & Schlomer, 2009).

Variation in ecological contexts alters the costs and benefits involved in these trade-offs, determining remarkable variation in LH strategies both between species and within the same species. A key factor affecting LH strategies is the pattern of extrinsic mortality; that is, mortality that cannot be prevented by altering the organism’s behavior. More generally, all (totally or partially) uncontrollable factors that negatively affect reproductive success can be considered sources of extrinsic risk (Quinlan 2007) or, in Ellis and colleagues’ (2009) formulation, extrinsic morbidity-mortality. When morbidity-mortality is high (e.g., because of high pathogen load), it is
adaptive to favor current reproduction by starting mating early, even at a cost for one’s future reproductive potential. In addition, high extrinsic risk means that investing in parental care has quickly diminishing returns: As (by definition) parental effort cannot decrease extrinsic morbidity-mortality, offspring’s fitness will not respond to parental care beyond a certain amount. Thus, environmental risk favors quantity versus quality of offspring and current versus future reproduction and selects for life histories that invest in mating at the expense of parenting (see Belsky, Steinberg & Draper, 1991; Chisholm 1993; Pennington & Harpending 1988). The same effects are caused by increases in environment unpredictability; by contrast, resource scarcity tends to slow down development, delay reproduction, and increase parenting effort. Although the different components of LH strategies are conceptually distinct, they are functionally related and often covary in real-life situations (Ellis et al., 2009); this is why some researchers place LH strategies on a single continuum from “fast” or r-selected (i.e., early maturation and reproduction, quantity over quality, mating over parenting) to “slow” or K-selected (the opposite pattern).

Importantly, in sexual species the two sexes predictably differ on most LH dimensions; they thus can be expected to employ somewhat different strategies in response to the same environmental cues. In most species, males tend to engage in higher mating effort and lower parental effort than females (Geary, 2002; Trivers, 1972). In addition, males usually undergo stronger sexual selection (i.e., their reproductive success is more variable) and tend to mature more slowly in order to gain the competitive abilities and qualities needed for successful competition for mates. Sexual asymmetries in LH strategies can be attenuated in species with substantial biparental care and monogamous mating systems. Compared with other mammals, humans show an unusual degree of paternal investment, and are clearly adapted for the possibility of monogamous, long-term relationships. However, human paternal care is highly variable and facultative (e.g., Geary, 2005), and strict monogamy is rare in human populations (Marlowe, 2000, 2003). Indeed, human mating is best characterized as strategically flexible (Gangestad & Simpson, 2000), with a widely documented tendency for men to engage in higher mating effort than women (e.g., Schmitt, 2005). As a result, the various components of LH strategies do not carry the same weight for men and women. The current vs. future reproduction trade-off is more pressing for women, since their reproductive rate is limited by the long gestation timing and the conspicuous energetic investment of pregnancy. In contrast, men can potentially sire many offspring in a very short time. Men’s crucial trade-off is that of mating versus parenting: The payoffs of high mating effort are potentially much larger for males, who can benefit directly from having access to a large number of partners; women can have only one child at a time (twin pregnancies aside), and thus benefit comparatively less from multiple matings. Therefore, factors that shift development toward fast LH strategies can be expected to have a larger impact on reproductive timing in women compared to men, and a larger impact on the mating vs. parenting balance in men compared to women. Finally, humans can enact “mixed” strategies, where parental investment and long-term commitment to one’s partner coexists with the occasional pursuit of short-term matings (see Del Giudice, 2009a; Gangestad & Simpson, 2000; Jackson & Kirkpatrick, 2007; Schmitt, 2005).

**Developmental Plasticity and the Role of Parental Cues**

Usually, organisms embody switching mechanisms that allow them to fine-tune their life histories according to the environmental cues they encounter
during development; in other words, LH strategies show *adaptive developmental plasticity* (Ellis, Jackson & Boyce, 2006; Chapter 7 of this title; see West-Eberhard, 2003, for a comprehensive account). Of course, adaptive plasticity does not entail infinite or arbitrary malleability: Rather, organisms assess their local environments and adjust their strategic decisions within a genetically-constrained reaction norm, following evolved rules that tend to maximize long-term fitness in different ecological conditions.

An especially valuable source of information about the local environment, particularly during the first stages of development, is provided by parents. Parental behavior and parental investment can vary according to the local ecology, to the parent’s own LH strategy, and to offspring condition (e.g., less investment in low-phenotypic-quality offspring); thus, offspring can use parents as a source of useful cues about the micro- and macro-ecological conditions they will (probabilistically) face in the future. This is especially true when the crucial features of the environment are tolerably stable, allowing for reliable prediction of future conditions. Even if information coming from parents is not completely reliable, offspring—or at least some offspring (see below)—may be better off not disregarding it, and better off still by using it, perhaps provisionally, to direct their strategy choice, with the possibility of later “revision.” So, in long-lived species like humans, one might expect multi-stage development of LH strategies, whereby individual strategies remain relatively flexible, rather than becoming fully established (i.e., fixed) at the beginning of life (Del Giudice, 2009b).

On the other hand, the relative unreliability of information about the future may also select for variation in plasticity, with some genotypes less responsive to parental cues and more similar to “fixed” strategists (Belsky, 1997a; 2000; 2005; Belsky, Bakermans-Kranenburg & van IJzendoorn, 2007; Wilson & Yoshimura, 1994). In general, LH theory shows that stochastic environmental variability tends to increase phenotypic variation (Roff, 2002); but even in the same environment there may exist different strategies (e.g. one privileging quantity, another quality) that enjoy equivalent fitness in the long term. In this case, strategy choice is expected to depend more strongly on genotypic differences. Mathematical models of plasticity predict an integration of genetic and environmental determination of individual strategies (Leimar, Hammerstein & Van Dooren, 2006). More generally, developmental plasticity coexists with genotypic variation; for example, different genotypes may be more or less plastic or may vary in the threshold required to switch strategy in response to environmental input (West-Eberhard, 2003). It may also be that individuals differ in terms of how long they remain open to environmental influences and thus plastic vis-à-vis reproductive strategy (see Belsky et al., 2007; Belsky & Pluess, 2009). In general, human reproductive strategies are likely to show extended plasticity,

with a multi-stage developmental process allowing for adjustment and revision, depending on the success of one’s strategy and on changes in the local environmental conditions.

**Life History Strategies and the Organization of Behavior**

When interpreted in a narrow sense, LH strategies refer mainly to reproduction-related traits, such as age at first reproduction, fertility, and mating effort. However, it is easy to see that the choice of a specific strategy can affect a much broader range of traits and behaviors (Belsky et al., 1991; Figueredo et al., 2004, 2006; Wolf et al., 2007). Imagine an organism that, following cues of environmental risk and unpredictability, adopts a strategy characterized by early reproduction and high mating effort. To succeed, the organism needs to out-compete same-sex
conspecifics and be chosen by members of the other sex. Especially for males, this is likely to involve status-seeking behaviors, plus considerable investment in traits and displays that the other sex finds attractive in short-term mates; in humans, these may involve verbal and creative displays, competitive sports, humor, and so on (Jackson & Ellis, 2009; Locke & Bogin, 2006; Miller, 2000). Moreover, the cues of environmental risk that drive the choice of the strategy will also prompt higher risk-taking in other domains (e.g., exploration, fighting, dangerous sexual displays) and a shorter time perspective, increasing preference for immediate over delayed rewards and impulsivity (Chisholm, 1999a; Daly & Wilson, 2005). Short time perspective and competitive attitudes should also decrease the willingness to engage in long-term cooperation and to behave altruistically (Belsky et al., 1991; Curry, Price & Price, 2008). And since the behaviors associated with a fast strategy will predictably increase the organism’s expected morbidity-mortality, the very fact of having adopted the strategy may act as a “self-produced cue” of increased hazard, leading to self-reinforcing feedback on behavior.

In synthesis, LH strategies play a powerful role in the organization of behavior. We can identify a cluster of related traits that are expected to covary along life-history dimensions: mating and sexual strategies, status- and dominance-seeking, aggression, cooperation, altruism, risk-taking, time perspective, romantic attachment and parenting styles. Correlations within this cluster have been documented in both nonhuman animals (e.g., Dingemanse & Réale, 2005) and in humans (Figueroed et al., 2006). Indeed, Figueredo and colleagues (2004, 2005, 2006; Chapter 8 of this title) identified a heritable general “K factor” accounting for a large proportion of variance in a suite of LH-related traits in humans. At the neurobiological level, LH strategies and transitions in animals are usually regulated by endocrine mechanisms, with sex and stress hormones playing a crucial role (e.g., Adkins-Regan, 2005; Hau, 2007; McGlothlin, Jawor & Ketterson, 2007).

The Development of Life History Strategies

In 1991, Belsky and colleagues presented an influential life-history model of psychological development, building on Draper and Harpending’s (1982) work on the effects of father absence on sexual behavior in adolescence and young adulthood. A core feature of Belsky et al.’s (1991) model was that caregiving behavior (which in turn shapes attachment security) acts as a parental cue to the safety and quality of the local environment: Felt security in the first 5–7 years would channel the child’s developmental trajectory along different LH strategies, with secure attachment leading to later reproduction and a quality-oriented style, and insecure attachment leading to earlier reproduction and quantity over quality. Furthermore, it was predicted that early experiences in the family that would influence felt security also would affect the timing of sexual maturation, with children experiencing less harmonious parent-child relations and exposed to father absence and/or marital conflict reaching puberty earlier than would otherwise be the case. Since this model was formulated, several revisions and integrations focused specifically on the role of attachment security have been proposed (Belsky, 1997b, 1999, 2007; Chisholm 1993, 1996, 1999b; Del Giudice, 2009a; Kirkpatrick, 1998; Simpson & Belsky, 2008). Here we synthesize the current state of the art, focusing on theory and describing key empirical findings pertaining to attachment.

Parental Cues and the Role of Attachment Security

The central idea of these developmental models is that parental behavior provides useful information to the child, allowing him/her to adaptively calibrate his/her LH strategy. Two basic
questions arise: What information? And how is the information encoded? Belsky et al. (1991) proposed that rejecting or insensitive parenting, often associated with marital discord and broader stressful ecological conditions, conveys information about: (1) resource scarcity and unpredictability, (2) low levels of social trust and cooperation, and (3) instability and low commitment in couple relationships. Chisholm (1999b) coined the term “socioassay” to characterize this information the child receives about the availability and quality of social relations. Chisholm (1993) also argued that the local mortality rate, a crucial LH parameter, is a key determinant of the quality of caregiving and social relations (see also Bereczkei & Csany, 2001), and is thus one of the major pieces of information indirectly received by the child.

How is all this information encoded? Parental sensitivity, acceptance/rejection, and familial stress are significant determinants of attachment patterns in infants and children. The general dimension of attachment security is then well suited to act as a “summary” of the quality and quantity of caregiving received by the child, which is the reason it figured prominently in Belsky et al.’s (1991) original model. Closely linked to the stress response system, the attachment system regulates the child’s feelings of distress, pain, fear, and loneliness; and while attachment security can change during the individual’s lifetime, it shows a prototype-like dynamic in which early security/insecurity (established in the first few years of life) can continue to affect behavior into adulthood (Fraley, 2002). Interestingly, insecurely attached adults have been found to report shorter estimates of their own life expectancy (Chisholm, 1999a), thus supporting the hypothesized link between attachment security and perceived environmental harshness.

While generally accurate, this outline needs some conceptual revisions. First, the models advanced by Belsky et al. (1991) and Chisholm (1999b) conflated the negative dimensions of the environment in a single, undifferentiated factor including risk, unpredictability and resource scarcity. Modern life history theory, however, suggests that different aspects of the environment may have different (even opposing) effects on LH strategies. Whereas extrinsic morbidity-mortality and unpredictability should shift strategies toward current reproduction and mating effort, as postulated in the original models, severe resource scarcity per se may actually have the opposite effect, favoring slow life histories and substantial biparental investment in children (see Ellis et al. 2009; Marlowe, 2000, 2003).

Second, maternal and paternal investment (and, consequently, attachment to mother and father) may provide the child with distinctive information about the environment (Del Giudice, 2009a); indeed, Ellis (2004) has long granted, following Draper and Harpending (1982), a special role for the father. Although data remain scarce, anthropological evidence suggests that maternal caregiving is more directly tied to environmental harshness than its paternal counterpart, which may be more dependent on mating system (e.g. polygyny vs. monogamy), the local sex ratio, and the intensity of male-male competition for status (Blurton Jones et al., 2000; Quinlan, 2007; Quinlan & Quinlan, 2007).

Third, parents may invest little in a given child because of “micro-ecological” factors such as parent’s mental illness, low phenotypic quality of the child, or the presence of step-parents. Even though such factors contribute to insecure attachment and early stress, they do not strictly convey information about the “macro-ecological” context à la Belsky et al. (1991); they
may actually provide information about the likelihood of receiving future investment by one’s own family (Ellis, 2004; Del Giudice, 2009a).

Perhaps due to the imperfect reliability of parental behavior as a source of information or the fact that having one’s development shaped by parenting may prove maladaptive in fitness terms, children differ in their sensitivity to the early familial environment (Belsky, 1997b, 2005; Belsky et al., 2007; Belsky & Pluess, 2009). Indeed, there are evolutionary grounds for hypothesizing that some children would be “fixed strategists” pursuing a particular LH strategy almost irrespective of their developmental experiences, whereas others would be “plastic strategists” pursuing conditional strategies strongly shaped by their developmental experiences. Consistent with this is emerging evidence that infants and toddlers with a highly reactive and negatively emotional temperament tend to be more affected by parenting than other children (reviewed in Belsky, 2005; see also Bradley & Corwyn, 2008), as do children carrying a particular dopamine receptor D4 allele (7-repeat DRD4; Bakermans-Kranenburg & Van Ijzendoorn, 2006) or alleles associated with low monoamine oxidase A (MAOA) activity (Caspi et al., 2002). These children appear to be more positively affected by nurturing and supportive rearing environments, as well as more negatively affected by harsh and unsupportive ones. Recently, another important genotype-by-environment (GxE) interaction involving attachment security in infants was discovered by Barry, Kochanska, and Philibert (2008). Whereas infants with one or two short alleles on the serotonin transporter gene (5-HTT) were affected, as expected, by maternal sensitivity, so that low sensitivity led to attachment insecurity, virtually all of those carrying two long alleles became securely attached irrespective of the quality of care experienced.

While genotypic factors may account for variable plasticity in response to parental influence, Boyce and Ellis (2005; Ellis, Essex & Boyce, 2005) have argued that children’s environmental sensitivity, which they label biological sensitivity to context, could itself be affected (at least in part) by the experience of early stress. In their model, high stress reactivity (viewed as a more plastic phenotype) would be adaptive both in supportive/favorable environments, where it would increase susceptibility to social and developmental benefits, and in very stressful/unfavorable environments, where it would prompt increased vigilance to danger and threats. This proposal is very interesting, and the idea that plasticity can be environmentally induced is definitely worth pursuing.

**Stages, Transitions, and Extended Plasticity**

We propose that human LH strategies develop in a flexible, multi-stage fashion. As already anticipated in the original formulation by Belsky et al. (1991),

enduring effects of early influences could be contingent on later ones, meaning that LH strategies would remain open, to some extent, to continual modification, though perhaps for some people more than others. A sequential process of assessment-adjustment could provide the best compromise between early commitment to a strategy (with the benefit of having time to develop the appropriate skills) and finely tuned tracking of changes in ecological and social conditions. In this process, it should be possible to identify some developmental switch points (see West-Eberhard, 2003; Ellis, Jackson & Boyce, 2006; Del Giudice et al., 2009) when plasticity is preferentially expressed and development is directed (or re-directed) along alternative pathways. At developmental switch points, genotypic variation is integrated with information from the environment (West-Eberhard, 2003), and the result of this integration shapes strategy “choice.”
What are the switch points in the development of human LH strategies? The answer is still partial, but our map is becoming more detailed (Figure 6.1). To begin with, some preliminary strategy-setting (affecting, for example, the degree of temperamental reactivity) may occur even before birth, for example following exposure to maternal stress hormones. Then, in the first years of life (when dependency on parents is maximal), attachment security can provide the child with indirect information about the local micro- and macro-ecology, thus entraining the development of conditional strategies. But when do these nascent LH strategies begin to be effectively implemented in children’s behavior?

The Juvenile Transition

Del Giudice (2009a; Del Giudice, Angeleri & Manera, 2009) argued that the first crucial switch point translating early stress into behavioral strategies coincides with the transition from early to middle childhood, labeled the *juvenile transition*, immediately following what Belsky et al. (1991) identified as essentially the sensitive period for establishing the nascent LH strategy. With the juvenile transition (which takes place around 6–8 years in industrialized societies), children dramatically increase their participation in social activities with peers, and they begin to effectively compete for place in dominance hierarchies and for ranking as socially attractive individuals. Middle childhood is characterized by a dramatic increase in competitive and social play (Pellegrini & Archer, 2005; Smith et al., 2005), by the onset of the first romantic (and sometimes sexual) attractions (Herdt & McClintock, 2000), and more generally by a peak in sexually differentiated behavior (Geary, 1998).

Figure 6.1 Stages and switch points in the development of human life history strategies.

The functional role of the juvenile transition vis-à-vis life history strategies is threefold: first, it co-ordinates the phenotypic expression of a suite of life history-related traits, including attachment, stress regulation, dominance seeking, cooperation, and nascent sexuality. Second, it does so in a sexually-differentiated way; for example, a mating-oriented strategy is expected to prompt an increase in high-risk, physically aggressive dominance-seeking in boys more than in girls (see below). Finally, human juvenility (i.e., middle childhood) provides an assessment
period before the actual onset of mating and reproduction; such an assessment period may be crucial for appraising the likely success of a chosen strategy, prompting strategic revision in case the strategy is unsuccessful or does not match the child’s social environment (Del Giudice, 2009b). Consistent with this claim is evidence that the degree of agonistic stress experienced in early adolescence affects the choice of mating strategies in adulthood (Davis & Werre, 2007). The intensity of social competition and the levels of trust, cooperation and aggression in one’s peer environment should be important factors contributing to the development of LH strategies in juvenility and adolescence.

When life history-related behavioral strategies are played out in the peer group, the relevant phenotypic traits become crucial in determining the outcomes: intelligence, attractiveness, and physical qualities such as strength and athletic prowess are required in different proportions by different strategies. Jackson and Ellis (2009) proposed that, especially for males, the social status acquired in adolescence (partly depending on one’s phenotype) should be a critical factor affecting the development of LH strategies. This approach is fully consistent with the model we present, although the critical phase of social feedback may already begin with juvenility.

Del Giudice and colleagues (2009) contend that the juvenile transition is mediated by the hormonal mechanism of “adrenal puberty” or adrenarche (the secretion of androgens 1

1 The main adrenal androgens are dehydroepiandrosterone (DHEA) and dehydroepiandrosterone sulfate (DHEAS), two chemical precursors of testosterone and estradiol that can be converted to active androgens and estrogens in the CNS (e.g., Labrie et al., 2001).

by the adrenal gland, beginning at about 6–8 years in industrialized countries; see Auchus & Rainey, 2004; Ibáñez et al., 2000). Adrenarche would act as a plasticity regulator, by integrating genetic and environmental information and shaping the expression of both sex-related and individual differences. From this perspective, life history strategies are primarily coordinated by a dynamic interplay of the stress and sexual endocrine pathways, with various neurobiological systems (e.g. the serotonergic system) being involved in their behavioral expression and fine-tuned regulation. The outcome of this hypothesized process is the emergence of coordinated individual and sex-related differences in the functioning of the stress system, the sexual system, and many behavioral systems directly or indirectly affected by them. This working model of the juvenile transition remains speculative in many respects, although much evidence is consistent with it: Del Giudice and colleagues (2009) summarize a number of empirical studies showing that the juvenile transition is linked to developmental discontinuity in aggression levels, and to the onset of anxiety- and aggression-related psychological disorders. Finally, a recent longitudinal study by Ellis and Essex (2007) found that early familial stress anticipates adrenarche in both males and females, consistent with the role we propose for adrenarche as a life-history switch point.

**Puberty**

The next switch point is provided by puberty, when individuals first enter the arena of actual mating and reproduction. The original prediction by Belsky and colleagues (1991) was that early stress would lead to earlier onset of puberty, as part of a strategy oriented to current reproduction. Following Draper and Harpending’s (1982) emphasis on father absence, many researchers have focused their puberty research on the effects of father absence and family structure on age at menarche, even though a central premise of Belsky et al.’s (1991) model was that the child
should be sensitive to more than just the presence/absence of father when it comes to calibrating a reproductive strategy.  

2 Other studies suggest that stepfather presence could have a specific role in predicting early menarche (see Ellis, 2004; Mendle et al., 2006).

There are now several studies assessing stressful family relationships more generally and looking at puberty timing in both sexes (see Belsky et al., 2007; Tither & Ellis, 2008 for reviews). The overall result of this evolutionary-inspired developmental research is that stressful and negative family relationships do seem to accelerate the onset of puberty (but only in girls; Belsky et al., 2007), and to predispose to earlier initiation of sexual activity. Fathers may play an especially important, even if not exclusive, role (Ellis, 2004; Quinlan, 2003); father absence per se, however, may not be the most important factor, since father’s psychosocial adjustment and parental investment appear to exert a sizeable moderating effect, consistent with Belsky et al.’s (1991) original theorizing. What is still debated is the extent to which the putative environmental influences being detected are genetically mediated or accounted for (e.g., Mendle et al., 2006, 2009; Rowe, 2000), though recent genetically-informed research indicates that this is by no means entirely the case (D’Onofrio et al., 2006; Ellis & Essex, 2007).

Tither & Ellis, 2008). In fact, even studies highlighting the genetic mediation of environmental influences may ultimately be consistent with the notion that, within a family, children vary in the extent to which their LH strategies are fixed or plastic (that is, developmentally regulated by family processes; Belsky, 2005).

Another important issue concerning puberty is the changing function of attachment when individuals enter reproductive age. Whereas attachment in childhood is primarily devoted to promoting survival (by securing parental investment and protection), in adults the attachment system serves a different evolutionary function—regulating long-term bonding between reproductive partners (Kirkpatrick, 1998; Jackson & Kirkpatrick, 2007). Whereas attachment in childhood plays the role of a key factor affecting the development of nascent LH strategies, in adulthood it becomes part of the individual’s manifest strategy.

Adulthood

Life history trade-offs extend well beyond puberty, and it seems likely that other switch points can be found across the human life course. For example, menopause most certainly represents a fundamental switch point for women, and there seems to be a tendency for men around the world to increase their parental effort when approaching middle age (Winking et al., 2007). Other factors may also contribute to strategic adjustment during adult life, even without qualifying as identifiable “switch points.” An event of special significance may be represented by the birth of one’s child: Not only does it signal (some degree of) reproductive success, but it is known to affect hormonal functioning in both sexes (e.g., Storey et al., 2000), and could thus directly interact with the endocrine systems which, in our model, regulate LH strategies. Dramatic changes in social dominance (especially for men) and in social support (especially for women) may also act as triggers for recalibrating one’s strategy in response to changing opportunities in the environment.

Sex Differences in Life History Strategies

When it comes to LH strategies, the sexes differ both in their available strategic options and in the related fitness costs and benefits. For this reason, the mechanisms regulating strategic variation are not sexually monomorphic and the same cues may exert quite different effects
depending on the person’s sex. What happens when environmental cues (e.g. rejecting parenting) signal elevated risk? In general, we expect LH strategies to shift toward present-oriented reproduction (i.e., early maturation, early sexual debut), increased mating, and reduced parental investment. However, there are reasons to expect males to adopt high-mating strategies even at moderate levels of risk (if able to mate with many partners), whereas females would favor higher investment levels in their offspring. Only at high levels of risk are females expected to adopt a male-like high-mating, low-parenting strategy (see Del Giudice, 2009a). Consistent with this account, romantic attachment styles in adults often show predictable sex differences, with males reporting higher levels of avoidant (dismissing) attachment, and females reporting higher levels of anxious (preoccupied, ambivalent) attachment.

This pattern is consistently found in community samples, whereas studies of undergraduates seem to yield smaller effects (see Del Giudice, 2009b; Del Giudice & Belsky, 2010). Sex-biased distributions of attachment have been found cross-culturally; across geographical regions, increased environmental risk predicts higher levels of avoidance, especially in females, and tends to reduce within-population sex differences (Schmitt et al., 2003). Avoidant attachment bears the hallmarks of a low-parenting strategy, favoring short-term relationship over intimate, long-term bonding (Belsky, 1997b; Kirkpatrick, 1998): Avoidant adults are more promiscuous and sexually unrestrained, less committed in couple relationships, tend to avoid intimacy, and are more likely to become sexually coercive (e.g., Allen & Baucom, 2004; Bogaert & Sadava, 2002; Brassard et al., 2007; Gentzler & Kerns, 2004). They report lower interest in long-term relationships (Jackson & Kirkpatrick, 2007), and tend to feel more attracted by persons other than the current partner (Overall & Sibley, 2008).

In contrast, anxious adults show higher dependency and are powerfully motivated to search for exclusive, intimate relationships. Whereas attachment anxiety in men is related to somewhat reduced mating success, in women it predicts earlier sexual debut, impulsive partner choice, and infidelity (see references above). Del Giudice (2009a) proposed that anxious attachment in women has been selected as a care-eliciting strategy, targeted at extracting continued investment and help from both partners and relatives. Attachment anxiety could play the role of a “counter-strategy” to male avoidance at moderate levels of risk. Moreover, both avoidant and anxious styles could predispose women to multiple, impulsive matings—consistent with the evolutionary hypothesis that women possess a conditional strategy of “facultative polyandry” (see Hrdy, 2000).

Sex differences in attachment styles are not only present in adults; notably, they seem to emerge during the juvenile transition, that is, around seven years of age (Del Giudice, 2008, 2009b; but see Bakermans-Kranenburg & van IJzendoorn [2009], and the response by Del Giudice & Belsky, 2010). The emergence of sex differences in middle childhood is a crucial link between early attachment and adult mating styles; since they are found with respect to child–parent relationships, they support the idea of a global reorganization of the attachment system in the transition from childhood to adulthood. According to Del Giudice (2009a; Del Giudice et al., 2009), sex-biased attachment patterns in juvenility are part of a more general sexual differentiation of LH strategies. Avoidant attachment in children is related to pseudo-maturity, overt/physical aggression, inflated self-esteem, and externalizing symptoms. These traits may be functional as part of a high-risk, dominance-oriented male strategy oriented
at gaining status and prominence in the peer group. In contrast, anxious attachment could function for females as a means of keeping oneself in close contact with the kin network (Del Giudice, 2009a; Goetz, Perilloux & Buss, 2009); more speculatively, emphasizing immaturity and dependency might work as an attractiveness display directed at males (Marquez & Rucas, 2008). In addition, attachment anxiety may predict increased relational/indirect aggression in the context of female peer competition (Campbell, 2009; Del Giudice, 2009b), but since most attachment-aggression studies have focused on overt physical aggression, this intriguing possibility remains to be explored. Finally, insecure attachment in juvenility predicts the early appearance of flirting and sexual contacts, even in pre-pubertal children (Sroufe et al., 1993); this further underlines the emerging functional coupling of the attachment and sexual systems.

Integration with Neurobiology

The present account of the development of attachment and LH strategies fits nicely with two recent neurobiological models of the stress response. Taylor et al. (2000) convincingly argued that the mammalian stress system works in a sexually dimorphic way: The classical “fight-or-flight” response is more typical of males, whereas females tend to manifest a “tend-and-befriend” response, characterized by protection of one’s offspring and increased affiliative behaviors. The “fight-or-flight” versus “tend-and-befriend” distinction closely mirrors the difference between (male-typical) avoidant and (female-typical) anxious insecure attachment, the latter being characterized by increased dependency and closeness-seeking.

Also relevant to our multi-stage theory of the development of reproductive strategies is Korte et al.’s (2005) “hawk-dove” model which regards individual variability in the physiology of the stress response as contingent on two alternative phenotypes. The aggressive “hawk” strategy is characterized by fight-or-flight behaviors, high androgen levels, low cortisol secretion, and high sympathetic/low parasympathetic activation. In contrast, the “dove” strategy is marked by freeze-hide behaviors and an opposite neurobiological profile; the two strategies are also hypothesized to be associated with different profiles in serotonin, dopamine, and vasopressin functionality. We think that the “hawk” phenotype could describe the stress response pattern of avoidant individuals (especially males): The interplay between stress and sex hormones during development may entrain a developmental pathway in which early insecure attachment interacts with androgen secretion via multiple feedbacks, leading to a profile of high androgen levels, lowered stress responsivity (e.g. lower cortisol secretion), and high aggression and impulsivity.

Conclusion

A great promise of the life history framework is its capacity to dramatically increase integration in the study of individual differences from a developmental perspective. There is, first, integration across life stages: Thinking of development as a sequence of switch points, with each phase linked to specific evolutionary functions, resolves the contradiction between continuity and discontinuity in development by relating them in the same explanatory frame. Next there is integration across behavioral domains: LH theory makes sense of covariation among different traits and behaviors, thus contributing to one of the main goals of individual differences psychology. Finally, this perspective permits increased integration between genetic and environmental determination of individual differences via the concept of developmental plasticity. The emerging map we have tried to draw highlights a fascinating and intricate landscape, full of uncharted pathways and opportunities for discovery; we anticipate that, in the
next future, the development of individual differences will become a productive and informative focus of inquiry in evolutionary psychology at large.

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A full understanding of the development and evolution through natural selection of alternative reproductive strategies requires two levels of explanation. First is proximal causation. How do alternative reproductive strategies develop over individual lifetimes? This question involves specifying how gene × environment × phenotype interactions structure the development of major patterns of individual differences. On the one hand, developmental experiences capture information that enables individuals to match reproductive strategies to environmental conditions encountered in their own lifetime. On the other hand, many allelic variations are maintained within populations, biasing development in given directions and increasing phenotypic diversity. While the critical role of both genes and environments is widely acknowledged, much less is known about how genetic and environmental influences are actually integrated in development to produce systematic adaptive variation. To address this issue, the first part of this chapter describes the concept of switch-controlled modular systems and their critical role in development and maintenance of alternative reproductive strategies. Second is ultimate causation. At an evolutionary level, how has the process of natural selection organized these modular systems? The second part of this chapter discusses the conditions under which natural selection favors regulation of alternative reproductive strategies through adaptive genetic variation versus conditional responsivity to developmental conditions.

How do alternative reproductive strategies develop over individual lifetimes?

To address this question, I present a framework for explaining adaptive individual differences that draws heavily on West-Eberhard’s (2003) model of development of alternative phenotypes. Central to this model are switch-controlled modular systems, which I illustrate in terms of the development of alternative patterns of sexual maturation and related reproductive behaviors. Despite my focus on reproductive strategies, the West-Eberhard model provides a general framework for understanding causes and development of individual differences.

Perhaps the most striking feature of human sexual development and behavior is its variation. Some individuals complete pubertal development in primary school while others are still relatively undeveloped when they start high school; some begin sexual activity and reproduction as teenagers while others delay having children until decades later; some pursue short-term sexual relationships with multiple partners while others commit to a single long-term partner for life. This variation begins with individual difference in maturation of the reproductive axis—when and how fast puberty occurs—which then feeds forward to many other reproductive characteristics. For example, girls who experience early pubertal development, compared with their later maturing peers, tend to have higher levels of serum estradiol and lower sex hormone binding globulin concentrations that persist through 20–30 years of age; have shorter periods of adolescent sub-fertility (the time between menarche and attainment of fertile menstrual cycles); experience earlier ages at first sexual intercourse, first pregnancy, and first childbirth; and tend to be heavier and carry more body fat in adolescence and early adulthood (reviewed in Ellis, 2004;
see also St. George, Williams, & Silva, 1994; van Lenthe et al., 1996). Variation in pubertal development is thus an important component of individual differences in reproductive strategies. How do genes and environments affect this variation? West-Eberhard (2003) provides a framework for conceptualizing genetic and environmental influences on development of alternative phenotypes. Central to her theory is the organized phenotype, which is initially provided by the parents in the form of a zygote and then changes during ontogeny in response to genomic and environmental influences. Developmental change is coordinated by regulatory switch mechanisms (R), which serve as transducers (mediators) of genetic, environmental, and structural influences on phenotypic variation (see Figure 7.1). These switch mechanisms control developmental switch points: “A point in time when some element of phenotype changes from a default state, action, or pathway to an alternative one—it is activated, deactivated, altered, or moved” (p. 67). This can involve a discrete structural change or a change in the rates of a process. In Figure 7.1, West-Eberhard diagrammatically presents the architecture of switch-controlled alternative phenotypes. I have adapted Figure 7.1 to illustrate the development of alternative life history strategies (fast vs. slow). The model distinguishes between regulatory switch mechanisms and the expression of alternative traits controlled by these switches. Genetic (r) and environmental (e) inputs interact with extant phenotypic qualities (e.g., condition) to determine the functioning of regulatory switch mechanisms and influence their thresholds (T). Once a threshold is passed (i.e., the switch occurs), the regulatory mechanism coordinates the expression and use of trait-specific gene products and environmental elements (a, b) that build either phenotype A or B (which are discrete at the individual level but usually form a normal distribution at the population level).

Regulatory switch mechanisms bear some similarities to the concept of evolved psychological mechanisms (e.g., Buss, 1999). Specifically, regulatory switch mechanisms are nervous system structures that (a) are designed to take as input only certain kinds of genetic, environmental, and phenotypic information; (b) process that information according to specific rules and procedures, which determine whether a threshold is passed; and (c) generate output in terms of changes in gene products and associated molecular functions and biological processes that, together with raw materials imported from the environment, implement the switch (i.e., coordinate and construct the alternative phenotype). Regulatory switch mechanisms divide the phenotype into modular systems: switch-controlled sub-units of the phenotype that display coordinated expression as a unit; are internally integrated (recurrence together in time or space of the same elements, indicating a common source of regulation); are temporally or spatially discrete relative to other systems; display stereotypy of form and location across individuals of the same species; and are semidissociable (able to be deleted or re-expressed as a unit) (West-Eberhard, 2003).

To illustrate the role of switch-controlled modular systems in the development of alternative reproductive strategies, consider the developmental event of gonadarche (maturation of the gonads) and its regulatory functions. Gonadarche is a switch point in human development that is regulated by the hypothalamic-pituitary-gonadal (HPG) axis. The HPG axis first develops and is temporarily active during periods of prenatal and neonatal development. Gonadarche is the secondary reactivation of the HPG axis after a period of relative quiescence during childhood. Gonadarche begins at approximately 9 or 10 years of age in girls and soon thereafter in boys. The actual developmental switch—gonadarche—includes a change from low-level, irregular secretion
of gonadotropin-releasing hormone (GnRH) to a pattern of distinct pulses. These pulses are released from neuroterminals in

1The neurotransmitter and neuromodulatory systems that control the GnRH secretory network.

2Threshold: change from low-level, irregular secretion of GnRH to a pattern of distinct pulses; threshold must be passed for gonadarche to occur.

3Environmental and genetic influences on regulatory mechanisms. Shown here influencing central regulation of HPG axis, but also influence peripheral switch points on the axis. Crossing arrows indicate that genetic and environmental influences moderate (depend on) each other.

5Specific modifier gene products and environmental elements (a, b) that build either Phenotype A or B but are not used in both.

6Species-typical gene products and environmental elements (c) that are shared by (expressed in) both Phenotype A and B.

Figure 7.1. Schematic diagram of the architecture of switch-controlled alternative phenotypes (adapted from West-Eberhard, 2003); applied here to gonadarche and its regulatory effects on the development of alternative life history strategies. Pathway A = fast life history strategy; Pathway B = slow life history strategy. ERα: estrogen receptor alpha; AVPVn: anteroventral periventricular nucleus; MPOA: medial preoptic area; LH: luteinizing hormone; GnRH: gonadotropin-releasing hormone.

the median eminence of the hypothalamus and bind to receptors in the anterior pituitary gland. This causes the gland to synthesize and secrete biologically potent gonadotropins: luteinizing hormone (LH) and follicle-stimulating hormone (FSH). At gonadarche, the GnRH surge markedly increases pulsatile secretion of LH and FSH, leading to a cascade of events, including gonadal maturation, increased production of steroid hormones, growth acceleration, weight gain, development of secondary sexual characteristics, and other processes that culminate in attainment
of adult levels of gonadal steroids and maturity of the reproductive system (see Ebling, 2005; Grumbach & Styne, 2003; Plant & Barker-Gibb, 2004, for overviews of the neurophysiology of puberty). The same control system that regulates maturation of the HPG axis also regulates its activity in adulthood (i.e., spermatogenesis, cyclic ovulatory function).

As shown in Figure 7.1, a switch point is controlled by a condition-sensitive, quantitatively variable regulatory mechanism (R) with threshold (T). In the case of gonadarche, the neurotransmitter and neuromodulatory systems that control the GnRH secretory network are the coordinating mechanism (cell assembly) that regulates maturation and functioning of the HPG axis (see Ebling, 2005). This cell assembly is made up of an array of neurons containing various neurotransmitters. Some of these neurotransmitters are generally stimulatory (e.g., glutamate, kisspeptin, galanin-like peptide, neuropeptide Y) and some are generally inhibitory (e.g., GABA, endogenous opiate peptides, corticotropin-releasing hormone) to the GnRH secretory network (Cameron, 2004; Ojeda et al., 2006). Appropriate pulsatile secretion of GnRH is the threshold that must be passed for the developmental switch to occur. The normal curve in Figure 7.1 represents the timing (i.e., age distribution) of the developmental switch (gonadarche) in the population. In terms of pubertal development, gonadarche can be thought of as a master switch, with subsequent decision points working as subordinate switches in a developmental sequence. The cell assembly that regulates this system is a major locus of operations for both genetic (r) and environmental (e) influences on not only the awakening of the HPG axis (gonadarche) but also operation of the axis after gonadarche occurs.

The Role of Environmental Factors

Environmental factors, such as nutrition and exercise, predation threats, or family stress and support, can potentially influence the reproductive axis at every developmental switch point (see Figure 7.1). For example, metabolic factors (nutrition, exercise) have a major influence on both the maturation and functioning of the HPG axis: Well fed populations experience early puberty and poorly fed populations experience late puberty (Parent, Teilmann, Juul, & Skakkebaek, 2003). Although it is not fully understood how energetic conditions influence reproductive development, an accumulating body of evidence now indicates that both metabolic hormones (e.g., leptin, insulin) and gastrointestinal hormones (e.g., ghrelin, PYY3-36), which signal the nutritional status of an individual to the hypothalamus, have multiple effects that are transduced across different levels of the HPG axis (Fernandez-Fernandez et al., 2006; Gamba & Pralong, 2006). That is, these hormones partially mediate the effects of energetic condition on pubertal maturation and gonadal functioning. Working through both peripheral and central nervous system pathways, these effects range from gonadal cell proliferation to modulation of the GnRH secretory network. Peripherally, variations in metabolic and gastrointestinal hormones can directly suppress (or bolster) the cellular machinery that controls gonadal steroid production (Ellison, 2001; Fernandez-Fernandez et al., 2006). Centrally, variations in these hormones can increase inhibitory (or stimulatory) drive to GnRH neurons, altering development and functioning of the HPG axis (Cunningham et al., 2004; Fernandez-Fernandez et al., 2006; Gamba & Pralong, 2006).

In sum, the HPG axis is a complex system that detects and responds to metabolic information at multiple levels. These metabolic effects, however, constitute only a subset of environmental influences on sexual maturation (see Ellis, 2004). The HPG axis is also responsive at multiple levels to information about psychosocial stressors. Information about environmental threats and
dangers is conveyed to the axis through activation of the stress response systems, which affect such neuroendocrine processes as GnRH pulsatility, GnRH surge secretion, pituitary responsiveness to GnRH, and stimulatory effects of gonadotropins on sex steroid production (Cameron, 1997; Dobson, Ghuman, Prabhakar, & Smith, 2003; Johnson, Kamilaris, Chrousos, & Gold, 1992; Rivier & Rivest, 1991). Whatever the sources of environmental influence, environmental effects are channeled through a common gateway: regulatory switch mechanisms. Different environmental factors, additively and interactively, influence when and if thresholds are passed and thus activate/deactivate switches that regulate development and functioning of the HPG axis.

The Role of Genetic Factors

Genes provide templates for molecules (and their receptor distributions) that become incorporated into the phenotype, depending on the responsivity of the phenotype to those molecules and the presence of necessary environmental building blocks (West-Eberhard, 2003). Together with environmental factors, a large number of genes influence the maturation and functioning of the HPG axis (see Figure 7.1). For example, 17 different single-gene mutations have been associated with delayed or absent puberty in humans (Herbison, 2007). More specifically, the cell assembly that controls the GnRH secretory network is influenced by genes that regulate the assembly’s neurotransmitter pathways. Some of these genes play a central role in GnRH secretion. For example, kisspeptins, which are the products of the Kiss1 gene, and their receptor, GPR54, directly affect GnRH neurons (Gottsch, Clifton, & Steiner, 2006). Genes encoding the two gonadotropins, LH and FSH, and their receptors also play a key role in development and activity of the reproductive axis (Huhtaniemi, 2006), regulating steroid-hormone biosynthesis and metabolism. In total, development and functioning of the HPG axis is influenced by complex and ordered networks of genes that regulate various physiological pathways; minor sequence changes in these genes as a result of mutations can cause abnormally early or delayed puberty (Banerjee & Clayton, 2007).

Although it is important to understand genetic regulation of species-typical pubertal processes (C), the West-Eberhard (2003) model is most relevant to understanding structured (functional) variation in reproductive strategies (A, B). Along these lines, heritability studies of Western populations suggest that at least half of the differences between individuals in timing of pubertal development is accounted for by differences in their genes (reviewed in Ellis, 2004; see also Ge, Natsuaki, Niederhiser & Reiss, 2007). This high heritability underscores the need to identify specific genetic influences (r) on normal variation in the development and functioning of the HPG axis. Identifying such influences has proven difficult, however, despite many candidate gene studies. Nonetheless, recent genome-wide association studies of European and American populations have found that variation in the LIN28B gene is associated with normal variation in pubertal development (specifically, age at menarche in girls and development of secondary sexual characteristics and middle-childhood growth rates in both girls and boys; He et al., 2009; Ong et al., 2009; Perry et al., 2009; Sulem et al., 2009).

Whatever the sources of genetic influence, genomic effects on regulation (r) are channeled through a common gateway: regulatory switch mechanisms (R). As with environmental factors, different genetic factors, additively and interactively, influence when and if the thresholds are passed that throw regulatory switches. Alleles at a range of genetic loci introduce variation into
regulatory mechanisms, contributing to individual differences in maturation and functioning of the HPG axis.

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**Gene–Gene, Environment–Environment, and Gene–Environment Interactions**

The key point is that regulatory switch mechanisms provide a common locus of operations for genetic and environmental influences on phenotypic development; that is, these mechanisms are the vehicle through which gene–gene, environment–environment, and gene–environment interactions occur. The HPG axis is characterized by a series of switch points across all levels of organization. Genetic (r) and environmental (e) inputs are integrated—additively and interactively—at these switch points (shown in Figure 7.1; crossing arrows indicate interactive effects). These inputs structure the operation of regulatory switch mechanisms (e.g., determine levels of pulsatile release of GnRH) and may affect the threshold (T) necessary for a developmental switch to occur and/or the organism’s ability to cross that threshold.

Importantly, regulatory switch mechanisms are influenced not only by gene products and environmental conditions, but also by extant phenotypic characteristics that modulate the regulatory mechanism’s functioning and sensitivity (e.g., metabolic efficiency, energy stores, leptin concentrations, efficiency of hormone-secreting organs). For example, because age at gonadarche is highly sensitive to energetic conditions, food-getting ability (a behavioral phenotype), metabolic efficiency (a physiological phenotype), and energy stores (an anatomical phenotype) all contribute to regulation; that is, these phenotypic traits mediate the effects of environmental conditions (food availability) and genes on the GnRH secreatory network. Further, the effects of genes, environments, and phenotypes are hierarchically organized: The preexisting phenotype is the transducer of both genetic and environmental sources of information (West-Eberhard, 2003). Specifically, genetic and environmental effects depend on the phenotype being organized to accept them, and the modified phenotype retains these effects as development proceeds.

The interactive effects of genes and environments on the HPG axis have been nicely demonstrated in research on alternative mating behaviors in male swordtail fish (*Xiphophorus nigrensis*). In the swordtail, three alleles at the *P* locus on the Y chromosome correspond to three modes in size distribution of mature males (small, intermediate, and large; Ryan et al., 1992). Although all three genotypes perform the range of species-typical mating strategies, they do so at different size-related frequencies. Specifically, small, intermediate, and large males generally sneak, sneak and court, and court females respectively. Size is the primary mediating mechanism in this species through which allelic variations influence mating strategies.

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In determining alternative mating strategies, the key developmental switch in male swordtail fish is gonadarche. Specifically, the three alleles at the *P* locus differentially influence timing of gonadarche (e.g., immunoreactive GnRH-containing neurons first appear at 5 weeks of age in genotypically small males versus 11 weeks of age in genotypically large males; Rhen & Crews, 2002). In addition to these allelic influences, timing of gonadarche is also sensitive to a number of environmental factors, such as temperature (Borowsky, 1987a) and agonistic interactions with other males (Borowsky, 1987b). These environmental influences can result in genotypically small males that are larger than genotypically intermediate males, and alternative mating strategies correlate more strongly with size than with genotype (Ryan & Causey, 1989). In addition, mating strategies of male swordtail fish are competition-dependent in relation to interaction with other
males. For example, males of intermediate size will sneak and chase females rather than court when in the presence of larger males.

In sum, both genomic and environmental factors influence timing of gonadarche, which in turn coordinates patterns of gene expression involved in the developmental cascade that induces sexual maturation and halts or dramatically reduces growth. Timing of gonadarche strongly influences size, and size is a major developmental factor in entrainment of alternative mating strategies. At the same time, mating strategies are facultatively adjusted in response to current physical and social dimensions of the environment. Thus, although there are strong genotypic influences on size and developmentally-linked mating strategies, the development of the alternative phenotypes in fact emerges through a complex series of gene–environment interactions. Importantly, these interactions occur through integrated effects of gene products and environmental conditions on developmental switches.

Although gene–environment co-regulation is normative, in some cases either genetic or environmental factors have a decisive impact on phenotypic determination. Genotype-specific regulation means that one of several alleles that influence a switch mechanism predominates over others (and over environmental factors) in the magnitude of its effect and thus has a decisive impact on phenotypic determination. Human sex determination, for example, depends on the presence or absence of a single gene, the H–Y antigen on the Y chromosome (Bull, 1983). However, genotype-specific regulation that is immune to environmental influence is rare in nature (West-Eberhard, 2003). Conversely, in the case of environment-specific regulation, one of several environmental cues that influence a switch mechanism predominates over others (and over allelic variations) in the magnitude of its effect and thus has a decisive impact on phenotypic development. For example, alternative mating strategies in male orangutans—growing large and dominant and courting females versus staying small and inconspicuous and sneaking—are built on a monomorphic genetic platform; i.e., the developmental switch is determined by the density of mature adult males and does not depend on allelic variations (see Tainaka, Yoshimura, & Rosenzweig, 2007). Nonetheless, the concept of pure environment-specific regulation can be misleading. Even if phenotypic alternatives are primarily due to environmental effects, this does not mean that a population is genetically uniform in its propensity to adopt one phenotype over another (Ellis et al., 2006).

*Humans.* Although we do not have detailed information on gene-environment interactions in human sexual development, there is compelling evidence that genetic effects on puberty are conditioned by environmental context. Specifically, heritability estimates of pubertal and sexual development are context-specific and can change dramatically when social or physical environments change (e.g., Dunne et al., 1997; Ellis, 2004). Comparison of correlations across multiple levels of kinship pairs—cousins, half-sibs, full sibs, mother-daughter pairs, identical twins—is a common method for estimating genetic influences on menarcheal age and typically yields heritabilities in the range of 45 to 55 (Chern, Gatewood, & Anderson, 1980; Doughty & Rodgers, 2000; Rowe, 2000). These heritability estimates, however, are scaffolded by environmental continuity between members of kinship pairs. Consider Chasiotis, Scheffer, Restmeier, & Keller’s (1998) investigation of mother-daughter correlations in age of menarche in comparable urban areas in East and West Germany. This study spanned the time period of reunification (which resulted in much greater social disruption and sociopolitical change for East Germans than for West Germans). In the East German sample, there was no significant
correlation between mothers and daughters in either resource availability (e.g., SES) in childhood ($r = -.04$) or age at menarche ($r = -.07$). By contrast, in the West German sample, there were substantial correlations between mothers and daughters in both resource availability in childhood ($r = .51$) and age at menarche ($r = .60$). Consistent with these data, low mother-daughter correlations for age at menarche ($r_s \leq .20$) were also recorded in a Czech Republic study in which mothers and daughters differed in having grown up in rural versus urban environments (Hajn & Komenda, 1985). These findings suggest that strong genetic effects on pubertal timing, as indicated by some heritability studies (e.g., Kaprio et al., 1995), depend on stability of environmental conditions. In total, genetic and environmental effects on regulation moderate each other.

**Alternative Phenotypes**

As noted above, timing of puberty is one component of a correlated suite of reproductive characteristics. In the life history literature, covariation between

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timing of puberty and related morphological, neuroendocrine, and behavioral characteristics is conceptualized as alternative reproductive strategies (i.e., alternative phenotypes that evolved together as coadapted, functional sets; see Figueredo et al., 2006; Tooby & Cosmides, 1990). The coherence of these strategies is underpinned by switch-controlled modular systems. In terms of Figure 7.1, I will refer to the early pubertal developmental pathway (fast reproductive strategy) as Phenotype A and the late pubertal developmental pathway (slow reproductive strategy) as Phenotype B. Please note that Figure 7.1 is an over-simplified representation of the causes of variation in reproductive strategies, as many factors besides age at gonadarche contribute to this variation. Nevertheless, timing of puberty is an important factor in development of individual differences in human reproductive strategies (reviewed in Ellis, 2004).

As West-Eberhard has stated, The regulatory mechanism coordinates the expression and use of *specific modifier* gene products and environmental elements (a, b) that compose phenotype A or B but are not used in both (p. 67).

Thus, depending on the timing of the developmental switch (gonadarche) and subsequent activity of the GnRH secretory network, certain patterns of gene expression differ in Phenotype A and Phenotype B. Figure 7.1 presents examples of these differences in gene expression, based on a rodent model of reproductive development (Cameron, Del Corpo, Diorio, McAllister, Sharma, & Meaney, 2008). At the same time there are non-specific modifiers (c): species-typical gene products and environmental elements involved in maturation and functioning of the HPG axis that are shared by (expressed in) both Phenotype A and Phenotype B (see Figure 7.1). These universal elements are not differentially affected by the regulatory switch. In sum, Phenotypes A and B, as well as the overlapping components of these phenotypes (C), are subunits of gene expression or gene-product use; i.e., the coordinated expression of these phenotypes—their linkage as coexpressed traits—are subunits of gene action. Use of specific modifier gene products and raw materials imported from the environment are coordinated by the operation of regulatory switch mechanisms, which are co-determined by impinging environmental and genomic information.

Although the West-Eberhard (2003) model diagrammatically represents the architecture of discrete, switch-controlled alternative phenotypes, discrete traits (i.e., qualitative or discontinuous variation) are relatively rare in nature; instead, phenotypic variation is almost always continuously distributed (Reznick & Travis, 1996), as is timing of pubertal development.
Gonadarche is a discrete developmental switch point, but the value of T and the organism’s ability to pass T varies across individuals as a function of allelic variations and environmental influences. Allelic influences on gonadarche, as in virtually all complex traits, are polygenic and thus bias the population toward a continuously variable distribution of phenotypes (West-Eberhard, 2003). Likewise, multiple environmental influences on regulatory mechanisms produce continuous phenotypic variation. Taken together, these multiple genetic and environmental influences produce the normal distribution in the regulatory mechanism box depicted in Figure 7.1 (see the normal curve in age at gonadarche). Finally, although some switches explicitly control expression of discrete phenotypes (e.g., sex determination), in the case of normally distributed phenotypic traits that are subject to multiple genetic and environment influences, switches occur between successive causal events in a developmental pathway. The result is that human reproductive strategies exist on a continuum. In total, although Phenotype A and Phenotype B are depicted in Figure 7.1 as discrete types, they should be regarded as prototypes that characterize contrasting ends of a continuous distribution.

**The Integrative Rodent Model of Adaptive Phenotypic Plasticity in Reproductive Strategies**

The role of gene expression in supporting development of alternative reproductive strategies has been elegantly demonstrated in the programmatic work of Michael Meaney and colleagues. Building on evolutionary models of the role of developmental experience in the regulation of reproductive strategies (Belsky, Steinberg, & Draper, 1991; see also Chisholm, 1999; Ellis, 2004), this work examines how early physical and social environments in rodents produce alterations in parent–offspring interactions, which in turn calibrate the development of stress- and steroid-responsive neural circuits and associated patterns of behavior (reviewed in Ellis et al., 2006). The Meaney laboratory has systematically characterized a cascade of behavioral, physiological, and epigenetic events through which phenotypic variation in offspring behavior is guided by ecological parameters within physical and social rearing environments (Cameron et al., 2008; Meaney, 2001; Meaney & Szyf, 2005; Weaver et al., 2004) (see Figure 7.2). Such ecological stressors as the presence of predators, infectious disease, and maternal-infant separations interact with heritable differences in maternal reproductive and stress physiology to produce robust individual differences in the character and frequency of maternal-infant interactions. Such interactions, Meaney and colleagues have shown, are capable of calibrating the differential expression of genes in the infant’s central neural circuitry (i.e., differences in how DNA sequences
are translated into cellular structure and function). These epigenetic differences underpin variation in defensive and reproductive strategies.

Within Meaney’s model of individual differences in rodents, two patterns of phenotypic variation—representing opposing poles along a spectrum of neurobehavioral development—are rough approximations of fast and
slow reproductive strategies (Phenotypes A & B). As depicted in Figure 7.2, ecological stress and instability cause low-quality parental investment, which provides input (e) to regulatory mechanisms in offspring that bias development toward high physiological and behavioral reactivity to stressors and a faster reproductive strategy (Phenotype A). This contingent strategy may function to promote vigilance for environmental dangers and early opportunities for mating and reproduction in dangerous or unpredictable environments. Conversely, more safe and stable ecological conditions support higher quality parental investment that fosters the opposite pattern of development in offspring (Phenotype B). This structured matching of phenotypes to environmental conditions—adaptive phenotypic plasticity—has human analogs in the observations of exaggerated stress reactivity (e.g., Felitti et al., 1998; Heim & Nemeroff, 1999) and precocious pubertal development and sexual activity (e.g., Ellis, 2004; Ellis et al., 2003) often found among children growing up in socially and economically adverse family and neighborhood contexts.

At the level of maternal reproductive and stress physiology, variation in social and ecological contexts, along with individual differences in maternal physiology, biases mother-infant interactions toward one of two distinctive patterns of infant care, each with clearly definable behavioral markers. Under low stress conditions, mothers display a pattern of frequent licking and grooming (LG) of pups, along with a typical arched back nursing (ABN) posture. By contrast, under conditions of stress and adversity, mothers show much less frequent licking, grooming, or arched back nursing of pups. In addition to such contextual influences (e) on these care-giving behaviors, both patterns are also heritable (r) and naturally occurring, and each pattern is typical of different strains of rodent. Female BALBc mice, for example, who display a predisposition to fearfulness and exaggerated hypothalamic-pituitary-adrenal (HPA) reactivity to stressors, characteristically evince low levels of LG-ABN (Anisman, Zaharia, Meaney, & Merali, 1998). On the other hand, C57 mothers, who show little behavioral evidence of fear and relatively low HPA reactivity, are those for whom high LG-ABN behaviors are typical. Heritable differences in maternal behavior appear to be highly stable from litter to litter (Champagne, Francis, Mar, & Meaney, 2003).

Structured by a combination of genetic diversity (r) and ecological conditions (e), variation in maternal LG-ABN behavior functions to program experience-sensitive neural circuitry in the young, resulting in striking and enduring differences in offspring stress reactivity, defensive behaviors, and reproductive physiology and behavior (Cameron et al., 2005, 2008) (see Figure 7.2). Pups born into stable, supportive environments with only short-term stressors become predisposed, through high levels of maternal LG-ABN (e), to patterns of low stress reactivity and low levels of fearfulness, as evidenced by relatively diminished adrenocortical reactivity and startle responses and greater resistance to stress-induced illness. These physiologic differences are accompanied by behavioral differences, such as increased open field exploration and decreased latency to eat in novel environments. This profile biases the developing pups toward a slower reproductive strategy (Phenotype B). Specifically, female pups that receive high levels of LG-ABN from their mothers experience later onset of puberty, tend to display agonistic behavior in response to mounting solicitations by novel males, enforce much longer intervals between matings, have lower rates of pregnancy following mating sessions (50%), and provide higher quality parental investment in their own offspring (high LG-ABN).
By contrast, pups born into conditions with longer-term and unpredictable stressors become biased, through low maternal LG-ABN, toward fearfulness and heightened biological reactivity to stressors (in both the adrenocortical and sympathetic adrenomedullary systems), as well as enhanced vulnerability to stress-related forms of morbidity. Pups in high stress, low LG-ABN environments also demonstrate pervasively higher rates of fear-induced behavior, increased burying behavior in response to threats, stronger startle reflexes, and decreased open-field exploration. The biasing toward a faster reproductive strategy (Phenotype A) is most strikingly demonstrated by the reproductive development and behavior of the female offspring of low LG-ABN mothers. These pups experience earlier onset of puberty, are substantially more sexually proceptive toward novel males, exhibit increased lordosis in response to male mounts, have sharply higher rates of pregnancy following mating sessions (over 80%), and provide lower quality parental investment in their own offspring (low LG-ABN) (Cameron et al., 2005, 2008). These individual differences in the first generation of offspring are transmitted into the second—a nongenomic intergenerational transmission—through perpetuated differences in maternal-infant behavior.

How does variation in mother-infant interactions translate into adaptive variability in offspring behavior? Recent research offers clear evidence for epigenetic regulation of stress- and steroid-responsive genes by maternal behavior in rodents. Epigenetic regulation refers to the modulation of gene expression through changes in the epigenome, that is, changes in the chromatin structure and methylation of DNA (which constitute the proteins and other molecules that are attached to DNA and determine whether or not genes can produce transcripts). Epigenetic changes thus mark genes for higher or lower activity. Mother-infant interactions (LG-ABN) throw developmental switches that permanently calibrate HPA responses to stress through tissue-specific effects on gene expression (a, b) (i.e., hippocampal glucocorticoid receptor messenger RNA expression, glucocorticoid feedback sensitivity, levels of hypothalamic corticotropin-releasing hormone messenger RNA; Liu et al., 1997; Meaney & Szyf, 2005; Weaver et al., 2004). These effects on the epigenome emerge within the first week of life, can be reversed with cross-fostering to mothers with the opposite pattern of LG-ABN behavior, occur only during this early sensitive period, and persist into adult life. Importantly, these epigenetic effects—alterations in DNA methylation and histone acetylation—appear to mediate the impact of quality of maternal care (LG-ABN) on physiological and behavioral responses to stress in offspring.

This epigenetic work has recently been extended to explain the effects of maternal behavior on development of alternative reproductive strategies (Cameron et al., 2008; Champagne et al., 2003, 2006). Both the anteroventral periventricular nucleus (AVPVn) and the medial preoptic area (MPOA) of the hypothalamus are rich in estrogen receptors (ER). Whereas levels of ER$\alpha$ expression in the AVPVn have important effects on activity of the HPG axis (stimulating GnRH gene expression), ER$\alpha$ expression in the MPOA substantially influences maternal behavior (via estrogen-induced oxytocin receptor binding). ER$\alpha$ expression in the AVPVn is increased in the adult female offspring of low versus high LG-ABN mothers (Figure 7.1: A-specific modifiers). This suggests that variation in maternal behavior influences the sensitivity (reactivity) of offspring to the effects of estradiol on pathways regulating GnRH and associated pulsatility of gonadotropins. This enhanced sensitivity increases GnRH biosynthesis and secretion, accelerating reproductive maturation and associated sexual behaviors (Phenotype A). By contrast, ER$\alpha$ expression in the MPOA and associated oxytocin receptor levels are increased in the adult
female offspring of high versus low LG-ABN mothers (Figure 7.1: B-specific modifiers). As a result of these changes in gene expression, the daughters of high LG-ABN mothers show increased maternal licking and grooming during lactation (Phenotype B). Most compelling, cross-fostering studies have confirmed that these maternal effects on the epigenome and related behavioral and physiological traits are transmitted across generations through non-genomic mechanisms. As shown in Figure 7.2,

“Stable variations in reproductive life histories of female mammals can, in part, derive from parental influences during both fetal and postnatal development resulting in tissue-specific, epigenetic programming of ERα expression in brain regions that regulate maternal behavior (MPOA) and HPG function (AVPVn)” (Cameron et al., 2008, p. 8).

The summarized work thus articulates a stepwise series of mechanisms—at varying levels of complexity and abstraction—by which ecological conditions  

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can produce systematic differences in a rodent pup’s experiences of maternal behavior, which throw regulatory switches that, in a developmental cascade, affect transcription of the pup’s stress- and steroid-responsive genetic material, the reactivity of its neural and neuroendocrine circuits, its timing of gonadarche, and its individual profile of defensive and reproductive behavior. In a seamless sequence of biological and behavioral processes predicted by evolutionary models (Belsky et al., 1991; Chisholm, 1999; Ellis, 2004), the developing pup’s survival and reproductive strategies are calibrated to maternal resources and the frequency and duration of threats in the environment into which it was born.

Although maternal LG-ABN has a major effect on the switch-controlled modular systems that regulate variation in defensive and reproductive behaviors, these environmental effects interact with genetic factors and extant phenotypic characteristics. Boyce and Ellis (2005; see also Belsky, 2005) have proposed that heightened stress reactivity reflects a form of enhanced, neurobiologically mediated sensitivity to context (i.e., openness or susceptibility to rearing experiences). Consistent with this theorizing, Cameron et al. (2008) found that changes in maternal behavior (cross-fostering of pups to mothers with the opposing maternal style) only caused changes in sexual behavior among rats from birth lineages characterized by low maternal LG-ABN/heightened biological reactivity to stress (an example of genes moderating an environmental effect, as depicted by the crossing arrows in Figure 7.1). This same effect had previously been observed for hippocampal synaptic development and hippocampal-dependent learning (i.e., spatial learning and memory) (Liu et al., 2000). In total, across both sexual and cognitive domains, lineages characterized by high biological reactivity to stress were the most susceptible to rearing experiences.

Both the hippocampus and the GnRH secretory system are particularly susceptible to the effects of glucocorticoids (principally cortisol) and other neuroendocrine responses to stress (Bremner & Vermetten, 2001; Dobson et al., 2003; Johnson et al., 1992; Sapolsky, 1996). Variation in cognitive and sexual development, therefore, are each affected by individual differences in activity and reactivity of the stress-response systems. Importantly, by the time a rodent or primate is born, its stress responses have already been calibrated by prenatal experiences (Oberlander et al., 2008; Van Den Hove et al., 2006; Weinstock, 1997) and genetic factors (reviewed in Boyce & Ellis, 2005). This extant phenotypic variation provides an important context for development and functioning of the switch-controlled modular systems (such as those that regulate cognitive or sexual development), including responsivity of regulatory switch mechanisms to postnatal
experiences (e.g., maternal LG-ABN) and genetic inputs. Ultimately, environmental conditions, genetic factors, and extant phenotypic characteristics co-determine the development and functioning of switch-controlled modular systems, and this co-determination occurs through integrative effects on regulatory switches.

**Putting the Pieces Together**

There is an essential human nature that underlies our unity as a species. The vast majority of the human genome—99.9% of chemical nucleotide bases—

1 The estimate of human genetic identity is based on the reference sequence constructed by the Human Genome Project, which is informative about the number of bases that are invariant across individuals. This identity is somewhat attenuated by differences across individuals in repetitions of DNA segments (i.e., copy-number variations).

—is exactly the same in all humans (Human Genome Project, 2001). This shared genetic structure, in interaction with species-typical environments, ensures reliable development of universal features of the human anatomy, physiology, and psychology (C). Central to this universal design are functional, modular systems. For example, the HGP axis governs production and release of gonadal steroid hormones, which regulate species-typical patterns of sexual development and behavior; the corticotrophin releasing hormone system and locus coeruleus-norepinephrine system modulate biological responses to stress, which regulate species-typical defensive behaviors; and the hypothalamic-neurohypophysial system regulates arginine vasopressin and oxytocin, which together play key roles in pair bonding and parental behavior.

From the perspective of the West-Eberhard model, these universal systems can be described at two levels. First, they comprise shared components (phenotypic traits, C) that are reliably constructed through species-typical patterns of gene expression and raw materials imported from the environment (non-specific modifiers, c). Second, they are hierarchically-organized and functionally integrated by regulatory switch mechanisms (R), which make use of and depend on species-typical gene products (genetically monomorphic modifiers of regulation, r\textsubscript{universal}) and systematic properties and conditions of the environments in which development regularly and predictably occurs (e\textsubscript{universal}).

Individual differences are variations on the theme. Although only a miniscule proportion (0.1%) of the approximately 3.2 billion nucleotide bases varies between individuals, this level of genetic variation still translates into approximately 9.2 million candidate single nucleotide polymorphisms (SNPs), of which 2.4 to 3.4 million have been validated using multiple techniques (International HapMap Consortium, 2005). SNPs, together with microsatellite mutations, are the most common and widely distributed classes of mutations that produce variation across DNA sequences, that is, allelic variations. These two types of small-scale mutations are thought to make up the bulk of human genetic diversity and potentially impact phenotypic trait variation. The products of these mutations (r), therefore, potentially impact switch-controlled modular systems. As discussed above (see The Role of Genetic Factors), such effects may occur at any switch-point in the system. Likewise, although there are many regularities in our species-typical environments, there is also adaptively significant variation (e.g., varying levels of nutritional resources, predation threats, quality of parental investment, social competition). This environmental variation (e) can also differentially impact switch-controlled modular systems across multiple levels of organization (see above, The...
Role of Environmental Factors). These genetic and environmental impacts are modulated by extant phenotypic characteristics and instantiated through their effects on regulatory switch mechanisms (R), which coordinate subsequent changes in expression and use of specific modifier gene products and environmental elements (a, b) that build alternative phenotypes (A,B) (West-Eberhard, 2003).

**How does natural selection organize the switch-controlled modular systems that underlie alternative reproductive strategies?**

There are broad and enduring individual differences in human reproductive strategies, both within and between populations, that can be arrayed on a slow-to-fast continuum of sexual development, mating, and parenting (Belsky et al., 1991; Chisholm, 1999; Ellis, Figueredo, Brumbach, & Schlomer, 2009a; Figueredo et al. 2006; Walker et al., 2006). Examples of slow versus fast life history traits are presented in Figure 7.1. For ease of presentation, slow and fast traits are grouped together in separate boxes. This grouping does not imply, however, that variation in all life history traits can be arrayed on a single slow-fast dimension.

What are the evolutionary origins of pervasive individual differences in reproductive strategies? One possibility is that this variation is simply random and non-adaptive (i.e., evolutionary noise), much as differences between people in the length of their toes is random and non-adaptive, owing to selection-irrelevant genetic variation, the random effects of sexual recombination, and nonadaptive phenotypic plasticity in response to experience. Such variation could still be heritable and somewhat predictable in response to environmental factors, but it would not be the product of natural selection and would have had little bearing on fitness in ancestral environments. Along these lines, theory and data from evolutionary quantitative genetics suggest that most genetic variation is due to non-adaptive or neutral forces such as mutation-selection balance (Hughes & Burleson, 2000). Another possibility is that alternative phenotypes are adaptively patterned (within species-typical developmental environments). If this were the case, then Phenotypes A & B should produce mean differences in survival and reproductive outcomes when all individuals are constrained to a single environment, but these differences should diminish when different phenotypes are allowed to covary with salient features of the environment, that is, when different individuals can employ strategies and inhabit niches that are matched to their distinctive phenotypes (see Mealey, 2001).

Natural environments are often complex and afford more than one way to survive and reproduce. Such multi-niche environments provide the ecological basis for the evolution of adaptive phenotypic variation within species. This adaptive variation can evolve in response to the physical diversity of environments (i.e., ecological niches that vary over time or space) or as alternative solutions to problems of social competition. In complex, multniiche environments, where selection is unlikely to converge on a single “best” phenotype, and where the fitness of alternative phenotypes is predictable on the basis of observable environmental cues, selection tends to favor adaptive phenotypic plasticity (reviewed in Ellis et al., 2006). Indeed, phenotypic plasticity is very common in nature, can be irreversible (i.e., fixed) or reversible (i.e., labile) during the lifetime of an organism, enables individuals to function as generalists or become specialized to a particular niche, enables adaptive coordination with environmental conditions, and can persist over the long-term without equal fitness payoffs (West-Eberhard, 2003).

Life history theory provides a solid basis for understanding environmental influences (e) on development of alternative reproductive strategies. As reviewed by Ellis et al. (2009a), harshness
(the rates at which external factors cause disability and death in a population) and unpredictability (the rates at which environmental harshness varies over time and space) are the most fundamental environmental influences on the evolution and development of life history strategies; moreover, these influences depend on population densities and related levels of intraspecific competition and resource scarcity, on age schedules of mortality, on the sensitivity of morbidity and mortality to the organism’s resource-allocation decisions, and on the extent to which environmental fluctuations affect individuals versus populations over short versus long timescales. Much research on contextual influences on variation in human life history strategies has supported core assumptions of this theory (Ellis et al., 2009a). These reliable contextual effects suggest that humans evolved in complex, multiniche environments that supported a range of life history strategies, and that the fitness of alternative strategies was predictable on the basis of environmental contextual cues (e) to harshness and unpredictability.

Relevant regulatory switch mechanisms, therefore, were shaped by natural selection to detect and respond to these cues. At the same time, however, there are potentially high costs of phenotypic plasticity (e.g., producing and maintaining the appropriate regulatory and assessment mechanisms for alternative development; see DeWitt, Sih, & Wilson, 1998); thus, in some cases genetically based polymorphisms will be selected for instead. Such polymorphisms are likely to be favored by selection when advantages of niche specialization are high (Wilson, 1994), when organisms can evaluate and select their niches (Wilson, 1994), and when reliable environmental cues for entraining or switching between alternative phenotypes do not exist (West-Eberhard, 2003). For genetic polymorphisms to be maintained by natural selection, they must evolve toward a state of equilibrium in which the average fitness of the alternative alleles is equal.

In terms of human life history traits, extant quantitative- and molecular-genetic research has documented substantial genetic influences (r) on age at menarche, age at first birth, interbirth interval, fecundity, age at last reproduction, and adult longevity (Kirk et al., 2001; Pettay et al., 2005; Rodgers et al., 2001b; for reviews, see Rodgers et al., 2001a; Ellis, 2004). Importantly, the patterning of genetic influences on these life history traits is not random. Both historical Finnish data (Pettay et al., 2005) and modern US data (Rowe, 2002) indicate significant genetic correlations among female life history traits. Likewise, behavioral genetic analyses have provided evidence of genetic covariation in cognitive and behavioral indicators of human life history strategy (Figueredo et al., 2004). This substantial degree of genetic correlation among different life history traits suggests that this genetic variation does not merely reflect residual “noise” left over by incomplete stabilizing selection but may itself be adaptively coordinated.

Despite conditions favoring either adaptive genetic variation or phenotypic plasticity, virtually all carefully studied phenotypic variants have been found to be both condition-sensitive and influenced by genetic variation (West-Eberhard, 2003). Wilson’s (1994; Wilson & Yoshimura, 1994) evolutionary model of the coexistence of adaptive genetic variation and adaptive phenotypic plasticity in multiniche environments may shed light on this dual regulation. All else being equal, the presence of multiple niches in a single environment will favor developmental specialists (i.e., narrow genetic reaction norms in which phenotypic development is minimally condition-dependent) over developmental generalists (i.e., broad genetic reaction norms in which phenotypic development is highly condition-dependent) when individuals can evaluate and select niches that increase their fitness. This is because specialists outperform generalists in their
preferred niche. However, multiniche environments are often characterized by negative density-dependence, meaning that as a given niche becomes more crowded (i.e., over-exploited relative to its size), the fitness benefits of specializing in that niche decrease. This is the cost of specialization. Indeed, as a given niche becomes over-crowded, more plastic individuals who can either developmentally entrain alternative strategies to exploit less saturated niches or facultatively change strategies to exploit different niches over time gain a selective advantage. Given fluctuations in the size of niches over time and space, and corresponding fluctuations in the density of competitors in those niches, natural selection should favor a mix of developmental specialists (adaptive genetic variation) and developmental generalists (adaptive phenotypic plasticity) rather than a single genetic or environmental mode of regulation. In such fluctuating environments, specialists experience feast and famine while generalists experience intermediate outcomes (adjusting phenotypic development to exploit less crowded niches, but never doing as well in those niches as the specialists do). The generalists do not replace the specialists, therefore, but instead co-exist in stable equilibrium (Wilson, 1994; Wilson & Yoshimura, 1994).

**An Agenda for Future Research**

Development of alternative reproductive strategies in humans provides a compelling illustration of gene–environment co-regulation. Nonetheless, extant human research examining specific environmental or genetic influences on variation in reproductive strategies has been limited. Consistent with life history models, descriptive longitudinal studies have established that stressful conditions in early childhood forecast earlier pubertal development and onset of sexual activity in adolescence (e.g., Ellis et al., 1999, 2003; Moffitt et al., 1992; Belsky et al., 2007); however, the causal status of measured environmental variables remains indeterminate due to possible third-variable confounds (i.e., gene–environment correlations). Implementation of causally informative research designs in this area is still in its formative stages (but see initial studies by D’Onofrio et al., 2006; Ellis, Schlomer, Tilley, & Butler, 2009b; Mendle et al., 2006, 2009; Tither & Ellis, 2008). More work is needed to identify the levels and types of environmental exposures that have the most causal influence on variation in human reproductive strategies. Life history theory and data underscore the importance of distinguishing between harsh versus unpredictable environmental conditions (see Ellis et al., 2009a).

Candidate gene and genome-wide association studies have begun to illuminate genomic influences on variation in the HPG axis and associated reproductive strategies. For example, as discussed above, variation in the LIN28B gene is reliably associated with normal variation in age at menarche; however, it accounts for less than 2% of this variation. Variation in the DRD4 gene has also been associated with various behavioral and cognitive indicators of alternative reproductive strategies. This has led some authors to propose that the DRD4 polymorphism has been maintained through balancing selection in multi-niche environments (Harpending & Cochran, 2002). However, the main effects of variations in the DRD4 gene are also small (and unreliable) (e.g., Kluger, Siegfried, & Ebstein, 2002). The inherent problem in all approaches focusing on the effects of single-gene variants is that the regulatory mechanisms controlling development of complex alternative phenotypes are influenced by multiple allelic variations. This highlights the need for molecular genetic studies to adopt a polygenic approach. The multivariate regression techniques...
employed by Comings et al. (2000a, 2000b), which examine the relative influence of individual genes within a larger group of genes on externalizing disorders, provide an example of such an approach in humans.

Most critical is the need to study gene–gene, environment–environment, and gene–environment interactions. Genuine progress in understanding the development of alternative reproductive strategies will depend on our ability to map these interactions. While such work is still in its infancy, extant research has generally focused on the moderating effects of allelic variations on relations between environmental factors and child-developmental outcomes. For example, although the main effects of variants in the *DRD4* gene remain dubious, the presence versus absence of the *DRD4* 7-repeat allele has been found to reliably moderate the effects of parental functioning on children’s attachment styles (Bakermans-Kranenburg & van IJzendoorn, 2007; Bakermans-Kranenburg, van IJzendoorn, Mesman, Alink, & Juffer, 2008)—a known precursor of variation in life history strategies. Specifically, children carrying the 7-repeat allele were more susceptible to parental rearing influences in both studies. Such results surely represent the tip of the iceberg of potential research on gene–environment co-regulation in development of alternative reproductive strategies. I expect that future research will confirm the centrality of gene–environment co-regulation, as well as gene–gene and environment–environment interactions, and move the field beyond its current focus on main effects.

A further important step is to incorporate the existing phenotype, which is the transducer of all genetic and environmental effects and their interactions. Genetic differences in susceptibility to rearing experiences, for example, must operate through variation in neuro-physiological (endophenotypic) and behavioral (phenotypic) processes. Along these lines, recent studies have found that the effects of early parent–child relationships on the timing of pubertal development are moderated by both biological reactivity to stress (Ellis, Shirtcliff, Boyce, Deardorff, & Essex, in press) and negative infant emotional reactivity (Belsky et al., 2007). Paralleling the rodent model reviewed earlier, these findings support the importance of differential susceptibility to rearing experience in human reproductive development (see Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, in press) and suggest potential mediating pathways through which susceptibility genes might operate. In total, a full understanding of development of alternative reproductive strategies requires analysis of gene × environment × phenotype interactions. Genetic and environmental factors not only influence phenotypic development but are also mediated and moderated by the extant phenotype.

Finally, research needs to get underneath the skin. Behavioral scientists could benefit from interdisciplinary collaborations with molecular biologists, geneticists, and endocrinologists to directly study the effects of rearing experiences and genetic factors on regulatory mechanisms, associated patterns of gene expression, and related neuro-endocrine processes. Research on the mediating effects of adrenal and gonadal hormones on relations between early life experiences and sexual and reproductive outcomes constitutes a preliminary step in this direction (see Alvergne, Faurie, & Raymond, 2008; Ellis & Essex, 2007). Ultimately, an adequate description of mechanistic pathways—including determinants of both regulation and form (Figure 7.1)—is necessary to truly understand how alternative reproductive strategies develop (and to effectively intervene to alter these strategies).
In conclusion, West-Eberhard’s (2003) model of switch-controlled modular systems provides the foundation for a comprehensive analysis of adaptive variation in reproductive strategies. It fully integrates genetic and environmental regulation, distinguishes between determinants of regulation and determinants of form, and applies to both species-typical development and functional variation. The model provides an evolutionary-developmental framework for understanding variation in human reproductive strategies—a framework that affords a clear agenda for future research.

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8 Ecological Approaches to Personality
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Many consider the maintenance of heritable behavioral variability among individuals across evolutionary time a dilemma for evolutionary psychology. Why have natural and sexual selection not eliminated individual differences in favor of some optimally adaptive set of universal trait values? The statistical associations among personality traits and a wide array of life outcomes clearly relevant to survival and reproduction make it unlikely that such individual differences in traits could be selectively neutral (Buss & Greiling, 1999; Figueredo, Sefcek, Vásquez, et al., 2005; Nettle, 2006).

We argue that a principled application of quantitative theoretical ecology can best explain interindividual behavioral diversity and that the diversification of behavioral phenotypes matches the diversification of socioecological niches within species-typical multidimensional niche space, particularly for social species. We show that this pattern manifests phylogenetically and ontologically in the evolutionary history of species and in the behavioral development of individuals. Furthermore, we demonstrate that selective pressures maintain, and in some cases increase, individual trait differences during both developmental and evolutionary time. Finally, we outline the multiple selective pressures hypothesized to bear on the evolution of individual behavioral differences.

Although we present several hypotheses regarding the centrality of some of these selective pressures, we do not champion any one. As in other branches of science, many evolved adaptations are the product of, not one, but multiple selective forces. For example, in Newtonian Physics, the “resultant” is the vector sum of two or more vectors representing multiple forces acting simultaneously on a single material particle. The trajectory of a projectile may be the vector sum of its own momentum (inertia), the downward forces of gravity, air resistance (friction), and wind velocity and directionality. The same principles hold in evolutionary biology: Multiple selective pressures act on any given trait simultaneously. Thus, there is seldom a one-to-one correspondence between a specific trait and a specific selective pressure.

Consider female mate choice. Both intrasexual and intersexual selective pressures clearly influence female mate choice. For example, females may prefer males with good genetic quality, epigamic selection, but may also prefer males that demonstrate intrasexual dominance as in lekking species, where females visit the most hotly contested central areas of the lek typically controlled by dominant males. In other species (e.g., territorial birds), females prefer males who control material resources, a characteristic partially the product of inter-male competition. Thus, distinguishing the products of intra-sexual competition from epigamic (intersexual) selection,
even in female mate choice, is a challenge because both may be responsible for the evolution of a particular trait.

*Exaptation* offers another example (Andrews et al., 2002). Here, a trait evolves in response to one set of selective pressures, but then responds to a new set of selective pressures which shape it toward other functions. In such cases, the trait may not lose its original function, but may do “double duty” over evolutionary time. For example, although bird feathers function in both thermoregulation and flight, the latter presumably evolved a bit later. Birds now use feathers for both functions, although some feathers appear more adapted to one function over the other. Except in the case of some secondarily flightless birds, the dominance of one selective pressure over the other is seldom complete.

We apply this principle to the multiple selective ecological pressures that may have shaped the evolution of individual differences in personality, without advocating the primacy of one or another, and develop a more holistic and integrated view of the entire process. More specifically, we review the theoretical arguments and empirical evidence that individual variation in personality and behavior is shaped by a combination of: (1) frequency-dependent niche-splitting, (2) developmental plasticity, (3) genetic diversification, (4) directional social selection, and (5) behavioral flexibility.

**Theoretical Evolutionary Ecology**

**Santa Rosalia Revisited**

Hutchinson (1957, 1959, 1978) defines an *ecological niche* as a hypervolume in multidimensional hyperspace, in which each dimension is one of the parameters describing the biotic (living) or abiotic (nonliving) factors in the species’ ecology. Biotic factors include the predators, prey, hosts, and parasites with which a given species interacts and also include cooperation and competition from conspecifics within the social environment. Abiotic factors include habitable ranges along spatial dimensions like latitude and altitude and along nonspatial physical parameters like temperature and humidity.

Whereas a *habitat* is the physical location an organism occupies, the collection of a species’ habitats constitutes its *geographic range*. Although one can describe both *habitat* and *geographic range* in the three standard spatial dimensions, these spatial dimensions are only one part of the definition of an *ecological niche*.

More finely-grained concepts reside within this general conceptualization. The limits of each dimension within which given species can survive define a species’ *fundamental niche*. In contrast, the portion of the fundamental niche that the population inhabits defines the species’ *realized niche*. A fundamental niche may be more extensive than a realized niche due to competition between species where the fundamental niches of two species overlap (Pianka, 1961). Most importantly, this concept extends to the circumscribed realized niches of individuals within a single species (Putman & Wratten, 1984), where within-species competition constrains an individual’s realized niche to distinct regions within the broader fundamental niche of the population. Thus, narrow niches occupied by individual specialists may compose the total niche breadth of a generalist species.

An application of these classical principles of quantitative theoretical ecology may explain inter-individual behavioral diversity. As in theoretical ecology, competition does not invariably lead to *competitive exclusion* or the extinction of one type and replacement by another. Instead, *niche-
splitting may occur, which is the partitioning of an ecological niche among competing types based upon the relative competitive advantages of the different types within various regions of the ecological hypervolume. Niche-splitting may, in turn, lead to character displacement, the physical and behavioral differentiation of the different types to specialize narrowly within their region of a previously shared hypervolume. These phenomena were originally used to describe the results of interspecific (between species) competition. Intraspecific niche-splitting, the fragmentation of the ecological hyperspace into more specialized niches within the species’ fundamental niche, however, may lead to intraspecific character displacement, the differentiation of individual traits to adapt to these diversified niches.

These strategies are not intentionally implemented in the interests of peaceful coexistence; instead they are consequences of depressed fitness experienced by individuals in the zone of greatest competition between competing types and the consequent relative fitness gains experienced by individuals at the outer extremes of the overlapping population distributions. In many birds, for example, mixed-species flocks gain fitness benefits through enhanced antipredator vigilance. Those that flock with members of their own species must perforce bear the cost of greater social competition (e.g., for food). In contrast, birds that flock with members of other species gain fitness benefits through enhanced antipredator vigilance in the context of a relative competitive release from conspecifics. Thus, mixed-species flocking with noncompeting allospecifics constitutes a form of optimal foraging under risk of predation.

For a given species in a given set of ecological conditions, an optimal response disposition (ORD) may exist from which deviations would not be selectively neutral. Selective pressure for a species-typical monomorphism at this optimum creates a centripetal force against substantial individual variation, as opposed to a selectively neutral adaptive plateau (a range over which the variants have equal fitness). This clustering of the entire population at the ORD produces intense social competition in the center of the distribution and relatively reduced social competition at the tails of the distribution. Reduced competition generates disruptive selection for individual differences as a centrifugal force, partially counteracting directional selective pressure toward the ORD. The competitive release experienced by individuals at the tails of the distribution compense for the cost of deviation from species-typical norm of response (Figueroed, Sefcek, Vásquez, et al., 2005).

A similar balance between competing forces occurs in the dynamics of optimal territory size in many birds. For example, unequal territory sizes in hummingbirds are a product of social competition, where the balance between the costs and benefits of territorial defense produce roughly equal numbers of flowers in adjacent hummingbird territories. At exactly this point, the benefits of expanding a territory become counterbalanced by the costs of defending it for any given individual. This outcome is the ideal free distribution. In the ecology of behavior, the progressive expansion of the tails of the distribution around the optimal central tendency create a kind of ideal free distribution of alternative behavioral phenotypes in the population (Figueroed et al., 2005). The ecological dominions of the competing individuals is not restricted to three-dimensional space, as in the case of the hummingbird territories, but are multidimensional partitions, or individual realized niches, within the larger hypervolume representing the fundamental niche of the species. The best metaphor for personality space is not a geological metaphor of a pre-existing plateau, created by physical plate tectonic forces, but instead a
biological metaphor of a coral reef formation, created by the outward pressure of the living coral organisms themselves.

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The Diversification of Niches
In the Ecological Psychology of Gibson (1977, 1979), affordances, the interactive possibilities of the organism with its environment, take a central role. By this view, an ecological niche is more than the absolute characteristics either of the organism or of the environment: instead, it is a set of affordances. The key to understanding interindividual variation is not encapsulated within the individuals, but instead is in their interactions with the contexts in which those individuals evolved and developed, a position consistent with Bronfenbrenner’s (1979) Ecological Systems Theory (Figueredo, Brumbach, Jones, et al., 2007).

This view predicts that diversification of behavioral phenotypes will match the degree of diversification of socioecological niches within the multidimensional niche space of the species. In species with marked individual differences (e.g., humans) niche diversification generates disruptive selective pressures (selection favoring individuals with divergent characteristics) promoting the diversification of behavioral traits at phylogenetic and ontogenetic levels.

Social factors are the subset of biotic factors lying on the dimension of the ecological niche generated by interactions with conspecifics. Just as biotic factors render the description of an ecological niche more complex than abiotic factors alone, social complexity generates additional individual niche-space multidimensionality. Social niches are therefore nested within the fundamental niche hypervolume of a social species: intraspecific competition constrains the resulting realized niches of individuals.

Because social interactions exacerbate intraspecific competition, Figueredo and colleagues (1995, 2005) predicted individual differences to be greater in social than solitary species, noting the majority of nonhuman animal species for which systematic variation in personality has been documented are social. Both Wilson (1994) and Figueredo (1995; Figueredo et al., 2005) argued that the diversification of individual traits to fit different social niches is partly due to social competition driving individuals into different realized social niches (frequency-dependent selection). Filling these diverse niches offers partial release from competitive conspecific pressure. The intraspecific character displacement produced by these selective pressures generates individual differences in the relative effectiveness of differing adaptive strategies within social groups (Buss, 1991, 1997; Buss & Greiling, 1999). Thus, different individuals become better suited to particular social niches than others, where greater behavioral specialization renders the various personality characteristics optimal under differing local conditions (MacDonald, 1995, 1998). The diversification of individual differences in a social species mimics the beneficial effects of mixed-species flocking in birds.

Most primates, for example, evolved sociality primarily for enhanced antipredator tactics (vigilance, alarm calling, and mobbing).

Starting with the expansion of Homo sapiens out of Africa and continuing into the modern industrial era, human behavior has increased the number of selective pressures on human niche space. Outward human migration alone increased the number of possible environments available for human habitation. This mass migration persisted throughout the late Pleistocene. Coinciding innovations in stone tools likely made this territorial expansion possible. Our species continued to
expand its range to a limit, primarily the size of the planet. Once our ancestors populated the whole of the Earth, they experienced most of the variations of abiotic and interspecific selective pressures that influence the niche space.

Despite limits on territorial expansion at the onset of the Holocene, human behavior continued to diversify the sources of selective pressures with an impact on the niche space. Alexander (1989, 1990) argued that humans became ecologically dominant. Ecological dominance allowed human populations to grow throughout the Holocene while changing the primary sources of selective pressures from abiotic and interspecific to intraspecific and hence social (Flinn, Geary, & Ward, 2005). A major source of ecological dominance was agriculture, which created conditions leading to an unprecedented increase in the size of the human niche space stemming primarily from an increasing efficiency of food production: fewer people and fewer acres could reliably sustain a given population size. Agricultural efficiency created conditions that produced an increasingly fragmented division of labor, greater population density, increased social complexity, and an increase in the intraspecific sources of selective pressures influencing the niche space.

The agricultural revolution eventually morphed into the industrial revolution. Innovations originating in the industrial revolution increased the efficiency of human food production and the diversity of human occupations. Modern division of labor is so fragmented that most individuals in the more developed countries do not produce food directly: they do not hunt, gather, or farm. Instead, they rely on numerous specialized occupations, many of which were invented during a single generation. This diversification in human occupations has accelerated the diversification and growth of human niche space.

According to population genetic theory, the variance of a selectively neutral trait decreases as a function of distance from the population’s origin (Wright, 1943; Malecot, 1969). Over the past half century, anthropological geneticists established Africa as our continent of origin. These same geneticists developed the isolation by distance model for selectively neutral traits. Recently, the supporting genetic evidence has been replicated using quantitative morphological data measuring cranial features. Cramon-Taubadel and Lycett (2008), who analyzed 57 male cranial variables in 28 geographically-distributed populations, determined that if one sets the starting point for human origins in three African locations, the isolation by distance model accounts for 24–34% of the variance, whereas using start points originating on other continents do not predict the distribution of the population’s variance estimates.

Using the same procedure, starting and way points, Wolf (2009) applied the isolation by distance model to data on the global distribution of the Big Five personality traits (Schmidt et al., 2007). This secondary analysis, which used 52 of the original 56 different globally-distributed personality samples for which the latitude and longitude of the sample could be determined, did not support the isolation by distance model, indicating that as populations of humans emigrated from Africa to more distant destinations the interindividual variance in the Big Five did not decrease with distance as this model predicts for selectively neutral traits. The variance in four of the Big Five did not decrease at all as a function of distance, whereas the variance for Openness to Experience increased with distance from Africa. Although this lack of support may be due to unmeasured migrations, imprecise measurement in the social sciences, problems with the sampling techniques, or other factors, these data are inconsistent with the contemporary distribution of personality traits being selectively neutral.
In summary, one needs to explore both macroevolutionary and phylogenetic levels to properly examine the diversification of multidimensional niche space produced by social living. In the special case of humans, the increasing complexity of human societies is relevant to the microevolutionary and historical levels. Furthermore, the behavioral development of individuals within social contexts brings an individual into contact with ever-widening spheres of social interaction within the hierarchy of socioecological systems (Bronfenbrenner, 1979; Figueredo, Brumbach, Jones, et al., 2007), which calls for even greater behavioral diversification at the ontogenetic level.

Because social niches are inherently unstable, other individuals do not constitute a fixed adaptive target for selection. Instead, individuals defining social niches are in ever-shifting dynamic equilibria created by populations of alternative phenotypes. The environment of evolutionary adaptation is therefore variable across generations. This is because, as Charlton Heston stated in *Soylent Green*, “it’s made of people!”

**Frequency-Dependent Selection**

Frequency-dependent selection occurs when the fitness of any given phenotype is either directly or inversely proportional to its prevalence in the population. If there is intraspecific competition for social niches and if that intraspecific character displacement produces specialized phenotypes exploiting particular niches, then frequency-dependent selection among competing individuals with any given phenotype occurs because, as the prevalence of any given phenotype increases, the competition for the social niche space to which that phenotype is adapted increases. Although a variety of mechanisms can generate alternative phenotypes (see below), we begin with genetic diversity.

An organism’s genome and environment affect personality development simultaneously. All human behavioral traits are at least partially heritable: Genetic influences are typically larger than shared environmental influences whereas non-shared environmental factors are consistently larger than either genetic or shared environmental effects (Turkheimer, 1998, 2000). The standard behavior-genetic approach assumes genetic and environmental influences are additive and ignores non-additive effects, such as gene-environment (G-E) correlations and gene-environment (GxE) interactions. G-E correlations occur when genetic predispositions influence, through active (causal) or passive (non-causal) mechanisms, an individual’s niche. At least three active niche-picking mechanisms generate G-E correlations: selection (active choice of specific environments), evocation (the organism elicits predictable reactions from its environment), and manipulation, (the organism uses specific tactics to alter its environment) (Buss, 1987). Passive mechanisms include genetic drift, which produce different allele frequencies between populations. GxE interactions occur when individuals react differently to the environment because of their genotype, or their genotype expression depends on the experienced environment. Frequency-dependent selection of alternative phenotypes requires G-E correlations. To occupy different social niches, genetically different individuals must locate the niche by one mechanism or another. Selection of the best-suited environments is the most obvious mechanism, but active evocation and manipulation permit an organism to modify its environment, thereby producing its own ecological niches (Day, Laland, & Odling-Smee, 2003; Laland, Odling-Smee, & Feldman, 1999; Laland & Brown, 2006).

The dimensions described by personality and life history theory define sociological theory’s multidimensional *social space* (e.g., Popielarz & McPherson, 1995). The theoretically ideal
attribute levels of a potential harvester, collector, or gatekeeper of the resource in question serve as the center of the niches within this social space. Market forces limit the “size” of, or acceptable parameters for inclusion within a given niche, because it is in the best interest of group members to protect the relative value of their resource by limiting it. As in theoretical ecology, competitive exclusion explains the growth and diminution of competitive niches within social space. Two processes ensure that individuals at the edge of the niche are lost more frequently and quickly than those toward the center: (1) competition among individuals occupying immediately adjacent niches; and (2) individuals on the edge of a niche tend to be as similar to those immediately outside the niche as to those immediately within it.

**Genetic Diversity and Developmental Plasticity**

Ecological contingencies that vary over evolutionary time select phenotypically plastic organisms that successfully adapt though learning over developmental time (Figueredo, Hammond, & McKiernan, 2006). Adaptive developmental plasticity cannot function without reliable and valid cues indicating optimal alternative phenotypes under localized conditions (West-Eberhard, 2003). In the face of environmental variability without reliable and valid cues, the adaptive solution is to produce genetically diverse individuals dispersed along the expected distribution of locally optimal trait values. Ecological cues, however, are typically neither completely unreliable and invalid nor completely reliable and valid. Instead, *ecological validity* coefficients ranging between zero and one characterize the reliability and validity of ecological cues (Figueredo, Hammond, & McKiernan, 2006). Under intermediate stochastic conditions, organisms evolve a combination of genetic diversity *and* developmental plasticity to collectively fill the available ecological niche space.

**Bet-Hedging Strategies of Genetic Diversification**

Genetic diversification in the biologically prepared behavioral predispositions of offspring may supplement, or at times even partially substitute for, developmental plasticity. Genetic diversification may serve as an adaptation to partially unpredictable environmental contingencies when the environmental cues that might otherwise selectively trigger developmentally plastic changes are relatively unreliable or invalid (Figueredo, Hammond, & McKiernan, 2006; West-Eberhard, 2003). Given the inherent instability of social niches, this occurs most frequently in social species. Recall that competition and cooperation with conspecifics creates a major portion of the hypervolume of the ecological niche. Given that genetic drift produces generations composed of random samples of previous generations, individual variability creates stochastic social environments due to the varying compositions of the population over time. The stochastic nature of the adaptive target supports selection for individual variability, further destabilizing social niches. Thus, social living produces selective pressures favoring bet-hedging genetic diversification as well as developmental plasticity.

Sexual recombination is a common means of genetic diversification in the service of bet hedging. Even so, the evolution of sexual reproduction remains one of the great mysteries of evolutionary biology (Hamilton, Axelrod, & Tenese, 1990). The costs of reproducing sexually are huge. In a sexually reproducing species, for example, offspring carry only half of the individual parent’s
genes. Given differential parental effort, the more investing sex bears a disproportional cost of sexual reproduction, producing selective pressure against sexual reproduction.

Sexual recombination of genetic material, however, increases resistance to environmental fluctuations, for example, in response to parasites (Hamilton, Axelrod, & Tenese, 1990). In facultatively sexual species, sexual reproduction appears under conditions of high population density. In the Cladoceran, *Moina macrocopa*, for example, a reduction of ingestion rate, a cue presumably reflecting food availability, triggers switching from asexually to sexually reproducing generations (D’Abramo, 1980). In the Rotiferan, *Bronchionus plicatilis*, changes in salinity and food level affect the number of sexual versus asexually reproducing members of a given population (Snell, 1986). Sex also helps remove deleterious mutations (Kondrashov, 1988; Smith, 1978). Following the Chernobyl Power Plant disaster, Tsytsugina and Polikarpov (2003) found that the severity of cytogenetic damage in the earthworm population correlated with the number of individuals switching to sexual reproduction. These three species switch between sexual and asexual reproduction, but such effects occur even in obligately sexual species.

**Dissassortative Mating as Bet-Hedging Genetic Diversification**

According to life history (LH) theory (Charnov, 1993; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Roff, 1992, 2002; Stearns, 1992), unstable, unpredictable environments, where the sources of morbidity and mortality are *uncontrollable* by genetically-influenced developmental processes (called *extrinsic* sources of mortality), select against slow LH strategies. These environmental conditions lead to highly variable population densities, which support this selective effect. In contrast, stable, predictable environments, where the sources of morbidity and mortality are *controllable* by genetically-influenced (and hence evolvable) developmental processes (called *intrinsic* sources of mortality), select against fast LH strategies. These environmental conditions lead to highly stable population densities, which support this selective effect.

Therefore, consistent with certain evolutionary biological theories regarding the origin and function of sexual reproduction (e.g., Maynard-Smith, 1978), conditions favoring faster LH strategies (unstable, unpredictable, and uncontrollable environments) put a selective premium on higher rates of genetic recombination and hence *exogamy*. In contrast, conditions favoring slower LH strategies (stable, predictable, and controllable environments) put a selective premium on lower rates of genetic recombination and hence *endogamy*, to preserve the integrity of locally well-adapted and perhaps co-adapted genomes. It follows that the assortative mating coefficients on heritable traits will be greater for slower LH strategists than for faster LH strategists.

To test these theoretical predictions, Figueredo and Wolf (2009) used data from a cross-cultural study on assortative pairing of both friends and lovers (Figueredo, 2007). Independently sampled pairs of opposite-sex romantic partners and pairs of same-sex friends rated both themselves and each other on LH strategy, mate value, sensational interests, and delinquency. These authors collected samples of pairs of opposite-sex romantic partners and pairs of same-sex friends in local bars, clubs, coffee houses, and other public places from three different cultures: Tucson, Arizona; Hermosillo, Sonora; and San José, Costa Rica.

Same-sex friends were included based on inclusive fitness theory (Hamilton, 1964) and genetic similarity theory (Rushton, 1989, 2009), which predict that one can accomplish genetic replication either directly by personal reproduction or indirectly by means of altruism directed towards genetically related or similar individuals. Slow LH strategists should therefore be higher on assortative pairing with social as well as sexual partners.
Figueroedo and Wolf (2009) correlated the mean LH scores among pairmates (friends or lovers) to the squared differences between the LH scores of pairmates. We statistically controlled these correlations for both length of relationship (aggregated over both pairmate reports) and mean age of pairmates (because longer relationships might partially be a proxy for older people), to adjust for the longer-term relationships generally favored by slow LH strategists. As predicted by theory, the adjusted pairmate means correlated significantly and negatively with the squared differences between pairmates on LH strategy, indicating that slow LH strategists practice systematically greater degrees of assortative pairing than fast LH strategists. Furthermore, the correlation was statistically equivalent across all three cultures, both friends and lovers, and the (non-significant but theoretically plausible) interactions of relationship type and culture. This indicates the difference between slow and fast LH strategist on assortative pairing is statistically identical both across cultures and across social and sexual relationships. The relationship remained invariant when the authors statistically controlled for the main effects and interactions (with relationship, culture, and relationship by culture) of the other three traits measured (mate value, sensational interests, and delinquency).

In summary, variable and heterogeneous environments pose unique sets of adaptive problems. Complex social environments are both variable and heterogeneous, suggesting that individual differences in human life history strategy are a product of both natural and social selection, the latter producing adaptations to the social environment (West-Eberhard, 1979). Consistent with this view, slow LH individuals assortatively mate more strongly than fast LH individuals. We interpret this as a bet-hedging adaptation favoring the genetic diversification of offspring in fast LH strategists and, hence, in unstable, unpredictable, and uncontrollable environments.

**Developmental Plasticity**

The term *developmental plasticity (sensu stricto)* describes permanent physiological change of an organism during development. This change may occur any time from conception to later in life and may be influenced either by external environmental factors or other internal physiological factors. Plasticity is monotonic, irreversible, and occurs through GxE interactions. Developmental plasticity differs from *behavioral flexibility* in that the latter entails temporary, reversible changes—the organism retains the ability to change throughout the lifespan (West-Eberhard, 2003).

Environmental influences moderate the function of genes by altering gene expression and hence the production of enzymes (West-Eberhard, 2003). GxE interactions involve modifications of a biochemical epigenetic code, by which histones (protein molecules that coil around strands of DNA) regulate gene expression. The biochemical environment within a cell, which includes metabolic products from other genes located either within or outside of the cell, chemically modifies histones in the chromatin, which either partially or fully activate or suppress the associated genes. For example, hormones transported from other cells may function to regulate gene expression.

Genes also set thresholds for developmental switches that influence organismic responses to environmental or genetic triggers (West-Eberhard, 2003). *Genetic accommodation* is the microevolutionary process by which natural selection sets genetically controlled thresholds for environmental influences. Waddington’s (1953) *genetic assimilation*, which exclusively denotes the *lowering* of the response threshold of a developmental switch, is a special case of genetic
accommodation. *Threshold selection* is the entire coevolutionary process setting the response parameters of the developmental switches, including both genetic and environmental influences.

The development of reproductive strategies in rats, influenced by their mother’s care, illustrates how GxE interactions can cause permanent developmental change. Pups of mothers that often lick and groom (LG) them exhibit greater sexual receptivity and higher quality of maternal care toward their own offspring (Cameron et al., 2008). Also, maternal LG predicts the later LG of their offspring (i.e., offspring of high LG mothers exhibit high LG behavior with their offspring). When low LG mothers raise pups of high LG mothers however, the pup’s level of LG as a mother decreases. Indeed, maternal LG influences expression of the gene ERα, which regulates maternal and sexual behavior. Thus, the ERα, which operates during the end of gestation, influences maternal LG behavior. Rhesus macaques exhibit a similar gene-by-environment interaction (Suomi, 2006); a short allele in the promoter region of the 5-HTT gene produces deficits in neurobehavioral functioning, control of aggression, and serotonin metabolism of monkeys reared as infants with same-aged peers but not in those reared with their biological mothers and their peers.

The timing of human pubertal development provides another example of developmental plasticity. Puberty is a physiological event that produces permanent biological change. The age of puberty is plastic, operating through a switch-controlled modular system. The neurotransmitter and neuromodulatory system, under the influence of internal (e.g., body fat composition) and external (e.g., stress) factors, control the GnRH secretory network, which controls puberty in human females. The timing of puberty responds to ecological conditions (e.g., resources, environment variability) and may have evolved to adjust physical development accordingly (Ellis, 2004). Specifically, prepubescent females with a history of father absence experience puberty earlier than females whose father was present, although it is not completely clear whether this is an environmental or a behavioral-genetic effect (Ellis, McFayden-Ketchum, Dodge, Pettit, & Bates, 1999).

The sensitivity of gene expression to environmental input is a general principle. Hubel and Wiesel (1970), for example, manipulated kittens’ visual exposure by suturing one eye closed during various points in development. This manipulation produced a sharp decline in the number of cells in the visual cortex driven by the sutured eye. Moreover, cells of the LGN were smaller and paler. The same manipulation had no effects on adult cats, suggesting there is a critical period during the development of the feline visual system. After this critical period, opening the eye for several years promotes limited recovery in the cortex and not at all in the LGN. Without proper environmental influence at a specific stage of development, the visual system does not develop properly.

A multitude of similar examples occur in the social insects (e.g., O’Donnell, 1998). Developmental switches, which the colony controls through differential feeding and housing, trigger developmentally plastic responses. In this way, the colony controls the number of individuals expressing a specific behavioral or physical morphology. This control creates an adaptive problem—how to produce an optimal number of castes and ratio of individuals who occupy those castes in the colony’s specific environment. Two classes of adaptive problems influence the evolution of adaptation solutions to the problem
of producing the optimal number of castes and the optimal ratio of individuals in each caste (Oster & Wilson, 1978). The first, ergonomic efficiency, is the colony’s ability to compete with conspecifics or allospecifics for a given geographic area’s resources. The second, risk avoidance, is the ability of the colony to deal with environmental variability in case specific resources become temporarily unavailable.

In general, environmental conditions promoting adaptations favoring ergonomic efficiency include a relatively stable environment or an aspect of the environment with reliable resources. Theoretically, the benefits of greater ergonomic efficiency outweigh the costs of retaining adaptations that avoid risk from historically absent adaptive problems. Conversely, environmental conditions promoting adaptations favoring risk avoidance include a relatively unstable environment. Although specialization is important when it comes to competition for specific resources in unpredictable environments, specialization may decrease the reproductive success of the colony. In essence, specialization becomes risky because the colony relies on specific resources that may or may not exist from one day to the next. For example, a highly specialized colony may reside in an environment that changes drastically from year to year. If the environment is not conducive to a colony’s highly specialized caste system, colonies or species that can survive and reproduce in the face of the variable adaptive problems inherent in that geographical location will outcompete it.

The Hymenoptera evolved two general strategies that solve the problem of environmental variability. The first is to create as many castes or morphs as there are tasks or adaptive problems in that geographic location. This strategy is called tychophobic, or “risk-fearing.” Each morph becomes highly specialized and increases the ergonomic efficiency of the colony in the presence of specific adaptive problems. Those individuals that develop as a part of a caste without a task, due to environmental fluctuations, become an ergonomic drain on the colony. They exist, however, as a hedged bet against risk. High caste differentiation thus causes problems of “structural unemployment” (as it is known in human economics), depending on the relative availability of specific resources. The benefit is that the colony is prepared for a variety of resource contingencies.

The second evolutionary strategy taken by many social insects is to produce fewer castes composed of less-specialized individuals. This strategy is called tychophilic, or “risk-loving.” In this case, the colony supports fewer idle workers, increasing the ergonomic efficiency of a colony. Each less-specialized individual, however, contributes less to the ergonomic efficiency of the colony. This persists because of environmental variance. Low caste differentiation permits the full colony workforce to exploit available resources. The risk is that the specific resources that the castes can exploit become temporarily unavailable.

Because these species are eusocial, the entire colony shares the costs and the benefits of ergonomic efficiency, as well as the risks described above. “Unemployed” workers are still fed. Irreversible adaptive specialization can spread similar costs and benefits to individual organisms in other, non-eusocial species. In these species, the costs and benefits are more severe because they often have no back-up social “safety net”.

Role specialization also occurs in humans. Sulloway (1996), who described niche picking within family units, suggested that the birth order of the siblings is a determinant of many personality characteristics. Although some reviews and reanalyses of Sulloway’s data cast doubt on these
conclusions (e.g., Harris, 1998; Townsend, 2000), empirical replications (e.g., Jefferson, 1998; Salmon, 2003), with admittedly small effect sizes, provide evidence supporting the proposed relationships between birth order and attitudes towards family, friends, and sexual fidelity. The basic principle of intrafamilial niche-picking is analogous to that of competitive exclusion. Later-born siblings, in an effort to garner adequate parental investment, adopt specialized “roles” unoccupied by other siblings. The last-born who has the advantage of no younger siblings to compete with, and a tendency of parents to give them a “handicap” in the parental allotment of time and resources relative to other siblings, is an exception to this.

The dynamics of these within-family niches serve as a microcosm for those within society as a whole. Sugiyama and Sugiyama (2003) proposed a complementary and contingent set of mechanisms that recognize and provide social cues indicating how well a behavioral strategy provides benefits to the self and others. In this view, individuals maximize personal returns (through reciprocal exchange) by occupying social niches with benefits to others. Other individuals track variants in the ability to provide these benefits, then confer greater social status and esteem on individuals who most excel in these roles. Greater social status and esteem ultimately translate into greater material aid in reciprocal exchange.

This mechanism is essentially identical to that proposed in Nesse’s (2007) theory of runaway social selection for displays of partner value and altruism. Social selection, which occupies an intermediate place between natural selection and sexual selection, refers to selection produced by the social behaviors of conspecifics. In this view, competition to be selected as a social partner produces runaway selective processes in the same way that competition to be selected as a sexual partner does in runaway sexual selection (Fisher, 1915, 1930). As above, individuals prefer social partners who display valuable resources and bestow them selectively on associates. This produces selective pressure for individuals to accurately identify: (1) socially valued resources, subject to the economic principles of supply and demand; and (2) preferred social partners, based on their resource and altruistic displays. As in Fisher’s original theory, these selective pressures tend to produce genetic correlations between the resource displays and the partner preferences.

Thus, niche specialization within societies mirrors the principle of niche partitioning within families, a process analogous to adaptive radiation in macroevolution and market specialization within various economies. The partitioning of social niche space allows more direct control and access to specific resources by individuals. Although the environment may induce these phenomena, the phenomena mimic the effects of disruptive genetic selection, leading to a perpetuation of individual differences in personality.

**Directional Social Selection**

An alternative to disruptive social selection is that personality is under directional social selection, an idea Rushton, Bons, and Hur (2008) recently championed. The proposal centers on the discovery of a General Factor of Personality (GFP) identified by a variety of studies (e.g., Figueredo et al. 2004, 2007; Musek, 2007) and cross-validated meta-analytically (Rushton & Irwin, 2008). These authors describe a positive manifold of relatively low but statistically significant phenotypic and genetic correlations among the Big Five (with Neuroticism reversed to indicate Emotional Stability). According to Rushton, et al. the GFP is a direct result of directional selection for a slow LH strategy. In independent studies using nationally representative samples, Figueredo et al. (2004, 2007) reported substantial phenotypic and genetic correlations of the GFP with a multivariate latent variable measuring life history strategy (the “K-Factor”) as well as with
a multivariate latent variable (the “Covitality” Factor) measuring general physical and mental health. More recently, Figueredo and Rushton (2009) reanalyzed these same data to examine how much of the genetic covariance between the GFP, the K-Factor, and the Covitality Factor, was of the non-additive variety. A common pathway ADE model indicated that approximately 47% of the covariance among these three lower-order factors was shared additive (A\(^2\)) genetic variance and 14% of the covariance was shared non-additive (D\(^2\)) genetic variance. Furthermore, the preponderance of the non-additive genetic variance was shared, as modeled by a higher-order “Super-K” LH factor, presumably indicating a common history of recent directional selection (see Penke, Denissen, & Miller, 2007). This more detailed analysis of the genetic correlations support the view that these three traits, a slow (K-selected) LH strategy, good physical and mental health, and the GFP, have been under the same recent directional selective pressures and may thus be coevolved and are mutually coadapted.

Although several groups have demonstrated the existence and even the heritability of the GFP, some challenge its ontological status (e.g., Konstabel & Virkus, 2006; McCrae et al., 2008; Weiss, Bates, & Luciano, 2008). These authors speculate that the GFP might be attributable to various methodological artifacts, among which socially desirable responding or an evaluative bias figure prominently. This is a reasonable argument: the Big Five do appear to have an evaluative dimension (Buss, 1991). Nevertheless, these criticisms are psychometrically, but not evolutionarily informed. If one seriously considers Nesse’s (2007) theory of runaway social selection, for example, then the generally acknowledged population preference for a personality profile reflecting the pattern of the GFP (see Figueredo, Sefcek, & Jones, 2006) inevitably produces selective pressure that create a GFP even when none previously existed. Moreover, the preference for the trait and the trait itself would acquire a genetic correlation with each other because of runaway social selection. Therefore, the existence of an evaluative bias does not, by itself, constitute a sufficient theoretical reason to discount the reality of the GFP, but instead provides grounds to propose its existence as a truly substantive phenomenon. In social evolution, an evaluative bias is not a methodological artifact, but a selective pressure.

Most recently, Rushton and collaborators (2009) reported an even stronger confirmation of the GFP featuring: (1) a cross-national twin design enabling the estimation of the heritability of the GFP; and (2) a multitrait-multimethod (MTMM) design enabling the statistical control of method variance. These features deal a fatal blow to the argument that the GFP represents no more than a methodological artifact. The cross-national twin design raises the important question of how a methodological artifact can have a heritability of 50%. There might be some plausible mechanisms for accomplishing this, such as a heritable disposition towards socially desirable responding, but this finding limits the possibilities for alternative, artifactual interpretations of the GFP. The MTMM design puts the alternative claim that the GFP is composed entirely of shared method variance among the component measures to rest.

We therefore conclude that, in addition to the various disruptive social selection pressures we have identified and reviewed, there is a directional social selection pressure favoring the emergence of the GFP. The relative magnitude of this pressure is, however, evidently not sufficient to completely counter disruptive social selection pressures that maintain interindividual variability in personality over evolutionary time.
Behavioral Flexibility

Temporal Stability: Striking a Balance between “Traits” and “States”

We predict that the temporal stability of behavior matches the temporal stability of environments. The adaptive value of permanent, lifelong changes generated by mechanisms of developmental plasticity (sensu stricto) presupposes four conditions: (1) intergenerational variability of environments; and (2) intragenerational stability of environments; or (3) spatial homogeneity of environments; or (4) relatively short lifespan. Without the intergenerational variability of environments, species-typical fixed action patterns rather than developmental plasticity would evolve. The ecology must be variable over evolutionary time for plasticity to be selected (Figueroedo, Hammond, & McKiernan, 2006). The adaptive value of permanent lifelong changes, however, presupposes the intragenerational stability of environments, because without it, a single adaptive strategy cannot be adaptive throughout the lifespan of the individual. Spatial homogeneity of environments and the supposition that individuals do not frequently migrate between adjacent heterogeneous environments also favor permanent lifelong changes. Alternatively, without sufficient environmental stability, a relatively short lifespan (as in many insects and fast LH species in general) favors developmental plasticity.

In contrast, the adaptive value of more ephemeral and reversible changes generated by the mechanisms of behavioral flexibility presupposes: (1) intergenerational variability of environments; and (2) intragenerational variability of environments; or (3) spatial heterogeneity of environments; or (4) relatively long lifespan. Just like developmental plasticity, the adaptive value of behavioral flexibility presupposes the intergenerational variability of environments. The adaptive value of behavioral flexibility, however, also presupposes the intragenerational variability of environments, because without it, selection favors the more permanent changes generated by developmental plasticity (sensu stricto). Furthermore, the spatial heterogeneity of environments favors behavioral flexibility, under the supposition that individuals frequently migrate among heterogeneous environments. Finally, a relatively long lifespan favors behavioral flexibility, as in humans and other K-selected species, because more long-lived organisms are more likely experience significant environmental changes or engage in repeated migrations among different environments over a lifetime.

Behavioral Signatures

Traditional personality theories assume largely stable, cross-situational trait-level dispositions. These theories focus on the average frequency of behaviors across situations, treating behavioral differences in specific situations as error variance (Mischel, Shoda, & Smith, 2004). In direct contrast to this foundational assumption, individual behavioral variability across situations is substantial (e.g., Mischel, 1968). These data led some researchers to assert that situations rather than traits determine behavior. These contrasting positions—traits versus situations—led to a long-standing person-situation debate among personality theorists.

Kenrick & Funder (1988) summarized the hard-fought resolution to this debate. When analyzed in the aggregate, human behavior exhibits trait-like characteristics. When analyzed in specific situations, behavior exhibits situational specificity. It appears that a situation-specific behavior, which from the aggregate view is error, is a function of traits, experiential history, and situational characteristics.
This confluence makes evolutionary sense. Specific situations are inherent to the study of personality because individuals cannot exhibit rigid global dispositions if they are to outcompete conspecifics by better solving situationalized adaptive problems. Instead, individuals must exhibit flexible behavior that solves adaptive problems presented by each specific situation.

Mischel and Shoda (1995) characterize situationalized behavioral tactics as behavioral signatures, which consist of behavioral tactics manifested as a function of characteristics contained in classes of situations. Thus, apparently distinct situations that contain similar adaptive demands elicit person-specific behavioral responses. In other words, within specific situational classes, behavioral responses follow stable “If...then” or Situation-Behavior relations that depend on the individual (his/her traits and experiential history) and the characteristics of the situation. Thus, two individuals may be equal in measured trait levels of extraversion, but exhibit unique behavioral tactics in apparently identical situations: Individual A may display extraverted behavior at work, but not in casual social settings, whereas individual B does the opposite. These two distinct patterns appear across situational classes as behavioral signatures. On average, one individual may also show higher extraversion than another, yet still show dramatic variability in extraverted behavior across situations.

Mischel & Shoda (1995) argue that redefining situations in terms of their psychological characteristics permits prediction of behavior across contexts. In other words, if personality consistency lies in stable situation-behavior relations, then defining situations in terms of their basic psychological features permits the prediction of an individual’s behavior across situations that share those psychological features. Thus, Mischel and Shoda predict that a proper classification of situations will reveal generalizable, situationally-specific behavioral strategies.

How then can we think about personality and behavioral signatures in a biologically meaningful way? It is unfortunate that Mischel and Shoda (1995) present no theoretically principled way to classify situations. A combination of empiricism and post-hoc intuition regarding shared psychological features appears to be the only current approach to classifying situations. Unhappily, intuitive taxonomies of situations produce unreliable and perhaps idiosyncratic classes—potentially independent of the biology of the species. Hence, there is no reason to expect that psychologically-defined situations will map onto biological constructs in a meaningful way. If the psychological taxonomy of situations is independent of biology, then little in personality theory relates to its biological underpinnings, even in the light of evolution.

Toward a Taxonomy of Adaptive Problems

Much of psychology relies on operational definitions (Bridgman, 1927) to achieve their constructs and measures of them. Although indisputable systematic critiques of operational methods and their use exist, an obvious flaw is that the method classifies by surface characteristics. The dangers of this approach are obvious. Consider the surface characteristics of magnetism, light, radio waves, and electricity—intuitive distinct phenomena belonging to unique categories. This intuition remained true until Maxwell noticed the equations characterizing these ‘distinct’ phenomena match perfectly. Given that, he correctly concluded that these phenomena, so distinct on the surface, belong to the same taxon. A similar situation existed with phenomena such as rust, burning, and metabolism until Lavoisier realized they are simply different manifestations of the same phenomenon. In short, for a proper scientifically useful taxonomy to exist, one needs deep well-developed theoretical principles upon which to build the classification
As a first step, we proposed that classes of adaptive problems may serve to organize a principled taxonomy of situations (Figueredo et al., 2007; Figueredo, et al., 2010). We now present an updated version of this taxonomy. Briefly, we can partition an environmental context into abiotic settings and biotic situations. We can then partition biotic situations into interspecific and intraspecific situations. Interspecific situations include prey situations, mutualistic situations, and threatening situations. Threatening situations include parasitic situations, pathogenic situations, and predatory situations.

Intraspecific situations can be partitioned into extrafamilial and intrafamilial situations, including parent–offspring situations and sibling–sibling situations. Both extrafamilial and intrafamilial situations can be further partitioned into intrasexual and intersexual situations. Intrasexual situations include male–male and female–female cooperation and competition. Intersexual situations include male–female and female–male cooperation and competition.

Classifying a context in terms of each of its component classes permits us to define a situation fairly precisely. Within this taxonomy, all intraspecific situations generate the selective pressures that various theorists have classified as attributable to social selection (Alexander, 1989, 1990; Flinn et al., 2005; Geary, 2005; Nesse, 2007).

Current trait-level taxonomies ground personality (e.g., the Big Five) empirically rather than theoretically. This lack of theoretical basis may explain why personality traits do not predict behavior on any single occasion consistently or well: The traits do not contain obvious design features that solve biologically meaningful adaptive problems. These trait labels may mislead scientists dedicated to uncovering the adaptive functions (and deep structure) of traits.

Our proposal leads us to a psychologically (but not biologically) radical proposal. Behavioral signatures, which are detectable based on intuitively classified “situations,” may be signs of behaviorally flexible traits rather than distinct classes of behavior. In other words, empirically detectable behavioral signatures may be highly variable surface manifestations of a smaller set of traits. We suggest that, because each adaptive problem demands a contextually sensitive behavioral solution, individuals evolved behaviorally flexible traits sensitive to particular adaptive circumstances. Hence, similar to pleiotropy found at the genetic level, a single trait or combination of traits may manifest as a behavioral signature conditional upon varying contextual affordances.

By this view, as-yet-undetected trait-level dispositions that when properly characterized, predict context-specific behavioral tactics across relevant adaptive problems (contexts). If we classify contexts by adaptive problems and thereby properly characterize traits, our problem becomes specifying an adequate measurement model for each latent trait. Under these circumstances, we expect that each trait will manifest itself as a function of genetic structure, experiential history, and in response to the affordances offered by the context in which the adaptive problem appears. In short, a single latent trait will, under such circumstances, predict the occurrence of behaviors that, on the surface, appear dissimilar or even unique.

Figueredo, Montero-Rojas, Frías-Armenta, & Corral-Verdugo (2009), for example, recently described certain individual difference traits that predict spousal abuse in some cultural contexts but not in others. High individual endorsement of the “culture of honor” (COH) predicts family deterrence of
the spousal abuse of female relatives in the high-COH state of Sonora, an ancestrally herding society, but not in the low-COH city of San José, Costa Rica, an ancestrally farming society (see Figueredo, Vásquez, Brumbach, et al., 2004), indicating that individual convictions, such as a personal code of honor, may be ineffective outside of a supportive social context. Even individuals holding a high COH, acting within a low-COH social context, did not project the kind of influence that they normally exhibit within a supportive, high-COH, social context. Conversely, the self-reported patriarchal values of individuals predict spousal abuse in San José, Costa Rica, a sexually and economically egalitarian society by Latin American standards, but not in Hermosillo, Sonora, a relatively sexually and economically inegalitarian society, where machismo and social conservatism predominate.

To solve an adaptive problem, an organism must first detect it. If it is to survive and reproduce, an organism must also detect, and perhaps perceive, setting and situation affordances (Gibson, 1976) that will support solutions to the adaptive problem (Tolman, 1938). Because multiple adaptive problems and a myriad of affordances simultaneously confront the organism, those problems and affordances must be prioritized. Only then can an organism respond to the most pressing problems adaptively. We call the outcome of psychological processes dedicated to the detection and selective prioritization of adaptive problems and affordances an immediate contextualized perspective. Interactions among the organism’s trait characteristics, experiential history, and current establishing operations (e.g., Pavlov, 1927), help establish behavioral (tactical) priorities within the context based on the organism’s immediate contextualized perspective.

Once behavior unfolds, one of three outcomes will occur: the behavior does not solve the adaptive problem, it partially solves the problem, or it completely solves the problem. If the behavior solves the adaptive problem partially or completely, then the problem and establishing operations disappear, previously relevant affordances become irrelevant, and the organism must repeat the prioritization, perspective taking, and problem solving process. Upon re-encountering a similar context, the organism will tend to repeat the ‘successful’ behavior (e.g., Guthrie, 1952). That is, evidence of reinforcement will be present (e.g., Thorndike, 1927).

If the behavioral tactic does not solve the adaptive problem, or does not solve it well enough, then extinction, punishment, or penalty will occur (e.g., Skinner, 1953). The organism may wait until the situation changes (extinction), leave the field (penalty), or continue to vary its tactics until striking upon an effective solution (punishment).

To test this proposal (and the implicit taxonomy upon which it rests), we must first acknowledge there is a problem—and that neither operational definitions nor intuitively classified situations help us classify biologically meaningful contexts. Second, we might look to other fields that have acknowledged similar problems and solved them. We might also look to psychologists who have given taxonomy significant thought (e.g., Meehl, 1994, 1996). Examining solutions to similar problems from other fields could provide multiple approaches that can guide critical tests. For example, perhaps the proposal that form is not a proper way to classify behavior will turn out to be wrongheaded and, like Mendeleev’s periodic table or Hennig’s phylogenetic systematics, form (surface characteristics of the behavior) classifies traits properly. On the other hand, the adaptive problems traits evolved to solve (function) rather than how those traits appear (form) may serve to classify
traits properly. Just as the biological systematists shifted from morphological (form) classification systems to systems based on more meaningful biological characteristics (genetic structure), personality psychologists may need to expand their theoretical perspectives to include apparently pressing taxonomic problems.

**Conclusion: an Integrated Overview**

We have reviewed theoretical arguments and empirical evidence that a combination of: (1) frequency-dependent niche-splitting, (2) developmental plasticity, (3) genetic diversification, (4) directional social selection, and (5) behavioral flexibility shape individual variation in personality. We have argued that extant theory and data are inconsistent with assigning the evolution of individual differences to any one selective pressure to the exclusion of the others. Instead, we argue that the ecological conditions intrinsic to the social circumstances of many species, including humans, favors a combination of these shaping pressures. Thus, we argue that the only single superordinate category that includes most of these convergent and divergent selective pressures is social selection (e.g., West-Eberhard, 1979, 1983; Nesse, 2007).

We predict that greater attention to the various evolved ecological functions of personality will repay mainstream research on personality psychology handsomely. The ecological approach to personality can do much to resolve the person-situation debate by viewing personality variation as a socially selected adaptation of individuals to the ecological contexts in which they evolved.

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**References**


Part III Evolutionary Genetics of Personality
Bridging the Gap Between Modern Evolutionary Psychology and the Study of Individual Differences

Lars Penke

One of the main aims of evolutionary biology is to explain the forms of adaptation found in nature—the complex functional design features that evolved through natural selection to increase the fit of organisms to their environment (Ridley, 2003). Evolutionary psychology is the scientific field that studies how organisms adapt behaviorally to their environments. Evolutionary approaches to understanding humans are as old as the theory of evolution itself, dating back to Charles Darwin. However, the forms of adaptation that modern human evolutionary psychology has addressed in the last 20 years have been somewhat limited: so far, its focus has been mainly on universal adaptations (Tooby & Cosmides, 1990a, 2005). Universal adaptation refers to aspects of the human genome that became fixated in the population by natural selection before our species began to spread over the globe about 50,000 years ago (Klein, 2008)—in the so-called ‘Environment of Evolutionary Adaptation’ (EEA, Tooby and Cosmides, 1990b)—and that have not changed systematically since, making them universal to all living human beings. Examples include opposable thumbs, upright gait, color vision, the capacities to easily acquire languages in childhood or to develop fear of spiders or snakes, a theory of mind, a desire for sex starting at puberty, the attachment system, or certain parental behaviors. The sum of all adaptations can be called the ‘adaptive design.’ Modern evolutionary psychology has developed a powerful methodology for the study of universal adaptations (Tooby & Cosmides, 2005), and since the theory of evolution is the only scientific meta-theory for the behavioral sciences that has been proposed to date, this makes it an invaluable asset to any area of general psychology.

However, modern evolutionary psychology has had (with some notable exceptions, e.g., Wilson, 1994; Gangestad & Yeo, 1997) a more limited impact on the study of individual differences like personality traits, cognitive abilities, psychopathologies, or morphological differences. The reason for that is already implied in the term ‘universal adaptations’: Since they are supposed to be part of the universal human design, they should not differ between individuals. It is important to understand that individuals of the same species never vary along any dimension of individual differences (like extraversion or intelligence) because they have different evolved adaptations (except for sex differences; see below): Adaptations are complex functional design features of a species that develop reliably in consistent environmental circumstances because they depend on the systematic interplay of complex genetic structures with environmental regularities (Tooby & Cosmides, 2005; Tooby, Cosmides, & Barrett, 2005). If adaptations were to vary between members of the same species, different individuals must show different complex systems of genetic structures. If individuals with different adaptations then engaged in sexual reproduction (which should be possible for them to qualify as members of the same species), these different genetic structures would be broken up and mixed during the process of recombination, disrupting their complex organization and consequently their adaptiveness (Tooby & Cosmides, 1990a). Thus, complex evolved adaptations themselves cannot vary between individuals.

However, adaptations are sometimes capable of producing different (morphological or behavioral) phenotypes under different conditions, and systems of adaptations are sometimes able
to tolerate some genetic variation. So while adaptations themselves are universal to all human beings, we can observe individual differences between humans that appear related to evolved adaptations: All humans show upright gait, but they differ in running speed and sense of balance. All humans see with two eyes of identical design and process what they see in their visual cortices, but they may differ in visual acuity or the ability to discriminate certain colors (e.g. if they suffer from color blindness). All humans are endowed with working memories that appear to rely on the same cognitive components (Myiake & Shah, 1999), but they differ in working memory capacity. Certain parieto-frontal circuits in the brain, including those that give rise to working memory, provide all humans with the adaptive ability to reason, but people differ in their information processing speed, neuronal white matter integrity, brain size, glucose metabolism efficiency, and other fundamental brain parameters that give rise to individual differences in general cognitive abilities, including reasoning ability (Deary, 2000; Deary, Penke, & Johnson, 2010; Jensen, 1998; Jung & Haier, 2007). All humans possess various adaptations for social exchange (Hammerstein, 2003), but their cooperative tendencies vary along a personality dimension of agreeableness (Denissen & Penke, 2008a). All humans come with a multitude of domain-specific adaptations for sexual reproduction (Buss, 2003; Geher & Miller, 2007), but they differ in their mate values and their sexual strategies and tactics (Gangestad & Simpson, 2000; Penke & Asendorpf, 2008). There is absolutely no reason to assume a one-to-one match between adaptations and dimensions of individual differences. The most likely relationship is that each dimension of individual differences that is fitness-relevant relates to a system of universal adaptations, and each adaptation relates to several individual differences. So despite occasional claims to the contrary (e.g. Bernard, Mills, Swenson, & Walsh, 2005; Kanazawa, 2004), universal adaptations cannot be studied by assessing related individual differences, and while dimensions of individual differences can be cross-culturally universal and even adaptive (a point to which I will return later), it is misleading to equate them with universal adaptations (as for example done by MacDonald, 1995, 2005).

In this chapter, I will explore how individual differences can be better integrated into modern evolutionary psychology. I will first introduce life history theory as arguably the most important evolutionary theory for the study of individual differences and then discuss different sources of individual differences from an evolutionary perspective. After a brief discussion of sex differences, I will focus on two different forms of conditional adaptations, those evolved mechanisms that react flexibly to the environment, as sources of non-heritable individual differences. I will then explicate how an evolutionary perspective can be taken on genetic differences between individuals. Finally, I will present a general evolutionary framework for the study of individual differences that integrates universal adaptations, genetic differences, and life history theory and outline some future directions for an evolutionary psychology of individual differences.

Life History Theory

From an evolutionary perspective, the most important dimension of individual differences is inclusive fitness (Penke, Denissen, & Miller, 2007a). It can be defined as the spread of one’s genes in the population over generations. Fitness is intimately intertwined with reproductive success in terms of surviving children, grandchildren, and genetic relatives. According to life history theory (Stearns, 1992; Roff, 1992; Kaplan & Gangestad, 2005), reaching high
reproductive success is a complex task that requires mastering a series of challenges over the lifetime, including successful growth and maturation, finding and courting mates, reproducing, raising children, supporting relatives, and maintaining a healthy phenotype for as long as one can provide kin with further helpful support of any kind. However, these different tasks often conflict with each other, and efforts (in terms of energy, time, money, attention, and other resources) that can be allocated to them over the lifespan are always limited. Consequently, trade-offs have to be made when allocation decisions are reached. Note that ‘decision’ in this context does not imply consciousness, and the word will be used in this sense throughout the chapter. According to life history theory, the two major trade-off dimensions are (1) extended growth vs. early reproduction and (2) number of offspring vs. amount of investment in every offspring. There are also other, more specific trade-off dimensions, for example courting many potential mates vs. committing to one, investing in own children vs. other genetic relatives, or putting a lot of effort in reproducing vs. maintaining the body long enough to make meaningful investments in grandchildren. The central function of the adaptive design is to make possible the most fitness-enhancing allocation decisions given these trade-offs (Kaplan & Gangestad, 2005).

Across species, it is helpful to characterize individual species along a continuum of broad life history strategy, ranging from so-called r-strategists (species that allocate efforts more towards early maturation and reproduction as well as offspring quantity, e.g. oysters, rabbits) to so-called K-strategists (who invest in extended growth, body maintenance, and offspring quality, e.g. elephants, whales) (Wilson, 1975; but see Bielby et al., 2007). The universal adaptive design of a species reflects its broad strategy in many regards, including for example its average body and brain size, life expectancy, and dependency of offspring at birth. According to all possible criteria, humans clearly fall at the K end of the continuum (Rushton, 2004). Thus, the adaptive design shared by all human beings predisposes them, compared to other species, to a life of relatively slow development, extensive learning, few children, and effortful parental duties. Within a species, however, there is usually not a single optimal set of life history decisions: Even if all members of a species are geared towards the same broad life history strategy, different individuals can allocate their efforts somewhat differently over the lifespan within the margins of their species-specific strategy and still end up with very similar fitness levels (i.e., they can follow different evolutionarily stable strategies: Maynard-Smith, 1982). Also, the most optimal strategies might be different for individuals with different genetic makeup or individuals who face different environmental opportunities or challenges. These degrees of freedom within the species-typical life history strategy allow for individual differences in fitness-relevant behaviors, and it is certainly the reason that life history theory has often been used as a theoretical framework for evolutionary approaches to individual differences (e.g. Buss, 2009; MacDonald, 2005; Figueredo et al., 2005). The current chapter stays in this tradition.

Two Versions of the Human Adaptive Design: Sex Differences

Without doubt, the individual differences that have received most attention from evolutionary psychologists are sex differences (Mealey, 2000). Biological sex (unlike psychological gender) is a very peculiar individual difference because it is not dimensional, but categorical. Some chromosome anomalies like Klinefelter’s (XXY) syndrome or XYY syndrome notwithstanding, humans normally come in two distinct versions or ‘morphs’— women and men (Tooby &
Cosmides, 1990a). The differentiation of these two distinctive forms of the human adaptive design is triggered in the first four weeks of prenatal development. If a Y chromosome is present, genes in called the so-called sex-determining region on the Y chromosome (SRY) initiate the deviation from the default female developmental program towards a male phenotype. Since the SRY genes have such major, irreversible effects on many different parts of the human phenotype that we can clearly distinguish two different human morphs, this genetic region can be called a genetic master switch. However, since there are no other clearly distinguishable human morphs that would qualify as equally categorical, the SRY region is probably also the only genetic master switch in humans that controls early and irreversible development into different morphs (Penke, Denissen, & Miller, 2007b).

The biological way to define sexes is that females contribute larger (and thus metabolically more costly) sex cells to sexual reproduction than males do, a difference called anisogamy. In mammalian species like humans, this initial asymmetric contribution is further exaggerated by the fact that women inevitably bear the costs of internal pregnancy, birth, and usually lactation, leading to a pronounced sex difference in minimal parental investment. In his seminal parental investment theory, Trivers (1972) proposed that this fundamental sex difference should lead to differences between men and women in all kinds of morphological features and behavioral tendencies that relate directly or indirectly to mating and reproduction. Indeed, this is the area where some of the strongest human sex differences have been found (Hyde, 2005), and they have already been studied extensively within an evolutionary framework (Buss, 2003; Mealey, 2000).

So in a nutshell, the human adaptive design comes in a male and a female version and the sexes show some universal differences on the morphological and psychological level that can be seen as adaptations to the stable feature of the social environment that women get pregnant and men don’t. These two distinctive adaptive designs facilitate qualitatively different life history strategies in men and women (e.g., men tend to prefer to have sex faster and with more different partners—Schmitt et al., 2003). So far, so good—but what else, beyond sex differences, can evolutionary psychology contribute to the study of individual differences?

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**Flexibility in the Adaptive Design: Conditional Adaptations and Phenotypic Plasticity**

The other major contribution that modern evolutionary psychology has made to the study of individual differences is the concept of *conditional adaptations*. Adaptations are conditional when they are capable of producing different (morphological, physiological, or behavioral) phenotypes depending on the requirements of the environment. Conditional adaptations are a special case of *phenotypic plasticity*, which refers to the fact that identical genotypes usually do not produce identical phenotypes (Pigliucci, 2005; West-Eberhardt, 2003). The natural example in humans is monozygotic (i.e., identical) twins, who often show astonishing, but never perfect resemblance (Segal, 2005). Aside from conditional adaptations, phenotypic plasticity can be due to factors that impede an organism’s development in accordance with its adaptive design, causing *developmental instability* (Polak, 2003) due to exposure to environmental factors that disturb development or somatic maintenance during adulthood. Examples include toxins, pathogens, extreme temperatures, maternal stress during pregnancy, or malnutrition. Over and beyond that, lab studies on model organisms have shown that even in identical environments, individuals with identical genomes do not produce perfectly identical phenotypes (Kirkwood et al., 2005). This has been taken as evidence that non-linear epigenetic interactions and pure molecular chance
events contribute a ‘chaotic’ component to phenotypic plasticity (Eaves, Kirk, Martin, & Russel, 1999; Finch & Kirkwood, 2000).

The phenotypic plasticity that is caused by conditional adaptations is different from developmental instability: It is not due to imperfect development, but is adaptive in its own right. Conditional adaptations, themselves part of the universal adaptive design, are sensitive to cues from the environment. These adaptations use the environmental information to adjust the phenotype towards increased fit to the environment. Conditional adjustments of the phenotype can happen in very different ways (see Penke, 2009): At one extreme, the whole developmental pathway of the organism can be fundamentally altered early on. An example of this is sex determination in some amniote vertebrates such as alligators, some turtles, or the Australian jacky lizard, which is dependent on the environmental temperature during the embryonic stage (Warner & Shine, 2008). In humans, where sex is genetically determined, such an extreme degree of adaptive developmental plasticity most likely does not exist. However, the possibilities of other, less extreme forms have received quite some attention in the evolutionary psychological literature. One prominent example is first language acquisition, which shows clear signs of adaptive design and is conditional to the language with which one has interactive exposure during early childhood, but is lifelong stable afterwards (Pinker, 1994). Another prominent example is the Belsky-Draper-Steinberg hypothesis (Belsky, Draper & Steinberg, 1991), which proclaims that the degree of environmental stress experienced during childhood can be an indicator of adult reproductive conditions (e.g. the prevalence of monogamy and paternal investment). This hypothesis proposes that humans possess a conditional adaptation that uses childhood stress as a cue to channel maturation and psychosocial development so that they fit the demands of the predicted optimal reproductive strategy in adulthood. Some prominent versions of this hypothesis, especially those relying on stepfather presence as the critical cue, have been disconfirmed by the empirical evidence (Ellis, 2004; Mendle et al., 2006; Neberich, Lehnart, Penke, & Asendorpf, 2010). However, different variants of the core Belsky-Draper-Steinberg hypothesis have subsequently been proposed (Del Giudice, 2009; Del Giudice & Belsky, this volume; Ellis, 2004, this volume), which might turn out to be more viable—and they all rely on adaptive developmental plasticity.

At the other extreme of phenotypic plasticity that is due to conditional adaptations are more direct adaptive conditional adjustments, which do not require developmental changes of the phenotype. Thus they are much more flexible and reversible than adaptive developmental plasticity, and they allow individuals to adapt to their current environment much faster. For example, people are able to quickly adjust their mating preferences, interests, and tactics adaptively to the opportunities of the local mating market (e.g. sex ratios, number of available potential mates, own and potential mates’ relative mate value—reviewed in Lenton, Penke, Todd, & Fasolo, in press; Penke, Todd, Lenton, & Fasolo, 2007). Emotions and affective states can also be seen as conditional adaptations that lead to adaptive conditional adjustments when triggered by the right cues (Tooby & Cosmides, 1990b; see also Miller, this volume). For example, someone might trigger the emotion of anger in an individual by being a persisting encumbrance to his or her goals, and this puts the actor in a transient altered state of consciousness called anger that facilitates taking actions against the encumbering person. Similarly, personal losses, social rejection, failed efforts, or other overly stressful life events might put people in a state of depression. It has been shown that the specific behaviors people show in this state fit the affordances of the triggering situation (e.g., passivity and rumination after failure, crying and seeking support after social loss or
rejection), which can be taken as indication of conditional adaptive design (Keller & Nesse, 2006).

Adaptive developmental plasticity and adaptive conditional adjustments can be seen as endpoints of a continuum of adaptive phenotypic plasticities that differ in how much the adaptive response requires developmental changes of the phenotype (Penke, 2009). This implies they also differ in how stable the relevant aspects of the environment need to be for an adaptive response: Even if childhood stress is a valid cue of current reproductive conditions, conditional adaptations in the tradition of the Belsky-Draper-Steinberg hypothesis will only yield an adaptive response if the reproductive conditions are sufficiently stable over the next 10 to 20 years later, until the individual reaches reproductive age. If they have changed substantially in the meantime (e.g. from socially tolerated promiscuity to strictly enforced monogamy), the developmental change triggered by the conditional adaptation can even be maladaptive. The same is also true for non-conditional adaptations, which can only be adaptive if the relevant environmental aspects resemble the EEA (Tooby & Cosmides, 1990b) and are thus dependent on an even higher degree of environmental stability (Penke, 2009). Adaptive conditional adjustments do not suffer from this problem, but lacking developmental time, they have limited power to change the phenotype to reach a better adaptive fit to the environment: The phenotypic changes triggered by adaptive conditional adjustments are usually only behavioral, not morphological (see Penke, 2009).

This trade-off between the possible degree of developmental phenotypic change and the ability to react to environmental change provides one reason that individuals do not show optimal adaptive plasticity (i.e., the ability to achieve perfect adaptive fit to the current environment instantaneously), which would of course be the desirable ideal (Penke et al., 2007a). A second reason for suboptimal adaptive plasticity is the noisiness of environmental cues: If the available environmental information is an unreliable indicator of the fitness pay-offs of different possible life history strategies, instant phenotypic plasticity can cause as much harm as it can do good (see Miller, 2007; Penke et al., 2007a). So even for humans, who are undoubtedly the species that has evolved the most advanced capacities for learning from, reasoning about, and flexibly adjusting to their environment (Kaplan, Lancaster, & Robson, 2003), perfect adaptive plasticity remains utopian.

**Genetic Differences**

Even though conditional adaptations can react to the environment, they are still part of the species-typical adaptive design, implying that the systematic, adaptive individual differences they produce are purely environmental in nature, implying zero heritability. This stands in stark contrast with the behavioral genetic literature, which has reported significant heritabilities for virtually all human traits that have been sufficiently studied (Plomin, DeFries, McClearn, & McGuffin, 2008). Indeed, genetic variance in human individual differences is so ubiquitous that Turkheimer (2000) has called its existence in any trait the first law of behavior genetics. While the molecular causes of genetic variance in most traits are still astonishingly poorly understood (Maher, 2008) and its mere existence tells us nothing specific about the evolutionary history or biological significance of a trait (Johnson, Penke, & Spinath, 2010), non-zero heritabilities are a clear indicator that traits
can and will react to evolutionary selection pressures as soon as the traits have any fitness-
relevant effects (Visscher, Hill, & Wray, 2008). Thus, any evolutionary approach to individual
differences is indefensibly incomplete if it cannot account for genetic variation among
individuals.

Unlike sex differences and conditional adaptations, however, within-sex heritable differences do
not sit well with modern evolutionary psychology’s strong focus on universal adaptations.
Indeed, in their seminal early paper, Tooby and Cosmides (1990a) discussed a variety of ways
that genetic variance can be viewed evolutionarily, but largely concluded that most genetic
variance in psychological traits is likely selectively neutral—merely noise in the system—or a side
effect of selection for pathogen resistance at best. Perhaps due to its convenience, this conclusion
remained rather unchallenged for about 15 years (but see Gangestad & Yeo, 1997; MacDonald,
1998; Wilson, 1994). However, on a closer look the neutrality argument relies on very strict
assumptions, including that not a single additional child is born in the next 15 generations
because people differ in a supposedly neutral trait (Penke et al., 2007a). These assumptions may
hold for some specific traits, like certain smell-, taste- and pheromone-related perceptual abilities
(Nozawa, Kawahara, & Nei, 2007), which might not have the adaptive benefits in modern
humans that they likely had in our ancestors. But the assumptions are hardly plausible for most
individual differences psychologists are interested in, since substantial effects on fitness-relevant
life outcomes have been shown again and again for personality traits (Ozer & Benet-Martinez,
2006; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007), general cognitive ability (Batty,
Deary, & Gottfredson, 2007; Deary, Whalley, & Starr, 2008; Gottfredson, 2004, 2007), physical
attractiveness (Langlois et al., 2000), and psychopathologies (Keller & Miller, 2006).

So if virtually all human individual differences of interest are heritable enough to be potentially
affected by selection and fitness-relevant enough that they cannot be selectively neutral, we have
to explain why these differences have not vanished—been driven to extinction or fixation—over
evolutionary time. In a nutshell, there are three possible reasons why non-neutral genetic
differences persist: 1) new genetic variants with positive effects on fitness have emerged fairly
recently, so that positive selection simply has not had enough time to fix them yet (recent
selective sweeps); 2) different competing genetic variants have the highest fitness pay-offs under
different conditions, so that there simply is not a single optimal genetic variant that could get
fixated (balancing selection); or 3) so many new genetic variants

with small negative effects on fitness emerge so that purifying selection is unable to get rid of all
of them (mutation-selection balance). Based on these possibilities, several possible mechanisms
have been proposed in the field of evolutionary genetics (Mitchell-Olds, Willis, & Goldstein,
2007; Roff, 1997; Roff & Fairbairn, 2007), which I will sketch in the following two sections (for
a more detailed discussion, see Penke et al., 2007a,b).

Recent Selective Sweeps

While many important parts of our genome have not changed since the Pleistocene EEA days
(and indeed have often been found to be conserved since way back in our mammalian and pre-
mammalian phylogenetic history), accumulating molecular genetic evidence suggests that it
would be a fallacy to assume that human evolution has stopped since (Cochran & Harpending,
2009; Hawks, Wang, Cochran, Harpending, & Moyzis, 2007; Nielsen et al., 2007; Williamson et
al., 2007). One main reason that selection pressures still act on our genetic variation is
environmental change: Since humans moved out of Africa, they experienced a broad variety of
climates, pathogens and dangers; it saw the advent of agriculture, domesticated animals, big cities, harems, social monogamy, and social health systems; and it invented literacy, penicillin, weapons of mass destruction, and the Internet, among many other things. Even though we still breathe air, digest food, and beget children, it is fair to say that our ecology has changed dramatically since the EEA. These ongoing radical environmental changes increase the odds that newly derived genetic variants (mutations) or existing, but selectively neutral genetic variants become more adaptive than the so far most widespread variants (the “wild type”). They also raise the probability that genetic variants that had not been phenotypically expressed (‘active’) in the EEA now suddenly affect the phenotype and its fitness for better or worse, thus becoming exposed to selection (so-called cryptic genetic variation, see Gibson & Dworkin, 2004). Genetic variants that are under positive selection because they are or have become fitness-enhancing can be called adaptive, since they are on their way to becoming fixated and possibly part of an adaptation. In reverse, all other genetic variants at the same genetic locus as the adaptive variant can be classified as maladaptive. As long as fixation of the more adaptive variant has not been completed, genes under recent selection can contribute to genetic differences we observe today—we observe them while they experience adaptive selective sweeps (Miller, this volume; Cochran & Harpending, 2009).

It is important to note that these ‘adaptations’ that are based on single or small sets of genetic variants are quite distinct from the complex adaptations that are usually studied in evolutionary psychology. Single adaptive genes may alter certain parameters of one or (more likely) several complex adaptations, such as their strengths, size, activation thresholds, sensitivities, reactivities, etc.—variation that complex adaptations might tolerate. Some possible examples of such parameters are given at the beginning of this chapter. The gradual fixation and accumulation of single adaptive genes over long evolutionary periods can lead to qualitative changes in and the emergence of new complex adaptations, but while complex adaptations need to be species-universal (at least within sexes) to be evolutionary stable, single adaptive genes can evolve quickly and contribute to genetic variation before they get fixated.

Various molecular genetic techniques can provide hints to whether a region in the genome has been under recent selection or not, either based on comparisons between human and non-human primate genomes (“comparative genomics”) or on searching for certain regional patterns within human genomes. The results, however, are heavily dependent on the quality of the genetic data and so far limited to larger genomic regions (Harris, 2008; Hawks et al., 2007; Hoffmann & Willi, 2008; Williamson et al., 2007). Still, the rapid development in this area makes it likely that we will have much more detailed knowledge about where selection acted on our genome in the near future.

Though recent selective sweeps appear to be good candidates for explaining currently observable genetic differences, recent empirical findings have tempered initial enthusiasm a bit. Take for example the MCHP1 and ASPM genes. Both of these genes are related to primary microcephaly, a neurodevelopmental disorder characterized by dramatic reduction in cortical volume, and both have been found to show signatures of recent adaptive selection (Evans et al., 2005; Mekel-Bobrov et al., 2005). To much surprise, however, subsequent studies failed to find any associations of these genes with current individual differences in brain size or cognitive, reading, or language abilities (Woods et al., 2006; Bates et al., 2008; Mekel-Bobrov et al., 2007).
One reason that genes under recent selection are not necessarily likely to explain much of the genetic differences among people is that the time selection needs to fix a genetic variant with consistent adaptive benefits in a population is not very long (judged by evolutionary standards)—only about 10,000 years (Keller & Miller, 2006). Since the last human ancestor that was shared by all modern humans lived much longer ago, selective sweeps are likely population-specific. Any sample we draw nowadays might be a snapshot of specific selective sweeps—the genetic variants that contribute to individual differences in one population now might have already been fixated in another and may never have been introduced by mutation in a third (see also Cochran & Harpending, 2009; Penke et al., 2007b; Penke et al., 2009).

So the proposition that recent selective sweeps explain a significant proportion of the genetic variance in human traits is based on the proposition that either traits are very much population-specific (which is at odds with the empirical finding that phenotypically very similar traits can be found in many different populations around the world, e.g. Caroll, 1993; McCrae & Allik, 2002), or that very different genetic make-ups underlie similar traits in different populations (an at least very counter-intuitive, though possible, proposition that would need strong empirical evidence). Still, recent selective sweeps (alongside random genetic drift) may explain genetic differences between populations. There is increasing empirical evidence that such differences exist, even between different European countries or even different regions within Italy or Switzerland (e.g. Heath et al., 2008; Lao et al., 2008; McEvoy et al., 2009; Novembre et al., 2008), though it is not well understood which phenotypic traits they affect. However, it is difficult to use selective sweeps as an explanation for the existence of cross-culturally replicable dimensions of heritable individual differences, like intelligence, extraversion, neuroticism, or agreeableness, unless we assume distinct underlying genetic make-ups. This is a provocative hypothesis for future research. But there are alternative explanations. One is that genetic variants can stay in a population much longer if their effects on fitness are not consistent, but change across different environments. In this case, they are under balancing selection, which will be discussed next.

**Balancing Selection**

Genetic variants can be maintained in the population indefinitely, as long as the average effect on fitness across all relevant environments is equal for all variants at a genetic locus. In this case, selection is unable to fixate any one of the variants, because each can be selected for under certain conditions. Evolutionary genetic models have shown that balancing selection is indeed a plausible mechanism for the maintenance of genetic differences, even in quantitative traits that vary dimensionally between individuals, like those usually studied by psychologists (Bürger, 2000; Roff, 1997; Turelli & Barton, 2004). Just like genes under recent selective sweeps, genes under balancing selection may affect parameters of complex adaptations. The major difference between them is that in the former case, one genetic variant is more adaptive (fitness-enhancing) for everyone in the population, while there is no single most adaptive genetic variant under balancing selection—it depends on the environment.

Note that 'environment' is defined very broadly in this context: For one, it includes the external physical and organic environments, which can vary in climate, food availability, dangerousness, pathogen and toxin prevalence, the rates at which cognitive and physical challenges are encountered, and many other properties. Take for example an individual with a genetic propensity to show risky, impulsive, novelty-seeking behaviors
(which have been related to the dopamine receptor gene DRD4; Ebstein, 2006): The early bird gets the worm, but the second mouse gets the cheese. Whether the novelty seekers or the hesitators get the rewards depends on the distribution of ‘worms’ vs. ‘mousetraps’ in the current environment (Pinker, 2009).

Secondly, the social environment can vary just as much as the physical and organic environments. Liars and cheaters will have advantages when the majority of people are honest and cooperative, but might fare worse than a minority of co-operators once they become the majority themselves (Mealey, 1995)—just as aggressive hawks only fare better than cowardly doves as long as they are more likely to encounter doves than hawks (Maynard-Smith, 1982). These are classic examples of negative frequency-dependent selection (called ‘negative’ because minorities are favored), the most established form of social balancing selection. Both the physical/organic and the social environments can vary spatial and temporally. The same individual can encounter different environments at different points over his or her lifespan, or his or her ancestors could have encountered different environments by staying in one place that changed over time or by moving to different places. In any case, what matters for balancing selection is that the fitness benefits of genetic variants, averaged across all environments that all their carriers encounter, stay the same. If this condition is met within a single lifespan, the genetic variants are effectively neutral in terms of lifetime fitness effects. If the condition is met across generations, the genetic variants are not neutral for the fitness of individuals, but balanced at the population level over time.

In other special cases of balancing selection, the ‘environment’ that affects a genetic variant is internal rather than external: it is constituted by other genes of the individual. One very plausible candidate for such a so-called epistatic interaction effect on fitness is the sex-determining genetic region SRY that was introduced earlier. A genetic variant might show an epistatic interaction with SRY genes in that it has fitness-enhancing effects in a male body but might be fitness-reducing in a female body and vice versa. Fitness-relevant interactions with sex (and thus epistatic interactions with the sex-determining SRY genes) are called sexually antagonistic pleiotropy, and it can be understood as a special case of temporal environmental variation across generations, with the male and the female bodies being the ‘environments’ that a genetic variant encounters over generations. An example could be genetic variants that predispose for antisocial personality marked by Machiavellism, narcissism and subclinical psychopathy, which appear to be more favorable for male than female mating success (Jonason, Li, Webster, & Schmitt, 2009). Similar interactions between a certain genetic variant and other parts of the genome or with overall genetic fitness (see below) are also possible, but far less well documented.

Balanced fitness effects of a trait in a single, stable environment will most likely not explain much genetic variance in individual differences, since such simple antagonistic pleiotropies or trade-offs are usually evolutionarily unstable—over time they will get replaced by genetic make-ups that avoids such direct conflicts (Roff & Fairbairn, 2007). An example might be the β₂-adrenergic receptor gene ADRB2, which appears to have opposing effects on general intelligence and some health conditions in early and late adulthood, but shows markedly reduced variability in some populations (Penke et al., 2010). Spatiotemporal environmental heterogeneity is the most plausible mechanism for keeping genetic variants under balancing selection over evolutionarily
long periods, with spatial variation usually working better than temporal variation (Hedrick, 1986, 2006).

Critical conditions for balancing selection to maintain genetic variation are that change in environmental selection pressures happen in a way that the average adaptive benefit of each genetic variant is equal across space and time, and that no genetic variant is affected long enough by unidirectional selection pressures to drive it to fixation or extinction. These equilibrium conditions, which are assumed in all mathematical models that support balancing selection (e.g. Bürger, 2000, 2005; Schneider, 2006; Roff, 1997; Turelli & Barton, 2004), might appear implausible in the light of all the radical changes that took place in human ecology during the last 10,000 years (see above and also Miller, this volume). Indeed, quite a few novel selection pressures, such as those for lighter skin pigmentation that came with populating the Northern hemisphere or those for lactose tolerance that came with the domestication of animals, were certainly so directional that they left hardly any room for balancing selective forces. A multitude of other cultural innovations led to widespread dominance of humans over ecological factors that once were hostile selection pressures, such as predators, food shortages, and the weather, essentially nullifying their selective effects (at least in the Western world) (Flinn & Alexander, 2007). Balancing selection is unlikely to explain any remaining genetic variation associated with traits related to these survival conditions.

However, other environmental factors—mostly those related to social cooperation and competition—can reach equilibrium states that allow for balancing selection even in the face of rapid modern cultural development: In every population, there will always be some niches for cheaters and co-operators (related to the personality traits of psychopathy and agreeableness), for risk-takers and hesitators (related to sensation seeking), for liberal and conservative attitudes (related to openness to experiences), for long-term planners and short-term opportunity seekers (related to conscientiousness), for monogamous and promiscuous individuals (related to sociosexuality and extraversion), for aggressive hawks and peaceful doves (related to extraversion and agreeableness), and many more (Denissen & Penke, 2008a; Penke et al., 2007a,b). However, the sizes of the niches for these traits might differ substantially across populations and even subpopulations. How well an individual with a genetic predisposition for any of these traits will fare in a certain population will depend on the traits of all the other individuals in the population (i.e., frequency-, density-, and competition-dependent selection), as well as on the existence of social institutions that support or constrain a certain social strategy and what is demanded by the distribution of resources and other environmental factors. As a consequence, the prevalence and success rates of different strategies and the traits that support them will differ between populations, but only quantitatively (i.e., in terms of allele frequencies, see Kidd, 2009), not qualitatively (i.e., in terms of the genetic architecture of the traits, as would be expected for recent selective sweeps).

There is another reason that the application of balancing selection to human genetic variance is special—and might be especially fruitful: Unlike crops that are planted on a field or lab mice that are kept under strictly controlled conditions, humans are not predestined to live in particular environments. They seek out their preferred conditions and adjust their surroundings to their own needs, which reflect their individual traits. In different disciplines this human tendency (or slight variations thereof) is known by different names: Niche construction (Laland & Brown, 2006), genotype-specific habitat selection (Hedrick, 1990), active gene-environment correlation (Plomin
et al., 2008), experience-producing drives (Bouchard, Lykken, Tellegen, & McGue, 1996), or simply personal freedom. But no matter what it is called, it has the same effect: Humans try to expose themselves as well as they can to the selection pressures that suit their traits best. Sociable people are more likely to move to densely populated cities (Jokela, Elovainio, Kivimäki, & Keltikangas-Järvinen, 2008), and cheaters might as well, in order to take advantage of the greater anonymity. Risk-takers will choose to become high-frequency financial traders and free-time sky surfers instead of accountants and lapidarists (e.g. Ozer & Benet-Martínez, 2006). Liberals as well as promiscuous people will shun conservative religious communities, and anxious individuals will sign more insurance contracts.

Of course, peoples’ abilities to influence the world they are living in will always be limited to some degree by environmental constraints and conflicting interests of other individuals. Cheaters may try, but people usually do not want to be exploited and might even care to punish their attempts (Boyd et al., 2003). Jobs and family situations might limit peoples’ chances to migrate to their favorite environments. Preferentially promiscuous people with low mate values might learn that they are better off in long-term relationships (Penke & Denissen, 2008). So, just as individuals cannot adjust themselves perfectly to their environment despite their conditional adaptations, they are also not able to select or adjust their environment perfectly to fit their traits. From an evolutionary genetic perspective, this means that as long as fitness-relevant trait differences exist, people will try to expose themselves to selection pressures that most favor their particular traits, though they may not always succeed. Effectively, the human tendency to strive for, but limited ability to permanently reach, optimal conditions for themselves exaggerates balanced selection pressures and it might thus help to generate the equilibrium state that is necessary for maintaining genetic variation in human traits by balancing selection (see Hedrick, 1986, 2006).

Individual differences that are heritable because their genetic foundations are under balancing selection can be distinguished from other traits in that they may have positive or negative effects on fitness-relevant life outcomes such as mating success, fertility, health, and longevity, depending on the environment in which they are expressed. Traits under balancing selection will also show a distinctive genetic make-up, with a limited number of genetic variants affecting the trait that have sizable phenotypic effects and intermediate frequencies in the population (neither extremely high nor extremely low). While more data on phenotypic and genetic relations are still needed, the current evidence suggests balancing selection as a plausible mechanism for the maintenance of genetic variance in personality traits like those hierarchically organized in the Big Five personality trait taxonomy (John, Naumann, & Soto, 2008), but not in general intelligence (e.g., roughly IQ) (for a detailed discussion, see Penke et al., 2007a,b). However, balancing selection might be a viable mechanism for genetic variance in lower-order dimensions of cognitive abilities after controlling for general intelligence, like the verbal-mental rotation and focus-diffusion dimensions identified by Johnson and Bouchard (2007; Johnson, Jung, Colom, & Haier, 2008). Similarly, balancing selection might not be a viable explanation for genetic variation in overall mental health, physical health, or physical attractiveness (Keller & Møller; Fink & Penton-Voak, 2002)—all traits with exclusively positive effects on fitness—but it might be for a potential psychosis-autism continuum (Crespi & Badcock, 2008). That is, balancing selection could explain the genetic variance that leads to different phenotypic expressions of a general liability for mental disorders in different individuals, but not the genetic variation in the general liability itself (Yeo, Gangestad, & Thoma,
It might also explain aspects of physical attractiveness that fall along a masculinity-femininity continuum (Penton-Voak, Jacobson, & Trivers, 2004), but not general differences in overall physical attractiveness. (Note that the masculinity-femininity continuum is distinct from the categorical sexual dimorphism discussed earlier—both sex morphs vary along this dimensions, though their optimal levels differ.) In contrast, the best explanation for the maintenance of genetic variation in general intelligence, health, and attractiveness is most plausibly a different one, which I will discuss next.

**Maintaining the Adaptive Design: Mutation-Selection Balance**

When genomes are copied to be transferred from one generation to the next, replication errors can occur. These errors are called mutations (or sometimes ‘derived alleles,’ as opposed to ‘ancestral alleles’), and they are the ultimate source of genetic variation among individuals. Mutations can occur in different forms, from point mutations of single nucleotides to copy number variations of parts of a gene to larger chromosomal aberrations (Frazer, Murray, Schork, & Topol, 2009). Despite sophisticated DNA repair mechanisms (Aguilera & Gomez-Gonzalez, 2008), new mutations are not uncommon in humans: Estimates indicate about 1.67 non-neutral mutations per individual per generation (Keightley & Gaffney, 2003), or a risk of about 80% to be born with at least one new, functional mutation (Keller, 2007). Only very rarely will these new mutations be advantageous and potentially favored by selection in selective sweeps (Eyre-Walker & Keightley, 2007). Most of the time, they will interfere with the adaptive design and thus have detrimental effects on fitness. Purifying selection will work against these harmful mutations, but its ability to eliminate them depends on the fitness effects that the mutations have. Mutations with strong effects and high penetrance, like those involved in many single-gene Mendelian disorders, can be eliminated quickly (sometimes in one generation, e.g. if they cause infertility or death before reproduction), but those with weaker effects and lower penetrance (‘recessive genes’) can be passed on from generation to generation and stay in populations for long periods (for example for an average of 10 generations, affecting a total of about 100 people, if the mutation reduces fitness by 1%, see Garcia-Dorado, Caballero, & Crow, 2003).

It follows that everyone carries a load of mildly harmful mutations. This mutation load is mostly inherited by offspring from parents, but a few new mutations arise in each generation. Thus, each particular mutation will be eliminated by selection eventually, but at the same time new mutations will arise, leading to an equilibrium state called mutation-selection balance. According to very conservative estimates, the average number of mildly harmful mutations carried by individual humans is about 500 (Fay, Wyckoff, & Wu, 2001; Sunyaev et al., 2001). This means that in all of us, some parts of our adaptive design are somewhat disrupted by mutations, but we differ in the number and the location of these disruptions. Mutation loads may account for a substantial portion of genetic variance in human traits, especially if the traits are dependent on many genes, which gives the traits a large mutational target size (Houle, 1998). Except for some mutational hotspots where greater variability is usually adaptive (e.g. for co-evolutionary arms races with pathogens), mutations occur randomly across all of the genetic loci that contribute to a trait’s mutational target size. It is very unlikely that any of these harmful mutations will ever reach an intermediate prevalence rate in the face of selection working against them (Turelli & Barton, 2004). The
mutations that underlie the genetic variance of traits with large mutational target sizes will thus
be numerous, but individually rare, evolutionarily transient, and with small effects on the traits
they affect.

The trait with the largest mutational target-size is, of course, fitness itself, because it is influenced
by all selectively non-neutral parts of the genome, which make up the adaptive design (Houle et
al., 1994). Thus, it can be assumed all mildly harmful mutations in the genome affect fitness and
thus that a large proportion of the genetic variance in fitness is maintained by mutation-selection
balance. A similar assumption can be made for complex traits that reflect the overall condition of
larger parts of the body and brain, and that have a strong, unidirectional relationship to fitness
outcomes like survival and reproductive success. For example, Keller and Miller (2006) made a
strong case that common psychopathologies like schizophrenia, bipolar disorder, and autism fall
in this category. In line with their hypothesis that these disorders are under mutation-selection
balance, a whole series of recent studies has found evidence for the involvement of many rare
 genetic variants in schizophrenia (Stefansson et al., 2008; Stone et al., 2008; Walsh et al., 2008;
Xu et al., 2008) and autism (Kumar et al., 2008; Morrow et al., 2008; Sebat et al., 2007; Weiss et
al., 2008; see also Abrahams & Geschwind, 2008). Indeed, it is plausible that a high load of rare
mutations results in a general susceptibility for psychopathologies, and whether and how this
predisposition gets expressed in specific clinical symptoms depends on other genetically
influenced traits and environmental factors (Penke et al., 2007a; Yeo, Gangestad, & Thoma,
2007; see also Crespi & Badcock, 2008). Similarly, it has been proposed that mutation-selection
balance explains the standing genetic variation in general intelligence (Miller, 2000), a
hypothesis that is in line with the existing phenotypic and genetic evidence (Penke et al., 2007a;
Deary, Penke, & Johnson, 2010). It is also possible that individual differences in mutation load
play a role in general health differences (Thornhill & Möller, 1997).

Finally, it has been argued that sexually attractive traits evolve to become dependent on large
parts of the genome through an evolutionary process
called ‘genic capture,’ which effectively makes these traits more reliable fitness indicators by
making them dependent on the overall condition of the individual, because only highly fit
individuals in good condition can afford to display these traits (Rowe & Houle, 1996; Tomkins et
al., 2004). Virtually all modern models of sexual selection now assume that sexually attractive
traits reflect mutation loads (Kokko, Brooks, Jennions, & Morley, 2003). Indeed, sexual selection
seems to be one of the most important evolutionary mechanisms that counteract harmful
mutations, at least in fruit flies (Sharp & Agrawal, 2008), but likely also in humans (Miller,
2000).

To summarize, a balance between rare, mildly harmful mutations and purifying selection is a
plausible evolutionary mechanism to explain genetic variation in broad human traits that are
influenced by large parts of the genome and thus large parts of the adaptive design. Certain
psychopathologies and general intelligence might qualify as such traits because they reflect the
overall functionality or system integrity of the brain, and general health and sexual attractiveness
might qualify because they reflect the overall condition of the body.

**Tying it all Together: a Life History Perspective on Sources of Individual Differences**

At the beginning of this chapter, I introduced life history theory as the most frequently used
framework for evolutionary approaches to individual differences. According to life history
theory, individual differences exist as manifestations of different strategies for allocating efforts to various fitness-related tasks over the lifespan. Except for neutral genetic variation and certain environmental influences with no effect on evolutionary fitness whatsoever, all other sources of individual differences that I have discussed in this chapter can be related to life history theory. Most of them relate to how individuals reach different strategic life history decisions for effort allocation, while one (mutation-selection balance) relates to how much effort individuals have available to allocate.

Universal, sexually dimorphic and conditional adaptations, as well as genetic variants under recent selective sweeps or balancing selection all have in common that they will contribute to the adaptiveness of strategic life history decisions as long as they are expressed in the right environment. For example, the basic motives to pursue life-history tasks such as mating, raising children, or helping kin can be seen as universal adaptations. These motives assure that people do not behave completely randomly over their lifespans, but instead are concerned with tasks that are necessary for successful propagation of their genes (Tooby, Cosmides & Barrett, 2003). However, preferences and desires regarding resource allocation to one specific task over another (like seeking and courting new potential mates vs. retaining a single mate) likely evolved to differ between the sexes (i.e., sexually dimorphic adaptations). Conditional adaptations allow for further systematic adjustments of allocation decisions to immediate environmental stimuli (e.g., the presence of babies, competitors, or potential mates—adaptive conditional adjustments) or developmental environments (e.g., faster pubertal development after experiencing chronic childhood stress—adaptive phenotypic plasticities). Furthermore, all these motives, preferences, desires, and other adaptations that support adaptive allocation decisions can differ to some degree in their strengths, activation thresholds, sensitivities, reactivities, or other parameters. It is very likely that most individual differences in these parameters are influenced by genetic differences, and as soon as a certain parameter setting leads to more adaptive effort allocations throughout the lifespan, its underlying genetic variants come under positive selection. From then on, whether these genetic variants remain adaptive and eventually become fixed (i.e., a selective sweep) depends on the stability of the relevant environmental circumstances. If the environment changes so that different parameter settings are more adaptive at different times or within different environmental niches, balancing selection may be operative.

In fact, environmental stability is the biggest determinant of the degree to which the various sources of individual differences discussed in this chapter are able to make contributions to the adaptiveness of life history decisions. These sources can be arranged along a continuum of environmental stability (Figure 9.1, see also Penke, 2009): When relevant environmental
aspects are stable over tens of thousands of years or longer, organisms can evolve universal adaptations that develop reliably in every individual every generation (or at least in every individual of the same sex, if the adaptive challenges are sex-specific). In this case, selective pressures have been stable for long enough to fix genetic variants and to allow for the gradual evolution of complex adaptations.

When the environment is less stable, some genetic variants that affect parameters of adaptations may not have had enough time to become fixed so that we might observe them as recent selective sweeps. Even less stable and homogenous environments might prevent certain genetic variants becoming fixed for very long periods because the selection pressures that the different environments exert on them are balanced. Environmental changes that occur over periods that are miniscule on an evolutionary scale—a few generations or even within the lifetimes of individuals—cannot have noticeable effects on the frequencies of genetic variants. However, if the environmental changes are recurrent over evolutionary times and fitness-relevant, conditional adaptations might still evolve: Adaptive developmental plasticities if the environmental changes recur across generations and adaptive conditional adjustments if they recur within generations.

Put differently, long-term stable environments allow for the evolution of universal adaptations that guide strategic life history decisions, but individual parameters of these adaptations (like trait sizes, sensitivities, thresholds, or strength of responses) can be adjusted to more transient environmental conditions by changes in the frequencies of relevant genetic variants, or such parameters of adaptations can be adjusted by purely environmental means (i.e., without primary involvement of genetic differences) if adaptations have evolved sensitivities to react to recurrent adaptive challenges (such as drought). Taken together, all these different sources of individual differences support the adaptive allocation of life efforts.

It should be noted that not all environmental factors will eventually elicit adaptive responses like the ones discussed here. Some factors will be irrelevant to fitness and merely add noise to the environmental cues that activate conditional adaptations (thus setting an upper limit on their maximal effectiveness—an example would be when mate choice preferences get distorted by arbitrary fashion trends), or possibly fix currently neutral genetic variants just by chance, which otherwise might have become the subject of selective sweeps or balancing selection in future environments (as happens with genetic drift). Other environmental factors can be fitness-relevant (sometimes highly so), but organisms are unable to react adaptively, either because
environmental factors change too rapidly (as in co-evolutionary arms races between pathogens and their hosts), or because no genetic variants have any adaptive advantage (for example against toxins or radiation), or

because the misfit between the existing adaptive design and novel environmental factors is simply too great (such as when an evolutionarily sudden abundance of food causes evolutionarily selected preferences for high-caloric food to become maladaptive). These environmental factors contribute to the fact that, despite all the sources of adaptive individual differences, people’s strategic life history decisions will never be perfect.

A different source of individual differences relevant for life history theory is the individual loads of mildly harmful mutations under mutation-selection balance that people carry in their genomes. Mutation loads represent idiosyncratic collections of random disruptions of any of the universal, sex-specific, and conditional adaptations that constitute the adaptive design (see Figure 9.1). Thus, mutations loads do not reflect very well how much any specific adaptation that steers a certain strategic life history decision is impeded by mutations. Instead, mutation loads reflect the overall genetic fitness or genetic quality (Penke et al., 2007a), an important determinant of an organism’s ability to develop according to its evolved genetic blueprint and despite environmental disturbances (i.e., developmental stability—Polak, 2003), to maintain its functional integrity over the lifespan (i.e., system integrity—Batty et al., 2007), and to secure resources from the environment that increase its competitiveness and mate value (i.e., condition—Tomkins et al., 2004). The role of overall genetic quality in life history theory is best understood as individual differences in how much effort is available for allocations between different life history tasks. Genetic quality sets upper limits for the quality of fitness indicators, maximal longevity, or the amount of investment people can make in offspring and kin. Of course, individuals of better genetic quality (and consequently developmental stability, system integrity, and condition) will face the same trade-offs in effort allocation between different tasks faced by individuals of worse genetic quality. However, those of better quality will, on average, be able to invest more in every single task. Thus, individual differences in overall genetic quality can explain why empirical studies usually find positive correlations between different life history traits like growth rate, fertility, and longevity, even though trade-offs in strategic life history decisions would predict negative correlations under the assumption that efforts are finite and allocation of more effort to one task means less for another (Tomkins et al., 2004).

**Future Challenges for an Evolutionary Psychology of Individual Differences**

In this chapter, I took an evolutionary perspective on sources of individual differences, including sex-specific and different forms of conditional adaptations, recent selective sweeps, balancing selection, mutation-selection balance,

neutral genetic variation, and non-adaptive phenotypic plasticity. These different sources of individual differences can be distinguished based on their fitness relevance, the degree and pattern of environmental stability that they require to be adaptive, the genetic architecture that they can be expected to have, and how they relate to the broader framework of life history theory. These sources can be seen as a rather comprehensive set of theoretical building blocks for evolutionary explanations of individual differences, thus bridging the gap between evolutionary psychology and the study of individual differences.
The individual differences dimensions studied in psychology tend to be derived from descriptive studies rather than underlying biological mechanisms, and thus these dimensions tend to be rather complex phenotypes that likely reflect the interplay of several sources of individual differences. Take for example the trait of sociosexuality. Sociosexuality reflects individual differences in the tendency to engage in short-term sexual relationships. It is closely linked to the strategic life history decision whether to allocate more effort in finding and courting new potential mates or to investing primarily in a single mate and potential offspring (Simpson & Gangestad, 1991; Penke & Asendorpf, 2008). Like every human trait, sociosexuality builds on a system of universal adaptations (like the sex drive and the adult attachment system). Some of the parameters in this system seem to have different settings in the male and female human morphs, leading to rather universal sex differences in, for example, the desire for sexual variety (Schmitt et al., 2003). Furthermore, individual differences in sociosexuality are influenced by adaptive conditional reactions to the environment (Gangestad & Simpson, 2000; Schmitt, 2005), including adaptive conditional adjustments to one’s own genetic and phenotypic quality (Gangestad & Simpson, 2000; Penke et al., 2007), a case of reactive heritability (Tooby & Cosmides, 1990a) which might partly explain its genetic variance. Other parts of the genetic variance in sociosexuality might be shared with personality traits like extraversion (Schmitt, 2004), which appear to be under balancing selection (Penke et al., 2007a).

However, even the genetic variance in those personality traits might in the end not be under balancing selection alone: Extraversion, for example, shows some relation to fluctuating asymmetry (Pound, Penton-Voak, & Brown, 2007), openness to experiences shows robust relationships to general intelligence (DeYoung, Peterson, & Higgins, 2005), and neuroticism relates to various psychopathologies (Saulsman & Page, 2004). All these associations imply certain links to genetic quality and mutation-selection balance, which might also explain the general personality factor that can be extracted because of the systematic overlap of broad personality traits (Rushton, Bons, & Hur, 2008; Rushton & Irwing, 2008; Miller, this volume), though a substantial part of the variance in this factor seems to be due to socially desirable responding, method biases, and other artifacts (Bäckström, Björklund, & Larsson, 2008; McCrae et al., 2008).

A major future task for an evolutionary approach to the study of individual differences will be to identify the different sources of individual differences for any given trait, to disentangle their interplay, and to quantify their relative impact. All not completely selectively neutral dimensions of individual differences must relate to some systems of universal adaptations, but it is important to know which systems these are, which parameters of these systems differ between people, and why they differ. Neuroticism, for example, relates to the sensitivity of people to social rejection from significant others (Denissen & Penke, 2008b), a key parameter in sociometer theory (Leary & Baumeister, 2000), which provides an adaptive explanation for the function of self-esteem as a gauge of social acceptance, and this explanation appears to be universally valid (Denissen, Penke, Schmitt, & van Aken, 2008). Conditional adaptations have been studied quite a bit in evolutionary psychology, but in recent years, cross-cultural studies that related population averages of traits to environmental conditions (Gangestad et al., 2006; Schaller & Murray, 2008; Schmitt, 2005) have proven especially valuable. Some traits appear to be influenced by various conditional adaptations, and for such traits it would be useful to know their relative impact on individual differences, since it would allow inferences about the nature of environmental variance in traits (Penke, 2009). The sensitivity or reactivity of conditional adaptations will differ between...
people due to genetic differences. These gene-environment interactions and transactions might be easier to disentangle by taking a reaction norm perspective on traits, where behaviors of people with different trait levels are systematically mapped to dimensions of relevant environmental factors (Denissen & Penke, 2008a; Pigullici, 2005; Penke et al., 2007a; West-Eberhard, 2003). Such a reaction norm perspective might also be helpful for identifying individual genes that underlie the heritable variance of traits, since gene-environment interactions (along with gene-gene interactions) are seen as some of the major obstacles in molecular genetic studies of quantitative traits (Maher, 2008; Frazer, Murray, Schork, & Topol, 2009). Furthermore, since a genetic variant cannot be under mutation-selection balance and balancing selection at the same time and they will result in quite distinctive genetic architectures (Penke et al., 2007a), it should be a fruitful approach to control for variance components for which there is strong evidence that they are under one selection pressure when looking for genetic variants under another selection pressure. This might be especially useful for genome-wide association studies (GWAS), where genetic markers across the whole genome are used to discover new genetic variants associated with quantitative traits in a purely explorative manner. GWAS are only able to detect effects of genetic variants that are rather common in terms of their population frequency (as expected if the variants are under balancing selection or possibly recent selective sweeps), but it is impossible for them to detect rare variants (as expected under mutation selection balance) (Frazer et al., 2009; McCarthy et al., 2008). So when, for example, searching for common genetic variants for openness to experience or spatial ability (which are supposed to be under balancing selection), it should help to control for the genetic variance shared with general intelligence (which is supposed to be due to rare variants of small effect sizes under mutation-selection balance). Similarly, it might be worthwhile to control for sexual attractiveness when looking for common genes for sociosexuality, or for fluctuating asymmetry or other markers of general condition when looking for genes for extraversion. Techniques are available to directly test genes that might be identified in this process for signatures of balancing selection (Hedrick, 2006). On the other hand, gene and genome re-sequencing studies are starting to become available, which allow us to directly test the impact of rare, small-effect mutations on quantitative traits like intelligence, common psychopathologies, attractiveness, and health (Bentley et al., 2008; Frazer et al., 2009). In addition, several molecular genetic tests already exist that allow the identification of signatures of adaptive evolution and recent selective sweeps (Bamshad & Wooding, 2003; Harris, 2008; Hoffmann & Willi, 2008; Williamson et al., 2007). For all these molecular techniques, the quality of the available data is constantly improving at rapid pace. Though the conclusions that we can currently draw on the genetic architecture and evolutionary history of traits cannot be considered definite in most cases, this will almost certainly change dramatically in the next years.

Taken together, these are exciting times, in which the need for an evolutionary psychology of individual differences is not only realized, but new methods and data from various fields are available for this endeavor. From an evolutionary perspective, lifetime reproductive fitness is the ultimate dimension of individual differences and aside from chance events, it is determined by how people strategically allocate the life effort they have available. Several of the sources of individual differences I discussed in this chapter—sexually dimorphic and conditional adaptations, recent selective sweeps, balancing selection, and mutation-selection balance—will interact to produce the interindividual variance in traits that relate to life history strategies. This
interplay is what we need to understand in order to create an evolutionary psychology of individual differences.

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References
Bäckström, M., Björklund, F., & Larsson, M. R. (2008). Five-factor inventories have a major general factor related to social desirability which can be reduced by framing items neutrally. *Journal of Research in Personality, 43*, 335–344.


Rushton, J. (2004). Placing intelligence into an evolutionary framework or how g fits into the r-K matrix of life-history traits including longevity. Intelligence, 32, 321–328.


Evolutionary Behavioral Genetics

It is a fascinating time to be a researcher interested in human evolution and genetics. Knowledge on molecular genetic variation is growing at a breathtaking pace, placing us in the midst of one of the remarkable revolutions of science—albeit one more akin to the empirically driven atomic revolution of the 20th century than to the theory-driven Darwinian one of the 19th century. In the last five years, researchers have collected data on up to several million of the most common DNA variants on tens of thousands of people. As a result, we know more about human genetics than that of any other animal—fruit fly and nematode worm notwithstanding. While some of this data was collected with the explicit aim to test evolutionary hypotheses, most of it awaits a unifying framework that only evolutionary theory can provide.

The principal goal of this chapter is to critically discuss how this new molecular genetic data, together with family and twin data traditionally used by behavioral geneticists, can be used to test evolutionary questions about the causes of human genetic variation. Our intended audience is the behavioral geneticist interested in evolution or evolutionary psychologist interested in genetics, and our treatment of these methods assumes a basic knowledge of population genetics and evolutionary theory and at least some familiarity with methods used in modern genetic analyses.

Before evaluating the methods that can be used to understand the evolution of human genetic variation, we briefly discuss the theoretical underpinnings of the new field we call evolutionary behavioral genetics. We argue that the central question in evolutionary behavioral genetics is: What evolutionary forces account for the genetic variation observed in human traits? For example, what evolutionary forces account for highly heritable disorders, such as schizophrenia? The fact that schizophrenia is heritable implies that alleles exist in the population that confer risk to the disorder. Why would such alleles exist and persist in the population in the first place? Similar questions could be asked regarding the heritability of any human trait, from extraversion to intelligence to athleticism to height.

Notice that the central question in evolutionary behavioral genetics is a much different one than typically asked by evolutionary psychologists. Whereas evolutionary psychology has typically been concerned with explaining the evolutionary forces that shaped human universals (adaptations), evolutionary behavioral genetics uses the theoretical lens of evolution to understand human variation, and in particular, human genetic variation. As discussed below, the main theoretical tool used by evolutionary psychologists to explain human universals, the theory of natural selection, is rarely the right tool to explain human genetic variation. This is for a very simple reason: while awesome in its ability to craft fine-tuned adaptations, natural selection tends to deplete rather than maintain genetic variation. Thus, investigating the central question in evolutionary behavioral genetics requires the use of a much more diverse, but equally fascinating, theoretical toolkit. These tools are largely drawn from the field of evolutionary genetics, although
the way to apply these tools to modern human genetic data remains very much a work in progress.

**Proximate Causes of Genetic Variation**

Almost every trait studied to date is heritable to some degree. *Heritability* is the ratio of genetic variation to total phenotypic variation, and therefore has a range of zero to one. Genetic variation is caused by differences in the DNA variants, or *alleles*, that people harbor at various locations across their chromosomes. *Alleles* can refer to any number of alternative sequences of base pairs that stretch hundreds or even thousands of base pairs in length. For example, 32% of the population may have the `TATGACCAGCAATC` allele, 15% the `AATGACCAGCAATG` allele, 6% the `TATGACAAGCAATG` allele, etc... Although it is sometimes useful to think of alleles in this way, as being combinations of many base pairs in a row, it is often easier to think of each varying base pair as its own allele. Such single nucleotide alleles are aptly named *single nucleotide polymorphisms* (SNPs). Note that the first, seventh, and fourteenth position of the above sequences are SNPs that vary between individuals. For example, 70% of the population may have a T (the major allele) and 30% an A (the minor allele) at the first SNP of the sequences. Due to the low likelihood of mutation at any single base pair (~$10^{-8}$), almost all SNPs have just two variants.

Over 95% of the 3.2 billion base pairs in the human genome are monomorphic: basically everyone in the population shares the same A, T, C, or G nucleotide at them. Such monomorphic base pairs contribute nothing to the genetic *variation* of any trait—even if critically important to creating universal adaptations. However, an estimated ten million (0.3%) base pairs harbor *common* alleles (by definition, SNPs with minor allele frequencies >1% in the population), and hundreds of millions of base pairs harbor rare alleles (SNPs with minor allele frequencies <1% in the population) (Kryukov, Pennacchio, & Sunyaev, 2007). SNPs are thought to serve as the principal substrate for the heritability of traits, although it has recently become apparent that structural variants, such as deletions and duplications, are much more common than previously thought (Feuk, Carson, & Scherer, 2006), and their role in the heritability of traits may also be significant.

Whereas *alleles* refer to variants in the population, *loci* (singular, *locus*), refer to locations along the genome where alleles may or may not exist. Loci that code for proteins are called *genes*. In colloquial usage, the term *gene* is often used where a geneticist would use the word *allele*, but technically the two terms have different meanings. A gene is a set of instructions for making a protein whereas an allele is one of two or more alternative variants of that set of instructions. While genes have traditionally been a central focus of evolutionists and geneticists, it has recently become apparent that an unknown but potentially large percentage of the genome is functional despite not coding for proteins (Birney et al., 2007). Thus, critiques of evolutionary psychology expressing doubt about how a small number of genes could possibly code for a large number of complex adaptations (Buller & Hardcastle, 2000), even if fundamentally misconceived (Hagen, 2005), should be updated to reflect the fact that a much larger percentage of the genome may be functional than previously thought.
Ultimate Causes of Genetic Variation

A proximate-level understanding of genetic variation does not shed light on the ultimate, or evolutionary, causes of genetic variation. Why does genetic variation exist in the first place? A moment’s consideration will reveal why this question is both fundamental and puzzling, especially when applied to genetic variation in traits related to Darwinian fitness. Consider an allele that affects a trait related to fitness. This allele must have a typical effect on the trait when averaged across all the bodies the allele finds itself in. If this typical effect increases fitness, it should “fixate” (reach 100% prevalence in the population) or by chance go extinct. On the other hand, if this typical effect decreases fitness, it should go extinct or (rarely) by chance reach fixation (Ohta, 1973; S. Wright, 1931). In no case should alleles that increase or decrease fitness on average exist for long at the non-zero frequencies required for them to contribute to genetic variation. Put another way, we should expect little genetic variation and low heritability in traits related to fitness (Fisher, 1930). Contrary to this expectation, the median heritability of fitness-related traits across many animal studies is quite far from zero—about 30% (Roff, 1997)—and several phenotypes thought in humans to lower fitness have heritabilities between 30–80% (Hughes & Burleson, 2000; Keller & Miller, 2006).

Forwarding testable and compelling theories for why heritability exists in fitness-related traits has been a central theme in evolutionary genetics. Given that most phenotypes of interest to psychiatrists and psychologists are probably related to fitness to varying degrees, explaining the genetic variation in fitness-related traits should take us some way toward generating testable hypotheses for the evolutionary existence of genetic variation in human psychological traits. In this section, we briefly discuss four evolutionary mechanisms that can explain the genetic variation in fitness-related traits, each of which leaves different, albeit messy, signatures in the genome. These mechanisms are in no way mutually exclusive: each may be important for different traits and all may simultaneously help to explain the genetic variation of a given trait.

Mutation-selection

Point mutations as well as deletions, duplications, translocations, and inversions are copying errors that occur during DNA replication. Those that arise in non-germline cells can result in diseases such as cancer but are of little interest evolutionarily because they are not transmitted to offspring. Mutations that occur during replication of sperm or egg cells, however, are central to the evolutionary process. Such mutation can be transferred to the fertilized ovum and eventually to every cell in the offspring’s body, including the offspring’s own germline cells and, potentially, any descendents of the offspring. This is what population geneticists mean when they say that mutations are ‘introduced’ into a population. It should be noted that the term mutation refers to the original germline mutation as well as the copies of that mutation that exist in descendents. An observant reader may have noticed that, by this definition, all genetic polymorphisms would be mutations because every genetic polymorphism originally arises as a mutational event. For this reason, we follow the usual convention that the term mutation refers to alleles with minor allele frequencies up to 1% and use the term polymorphism to refer to alleles (e.g., SNPs) with minor allele frequencies greater than 1%.
Most new mutations arise in chromosomal locations that have no phenotypic effect (so-called ‘junk DNA’), but those that do affect the phenotype almost always degrade its tightly coordinated performance, and such mutations are kept at low but calculable frequencies by natural selection (Falconer, 1989). For example, a mutation that decreases fitness by 1% will tend to exist in an average of 100 individuals and persist in a population for about 10 generations in multiple co-existing copies (García-Dorado, Caballero, & Crow, 2003). Thus, such deleterious mutations almost never reach a frequency of 1% where they would be termed polymorphisms. Mutation-selection models describe the equilibrium between new deleterious mutations being introduced into the population and their removal, often tens to hundreds of generations later, by selection.

The importance of mutation-selection in explaining trait variation has long been debated, but many evolutionary geneticists now consider it to be a primary factor in explaining the heritability of fitness-related traits (Charlesworth & Hughes, 1999; Houle, 1998). Although deleterious mutations are rare per locus, hundreds or even thousands of loci can influence complex traits, and so the cumulative number of mutations could be high enough to explain the heritability of complex traits. Indeed, it is likely that every human alive is affected by hundreds to thousands of rare (usually partially recessive) deleterious mutations that individually have minor effects on the phenotype (Fay, Wyckoff, & Wu, 2001). These deleterious mutations must cause maladaptive noise of some sort, but what does such variation look like? Mutations do not simply affect fitness directly—how could they?—rather, they decrease fitness by degrading the proper functioning of adaptations. Figure 10.1 presents the ‘watershed’ model of mutations (Cannon & Keller, 2006; Keller & Miller, 2006), showing how three classes of mutations (triangles) lead to abnormal genetic products, which in turn disrupt ‘upstream’ phenotypes (e.g., neuronal pruning in the dorsal medial amygdala), which in turn disrupt further downstream phenotypes (e.g., a fear conditioning adaptation). The ultimate downstream trait must be some fitness-related trait (e.g., survival to sexual maturation), which captures variation from many upstream traits and therefore represents a large ‘target’ for mutations.
Figure 10.1. Deleterious effects of three types of mutations and extended homozygosity on genes, upstream phenotypes, and downstream phenotypes. Shading represents higher mutational ‘target sizes.’ Certain downstream phenotypes could be mental disorders.

Mutation-selection has been used to help explain why some disorders are both common and heritable despite being profoundly harmful to survival and reproduction (Gangestad & Yeo, 1997; Keller & Miller, 2006; Kryukov et al., 2007; McClellan, Susser, & King, 2007; Penke, Dennison, & Miller, 2007). Mutational models of disorders posit that mutations increase the risk for having adaptations that malfunction. By this view, common disorders are heterogeneous groups of dysfunctions in the thousands of upstream mechanisms affecting normal adaptive functioning. The final common pathways of various constellations of these upstream dysfunctions may increase risk for ‘physical’ disorders (e.g., chronic pain syndrome, fibromyalgia, irritable bowel syndrome, etc.), mental disorders (schizophrenia, bipolar disorder, attention-deficit hyperactivity disorder, autism, mental retardation, etc.) and other fitness-related traits (e.g., low intelligence, low attractiveness, poor athletic ability, etc.) that may appear to be singular diseases or dimensions, but that are highly heterogeneous etiologically.

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Mutation-drift

The primacy of neutral evolution was most famously argued by Kimura (1983), but recent genetic evidence appears to confirm its truth—at least with respect to genetic sites rather than traits (Birney et al., 2007). The vast majority of base pairs across the genome either has no phenotypic effect whatsoever or contributes to variation in traits that have no effect on fitness. Over evolutionary time, the frequencies of alleles at such neutral sites are governed by “mutation-
drift.” This process is exactly the same as mutation-selection except that no allele is ‘preferred’ by natural selection, and therefore alleles ‘drift’ according to pure stochastic chance. The vast majority of new neutral mutations never gain an appreciable frequency in the population and eventually are lost. However, a small minority do become common and may, over a long period (depending on population size), “fixate,” or reach 100% frequency in the population. Of course, at any snapshot of time, given the billions of opportunities, millions of such neutral alleles are at intermediate frequencies. For this reason, the vast majority of common SNPs and other structural variants are thought to be governed by mutation-drift, whereas rare alleles (i.e., mutations) are more likely to be deleterious (Kryukov et al., 2007).

Researchers and tax-payers have invested considerable capital attempting to relate common SNPs to common diseases. However, as noted above, common SNPs are probably common precisely because the vast majority of them have no functional effect and are thus unlikely to be associated with fitness-related phenotypes, including disease (A. F. Wright, Charlesworth, Rudan, Carothers, & Campbell, 2003). Such considerations argue against the long-term success of gene-hunting studies using SNP data (see section on whole-genome association studies). Nevertheless, some alleles affecting modern diseases may be common because they did not have a deleterious effect ancestrally. Some of the heritability for depression, for example, might be explained by alleles that were neutral in ancestral environments but that increase the risk of depression in modern environments. Social isolation is an important risk factor for depression, especially in women (Kendler, Myers, & Prescott, 2005). Humans evolved in small hunter-gatherer societies where social isolation would have been uncommon. Alleles that increase shyness and therefore the risk of social isolation, and hence depression, in modern environments may not have led to social isolation or depression in ancestral environments. Invisible to natural selection, such alleles might have drifted by chance to intermediate frequencies in ancestral environments and might help explain why susceptibility alleles for depression are so common today. This example demonstrates how the mismatch hypothesis (Gluckman & Hanson, 2006) can also help explain the existence of genetic variation in traits seemingly related to fitness.

**Directional (positive or negative) Selection**

Against the backdrop of detrimental mutations that are constantly being purged by natural selection, new mutations occasionally arise that increase fitness. Via natural selection, these beneficial mutations can spread throughout a population, but once they fixate, they cause no genetic variation. For this reason, directional selection does not maintain genetic variation at equilibrium. Nevertheless, at any given time, many alleles are not at equilibrium but rather are rising (due to positive selection) or falling (due to negative selection) in frequency. This increase in beneficial alleles and decrease in formerly beneficial ones can cause a large amount of genetic variation in traits because such alleles are not necessarily rare (loci that house rare alleles tend to contribute less to variation than loci that house common alleles).

The ancestral-susceptibility model (Di Rienzo & Hudson, 2005) proposes that many current risk alleles are common because they were beneficial in ancestral human populations but are now being driven to extinction due to rapid changes in human environments. Indeed, molecular evidence suggests that human evolution has recently sped up, as rates of newly arisen SNPs replacing old ones (reflecting natural selection) are over 100 times higher in the last 10,000 years relative to the rates which characterized most of human evolution (Hawks, Wang, Cochran,
Harpending, & Moyzis, 2007). In addition, several risk alleles for common diseases, such as Alzheimer’s and hypertension, are ancestral (Di Rienzo & Hudson, 2005), and Lo et al. (2007) found that schizophrenia risk alleles in the GABA-A receptor β2 gene have been under recent negative selection. Thus, there is evidence that some of the genetic variation underlying common disorders is due to out-of-equilibrium alleles that are rising or lowering in frequency due to natural selection, although it remains unknown how general this explanation is for extant genetic variation.

**Balancing Selection**

Balancing selection occurs when two or more alternative alleles at a locus are actively maintained in a population by natural selection. This generally occurs when the fitness of an allele increases as it becomes rarer. Heterozygote advantage—where heterozygote individuals at some locus have higher fitness

than either homozygote—is a special case of this. For example, heterozygous individuals at the β-hemoglobin locus in equatorial Africa are protected against malaria, whereas homozygous individuals are either vulnerable to malaria or at risk of sickle-cell anemia (Allison, 1954). Each allele—and sickle-cell anemia—is maintained: if one allele becomes infrequent by chance, it more often finds itself paired with the opposite allele, increasing its fitness and frequency.

Antagonistic pleiotropy is another process that might lead to a balanced polymorphism. In this process, pleiotropic genes (which affect more than one trait) have a fitness-boosting effect on one trait but a fitness-lowering effect on another, which could potentially lead to the maintenance of two or more alleles at a locus and hence genetic variation within populations. For example, alleles that enhance reproductive fitness but reduce longevity, and vice-versa, have been found in fruit flies and nematode worms (for a review, see Leroi et al., 2005).

In principle, balancing selection can maintain genetic polymorphisms indefinitely. However, several theoretical and empirical studies in the last twenty years seem to suggest that balancing selection is evolutionarily transient, causing genetic variation for only short periods of time. With respect to antagonistic pleiotropy, either the positive effect or negative effect of an allele is generally stronger, leading to fixation or extinction of the highest fitness allele and no balanced polymorphisms (Curtisinger, Service, & Prout, 1994; Prout, 1999). For example, chance unequal crossovers during meiosis near loci governed by heterozygote advantage will result in both alleles residing on the same chromosomal arm. Such a ‘heterozygote’ allele will be strongly selected for and will therefore destroy the balancing selection. Essentially the genome tends to eventually re-arrange itself if fitness favors such an outcome, destroying this delicate balance created by the heterozygote advantage. Moreover, a recent whole-genome scan designed to detect signatures of ancient balancing selection found no loci governed by it aside from those few already known to exist (Bubb et al., 2006). Nevertheless, recent balancing selection (such as that responsible for sickle-cell anemia) may be more common, and may help explain genetic variation in fitness-related traits. Mealey (1995), for example, made a convincing case that antisocial personality disorder is a recently arisen psychological morph maintained in this way, and Tooby and Cosmides have theorized that the bulk of human behavioral genetic variation is a side effect of pathogen-driven balancing selection favoring biochemical diversity (Tooby, 1982; Tooby & Cosmides, 1990). All such balancing selection theories predict that alleles underlying traits maintained in this way will be common and therefore detectable using current approaches (section on whole-genome association studies). Thus, the
lack of success in gene hunting might indicate that balancing selection is a rare process in the human genome.

**Testing the Evolutionary Mechanisms Accounting for Genetic Variation**

In this section, we critically discuss several methods that evolutionary researchers might use to test hypotheses on which evolutionary mechanisms cause genetic variation in traits of interest. These methods include traditional behavioral genetic approaches (e.g., twin analyses) as well as approaches based on newer molecular data. We stress two things. First, our own ideas regarding how to test the evolutionary mechanisms above and the strengths/weaknesses of these methods remain a work in progress. Second, the conclusions that can be drawn from the methods we review fall short of the type of “strong inference” (Platt, 1964) that allows researchers to definitively exclude one or more alternative explanations. This is partly due to the non-experimental nature of human genetic data, and partly because each evolutionary mechanism discussed above (previous section) could simultaneously contribute to the genetic variation of a given trait. The true challenge in the years to come will be weighing findings appropriately in order to understand the degrees to which different mechanisms account for the genetic variation underlying different traits.

**Genetic Correlations**

Traditional behavioral genetic approaches use “genetically informative” relatives such as twins and adoptees to understand the roles of genetic and environmental factors in behavioral variation. Also exciting, and a major interest in behavioral genetics currently, is elucidating genetic correlations between traits (Kendler, Prescott, Myers, & Neale, 2003), which occur when the same genes affect two or more traits. Such shared genetic effects (called pleiotropy) induce trait correlations that are genetic in origin, and can be discerned by comparing, for example, the cross-trait identical twin correlation to the cross-trait fraternal twin correlation. A high ratio of identical to fraternal cross-trait correlations suggests that the correlation is partly or wholly genetic in origin.

Genetic correlations can provide clues about the evolutionary mechanisms responsible for traits’ genetic variation. Mutation-selection predicts that traits related to fitness will demonstrate positive (low-fitness end with low-fitness end) genetic correlations with each other. This is because mutation-selection keeps deleterious alleles at very low frequencies where they individually contribute little to a trait’s genetic variation; only the cumulative effect of very many mutations—and thus very many genes—could maintain substantial genetic variation in a trait. If mutation-selection maintains substantial genetic variation in two fitness-related traits, simple probability dictates that the traits share many genes in common (Roff, 1997), and will therefore show a positive genetic correlation (high-fitness end of trait 1 with high-fitness end of trait 2).

Fitness-related traits whose genetic variation is explained by antagonistic pleiotropy, on the other hand, should show negative genetic correlations (high-fitness end of trait 1 with low-fitness end of trait 2). For example, alleles increasing fitness via increased creativity might also decrease fitness via increased risk for schizophrenia (Nettle & Clegg, 2006). Although theoretical treatments have cast doubt on the ability of antagonistic pleiotropy to maintain genetic variance indefinitely (see section on balancing selection), mutations with antagonistic effects on two
fitness-related traits will tend to reach higher frequencies and persist for longer than
unconditionally deleterious mutations. Thus, antagonistic pleiotropy may be an important
contributor to variation in fitness-related traits, even if it does not maintain balanced polymorphisms indefinitely.

There is evidence in the animal literature supporting the idea that both mutation-selection and
antagonistic pleiotropy play roles in genetic variation of fitness-related traits in nature. Fitness-
related traits (e.g., survival to sexual maturity) do tend to show less positive genetic correlations
than do other types of traits (e.g., morphological measurements), consistent with antagonistic
pleiotropy, but most genetic correlations (~ 60%) between such traits are nevertheless positive, consistent with some degree of mutational variation (Roff, 1997). In humans, there is wide
agreement that mental disorders typically show positive genetic correlations (reviewed in Keller,
2008), consistent with a mutational role. The mutation-selection model would similarly predict
that intelligence, athleticism, physical health, facial and bodily attractiveness, and any other trait
related to fitness/mate value will show positive genetic correlations; the antagonistic pleiotropy
model predicts that they will show negative genetic correlations (Miller, 2000). With a few
exceptions (Arden, Gottfredson, Miller, & Pierce, 2009), such studies have yet to be done. This
appears set to change. In collaboration with the Genetic Epidemiology unit of the Queensland
Institute for Medical Research, a consortium of evolutionary psychologists and behavioral
geneticists is currently collecting evolutionarily relevant data in a large community twin sample
in order to assess genetic correlations among several ostensibly fitness-related traits.

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Relative Degree of Additive and Non-additive Genetic Variation

Non-additive genetic variation is caused by statistical interactions between alleles at either the
same locus (dominance) or different loci (epistasis). If the combined effect of two or more alleles
is different than what would be predicted from adding the effects of each one individually, then
some degree of non-additive genetic variation will result. Traits most related to fitness have a
higher ratio of non-additive to additive genetic variation (around 1) than traits under less intense
selection (around 3.3) in non-human animals (Crnokrak & Roff, 1995). This is consistent with
theoretical predictions, because selection depletes additive genetic variation faster than non-
additive genetic variation (Fisher, 1930; Merilä & Sheldon, 1999). Psychoticism, neuroticism,
extraversion, somatization, and panic/phobia show relatively high levels, and major depression
shows modest levels, of non-additive genetic variation (reviewed in Coventry & Keller, 2005),
which is consistent with the hypothesis that these traits have been subject to natural selection
ancestrally.

Four important caveats should be kept in mind in using the level of non-additive genetic variance
as a standard of evidence for inferring the intensity of selection:

1. There is high variation in estimates of the non-additive:additive ratios for fitness-related traits
   in nature, which typically fall between 25 and 7.5 (Crnokrak & Roff, 1995). Thus, a single
   estimate of this ratio in humans is not compelling evidence for inferring strength of selection.

2. Non-additive genetic variance can be seriously underestimated by twin and twin-plus-sibling
designs (Keller & Coventry, 2005), and even those designs that include the necessary relative
types (e.g., parents) to estimate it tend to estimate it imprecisely (Medland & Keller, 2009).
   Because of this, we have surprisingly little understanding of the true levels of non-additive
genetic variation underlying phenotypes known to be highly heritable, such as IQ and most
1. There is high variation in estimates of the non-additive:additive ratios for fitness-related traits in nature, which typically fall between 2.5 and 7.5 (Crnokrak & Roff, 1995). Thus, a single estimate of this ratio in humans is not compelling evidence for inferring strength of selection.

2. Mental disorders.

3. As with any statistical interaction, non-additive genetic effects are sensitive to scale: a change in scale of a purely arbitrary and neutral character (such as skin conductance) can cause the appearance or disappearance of non-additivity (see Lykken, 2006). Such sensitivity to scale is problematic for most psychological traits, which tend to be measured on arbitrary scales.

4. The three caveats above highlight that the level of non-additive genetic variation provides weak evidence as to the strength of selection on traits. It provides even weaker information on which evolutionary mechanism maintains a trait’s genetic variation. High levels of non-additive genetic variation can arise from either mutation-selection or certain types of balancing selection (e.g., heterozygote advantage), but other types of balancing selection (e.g., frequency dependent selection) can lead to high levels of additive genetic variation (Merilä & Sheldon, 1999).

In conclusion, high levels of non-additive genetic variation provide weak evidence that a given trait has been under some type of natural selection, but most estimates that exist must be taken with a grain of salt, and even good estimates cannot elucidate what mechanism maintained the trait’s genetic variation.

**Inbreeding Depression**

Inbreeding depression refers to a decline in the value of traits among offspring of genetic relatives. The first person to study the phenomenon scientifically was Darwin (1868, 1876), who, with characteristic insight, grew concerned that the poor health of his children might be due to his marriage to his first cousin, Emma Wedgwood (Bowlby, 1992). A century of subsequent research on domesticated and wild animals consistently corroborated what Darwin suspected: Inbreeding leads to lower values on fitness-related traits (Crnokrak & Roff, 1999). Because inbreeding depression is stronger among traits that have been under directional selection ancestrally, the degree to which a trait is affected by inbreeding can be used as a rough gauge for how strongly selection acted on the genes influencing that trait over evolutionary time (DeRose & Roff, 1999).

Inbreeding increases homozygosity (aa or AA rather than Aa), which may lower fitness by decreasing the probability of advantageous heterozygous alleles maintained by balancing selection or by exposing the full deleterious effects of partially recessive mutations (Figure 10.1) thought to be sprinkled throughout every genome (S. Wright, 1977). Some evidence supports the latter mutational mechanism (Charlesworth & Charlesworth, 1999; Crow, 1999). For example, if the mutational mechanism is correct, populations that have gone through many generations of inbreeding should have higher fitness once they outbreed because partially recessive mutations can be exposed and purged from the population during the inbreeding period. An increase in fitness following inbreeding has occurred in several experimental organisms (Barrett & Charlesworth, 1991; Strong, 1978; Templeton & Read, 1983), although for certain traits in *Drosophila* (and ostensibly in other species), balancing selection also appears to play some role in inbreeding depression (Charlesworth & Hughes, 1999).
Traditionally, inbreeding depression studies have used pedigree information. Such studies in humans have found evidence that inbreeding reduces IQ (Afzal, 1988; Morton, 1979) and general health (Rudan et al., 2006), and increases the probability of learning disabilities (Rudan et al., 2002), osteoporosis (Rudan et al., 2004), schizophrenia (although see Saugstad & Ødegard, 1986), cancer, depression, gout, peptic ulcers, and epilepsy (Rudan et al., 2003). A more direct method of studying inbreeding depression uses whole-genome SNP data to quantify how homozygous individuals’ genomes are (e.g., Lencz et al., 2007). Such measures of genomic inbreeding are quantified as the percent of each person’s genome that exists in long stretches, or runs, of homozygosity. A given run of homozygosity probably reflects the pairing of two stretches of a chromosome that are “identical by descent,” meaning that the two chromosomal stretches making up the run come from the same common ancestor at some point back in the family tree. Such “identical by descent” stretches guarantee that everything (or nearly so) in the run of homozygosity, including rare mutations that existed in that stretch of the common ancestor’s chromosome, is also homozygous.

Genomic inbreeding measures are preferable to those based on pedigree information for three reasons. First, individuals who breed with known relatives are probably not representative of the general population, which introduces an alternative explanation to inbreeding findings based on pedigree information. Genomic measures of homozygosity, on the other hand, elucidate even distant and unintended inbreeding. Second, self-reported pedigree information can be inaccurate. Third, even when pedigree information is accurately reported, the true level of homozygosity in inbred offspring is unknown. For example, the percent of the genome in homozygous runs among progeny of first cousins averages 6.25%, but the 95% confidence interval around this is 1.4% to 11.0% (Carothers et al., 2006).

Few results exist as yet on how runs of homozygosity are associated with human traits. Lencz et al. (2007) found that schizophrenia cases have more runs of homozygosity in their genomes than controls, but their study only focused on locations were multiple runs were observed in the sample, limiting the study’s generalizability. Our own lab is currently in the early stages of investigating the effect of runs of homozygosity on IQ and schizophrenia, and we hope to present results of these investigations in the near future.

**Direct Assessments of Mutational Loads**

There are several indirect methods for investigating whether mutations (and thus mutation-selection) contribute to trait variation. These include investigating the effects of brain trauma, ionizing radiation, parental inbreeding, and paternal age on phenotypes of interest (for a full explanation for why these methods provide evidence for a mutational model, see Keller & Miller, 2006). However, one of the most exciting developments in genetics over the last few years has been the ability for researchers to directly assess certain types of mutations. At first blush, such direct assessment seems easy: just measure all the base pairs in a genome and note where rare or unique single base pair polymorphisms (point mutations) exist. An overall ‘mutational load’ could then be related to phenotypes of interest. For technical reasons, however, we are still 3–5 years away from being able to measure all 3.2 billion base pairs in large genome-wide scans, and those base pairs that are currently being measured focus on common SNPs rather than rare mutations. Thus, detection of single base pair mutations in large samples is not yet possible.
Nevertheless, current technology does allow measurement of rare deletion and duplication mutations. By combining intensity data (the strength of the signal for each allele at a SNP) across many SNPs in a row, researchers can infer whether a deletion (indicated by low intensity and apparent homozygosity across contiguous SNPs) or a duplication (indicated by high intensity and normal heterozygosity across contiguous SNPs) exists at a particular genetic location (see, e.g., Korn et al., 2008). Importantly, even though common SNPs are used to assay them, this technique allows detection of both common and rare (i.e., mutational) deletions and duplications. Using such a technology, it appears that deletion and duplication mutations are important contributors to variation in HDL cholesterol levels (Cohen et al., 2004), autism (Sebat et al., 2007), Parkinson’s disease (Simon-Sanchez et al., 2008), mental retardation (reviewed in Lee & Lupski, 2006), Tourette’s syndrome (Lawson-Yuen, Saldivar, Sommer, & Picker, 2008), and schizophrenia (Walsh et al., 2008). Evidence on the effects of rare deletions and duplications on bipolar disorder, obsessive-compulsive disorder, major depression, anxiety disorder, and other psychiatric and non-psychiatric conditions will probably be released within the next two years.

There are two important conclusions to take away from the studied effects of deletions and duplications on illness. First, deletion and duplication mutations play important roles in the etiology of these disorders, but it is difficult to put a quantitative estimate on how big of a role such mutations play. Presumably, once all classes of mutations can be accurately measured, investigators will be able to estimate the total contribution of mutations to trait heritability. Second, over the years, several evolutionary thinkers have postulated that disorders such as schizophrenia (Polimeni & Reiss, 2002) and autism (Gernsbacher, Dawson, & Mottron, 2006) might themselves be heritable, complex, adaptations maintained in the population by balancing selection. Finding that these same disorders are influenced by mutations to any degree rules against such hypotheses (Keller & Miller, 2006). This is because mutations disrupt complex adaptations; it is highly improbable that deleterious mutations or other developmental insults would lead to full-fledged, complex adaptations by chance.

Whole-genome Association Studies

Alleles maintained by balancing selection should be relatively common (minor allele frequencies \(>1\%\)) in the population at each locus (Barton & Keightley, 2002; Mani, Clarke, & Shelton, 1990). This prediction is true even if the balancing selection maintains one functional allele and any one of many potential loss-of-function alleles (Reich & Lander, 2001). Mutation-selection models, on the other hand, predict the opposite: One very common (most adaptive) allele at a given locus and many (hundreds or even thousands) extremely rare, lineage specific mutations in the population at that locus. As noted above (section on mutation-drift), the success of whole-genome association studies depends on common alleles being associated with traits of interest. For this reason, whole-genome association studies should be more successful for traits whose variation is maintained by balancing selection, directional selection, and mutation-drift than on traits whose variation is maintained by mutation-selection.

Whole-genome association studies have been successful at finding alleles that explain significant variation (e.g., cumulative \(\geq 5\%\)) for certain traits: lung cancer (Spinola et al., 2006), breast cancer (Easton et al., 2007), prostate cancer (Yeager et al., 2007), heart disease (Samani et al., 2007), macular degeneration (Li et al., 2006), nicotine dependence (Bierut et al., 2007), type 2 diabetes (Scott et al., 2007), and obesity (Herbert et al., 2006). These studies show that genome-wide
association studies work as advertised when common alleles are responsible for some portion of genetic variation. However, despite great investment in treasure and effort, similar success has not occurred for many other disorders of interest, including any psychiatric disorder. In a paper that came out before the results of whole-genome association studies were known (Keller & Miller, 2006), we predicted that if the genetic variation underlying most mental disorders was largely mutational in nature, as we argued, then whole-genome association studies would have little success in finding mental disorder risk alleles of major effect. So far, this prediction has been born out in the data. Our interpretation of the pattern of whole-genome results to date is that they are being found for phenotypes that show large gene-by-environment interactions, such that common alleles that today are risk factors for nicotine dependence, obesity, diabetes, cancer, and heart disease were not risk factors for these diseases ancestrally, and did not decrease fitness in the environments in which humans evolved.

Conclusions
Evolutionary psychology has traditionally been concerned with understanding human universal adaptations. Behavioral variation was interesting to the degree that it was facultative (contingent on the situation) and elucidated universal adaptations, whereas genetic variation was deemed mostly as uninteresting side effects or as defenses against pathogens (Tooby & Cosmides, 1990). The field of behavioral genetics has traditionally focused on understanding the genetic and environmental contributions to trait variation, but has lacked a meta-theory that can suggest interesting new tests or that ties disjointed findings together in a cohesive way. Thus, to date, evolutionary psychology and behavioral genetics have largely talked past each other; what is chaff to one field has been wheat to the other (Mealey, 2001). But evolutionary psychology and behavioral genetics continue to ignore each other to their own detriment. We believe that much more dialogue and cross-fertilization between these fields is not only possible, but would mutually strengthen and benefit both fields. Evolutionary genetics is the bridge between evolutionary psychology and behavioral genetics that makes this “consilience” (Wilson, 1999) possible, and new data sources in molecular genetics offer many exciting ways to test questions of interest to researchers in this area. Evolution leaves fossils within DNA every bit as real and exciting—and sometimes confusing—as those buried in the soil. It is time for evolutionary psychology to take genes seriously, and for behavioral genetics to take evolution seriously.

References


11 Twin, Adoption, and Family Methods as Approaches to the Evolution of Individual Differences

Nancy L. Segal

My boys were thrilled to learn that they are definitely identical twins. Why, I don’t really know, but it is a big deal to them.

(Mother of twins)

Introduction

Individual differences in behavior have been of great concern to behavioral geneticists whose interests span a wide range of phenotypes. Individual behavioral variation has, however, received less attention from evolutionarily minded investigators. These two approaches have generally proceeded apart, yet this has started to change as each stands to gain from contact with the other (Segal, 1993; Mealey, 2001; Buss, 2007).

The present chapter provides a brief overview of these two disciplines and their current relationship with one another. This is followed by a review of the biological bases of twinning, twin and adoption research methods, and studies that have used behavioral-genetic methods to address selected evolutionary questions. The implications of the findings for understanding individual differences within an evolutionary framework are presented at the end of each section and summarized at the end. The chapter concludes with a look at future research directions.

Behavioral Genetics and Evolutionary Psychology: Apart and Together

Behavioral-genetics (BG) is a discipline linking biology and behavior in its search for factors underlying individual differences in behavior (Plomin, DeFries, McClearn, & McGuffin, 2008). Investigators apply an array of genetically informative research methods (e.g., twin and adoption studies) in order to accomplish this goal. These methods allow behavioral variance to be partitioned into genetic, non-shared environmental and shared environmental sources of influence. Gene × environment interactions (the variable expression of genotypes in different environments) and gene-environment correlations (the association of particular genotypes with certain environments) are also of interest (see, for example, Dick, Arawal, Schuckit, Bierut, Hinrichs, Fox et al., 2006).

BG includes a range of informative models and research designs for disentangling genetic and environmental effects (Segal, Chavarria, & Stohs, 2007). These models variously include monozygotic (MZ) twins and dizygotic (DZ) twins (both reared apart and together), full siblings, half-siblings, adoptive siblings and other relatives, to be described below. If genes contribute to variation in a given trait, then family resemblance for that trait is expected to be positively associated with the percentage of genes shared by individuals. For example, if genes influence general intelligence or extraversion, then MZ twins (who share 100% of their genetic inheritance) are expected to be more alike than DZ twins (who share 50% of their genetic inheritance, on average, by descent). The majority of studies have supported such predictions.

Behavioral genetics is now part of the mainstream of psychology, but this has only been true since the 1980s (Plomin, et al., 2008). Prior to that time, BG was mired in controversy over accusations of genetic determinism and the Nazi legacy of the biological superiority of some
populations over others. However, advances in genetic research and the growing number of twin and adoption studies showing genetic effects have made findings from behavioral genetic research difficult to ignore. Molecular genetic analyses have also yielded associations between specific genes and complex quantitative behaviors, such as Alzheimer’s disease (Owen, Liddle, & McGuffin, 1994) and general intelligence (Harlaar, Butcher, Meaburn, Sham, Craig, & Plomin, 2005). The effects of single genes on complex traits are, however, small.

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There is also increased understanding of the fact that genes work in probabilistic, rather than deterministic, fashion. Modifying environments can alter gene expression, although some genetically influenced traits are more difficult to modify than others. However, even the expression of single genes that are closely tied to certain outcomes (e.g., the gene coding for PKU or phenylketonuria) can be altered. In the case of PKU, providing the affected person with a phenylalanine-free diet from birth to adolescence offsets the mental retardation that would otherwise result. However, there has also been a recent focus on neuropsychological and psychosocial issues in the treatment of children and adults affected with PKU (van Spronson & Burgard, 2008).

Sociobiology, a precursor of evolutionary psychology (EP), was the focus of considerable attention in the 1970s and 1980s (Wilson, 1975; Freedman, 1979; Crawford, Smith & Krebs, 1987). Wilson (1975) defined sociobiology as “the systematic study of the biological basis of all social behavior” (p. 595). Sociobiology emphasized fitness maximization (organism survival and gene transmission by the production of offspring), whereas EP focuses on identifying psychological mechanisms that evolved to meet environmental demands and challenges our ancestors faced. The importance of identifying psychological mechanisms was encouraged by the work of Hamilton (1964), Dawkins (1976), Trivers (1971, 1974) and others emphasizing evolution at the gene level. Thus, EP represents the culmination of joining what has been termed “the gene’s eye view” with the search for psychological processes underlying behavior, expressed in given environments (see Caporeal, 2001). Furthermore, EP is not limited to the social domain, but spans a wide array of human behaviors including intelligence, consciousness and motivation.

A significant contribution of EP is that it highlighted the importance of examining ultimate explanations (i.e., explanations that emphasize behavioral and cognitive functions in terms of survival and reproduction). However, EP is also very concerned with proximal explanations (i.e., explanations emphasizing immediate causal events underlying behavior and cognition). For example, EP is concerned with identifying proximate cues that activate psychological mechanisms. Clearly, both types of explanations are essential to research guided by evolutionary perspectives (Mealey, 2001). Historically, however, EP has been more concerned with species-typical or sex-typical adaptations, with some notable exceptions such as those due to frequency-dependent selection (e.g., Mealey, 1995) and those due to variation in selection pressures over time and space (e.g., Buss & Greiling, 1994; Wilson, 1994).

Like critics of BG, critics of EP have espoused the misguided view that its proponents regard inherited effects as deterministic. Other criticisms have included the naturalistic fallacy (that all naturally occurring behaviors are

necessarily positive) and “just-so stories” (that any results can be interpreted in ways that support evolutionary reasoning). However, careful execution and appraisal of findings avoids such difficulties.
Evolutionary psychology provides an informative theoretical framework for assessing the meaning and significance of behavioral findings. Many scientists have used EP to bring fresh understanding to subjects such as parent-child conflict (Haig, 1992), offspring sex ratio (Manning, Martin, Trivers, & Soler, 2002), judgments of sexual desirability (Hill & Buss, 2008) and sense of humor (Weisfeld, 2006). However, Hamilton’s 1964 reasoning about altruistic acts (behaviors benefitting a recipient at some cost to the actor) may have had the greatest impact on research in EP. Hamilton asserted that altruism should be directed more often toward close kin (i.e., individuals likely to carry shared alleles) than distant kin, as an indirect means by which one’s genes are preserved in future generations. He defined inclusive fitness as an individual’s reproductive success, enhanced by effects on relatives other than offspring, where each effect is multiplied by the actor’s relatedness to the recipient (Hamilton, 1964). Identification of proximal events underlying the expression and maintenance of closeness, cooperation and other beneficial interactions between relatives has been of great interest, although somewhat speculative (Burnstein, 2005; Park, Schaller, & Van Vugt, 2008).

Behavioral genetics and evolutionary psychology have a number of overlapping goals and interests, despite differences in their methods and concepts (Mealey, 2001). The importance of applying evolutionary perspectives to human behavioral analyses was emphasized as early as the 1960s (prior to the emergence of EP as a discipline), in Freedman’s (1968) seminal infant studies. Points of intersection between BG and EP have been described since then in a series of scholarly papers; see, for example, Buss (1987, 1990), Crawford and Anderson (1989), Belsky, Steinberg and Draper (1991), Segal (1994, 1997), Bailey (1997), Segal and MacDonald (1998), Buss and Greiling (1999), Mealey (2001), and Keller and Miller (2006). Key concepts are that (1) behavioral genetics offers informative methods for testing evolutionary-based hypotheses, especially those concerning kin relations, and (2) evolutionary psychology provides behavioral genetics with an additional informative theoretical perspective for assessing results. Mealey’s (2001) overview continues to be the best recent discussion of themes shared by behavioral genetics and evolutionary psychology. She notes that for evolutionary psychologists, “kinship, via the effect of inclusive fitness, constitutes a core construct of relevance to all social interaction” (p. 23). Mealey defined a number of goals toward which behavioral genetics and evolutionary psychology can both aim. For example, one goal is to decide how heritable traits showing adaptive variation map onto life history strategies. This might be accomplished by the use of adoption and other family methods.

The biological bases of twinning and an overview of twin, sibling, and adoption research designs are presented below.

**Behavioral Genetic Methodology**

**Biological Bases of Twinning**

Monozygotic (MZ or identical) twins result when a single fertilized egg (or zygote) divides between the first and fourteenth post-conceptional day. As such, MZ twins should share all their genes in common, but this is not strictly the case. Most recently, copy number variations (CNVs) (structural variations affecting the number of DNA segments) have been shown to explain some MZ co-twin differences in physical conditions, e.g., Parkinson’s disease (Bruder, Piotrowski, Gijsbers, Andersson, Erickson, de Stahl et al., 2008). However, CNVs may also exist in trait-concordant MZ co-twins, so their presence must be interpreted cautiously. MZ co-twins may also differ for other reasons. Epigenetic events (processes associated with gene expression) may
underlie MZ co-twin differences in selected characteristics (Fraga, Ballestar, Paz, Ropero, Setien, Ballestar et al., 2005; Wong, Gottesman, & Petronis, 2005; Kaminsky, Tang, Wang, Ptak, Oh, Wong et al., 2009). MZ female co-twins may also differ in X-linked traits, such as color-blindness (Walls, 1959) and Lesch-Nyhan syndrome (De Gregorio, Jinnah, Harris, Nyhan, Schretlen, Trobley, & O’Neill, 2005), for reasons associated with differential X chromosome inactivation (Trejo, Derom, Vlietinck, Ollier, Silman, Ebers, et al., 1994). Differences in intrauterine conditions and post-natal experiences may also explain the less than perfect similarity between MZ co-twins (Segal, 2000, 2007). Nevertheless, most MZ co-twins are highly matched in physical appearance and behavior, and are more alike in virtually every measured trait when compared with DZ twins and other relatives.

Monozygotic twins occur naturally in approximately 0.35–0.42% of births world-wide (Bulmer, 1970; Machin & Keith, 1999). However, a recent three-year prospective study found that MZ twins occurred in 31/2,187 pregnancies or 1.42% (Shipley, Graham, Krecko, & Tucker, 2003). This increase has been explained by the greater availability of various artificial reproductive technologies (ART). Explanations have implicated the use of gonadotropins, manipulation of the zona pelucida (the membrane that develops around the oocyte), and the stage at which embryos are transferred (Moayeri, Behr, Lathi, Westphal, & Milki, 2007).

The natural DZ twinning rate varies widely as a function of country and race. Dizygotic twins occur in approximately 2.3% of births in Japan, as compared with 44.5% of births among the Yoruba tribe of Western Nigeria.
Figure 11.1. A. MZ twins who look very much alike. B. MZ twins who look very different. C. DZ twins who look very much alike. D. DZ twins who look very different. (Sources: A: Photo by Dr. Nancy L. Segal. B: Photo by Dr. Nancy L. Segal. C: Courtesy of the twins’ family. D: Courtesy of the twins’ family.)
Dizygotic twins result when women release two eggs simultaneously, both of which undergo fertilization. Such twins share half their genes, on average, by descent. The use of ART has had a far greater impact on the increased rates of DZ twinning than MZ twinning, in the United States and in other western nations. ART elevates the chances of DZ twinning due to ovarian simulation from drug treatment or the simultaneous transfer of multiple embryos. Prior to the advent of ART, DZ twins represented about two-thirds of twin pairs, but that figure is now higher. The overall twinning rate in western nations increased from 1/53 births in 1980 to 1/32 births in 2002 (Martin, Kochanek, Strobino, Guyer, & MacDorman, 2005). It is estimated that one-third of this increase (mostly among DZ twins) is due to mothers’ older age at conception, and the other two-thirds is associated with ART (Martin & Park, 1999). By way of contrast, the twinning rate increased 42% since 1990 (from 22.6 twins/1,000 births) and 70% since 1980 (from 18.9 twins/1,000 births) (Martin, Hamilton, Sutton, Ventura, Menacker, Kirmeyer, & Munson, 2006).

Some DZ twins have different fathers, so share an average of one-fourth of their genes, as do half-siblings; such twins are referred to as superfecundated twins. The frequency with which such pairs occur is unknown, but they may be less rare than has been presumed (Segal, 2000).

Accurate classification of twins as MZ or DZ is a crucial first step in twin research. This is best accomplished by comparative analyses of co-twins’ DNA markers. Physical resemblance questionnaires show agreement with results from extensive serological analysis and can be substituted for DNA analysis, if necessary (Segal, 2000). It is not acceptable to classify twins based on looks alone. Most MZ twins look very much alike, but co-twins in some pairs may not appear to be identical due to birth trauma or other events affecting only one twin, or affecting each twin differently. Most DZ twins look no more alike than ordinary siblings, but some DZ twins look very similar, while other DZ pairs look extremely dissimilar. Examples of MZ and DZ twins who vary in physical resemblance are displayed in Figure 11.1.

**Classic Twin Design**

The classic twin design was introduced by Sir Frances Galton in the late 1800s even though the biological bases of twinning had not been worked out at that time (see Galton, 1876). Galton reasoned that twins who look alike share all their heredity, while twins who do not look alike share a smaller proportion of their heredity. Basic twin research compares similarity between MZ and DZ twins with reference to traits of interest. Greater MZ than DZ twin resemblance shows genetic influence. A necessary requirement is fulfillment of the equal environments assumption (EEA), or the demonstration that trait-relevant environmental effects are the same for the two types of twins. Studies show that the EEA has been upheld for the majority of measured traits (Plomin et al., 2008). Recent work suggests that measurement invariance issues and the distribution of the underlying equal environments trait should both be examined when evaluating the EEA (Mitchell, Mazzeo, Bulik, Aggen, Kendler, & Neale, 2007).

**Informative Variants of the Classic Twin Design**

There have been approximately ten variants of the classic twin design (see Segal, 1990, 2000). The present chapter reviews the three that are most relevant to joint ventures between behavioral genetics and evolutionary psychology: “twins as couples,” twins reared apart, and twin-family models.
Twins as Couples. The “twins as couples” design compares social-interactional outcomes and processes between MZ and DZ twin pairs. This can be accomplished by having co-twins complete joint games, tasks, or problems that allow for cooperation or competition. Such analyses offer a look at how relative genetic relatedness affects social relations.

Twins Reared Apart. The rare pairs of twins reared apart from birth are valued participants in behavioral research. Monozygotic twins separated at birth and raised in uncorrelated environments provide pure estimates of genetic influence; DZ twins separated at birth provide an important comparison group. Most importantly from an evolutionary perspective, twins reared apart allow tests of hypotheses concerning kin relationships and associations between life history strategies and behavioral outcomes.

Twin-Family Models. Monozygotic twins who both marry and have children yield unusual families of interest to behavioral geneticists, evolutionary psychologists, and researchers with intersecting interests. Monozygotic co-twins become the genetic “parents” of their nieces and nephews who, in turn, become their genetic “children.” The children in the two families are equivalent to genetic “half-siblings” because the two mothers (or two fathers) are genetically identical. In contrast, when DZ twins marry and have children, the ordinary parent-child, aunt/uncle-niece/nephew and first cousin relationships remain unaltered. This twin design has been applied by behavioral geneticists to study influences on birth weight, spatial skill, psychopathology, and behavioral problems. Only two studies, reviewed below, have used this design to test evolutionary predictions.

Adoption Studies

Adoption studies help disentangle genetic and environmental effects in ways that classic twin studies do not. They do this by comparing resemblance between unrelated individuals living together or related individuals living apart. The majority of studies have shown substantially less behavioral resemblance in intelligence and personality between adoptive parents and children, and between adoptive siblings than between biological relatives whether reared apart or reared together (Plomin et al., 2008). This pattern of findings demonstrates genetic influence on the behavior under study.

In the present chapter, attention will be paid to a special class of adoptive siblings, called virtual twins (VTs). Virtual twins are same-age unrelated siblings reared together since birth (Segal, 2006a, 2007; Segal, McGuire, Havlena, & Hershberger, 2007; Segal, McGuire, Miller, & Havlena, 2008). As such, they replay the rearing situation of twins, but without the genetic link. Results from VT studies provide a more informative contrast with results from twin studies than do results from ordinary adoptive sibling studies. This is because most adoptive siblings differ in age and time of entry into their home, factors that remain constant for VTs. Research thus far shows very modest resemblance between VT co-twins in body size (Segal, Feng, McGuire, Allison, & Miller, 2008), general intelligence, special mental abilities, and decision-making, relative to MZ and DZ twins (Kershaw, 2008).

Research that has applied these twin and adoption designs to assess evolutionary-based hypotheses is reviewed below. The different studies have variously examined social relationships, bereavement, and reproduction-related behaviors.
Extant and Ongoing Studies Applying Behavioral Genetic Approaches to Selected Evolutionary Questions

Twin Relationships

Most psychological twin studies have found that MZ twins are closer socially than DZ twins (see Segal, 2000). This twin group difference has been upheld regardless of differences in theoretical perspectives, methodological approaches, research designs, and participant samples. Evolutionary-based hypotheses would anticipate greater cooperation and affiliation between MZ than DZ co-twins, given their closer genetic relationship. It is, therefore, worth noting that many researchers exploring twin relationships have not considered evolutionary viewpoints, yet have produced findings consistent with evolutionary predictions.

A large number of questionnaire and interview studies have found greater social closeness and compatibility between MZ than DZ twins, regardless of age and sex. Studies of young twins include those by Danby and Thorpe (2006) and Thorpe and Gardner (2006). Studies of older twins include those by Reiss, Neiderhauser, Hetherington and Plomin (2000). Reiss et al. reported greater “positivity” and less “negativity” between adolescent MZ co-twins, relative to DZ co-twins and other pairs of siblings. Foy, Vernon, and Jang (2001) observed comparable levels of intimacy between MZ and DZ co-twins, but noted that MZ twins were more likely to name one another as their best friend. Tancredy and Fraley (2006) showed that using the co-twin as an attachment figure was more likely among twins than non-twins, and that MZ co-twins showed greater attachment to their co-twin than did DZ twins. Genetic relatedness was also shown to enhance social investments in full siblings, relative to half-siblings. Maternal half-siblings showed greater investment than paternal half-siblings, implicating co-residence as a factor affecting social relatedness (Pollet, 2007).

Some twin studies have examined more specific features of twins’ social relations. Neyer (2002) found that the quality of DZ twins’ relationships depended on frequency of contact, while this effect was minimal or absent in MZ twins’ relationships. Neyer (2002) also reported greater spatial closeness between MZ than DZ twins, conceptualized as the effort expended to meet the co-twin, rather than as the actual physical distance between them. In a later study of personal networks, Neyer and Lang (2003) found that partners’ genetic relatedness predicted subjective closeness and social support. This finding concurs with studies showing that emotional closeness and obligation mediate the relationship between genetic relatedness and willingness to behave altruistically (Korchmaros & Kenny, 2001, 2006). Emotional closeness and obligation have also been shown to mediate the association between genetic relatedness.

Behavioral-genetic and social-genetic studies of MZ-DZ differences in their relations with one another are important steps toward understanding associations between genes and behavior. However, questions left unanswered by some, but not all, of these studies concern the mechanisms underlying differences associated with genetic relatedness. Social closeness would appear to be a prerequisite for cooperation and altruism between interactants, but what triggers such feelings is of great importance to evolutionary psychologists. The next three sections describe mostly evolutionary based studies that have observed twins interacting in (1) naturalistic and semi-naturalistic settings, (2) experimental game situations and (3) various family arrangements. Some of these studies have attempted to identify the proximal mechanisms that
facilitate cooperation, behavior that ultimately translates into transmission of common genes.

**Cooperation, Competition, and Altruism**

Twin studies of cooperation, competition and altruism have enabled tests of evolutionary hypotheses concerning genetics and social relatedness. Defining these terms within an evolutionary framework is an important first step. **Cooperation** refers to behavior by an actor that benefits both the actor and recipient; **competition** refers to behavior by partners directed toward achieving an individual (unshared) goal, with benefits to the winner and costs to the loser; while **altruism** denotes behavior by an actor that benefits a recipient at some cost to the benefactor (Kurland & Gaulin, 2005). An extensive review article on morality elaborates on these and related concepts (Krebs, 2008).

The studies reviewed below are set within a specific area of behavioral genetics, namely social genetics (SG). Social genetics is concerned with how the genotypes of the interactants affect the social-interactional processes and outcomes that emerge during their joint activities (Scott (1977; Hahn, 1990). Genes influence aspects of social behavior that can affect the chance that copies of those genes will be transmitted into future generations (Fuller, 1983), highlighting the relationship between social-genetic and kinship-genetic perspectives. The twins as couples design is well-suited to testing the hypothesis that MZ twins should show greater within-pair cooperation than DZ twins.

A marvelous experimental study of cooperation and competition within MZ and DZ twinships is also one of the oldest and most overlooked. Von Bracken (1934), a German investigator, applied the twins as couples design to investigate genetic effects on social interaction in MZ and DZ twin children. He observed the twins as they independently performed arithmetic and coding tasks under two conditions: alone and in close proximity to their co-twin. MZ twins produced more similar output when working near one another than when they were apart, in attempts to synchronize their efforts. In contrast, DZ twins who believed that they were matched in ability were highly motivated to outperform one other; those DZ twins who believed that they were unmatched in ability were not motivated to work very hard, knowing how they would perform vis-à-vis their co-twin. This study served as a model for much of the evolutionary-based research that came out of my own laboratory.

Segal (1984, 1997a) conducted a filmed, semi-naturalistic study of young twins’ behaviors during a puzzle completion task. Cooperative behaviors (e.g., balanced contribution to the activity; equidistant placement of puzzle pieces) were displayed more often by MZ than DZ twin pairs, as hypothesized. These filmed sequences were also assessed by independent judges with reference to the following behaviors: mutuality, cooperation, accommodation, role division, involvement and contribution. As anticipated, MZ twins scored higher than DZ twins on all measures with the exception of role division (Segal, 2002).

As part of that same study, Segal (1984) administered a differential productivity task (DPT) to the twin children. Twins were instructed to trace rows of small figures with a red pen, with the instruction that the more work they completed the higher their score. This activity was also completed under a second condition in which points earned were donated to their co-twin. Twins in both MZ and DZ pairs worked harder for themselves than for their co-twin; however, MZ
twins worked harder for their co-twins than did DZ twins. These findings suggested that MZ twins may be more altruistic with one another by dampening their selfish tendencies to a greater extent than DZ twins, a concept discussed by Axelrod and Hamilton (1981). That is to say, MZ twins may achieve more cooperative relationships than DZ twins by competing less.

Replication of Segal’s first study was attempted by Loh and Elliott (1998) using twins from Singapore. The cooperative and competitive behaviors of young twins were compared in two different game situations, one that assured equality of outcome and one that did not. It was observed that the MZ twin pairs were more cooperative than the DZ twin pairs during the game in which reward equality was uncertain. However, MZ twins competed to a greater degree than the DZ twins when conditions provided matched outcomes for both players. The investigators proposed that the equal rewards situation offered MZ twins a chance for “dominance testing” (p. 408), given that there was little challenge to their relationship.

The findings presented above, made possible via behavioral-genetic methods, highlight the significance of partners’ genotypes in the nature of social interactions. However, the underlying mechanisms are left unspecified. To identify them would require organizing the pairs according to resemblance in attributes such as physical features, intellectual abilities, information-processing skills and personality traits. For example, to the extent that physical appearance provides clues to genetic relatedness, MZ co-twins who look most alike should be more cooperative than MZ co-twins who look less alike. Results of such efforts, which represent another bridge between BG and EP, have been reported in studies reviewed below. Nevertheless, some implications of the present findings can be considered at this time.

It seems unlikely that there exists a special mechanism for twin recognition, given the rarity of twinning in the human population (Segal, 2005). A more plausible view is that the general processes regarding kin recognition and responses to kinship cues also apply to MZ and DZ twins. Research on evolved kin recognition mechanisms has described two possible cues to genetic relatedness between siblings: perinatal association with the biological mother and length of coresidence (Lieberman, Tooby, & Cosmides, 2007). However, these characteristics are matched for members of all twin pairs, so could not distinguish between an MZ or DZ co-twin. Other processes (e.g., phenotypic matching) may be necessary for drawing finer distinctions within relative categories. MZ twins are highly matched across most measured physical and behavioral traits, and probably possess similar sensitivities to relevant kin recognition cues. Freedman (1979) phrased it best when he suggested that recognition of common features may foster a “sense of ‘we’ between ourselves and our fellow tribesmen. Recognition of this sense triggers a series of emotions whose net effect is tribal unity and the increased chance for altruism” (p. 129).

**Game-Theoretic Approaches**

A recent area of interest for behavioral geneticists, evolutionary psychologists, and economists lies at the juncture of game theory, cooperation, coordination, altruism, and genetics.

There have been surprisingly few behavioral genetic analyses of interactive behavior during experimental games. The first such study by Segal and Hershberger (1991) administered a prisoner’s dilemma game to MZ and DZ twin pairs between the ages of 10.92 to 82.67 years.

The participants were instructed to apply an individualistic strategy, emphasizing interest in their own personal gain (Deutsch, 1973). Monozygotic co-twins displayed greater cooperation during
this task compared with DZ co-twins, as expected. Females showed more frequent cooperative efforts than males, although this difference did not reach statistical significance. Mutual cooperation increased across trials for MZ twins, but decreased across trials for DZ twins.

It was also observed that MZ twins made more competitive than cooperative choices. This outcome does not contradict their tendencies toward within-pair cooperation, but is reminiscent of MZ twins’ possible “greater restraint of selfishness,” discussed above. Based on intelligence tests and interviews with twins or parents, it was also found that increased IQ scores, increased social closeness, and increased tendencies toward sharing correlated with increased mutual cooperation.

The ultimatum game is another experimental task that has been widely used to assess one partner’s willingness to divide a sum of money and the other partner’s willingness to accept the proposed sum. The game is played once and anonymously. Wallace, Cesarni, Lichtenstein, and Johannesson (2007) reported that MZ twins made more similar rejection responses than DZ twins during this game, demonstrating genetic influence. The heritability of this behavior was estimated to be 42%. Another set of studies, independently conducted by separate research teams in Sweden and in the United States, provided comparable results from twin studies using a similar task, the trust game (Cesarini, Dawes, Fowler, Johannesson, Lichenstein, & Wallace, 2008). Heritability estimates were 10% and 20% for trust, and 18% and 17% for trustworthiness for the Swedish and American samples, respectively. An informative next step in this research program would involve pairing MZ and DZ twins with their co-twin in this situation. Additional pairs of individuals varying in genetic relatedness (e.g., full siblings, adoptive siblings, biological parent-child pairs, adoptive parent-child pairs) would enable further tests of the same class of questions.

Coordination is an interactive behavior that differs from cooperation in a key respect. Specifically, coordination signifies mutual efforts by partners that fulfill a common goal. Tacit coordination (TC) has recently been investigated with reference to evolutionary considerations. Tacit coordination refers to situations in which “two parties have identical interests and face the problem not of reconciling interests but only of coordinating their actions for their mutual benefit when communication is impossible” (Schelling, 1960, p. 54). Segal, McGuire, Miller, and Havlena (2008) conducted the first twin-adoption study to determine whether TC varies as a function of the genetic relatedness of the social partners. The sample included 7 to 13-year-old MZ twin pairs (n = 53), DZ twin pairs (n = 85), and virtual twin pairs (n = 42; same-age unrelated siblings). Each child completed twenty questions (e.g., name an article of clothing, name a book) under both individual and coordination conditions. In the individual condition, they were instructed to simply answer the questions; in the coordination condition they were instructed to answer so that they and their co-twin or co-sib would arrive at the same solution. Co-twin agreement on questions completed under these two conditions was compared across the three sibling groups. Monozygotic twins demonstrated significantly greater overall agreement than both DZ twins and VTs, as expected.

The few extant twin studies using experimental games demonstrate genetic influence on social exchange behaviors. Cesarini et al. (2008) suggested that evidence of genetic variance in cooperative strategies favors evolutionary models of cooperation that include polymorphic equilibria (stable distributions that are unaffected by small disturbances). The finding of MZ-DZ
differences in coordination also raises the question of what behavioral mechanisms may be involved. Segal et al. (2008) suggested that recognition of both coordinated behaviors and concordant solutions by the interactants themselves may be a possible mechanism underlying kin recognition, facilitating within-pair cooperation. This is because coordinated strategies may be less ambiguous signs of similarity than perceived resemblance in many physical and behavioral traits. Twin studies combining coordination and cooperation tasks could effectively answer this question. Future studies might also include incentives or manipulations involving reputation, as these can modify behavior in experimental games (Parks, 2000; Jared & Bering, 2008).

Social Closeness: Twin-Family Study and Twins Reared Apart

The twin-family study design capitalizes on the naturally occurring situation that includes adult twins who are married and have children (see above). Twin-family studies have been used to investigate the transmission and expression of birth weight (Magnus, 1984), non-verbal skills (Rose, Harris, Christian & Nance, 1979), schizophrenia (Gottesman & Bertelsen, 1989), child development outcomes (D’Onofrio, Turkheimer, Eaves, Corey, Berg, Solaas, & Emery, 2003; D’Onofrio, Turkheimer, Emery, Slutske, Heath, Madden, & Martin, 2005, 2006), conduct disorder (Haber, Jacob & Heath, 2005) and age at menarche (Mendle, Turkheimer, D’Onofrio, Kynch, Emery, Slutske, & Martin, 2006). A twin-family database for behavior genetics and genomic studies has also been established (Boomsma, Willemse, Vink, Bartels, Groot, Hottenga, et al., 2008). However, only two studies have used this design to explore evolutionary-based hypotheses. The first one, reviewed below, compared social closeness toward nieces and nephews by MZ and DZ twin aunts and uncles. The second study, concerned with age at menarche in children of twins discordant for rearing by a step-father, is reviewed in a later section.

Segal, Seghers, Marelich, Mechanic, and Castillo (2007) administered an Internet survey to assess social closeness experienced by aunts and uncles toward their nieces and nephews in MZ and DZ twin-families. The study included 248 adult MZ twins and 75 DZ twins, from intact and non-intact twin pairs. Composite closeness scores, derived from a 15-item Closeness Questionnaire (CQ), were significantly higher for MZ than DZ twins, supporting a relationship between relative genetic and social relatedness. In addition, scores from the CQ were significantly higher for twins with female co-twins than for twins with male co-twins. It is possible that paternity uncertainty explains the direction of this difference, namely that twins could not be certain that their brothers truly fathered their nieces and nephews (see Gaulin, McBurney & Brakeman-Wartell, 1997). Factor analysis of the CQ yielded three factors: comparative closeness, perceived closeness, and perceived similarity. We also showed that zygosity had significant effects on perceived closeness and perceived similarity, while sex of the co-twin had significant effects on all three factors.

Future studies along these lines might reverse the direction of inquiry. Specifically, my laboratory hopes to obtain closeness ratings from the children of MZ and DZ twins with reference to their aunts and uncles. We expect that the children of MZ twins will feel closer to their aunts and uncles, relative to the children of DZ twins; anecdotal evidence suggests that this is the case.
Insightful tests of associations between genetic relatedness and social closeness are also provided by MZ and DZ twins raised apart from birth (MZA and DZA) and reunited as adults. Biographical summaries included in early reared-apart twin studies show that 40 of the 76 pairs demonstrated close social relations after meeting, while 14 pairs did not and 22 pairs were difficult to judge (Segal, Hershberger, & Arad, 2003). However, the first formal analysis of social closeness in reared apart pairs was conducted using both MZ and DZ twins from the Minnesota Study of Twins Reared Apart (Segal, et al., 2003).

Participants included 44 MZA twin pairs and 33 DZA twin pairs, as well as 7 individual twins and triplets. Twins ranged in age from 16–70 years (Mean = 45.28 years, SD = 13.68). Age at separation ranged from 0 to 54.08 months (Mean = 8.03 months, SD = 12.64). Twins were administered a comprehensive Twin Relationship Survey that included items about their adoption and reunion experiences. Comparative ratings of initial and current closeness and familiarity were assessed. A second set of analyses included comparisons of ratings by twins with reference to the unrelated siblings with whom they were raised.

As expected, MZA twins indicated significantly greater closeness and familiarity toward one another than DZA twins. Twins, as a group, also experienced greater closeness than familiarity toward their co-twin, and current ratings exceeded initial ratings (retrospective recall of feelings at reunion).

Analysis of twins’ current closeness and familiarity ratings with references to their adoptive siblings offered an informative contrast to the reunited twin data. Feelings of both closeness and familiarity were stronger for co-twins than for adoptive siblings. The subset of twins who were raised with adoptive siblings (n = 41) rated co-twin closeness and co-twin familiarity equally, but they rated adoptive sibling familiarity higher than adoptive sibling closeness. The twin-sib analysis is especially powerful, given that twins had known their adoptive siblings all their lives, while they had known their twins for a relatively short time. Associations between co-twin similarities in self-ratings of personality, interest and values, and social closeness yielded few meaningful findings. We concluded that twins’ perceptions of their similarities may be a better barometer of their evolving social relations than similarities in self-rated traits.

Results from these studies support an evolutionary view of social closeness, given that the ratings varied as a function of the genetic relatedness of the participants. Dawkins (1976) theorized that people do not reason consciously over whom to direct cooperative behaviors. Rather, he held that individuals are affected by their genes to act in ways that would be consistent with the results of such calculations. It could be also argued that frequency of contact might explain the twin group differences in the first study; however, distance between twins (in miles) was not associated with closeness toward nieces and nephews. Some studies have found that greater contact between MZ than DZ twins is positively associated with personality similarity (Rose, Koskenvuo, Kaprio, Sarna, & Langanvainio, 1988). However, it may also be that twins who feel closer (or more similar) to one another are more likely to be in contact than those who do not.

The greater closeness felt by twins toward the children of their female than male co-twins is consistent with predictions based on the concept of paternity uncertainty. Human birth processes assure a mother that a child she bears belongs to her in a biological sense, but this is not true for a father. Hidden ovulation, internal fertilization, and continuous female receptivity mean that a male can never be completely certain that he is the biological father of a child delivered by his
partner. In the context of the study reviewed here, twins should be more confident of their relationship to the children of their twin sister, relative to their twin brother. This prediction was sustained, even though the genetic relatedness of nieces and nephews and their aunts and uncles is the same, regardless of whether children are born to twin sisters or twin brothers.

The second study demonstrated that shared time is not requisite to feelings of social closeness. This is underlined especially by the higher closeness ratings provided by twins for their co-twin, relative to their adoptive sibling. The fact that the MZA twins felt closer and more familiar than DZA twins also weakens explanations resting mostly on social contact. That is, the MZA-DZA difference completely parallels findings for twins reared together. Note that most reared-apart twins are unaware of their zygosity when they meet. Thus, it seems that these twins are responding to relatedness cues, albeit unidentified at present. I am unaware of any socialization explanation that can account for these findings.

Responses to the loss of a twin offer another source of information on associations between genetic and social relatedness. Relevant evolutionary-based studies are examined in the next section.

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Loss of a Twin and Other Relatives

Comparative studies of responses among bereaved MZ and DZ twins provide another approach to the class of questions concerning genetic and social relatedness (Segal, 2000). Based on evolutionary reasoning, loss of an MZ or DZ co-twin is expected to mirror the quality of relationships expressed by intact twin pairs. Specifically, loss of an MZ co-twin, especially during the reproductive years, represents a potential loss of one’s own children; loss of a DZ co-twin represents a potential loss of nieces and nephews. A number of studies (even those not guided by evolutionary perspectives; see Sanders, 1980) have shown that grief response varies as a function of genetic relatedness to the deceased. This finding was effectively captured by Barash’s (1979) assertion that in the despair of parental bereavement following the loss of a child, we hear “the wail of frustrated genes” (p. 99).

One might expect parents with many children to feel the loss of a child less intensely than parents with fewer children. A competing hypothesis would be that family size is unrelated to the level of grief following the loss of a child. These questions capture the difference between expected fitness and attained fitness (Pianka, 1978). Bereavement response reflects a loss in expected fitness, but whether this may be affected by attained fitness is not entirely clear. The few studies that have addressed this issue did not support the view that having multiple children diminishes depressive symptoms or the sense of loss; see, for example, Wilson, Fenton, Stevens, and Soule (1982) and Littlefield and Rushton (1986).

Grief following loss has been conceptualized as an adaptive response by some investigators. For example, unfavorable conditions may communicate needs to be with others or help guide future behaviors toward productive ends (Littlefield & Rushton, 1986; Hofer, 1994; Nesse, 1994). However, Archer (1988, 1999) has questioned whether grief can be considered to be adaptive because of its associations with emotional and physical stress. He suggested that grief may be better viewed as a byproduct of a characteristic that does augment fitness, e.g., investment in close personal relationships. It is, however, possible that stressful behaviors associated with separation could be adaptive in some contexts. Nesse and Williams (1994) have suggested that the capacity for sadness may have evolved to limit the occurrence of additional losses.
These different views generate several predictions. To the extent that grief is adaptive, moderate levels of grief-related behavior might motivate bereaved individuals to reconnect with old friends, adopt new projects, or establish a fund in memory of the deceased. In contrast, extreme levels of grief may be too physically and emotionally debilitating to allow such individuals to pursue productive activities. The minority of individuals experiencing little or no grief following the loss of a close genetic relative would also be unlikely to engage in new activities or tasks. However, lack of response would be atypical, suggesting that such individuals may lack the capacity for close social relations. The studies reviewed below demonstrate a positive association between genetic relatedness and level of grief, but there is variation within relationship classes.

The first study to examine bereavement responses in light of evolutionary predictions was conducted by Littlefield and Rushton (1986). Parental grief was higher for females than males, higher for healthy children than for sick children, and higher among maternal relatives than paternal relatives. Other studies have reported a similar pattern of findings, but without attaching evolutionary interpretations (see Segal et al., 1995).

The Fullerton Twin Loss Study, initiated at the University of Minnesota in 1983, is currently ongoing at California State University, Fullerton (Segal, 2000, 2007). Over 650 bereaved twins have completed an extensive twin loss survey designed to fulfill two goals. The first goal is to compare differences in bereavement-related behaviors between surviving MZ and DZ twins. The second goal is to examine differences in the twins’ responses to losing a twin versus other relatives and non-relatives. Hypotheses generated by evolutionary psychology are that (1) bereaved MZ twins will grieve more intensely than DZ twins, and (2) grief intensity following the loss of a twin will exceed grief intensity following the loss of non-twin relatives and acquaintances.

The Twin Loss Survey includes multiple components concerned with grief at the time of loss, grief at the time of participation, cause of death, and available support systems. Results from this study that are summarized below vary with respect to participant number, given the continually expanding sample size.

A consistent finding is that grief intensity is significantly higher for MZ (n = 394) than DZ twins (n = 202). The mean age at the time of loss was 39.98 years and ranged from 15 to 87 years. Ratings for the combined twin sample, and for MZ and DZ twins separately, showed that the loss of the twin was rated as significantly more devastating than the loss of most other relatives and non-relatives. One exception was the rating for the deceased spouse for whom ratings equaled those for the deceased co-twin. This makes sense in an evolutionary framework, given that a spouse represents a vehicle by which one’s own genetic transmission occurs. Too few twins have lost children (fortunately!), precluding analysis of loss in this kinship category (Segal & Harris, 2008).

In a related study, Segal and Ream (1998) studied changes in grief intensity ratings (within two months of the loss and at the time of participation) provided by the twin individuals. Consistent with our hypotheses, surviving MZ twins showed significantly smaller reductions in grief intensity over time than did DZ twins. The combined twin sample also showed significantly less reduction in grief for their deceased co-
twins, relative to other deceased relatives. However, a significantly smaller level of grief reduction by females than males with reference to their deceased co-twin, was not detected. We suggested that the small number of male volunteers may have been particularly bereaved, reducing gender differences in this measure.

MZ twins also scored higher across most bereavement scales of the Grief Intensity Inventory (GEI) than did DZ twins (Segal, Wilson, Bouchard, & Gitlin, 1995), consistent with the above. Segal, Sussman, Marelich, Mearns, and Blozis (2002) compared retrospective and current GEI scale scores for surviving MZ and DZ twins by means of discriminant function analysis and profile analysis. The data yielded supportive findings, but for the retrospective group only. This was somewhat surprising because earlier analyses showed less change in grief intensity over time for MZ than DZ twins (Segal & Ream, 1998). The use of different assessment scales in these two analyses may explain the different outcomes.

Segal and Blozis (2002) assessed coping and health characteristics immediately following loss, retrospectively and currently. The sample included 200 MZ twins and 45 DZ twins. Path analysis showed that zygosity had a significant effect on current grief, implicating correlates of grief in the bereavement process. The closeness of the twin relationship was associated with grief intensity, which affected physical symptoms and coping efficacy in predicted directions. This set of results concurs with evolutionary expectations.

It should be noted that MZ-DZ differences in bereavement response, while consistent, were not large. This may be explained, in part, by the self-selected nature of the sample. Most volunteer twin studies attract an excess of MZ twins, as did the present series. DZ twin volunteers may represent the subset of DZ twins who are especially grieved. It is, therefore, likely that a broader sampling of DZ twins would yield more marked twin group differences.

The present findings, while fulfilling evolutionary expectations of associations between genetic relatedness and bereavement severity, are also compatible with other explanatory frameworks. For example, social expectations are that people would grieve more intensely for close relatives (e.g., parents and children) than for more distant relatives (e.g., greatgrandparents and cousins). However, socially-based explanations fail to address the question of why bereavement varies systematically as a function of relatedness to the deceased, and why this pattern seems universal. In contrast, evolutionary psychology brings another level of analysis to these questions, namely the functional significance of individual bereavement differences.

Twin studies have also explored factors affecting age at menarche and other reproductive-related behaviors. However, it is only recently that studies have applied evolutionary themes to interpretations of the findings.

**Age at Menarche and Other Reproductive Characteristics**

Menarche is a significant developmental event in the lives of young women. Genetic influence on menarche has been demonstrated by a sizeable series of twin and family studies. Intraclass correlations range from 65 to 97 for MZ twins and from 18 to 50 for DZ twins, demonstrating genetic influence (see Segal & Stohs, 2007). Most recently, Segal and Stohs (2007) conducted the first study of menarche that combined MZ and DZ twins reared apart and together. Genetic influence was indicated by the higher MZ than DZ intraclass correlations, regardless of rearing status (MZA:. 56, DZA: 16, MZT:. 70, DZT:. 41). However, shared environmental effects were suggested by the greater resemblance between twins reared together than apart.
Some evolutionary psychologists have regarded age at menarche as partly reflecting girls’ reproductive life strategy, based on their rearing circumstances. It has been reasoned that the rearing of girls in father-absent homes should accelerate pubertal timing. More specifically, age at menarche for girls from such unstable environments would be adaptive in the sense that they could be more successful by leaving home and raising children (Belsky, Steinberg, & Draper (1991; Ellis, 2004). However, not all research inquiries along these lines have confirmed this evolutionary hypothesis. Rowe (2002) challenged this view, showing genetic influence on age at first intercourse, a finding confirmed in more recent studies (Guo & Tong, 2006; Mustanski, Viken, Kaprio, Winter & Rose, 2007). Segal and Stohs (2007) examined associations between family rearing factors and age at menarche in twins reared apart. They found that twins who felt understood by their parents experienced earlier menarche than those who did not, a relationship that did not confirm Belsky’s hypothesis. However, their finding was consistent with research linking favorable conditions to earlier menarche (see Ellis, 2004). Note that their sample was composed largely of adoptees, individuals who were desired by their parents. Adoptive parents also tend to be self-selected for higher age and socioeconomic status, relative to non-adoptive parents. Segal and Stohs (2007) suggested that the twins’ parents may have accelerated their daughters’ pubertal timing via resource provision. Other measures of childhood adjustment and satisfaction (e.g., happiness and anxiety relative to peers) were not significantly associated with age at menarche.

Mendle, Turkheimer, D’Onofrio, Lynch, Emery, Slutske, and Martin (2006) used the twin-family design to examine age at menarche in the children of twins (cousins) who were discordant for being reared by a step-father. They found no difference in age at menarche between cousins raised by a step-father or biological father. Furthermore, controlling for mothers’ age at menarche eliminated differences in age at menarche linked to step-fathering in unrelated girls. Organizing the families by the zygosity of the twin mother and children’s concordance-discordance for having a step-father yielded uninterpretable results, given the reduction in sample size per cell.

Factors affecting age at first intercourse (AFI) have been of interest to behavioral geneticists. Genetic influence on AFI has been demonstrated by studies of twins reared together (Dunne, Martin, Statham, Slutske, Dinwiddie, Bucholz, et al., 1997; Waldron, Heath, Turkheimer, Emery, Bucholz, Madden & Martin, 2007) and apart (Segal & Stohs, 2009). Heritabilities for AFI range between 0.00–0.72 for males and 0.15–0.49 for females, with higher heritabilities observed for younger samples. Segal and Stohs (2009) brought an evolutionary perspective to AFI by exploring associations between selected life history characteristics and AFI in reared apart twins. They found that feeling less fulfilled in the rearing home and feeling less happy than peers correlated with earlier AFI. The data suggest that individuals who are less content during development are more focused on their reproductive capacities than those who are more content. AFI was unrelated to the quality of the parent–child relationship, in contrast with age at menarche.

The studies reviewed here are good illustrations of how behavioral-genetic methods can be used to assess evolutionary-based hypotheses. Note that both twin studies on age at menarche failed to confirm hypotheses linking age at menarche to selected features of the rearing family environment. This is especially important, given the claim by some critics that evolutionary predictions are not amenable to falsification (see Conway & Schaller, 2002).
Fluctuating Asymmetry

*Fluctuating asymmetry* (FA) refers to the body’s deviation from consistency or symmetry. It has been reflected by increased right-left differences in body measures such as wrist circumference, ear length, and facial width. A number of investigators (e.g., Whitlock, 1996; Moller; 1999) have asserted that individuals showing relatively high FA may have experienced developmental instability due to biological and/or environmental stressors. Research evidence is consistent with this view. Mealey, Bridgstock, and Townsend (1999) showed that higher FA is associated with reduced physical attractiveness within MZ twin pairs. In addition, higher FA individuals have been shown to have fewer sexual partners than people with lower FA. Note that both physical attractiveness and number of sexual partners are associated with reproductive success (Gangestad & Thornhill, 1996). A recent study found that more symmetrical males made lower offers in an ultimatum game that less symmetrical males, a finding that did not characterize females (Zaatari, & Trivers, 2007).

A co-twin control study by Mealey, Bridgstock and Townsend (1999) is exemplary in its efforts to circumvent potential difficulties with research in this area. First of all, fitness itself may be genetically influenced (see Johnson, Gangestad, Segal, & Bouchard, 2008, cited below). Secondly, FA is only one measure of fitness and needs to be separated from other fitness measures, such as the waist-to-hip ratio in females (Singh, 1993), skin condition, and energy level (Miller, 2001).

Mealey et al. (1999) found that the lower FA twin was judged to be more attractive than the higher FA co-twin. Most importantly, the degree of difference in perceived attractiveness was directly related to the degree of difference in asymmetry. However, questions concerning whether or not lower FA individuals show fitness advantages, relative to higher FA individuals, remained. Subsequently, Mealey (2002) used the twins to examine the development of parental favoritism. The specific question was whether lower FA twins were favored by their parents, relative to their co-twins. The Parental Bonding Instrument was used to gather the twins’ perceptions of treatment by their parents from childhood through age sixteen years. This analysis did not yield any significant relationships, with one exception that was in the direction opposite to expectation. Mealey (2002) suggested that childhood FA may differ from later FA, due to modifications during developmental growth periods.

Two recent studies have addressed additional questions surrounding FA. Johnson, Gangestad, Segal, and Bouchard (2008) demonstrated genetic influence on FA by means of a reared apart twin sample. FA heritability across a ten-trait trait composite ranged from. 27 to. 30 even when the FA of individual traits did not differ significantly from zero. In another study, Johnson, Segal and Bouchard (2008) attempted to replicate the negative association between FA and general intelligence reported by Furlow et al. (1997) and by Luxen and Buunk (2006). However, this association was not detected using data from their reared apart twin sample. They noted that previously published studies used samples that were quite small, relative to their twin sample, a factor that may explain the different sets of findings.

Twin studies allow for informed analyses of factors affecting FA, over and beyond what non-twin studies reveal. They accomplish this in several ways. First, by holding the genotype constant it is possible to examine within-pair correlations between degree of FA and fitness-related traits, as in Mealey’s study. It would not otherwise be possible to disentangle individuals’ other attributes
(e.g., hair color or skin color) from those affected by FA. Her study of parental favoritism was unsuccessful, but the research design was appropriate and deserves repeated application. Second, twin studies enable analyses of shared trait variance. Johnson et al.’s (2008) finding of genetic effects on FA across multiple traits (even when genetic effects on FA are low for single traits) indicates shared genetic variance. The source of this variance is uncertain, but Keller and Miller (2006) suggested that mutations might have pleiotropic effects, thus affecting multiple features of the organism.

**Future Research Directions**

**Suggestions for Future Studies**

Behavioral genetics offers a wide array of genetically and environmentally informative kinships for testing evolutionary-based hypotheses. This section reviews some of these kinship groups and describes ways in which they may be inserted into ongoing research programs.

Virtual twins could be added to samples used to test various associations between genetic and social relatedness. VTs have never been recruited for experimental game situations (other than Segal et al.’s study of tacit coordination). Therefore, studies combining MZ twins, DZ twins and VTs in an ultimatum game would be of interest with respect to similarity in amount of offers and acceptance and rejection behaviors.

Female VTs have never participated in studies of pubertal development. Tracking age at menarche in these age-matched girls reared together could help resolve controversies surrounding the impact of the rearing environment (e.g., father absence or step-father presence) on rate of puberty. The effects of rearing by an unrelated male on age at menarche would be especially interesting using VT pairs composed of a biological child and an adoptee. Such co-sibs would have the same rearing father, but only the biological child would have a genetic relationship with this parent.

Twin research could advance understanding of associations between fluctuating asymmetry and reproductive traits. Organizing MZ twins according to FA and obtaining information on actual life history events such as age at first marriage, number of children and longevity would be informative. Additional studies with reared apart twins would also be beneficial. Segal is currently following the development of young reared apart twins from China in a prospective study (Segal, Chavarria, & Stohs, 2008). The children are quite young, but will eventually offer insights into factors affecting social relationships; most of the pairs meet periodically even though they live substantial distances apart. These MZA pairs also offer opportunities to assess gene-environment interactions, i.e., how differing family environments affect developmental outcomes. Like the VTs, they can also help shape understanding of how environments impact age at menarche and related measures, while controlling for genetic differences.

A reared apart twin study could also inform Sulloway’s (1986) theory of birth order. Sulloway reasoned that siblings benefit from securing a specific niche within a family and that this niche is closely tied to birth order. For example, he claimed that firstborn children should be relatively traditional, while laterborn children should be relatively non-conforming. Segal (2008) and colleagues attempted to examine these relationships using twins from the Minnesota Study of Twins Reared Apart (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990) and the Swedish
Adoption Twin Study of Aging (SATSA; Pedersen, Plomin, Nesselroade, & McClearn, 1992). They found that co-twins in different-birth order positions did not differ from one another, although the sample sizes were too small to allow firm conclusions. It is also worth noting that the twins’ personality self-ratings were made without reference to their co-twin, so were less sensitive to birth order effects than self-ratings that do consider the sibling (Sulloway, 2001). Of course, many MZA twins cannot rate their personality traits relative to those of their co-twin because they have spent little or no time together. Nevertheless, the possibility that MZA co-twins’ personalities may be modified by birth order in their respective rearing families is theoretically interesting. Comparing personality traits in separated twins who assume different birth order positions remains a potentially informative test of Sulloway’s evolutionary reasoning.

**Closing Comments: Behavioral Genetics and Evolutionary Psychology**

The present chapter has attempted to show that unions between behavioral genetics and evolutionary psychological approaches to behavior are possible and fruitful. The focus was on social relatedness, developmental theories of sexual strategies, and evolutionary hypotheses about fluctuating asymmetry, yet other behaviors, such as personality (Buss, in press), psychopathology (Keller & Nesse, 2008), and gender identity (Segal, 2006) could have been considered. Clearly, appreciation for individual differences has been growing among evolutionary psychologists. Several key points emerged throughout the discussion and are summarized below:

1. Behavioral genetics offers a powerful set of research designs for assessing evolutionary-based hypotheses.

2. Genetically-based individual differences have been well-documented across virtually all measured traits, including those of interest to evolutionary psychologists.

3. Behavioral-genetic methods are able to falsify, as well as to confirm, hypotheses grounded in evolutionary psychological theory.

4. Genetically-based individual differences cannot be overlooked by evolutionary psychologists when conceptualizing about human behavior.

5. Behavioral geneticists need to consider the functional significance of variations in heritability across different traits.

Human populations are rich with pairs of individuals who vary in genetic relatedness. This is largely due to the greater use of artificial reproductive technologies and the increased number of blended families. Capitalizing on these naturally occurring situations can advance research efforts in behavioral genetic and evolutionary psychological domains, both individually and collectively.

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**References**


Implications for immune relatedness and concordance for autoimmunity. *Molecular Medicine, 1*, 62–70.


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One of the major conclusions emanating from research on personality in the past half century is that individual differences are, almost universally, substantially heritable. In a state-of-the-art review, Bouchard and Loehlin (2001) estimated heritabilities for the Big Five traits in the 4 to 6 range. Personality traits surely conform to Turkheimer’s (2000) first “law” of behavior genetics: Individual differences in behavioral traits are partially heritable.

During the 1970s and ‘80s, behavior geneticists focused intently on the question of heritability—its existence, its magnitude, and the extent to which it reflected additive vs. non-additive effects. In the past 20 years, research has increasingly turned to issues that the heritability of personality begs: What genes are responsible for heritable variation? Do important gene × environment interactions exist? To what extent is covariation between phenotypic traits explained by pleiotropic effects of genes? Does the structure of individual differences at the genotypic level mirror their structure at the phenotypic level?

One major issue sits squarely at the crossroads of behavior genetics and evolutionary biology: What evolutionary processes maintain heritable variation in personality? Prior to the past two decades, this question drew little attention; increasingly researchers have turned to address it. As Bouchard (1994) argued, understanding genetic variation in evolutionary context will “turn the behavior genetics of human personality from a descriptive discipline to an explanatory one” (p. 1701).

This chapter has two major aims. First, to describe the primary scenarios under which genetic variation in phenotypic individual differences is maintained, and illustrate how they have been or could be applied to an understanding of genetic variation in personality traits. (See also Penke et al., 2007a). Most of the chapter is organized around these scenarios. Second, to stress the need for psychologists interested in these matters to develop plausible ways to test particular models. At this point, there is no dearth of general proposals about the evolutionary processes that account for genetic variation in personality. Currently, however, there are few compelling tests of the possibilities in the literature.

For purposes of this chapter, I refer to personality traits in very low-level theoretical terms (e.g., as dispositions to respond in particular ways). No doubt, an understanding of the role genes play in giving rise to individual differences and how those differences relate to behavior across varying circumstances ultimately requires a deep understanding of developmental systems that give rise to neural and physiological systems, which behave dynamically and interactively with ongoing conditions. But the emphasis of this chapter is on evolutionary processes that maintain genetic variation, however it becomes associated with behavioral variation and whatever psychological processes explicate “personality.” Similarly, I say little about how these traits specifically address major adaptive problems in human evolution (see, e.g., Buss, 2009). In various places, I refer to specific conceptualizations of major personality dimensions, such as the Big Five or Eysenck’s Extraversion, Neuroticism, Psychoticism, and Lie. Empirical tests of evolutionary explanations require specification of traits and measures of them. Most of what I
present here, however, does not assume commitment to one conceptualization over another. (See Bouchard & Loehlin, 2001, on how dominant conceptualizations relate to one another.)

The Fundamental Issue at Stake

Fisher’s (1958) fundamental theorem implies that consistent selection will, over time, remove additive genetic variation (genetic effects owing to individual genetic variants) absent any opposing evolutionary force (i.e., under ideal conditions). Yet individual differences in personality clearly do relate to genetic variants. It follows that the ideal conditions leading to depletion of genetic variation are violated in some way. But in what way (or ways)?

A possibility to consider at the outset is selective neutrality. Allelic variants in some genes may not be subject to selection simply because they don’t affect fitness. For these genes, random drift (changes in allele frequencies due to chance) may maintain allelic variation. In fact, many genetic variants are not expressed phenotypically and hence not subject to selection (see discussion of non-coding sites below). And, for various reasons other genetic variants may have such weak effects on fitness that they are near-neutral (see discussion of non-additive effects below). As Penke et al. (2007a) argued, however, it is unlikely that the genetic variants expressed in personality traits are generally invisible to selection.

That leaves a relatively small number of other broadly defined scenarios. The first is that mutation is a potent force opposing selection. The second is that selection has not been consistent; rather, it has been variable in some fashion, and variable selection maintains genetic variation. The third is that genetic effects on fitness are not additive. Each of these will be taken in turn.

SCENARIO 1: MUTATION IS AN OPPOSING EVOLUTIONARY FORCE

The Rate of Mutation

Mutational events are ubiquitous in biological organisms. At the individual base pair level, the rate of mutation is very small. Debate over the precise rate persists, but one figure often cited is about 1 error per 1 billion nucleotide copying events in eukaryotic nuclear DNA, which translates in humans to about 1 error per 40 million bases in germ cells (e.g., Nachman & Crowell, 2000). Most of the 3 billion or so base pairs in the human genome are non-coding and, at least until recently, it has been thought that the large majority of the non-coding DNA is not subject to selection (i.e., is “junk,” not expressed; but see below). The rate of de novo mutation at functional sites is hence thought to be about 3 per genome per generation, though geneticists acknowledge that current methods permit little confidence in a point estimate (e.g., Kightley & Charlesworth, 2005).1

1 If there are 75 errors per average gamete (1 per 40 million out of a total of 3 billion) x 2 gametes, then there are 150 mutations per diploid genome. (Using similar numbers, Nachman and Crowell [2000] estimated 175 mutations.) If one assumes that 2% of the base pairs are functional, then the mean rate of new mutation at functional sites is ^3 per individual.

Balance Between Mutation and Directional Selection on Fitness

Most mutations at functional sites are deleterious: With respect to fitness, their effects are biased downward; virtually all reduce fitness. As Fisher
(1958) himself recognized, selection and mutation are expected to be in an equilibrium state of balance: The rate at which deleterious mutational effects are removed by “negative” or “purifying” selection per generation (through differential reproduction) is, at equilibrium, equal to the rate at which deleterious effects are introduced through mutation. If the mean deleterious effect of a mutation at a functional site corresponds to a 1–2% reduction in fitness (e.g., Lynch et al., 1999), then an assumed rate of mutation of 3 per genome would result in ~3–6% reduction in fitness. (This calculation assumes that effects of mutations on fitness are additive. In fact, they probably exhibit positive epistasis [e.g., Rice, 1998]—increased effects as total number of mutations increase—but for illustrative purposes, simple additive effects can be assumed here.) In such an instance, at equilibrium the effect of selection would be to yield a next generation that has, per individual, fewer “old” mutations (ones inherited from parents)—specifically, on average accounting for a 3–6% increase in fitness.

Because most mutations are only mildly deleterious, they often aren’t selected out immediately. The mean number of generations an individual mutation persists before being eliminated is close to the reciprocal of its effect on fitness—e.g., if that effect is 1%, 100 generations. Although, at equilibrium, individuals possess, on average, three new deleterious mutations not possessed by their parents, they inherit many additional mutations that were introduced in previous generations, some very old. Indeed, as highly deleterious ones tend to be removed quickly, most inherited ones are mildly deleterious.

Not all individuals carry the same number of mutations, however. Variation exists. The standardized variance in fitness (where mean fitness is 1) maintained at balance is the mean reduction in fitness due to new mutations (see Burt, 1995). If that is 3%, then that standardized variance is 0.03.

An important insight achieved over the past two decades is that vast amounts of variation in fitness may be maintained at mutation-selection balance (e.g., Rice, 1988; Houle, 1992; Lynch et al., 1999; Burt, 1995). A standardized measure of genetic variation in traits measured on a ratio scale (as one could, in theory, measure fitness on) is the additive genetic coefficient of variation (CVA): The square root of the trait’s genetic variance divided by the trait mean, multiplied by 100 (to remove decimal places and convert a proportion into a percentage). If the standardized genetic variance in fitness is 0.03, then the CVA of fitness is \( \sqrt{0.03 \times 100} = 17 \). (See also Burt, 1995.) This amount of variation is very substantial. By comparison, the CVA of human height is about 4—several times smaller. If height had a CVA of 17, NBA centers would be 9 footers rather than 7 footers, and men shorter than Verne Troyer (renowned for his role as Mini-Me—2 feet, 8 inches tall) would be more common than 7 footers are today.

Individual fitness is a theoretical variable, never one actually measured in empirical studies. Certain measurable traits, however, relate to fitness and, in theory, are under directional selection: Longevity, mating success, number of offspring, number of grand-offspring. As first reported by Houle (1992), heritability studies show that, across a variety of organisms, these “fitness traits” tend to have high CVAs (often 10 or greater) compared to traits under stabilizing selection (often under 5; see below). For this reason, sexually selected traits also tend to have CVAs greater than those of traits under stabilizing selection (Pomiankowski & Møller, 1995).

In an important paper, Keller and Miller (2006) argued that genetic variants affecting serious psychopathology (e.g., schizophrenia) are generally mutations, each individually very rare, but with cumulative effects through multiple pathways adding up to an appreciable level. By this
view, very few major gene effects on serious psychopathology should be found (but see discussion below on CNVs).

Relatedly, Penke et al. (2007a) argued that variation in human intelligence too reflects the cumulative effects of rare mutations; smarter people, they argued, have fewer rare deleterious mutations. Brain size is modestly associated with psychometric intelligence ($r$ in adults $\sim .4$; McDaniel, 2005). If it too is a fitness trait, then it should be expected to possess a high CVA. In fact, the CVA for brain size is unusually low (Miller & Penke, 2007). To account for this and other findings, my colleagues and I suggest that the positive association between intelligence and genetic variance in fitness (e.g., mutation load) is not uniformly linear. We offer a condition-dependent allocation model (see below) to account for it (Gangestad et al., in press). Whether or not our model is correct, it seems that new understandings of the association between intelligence and heritable fitness are needed to account for all relevant findings.

In theory, some human personality traits too may directionally relate to fitness (or, more importantly, did ancestrally). On average, for instance, individuals who are socially dominant or vigorous may have been fitter (and left more descendants) than less socially dominant individuals (e.g., due to condition-dependence; see below and Gangestad et al., in prep.). If so, then substantial variation on such traits could be maintained at mutation-selection balance. I discuss mixed evidence on this matter later.

**Balance Between Mutation and Stabilizing Selection on Traits**

Distributions on many traits may tend to evolve such that the optimum level is the population mean. People’s height varies. Individuals with the highest fitness (ancestrally) may have achieved close to the mean height. Strong deviations from the mean in either direction may have been associated with lower fitness. The same might be true of many morphological features (e.g., BMI, birth weight, facial features). Selection against extremes on a trait and for mean levels is referred to as stabilizing selection.

Just as variation on fitness traits (those under directional selection) is maintained under mutation-selection balance, so too is variation on traits under stabilizing selection. Were mutations allowed to accumulate in a population unabated, variance on traits affected by the mutations would increase. Selection against extremes, however, tends to eliminate mutations that push individuals toward extreme levels and thereby reduce variance. At equilibrium, the variance-increasing effects of newly introduced mutations equal the variance-decreasing effects of selection against extremes. As noted above, the amount of variation on traits under stabilizing selection maintained at mutation-selection balance tends to be smaller than the amount of variation on fitness traits maintained; their CVAs are generally under 5 (Houle, 1992; Pomiankowski & Møller, 1995).

In theory, a number of major personality traits are probably under stabilizing selection, with at least some proportion of variation maintained by mutation-selection balance (e.g., Bouchard & Loehlin, 2001; for a contrasting view, see Penke et al., 2007a, who only seriously considered mutation-selection balance in the context of traits under directional selection). High levels of avoidance of social conflict (agreeableness) or cautiousness entails costs. But individuals who have exceptionally low thresholds to engage in social conflict over very minor resources, or throw caution to the wind at every bend, pay fitness costs too (e.g., Buss, 1991; MacDonald, 1995).
A Continuum of Mixed Directional and Stabilizing Selection

Perhaps few traits are “purely” under directional selection or stabilizing selection. Both forms can simultaneously act on a trait. In the case of pure stabilizing selection, the association between trait level and fitness is curvilinear (often assumed quadratic; see Crow, 1986), with the maxima associated with the mean trait level. If directional selection as well as stabilizing selection operates, the association remains curvilinear, but the maximum is not centered at the mean. As directional selection increases in strength relative to stabilizing selection, the maximum becomes further displaced from the mean.

What determines the extent to which directional or stabilizing selection operates? One approach to addressing this question invokes life history thinking about trade-offs. Individuals accrue resources (e.g., energy) from the environment and convert them into fitness-enhancing activities. An individual’s

“condition” partly refers to the extent to which they can or have accrued fitness-enhancing resources (e.g., Rowe & Houle, 1996). Rowe and Houle (1996) argue that it is tied strongly to capacity for fitness; individuals with the greatest ability to accrue resources possess relatively high potential for fitness. Ability to expend resources and energy efficiently and at low cost—e.g., low oxidative stress and resulting tissue damage, or ability to expend resources on reproductive activities at relatively low cost to survival (e.g., native resistance to extant pathogens)—may be just as important to capacity for fitness and, hence, a complete theoretical explication of “condition.”

Broadly speaking, fitness-enhancing activities that are energetically costly will tend to be associated with condition, as individuals in better condition can more effectively expend resources on them. Adaptive traits that are a function of energetically costly activities, then, will typically be associated with condition, i.e., under directional selection. Moreover, the more strongly the trait is condition-dependent, the more strongly it will typically be under direction selection (e.g., Rowe & Houle, 1996).

Capacity for fitness, however, is affected by factors other than condition. Resources can be allocated to a variety of fitness-enhancing activities, and some strategies for allocating resources outperform other strategies (e.g., Houle, 1991). Indeed, life history theory effectively seeks to understand the nature of the allocation strategies that selection favors (for one overview, see Kaplan & Gangestad, 2005). Given one’s condition, there’s a point to growing to a certain size, for instance; “overshooting” that size can result in a reduction in fitness, as some of the energy used to increase size could have been better spent on other fitness-enhancing activities (e.g., direct reproductive activities, somatic repair, immune function, etc.). Fitness, then, is affected not only by individual differences in resource-accrual abilities and energetic efficiency; it is also affected by differences in allocation strategies. No centralized “allocator” need be assumed. Hormonal systems and other physiological processes may affect allocation of effort to particular growth parameters (e.g., bone growth, muscle development) or other features (e.g., immune functions) and are “tuned” to respond to cues and inputs in ways that, de facto, yield allocations (e.g., Finch & Rose, 1995). Absent a centralized processor, when particular circumstances to which endocrine and other physiological systems are responsive are encountered, changes in allocation occur.

Traits that are not primarily affected by condition but rather are largely affected by allocation decisions may tend to be under stabilizing selection. If height is one such feature, then individuals
who are particularly tall may possess mutations that yield “overallocation” to upward growth at a
cost to other fitness-enhancing traits, and individuals who are particularly short may possess
mutations that yield “underallocation” to upward growth, overallocation
to other features. Variation in a variety of morphological features may be understood in similar
ways (e.g., Houle, 1991; Schulter et al., 1991).

Though this approach perhaps offers a reasonable way to think about variation in many traits, it
falls short of explaining variation in some behavioral traits whose costs are not primarily in
currencies of energetic expenditures. Behavioral caution, for instance, may not be energetically
costly. Too little caution has a cost of increased risk and potential mortality or morbidity. Too
much caution has a cost of decreased opportunities (e.g., to garner mates, to accrue a resource).
With some behavioral traits, variation maintained by mutation-selection balance may be thought
of in terms of overshothing or undershothing development of a behavioral propensity with
benefits and costs expressed in currencies other than energetic expenditure (see also Nettle,
2006).

Again, few traits may be under purely directional or stabilizing selection. To the extent that (a) a
trait is at least moderately energetically costly and hence partially condition-dependent, but (b)
also affected by variation in extent of allocations to it, it will be under both forms of selection.
Although I’ve discussed height as an example of a trait under stabilizing selection, it may well
exemplify a trait under both. Growing is costly, such that the optimal height for individuals in
best condition is greater than that of individuals in poor condition. At the same time, within level
of condition, variation in over- vs. under-allocation to height exists. The result may be a
curvilinear association with fitness (ancestrally) with maximum fitness achieved with a height
greater than average (see Nettle, 2002). The same may be true of some condition-dependent
personality traits. For instance, individuals in better condition can probably afford to be less
submissive. But for a given condition, one can either undershoot or overshoot one’s optimal level
of submissiveness.

**How Does a Trait Become Associated With Condition or Fitness?**

The discussion above implies an important point: The processes through which a trait may be tied
to genetic variation in condition or fitness and thereby affected by selection are multiple. In broad
terms, there are at least two major routes. First, a genetically influenced trait may directly
contribute to condition. Second, a trait may be affected by condition, mediated by evolved
contingent responses to condition (as a result of different levels of investment in the trait being
optimal for individuals of differing condition). The former traits *cause condition*. The latter traits
are *condition-dependent*.

Suppose a mutation affects efficiency in energy production. It thereby affects its carriers’
condition and, thereby, fitness. This is an example of a

variant causing condition. By contrast, differential allocations to trait development (e.g., size,
intelligence, immune function, testosterone-mediated traits) as a function of condition lead traits
to be *condition-dependent*. In biological organisms, condition-dependence is widely found (e.g.,
Rowe & Houle, 1996)

The genetic variation underlying the traits in these scenarios is in fact the same variation: The
mutation that affects energy efficiency and thereby condition is a genetic variant that underlies
any trait that is condition-dependent, as mediated through energy allocations. Despite common shared variation, however, these traits are distinct, and are the product of distinct causal processes.

In some instances, it’s not obvious which causal process produces a trait’s association with condition. As noted above, for instance, intelligence ($g$) has been claimed to be associated with fitness (e.g., Furlow et al., 1997; Miller, 2000; Prokosch et al., 2005; Arden et al., 2009). One possibility is that $g$ is associated with fitness because mutations at many loci deleteriously affect patterns of genetic expression in the brain, leading to reduced fitness via compromised intelligence (e.g., Penke et al., 2007a). Alternatively, allocations of energy and other resources to brain growth and development may be contingent on condition (as affected, for instance, by mutations, which need not be expressed in neural tissue per se), leading intelligence to be associated with condition (e.g., Gangestad et al., in prep.).

**Recent Issues Concerning Mutation Rates and Mutation-selection Balance**

At the outset of discussing mutations, I presented what has been, until recently, a fairly standard approach to understanding the mutation rate: A rate of mutation per copied nucleotide gives rise to a rate of mutation in gametes, which, in conjunction with a proportion of total nucleotide site that are functional, in turn gives rise to a rate of new mutation per individual—in humans, perhaps ~3 new mutations.

_The functional significance of non-coding sites._ Two recent developments raise serious questions about the adequacy of this approach. First, the common assumption that 98% of the nucleotide sites in the genome are not functional has been questioned. The vast majority of nucleotide sequences (98+%) in the human genome is “non-coding” (i.e., do not directly contain information affecting protein synthesis). By some views, it has been thought to be “junk”—of no consequence to the phenotypes of organisms. But mounting evidence indicates that non-coding DNA plays crucial roles in regulation of gene activity, epigenetic modifications of genes (e.g., imprinting), and DNA splicing and arrangement (e.g., Mallik & Lakhotia, 2007; Amaral & Mattick, 2008; Mariusz Nowacki et al., 2009; Feng et al., 2009; Mattick, 2009; Vinces et al., 2009). The more complex the organism, the more involved are these roles.

Of course, evidence that _some_ non-coding DNA is functional is not evidence that _all_ is. Non-coding sequences is less phylogenetically conserved than is coding DNA, consistent with variation being neutral and subject to random drift (Chiaromonte et al., 2003; Siepel et al., 2005; Lunter et al., 2006). Comparative analyses (e.g., of the mouse and human genomes) suggest that perhaps 5% of the human genome is functional and subject to selection against mutations. These analyses, however, address only sequences that are similarly functional in all species compared. Some non-coding DNA may be functional in some species, not functional in others, or subject to different selection across species. Consistent with this scenario, one analysis found evidence that, as the phylogenetic distance between compared species narrows, the estimated proportion of functional genes increases (e.g., Smith et al., 2004). Taking these findings into account leads to an estimate that ~10% of non-coding sites are functional (Ponting, 2008).

These developments vastly alter estimated rates of mutation. If the 10% of the genome is functional (a five-fold increase over 2%), the estimated total number of new mutations per functional genome is five-fold greater as well, e.g., 15 rather than 3. For an organism to be able to
persist in the face of the resulting mutation load, the mean effect of these mutations on fitness must be much less than 2%. In fact, mutations at functional non-coding sites, though more prevalent than mutations at coding sites, may be very weakly deleterious (e.g., Smith et al., 2004). If so, these mutations will accumulate in the genome. A very substantial proportion of the total mutations at functional loci in the population may consist of very mildly deleterious mutations in non-coding DNA. Interestingly, large-scale whole genome association studies of quantitative phenotypic features (e.g., height) find that genetic variation is explained by very weak effects of many loci, most of them non-coding variants (Martin, 2009).

The discovery of copy number variations. A second important development concerns copy number variations or CNVs. It was once thought that the “normal” human genome could be defined by a shared reference genomic structure, one specifying all single nucleotide sites. In an extreme form of this view, all genetic variation between any two (“normal”) individuals would consist merely of the aggregate of base differences at all 3 billion or so single nucleotide sites (e.g., A vs. T, C vs. G). Geneticists have long recognized the existence of exceptions—insertions, deletions, or inversion of long chromosomal segments in individual genomes. Recent discoveries, however, show that “exceptions” are anything but unusual (e.g., Iafaré et al., 2004; Sebat et al., 2004). A substantial portion of the genome is subject to “copy number variation”—differences across individuals in number of copies of a chromosomal segment at least 1000 bases long (in rare cases over 1 million bases long). Some individuals might have two copies of such a sequence (one inherited from each parent), whereas others have one copy (with no copy—a “deletion”—inherited from one parent), and yet others more than two copies. Thus far, several thousand such sequences have been found in the human genome, comprising at least 12% of it (e.g., Redon et al., 2006; Wong et al., 2007; Kidd et al., 2008). Variation across individuals, then, consists not only of differences at single nucleotide sites, but also in number of copies of particular DNA strands (referred to as “structural variation”). Because CNV strands consist of many bases, CNVs may account for more total inter-individual genetic variation than single nucleotide variants combined (Beckmann et al., 2007).

CNVs originate through mutational events of sorts, but not, of course, mutations defined as single nucleotide substitutions (also known as point mutations), as treated above. One important cause of a new CNV is non-allelic homologous recombination (NAHR; see Kim et al., 2008; Bailey & Eichler, 2006, for a discussion of this and other processes). Through recombination, homologous chromosomes inherited from the two parents (e.g., chromosome 16 from each parent) are spliced and recombined to create new homologous chromosomes. NAHR is a mistake that occurs when a segment of DNA incorrectly matches up with a segment on the homologous chromosome during recombination, resulting in the creation of a new chromosome that contains a deletion, duplication, or inversion. Chromosomal regions containing segmental duplications (SDs) are prone to NAHR. SDs are effectively copy number variations (specifically, duplications) that have gone to fixation; they are characteristic of nearly all individuals. Specific regions of the human genome are very rich in SDs. NAHR is particularly likely to occur in the presence of SDs because a long segment of DNA can readily “mismatch” with a similar DNA sequence (its duplication) on a different allele during the crossover process involved in recombination, leading to alteration in structure in the recombined chromosome. In short, duplications beget more duplications as well as deletions; duplications yield genomic instability. As expected, then, CNVs are not randomly distributed across the genome; they are 4–12 times more likely to appear in
genomic regions ich in SDs (e.g., Iafarte et al., 2004; Sebat et al., 2004; Wong et al., 2007; see also Bailey & Eichler, 2006).

The rate at which mutations creating CNVs occurs is much greater than the rate at which single nucleotide mutations occurs—indeed, on average, several orders of magnitude greater (perhaps 1 in 10,000; e.g., Sebat, 2007). CNVs (perhaps deletions more so than duplications) tend to be deleterious (Locke et al., 2006). Individuals carry a substantial number of deleterious CNVs in addition to deleterious point mutations. One study found 100 CNVs per individual, though the proportion that is deleterious remains unknown (Wong et al., 2007).

In the words of one pair of authors, psychiatry was recently hit by a “copy number variant tsunami” (Joober & Boksa, 2009). Whereas psychiatric geneticists have had difficulty identifying single nucleotide variations that robustly associate with major disorders such as schizophrenia, autism, or bipolar disorder (but see Crespi, 2008, for a review of some such variations), recent research has convincingly linked these disorders to CNVs (particularly ones rare and newly arising in the affected individual): for autism, see Sebat et al., 2007; Weiss et al., 2008; Szatmari et al., 2007; for schizophrenia, see Stefansson et al., 2008; International Schizophrenia Consortium, 2008; Walsh et al., 2008; Kirov et al., 2009; for bipolar disorder, Zhang et al. 2009; see also reviews by St. Clair, 2009; Cook & Scherer, 2008. Naturally, single nucleotide variations may also predispose these disorders, but largely as a function of rare mutations at sites distributed throughout the entire genome (Keller & Miller, 2006).

One reason why CNVs have substantial effects on psychiatric disorders is they reflect “big” mutations (variations at many nucleotide sites). But CNVs may be especially important to psychiatric disorders for additional reasons, as discussed below.

Segmental duplications, CNVs, and human evolution. CNVs, again, are overrepresented in regions rich in SDs (which themselves tend to be near centromeres and telomeres—toward the center and far ends of chromosomes, respectively), regions in which multiple copies of sequences of DNA are found. SDs, it is thought, were often duplications that selection favored. A duplication of a DNA sequence may bolster level of expression of a gene (or genes) contained within the segment, and at times increased level of gene expression is favored. In addition, once a segment of DNA has been duplicated, selection may favor changes in that segment, such that it can serve functions partly distinct from the original copy (while the functions served by the original are preserved). Selection that copies-pastes-modifies is a common route to adaptation (e.g., Taylor & Raes, 2004).

SDs appear to have been particularly important in the evolution of the great apes and humans. A rapidly advancing science of comparative genomics finds that a burst of increased SDs occurred in the common ancestor of great apes. SD content has continued to expand in chimpanzee and human genomes (Marques-Bonet et al., 2009). Though comprising only about 5% of the human genome, SDs account for more divergent evolution between chimpanzees and humans than all single base-pair changes combined.

Segmental duplications near centromeres (e.g., on chromosomes 1, 9, 16) appear to be especially core to the lineage-specific expansions of the human genome. Consistent with these duplications being adaptive, SD regions are richly inhabited by signatures of positive selection for
substitutions within them (perhaps reflective of a copy-paste-modify adaptive process; Bailey & Eichler, 1996).

If much of human evolution occurred through changes in SD-rich regions, these regions are likely to play critical roles in the development and expression of many traits derived in the human lineage, phenotypically distinguishing us from close relatives. Some of these unique features are ones that coevolve in response to other species or individuals. As should be expected, then, human SDs are particularly rich in genes involved in immune function, olfaction, and reproduction, systems responsive to coevolutionary processes or changes in diet. Perhaps more interesting from an evolutionary psychological perspective, SDs also appear to contain genes involved in neuronal development or expressed in neural tissues, perhaps central to human-specific cognitive features (e.g., Dumas et al., 2007; Sikela, 2006; Popesco et al., 2006).

Though substantial levels of SD have likely been selected in the recent human lineage, SDs once again carry a special cost: They predispose genomic instability and hence deleterious CNVs. The functions that these CNVs likely disrupt are ones facilitated by DNA in SDs, including psychological ones. From an emerging understanding of the evolution of the human genome, then, it is perhaps no coincidence that CNVs are important to an understanding of major psychiatric disorders (see above).

Do CNVs affect “normal” variations in personality? No study has yet closely examined these associations. Later, I discuss in more detail a study by Shifman et al. (2007), a genome-wide scan for genetic markers associated with neuroticism. One tantalizing finding of that study is that genetic markers in CNV-rich regions of the genome were significantly overrepresented in the set of markers with greatest associations; variations within these regions may be particularly important to neuroticism. Might the same be true of many other personality traits? Future research will tell.

Scenario 2: Selection is Variable

Absent an opposing evolutionary force, consistent selection removes additive genetic variance—selection consistently favoring particular phenotypes over others. When selection varies, genetic variation can be maintained. Selection can vary across space, time, and allele frequencies.

Selection That Is Variable Across Space

Assume that individuals in a population are spatially distributed across regions that systematically vary in some way: e.g., in mean temperature, mean rainfall, density of predators, or density and type of pathogens. Selection in different regions may favor different phenotypes and, as a result, different alleles at some loci. Interbreeding of individuals at regional margins or migration across regions will result in alleles favored in one region to “flow” to others.

Some researchers have argued that human groups may systematically vary in personality partly as a result of spatially varying selection. For instance, populations may differ in the extent to which they exhibit a relatively “fast” or “slow” life history. Slow life histories are associated with a prolonged juvenile period, substantial investment in somatic repair and other mortality reduction tactics (and hence health), marked parental investment (including paternal investment), and relatively great caution (e.g., Figueredo et al., 2005). Fast life histories are associated with an earlier transition to the reproductive phase, less parental investment (particularly paternal investment), and relatively great risk-taking. When “extrinsic” adult mortality risks (risks independent of individuals’ own resource allocation strategies) are high, faster life histories tend
to be favored, whereas low extrinsic mortality risks tend to favor slower life histories (e.g., Promislow & Harvey, 1990). A psychometric measure purported to tap individual differences in slow vs. fast life histories covaries with personality (in particular, with a higher-order factor defined by high aggressivity, high impulsivity, low agreeableness, and low conscientiousness, and, to a lesser extent, lack of emotional stability). In theory, some variation in life histories and associated personality characteristics may exist as a function of human ecologies. Particularly in modern societies into which people from varying geographies have migrated, some within-population variation could derive from temporally variable ancestral selection (e.g., Camperio Ciani, this volume).

Does personality vary strongly across geographical regions in modern populations? Schmitt et al. (2009) examined differences across nations and geographical regions in the Big Five personality traits. Differences across regions were small, with percentage of variance accounted for ranging from 1% (Extraversion) to 6% (Conscientiousness) (mean = 3%). The region that stood out as consistently most different was East Asia (Hong Kong, Japan, Korea, Taiwan), where agreeableness, conscientiousness, openness, and extraversion were lowest, and neuroticism highest. The highest agreeableness and conscientiousness levels, and lowest neuroticism levels, were found in Africa. If associations between life histories and personality argued by Figueredo et al. are assumed, these data suggest that East Asians have the fastest life histories and Africans the slowest, a pattern that contradicts some proposals (e.g., Rushton, 1996) and other life history data (e.g., life expectancies). Again, however, effect sizes are small. In a study notable for demonstrating reliable differences between small island and mainland Italian populations in Big Five traits, they accounted for, on average, less than 1% of the variance (Camperio Ciani et al., 2007).³

³ The authors favored the interpretation that more extraverted and open individuals were more likely to emigrate from the islands, leaving less extraverted and open people there, over a local adaptation interpretation. A third possible cause is inbreeding depression (Rushton et al., 2008).

The vast majority of personality variation is within-culture, not between-culture.

Selection That Is Variable Across Time

Selection that varies temporally can lead to genetic variation for two reasons. First, suppose selection iteratively shifts, back and forth, from favoring allele a over allele b to favoring b over a, and vice versa. If the temporal phases between shifts are sufficiently short, selection may not eliminate the disfavored allele from the population before it is favored once again. Allele frequencies change over time, but variation is continuously maintained. Second, even if the temporal phase between shifts is long enough to fix one allele in the population, the shift, once it takes place, sets the stage for positive selection for new alleles arising through mutation, which then may sweep through the population, with variation maintained until these new alleles are fixed. Thus, for instance, suppose selection drives out allele b, fixing a in the population. When selection changes, perhaps no individuals possess b. But b or a new allele c can arise through mutation and be favored. As it sweeps through the population, multiple alleles are present once again (see Roff, 2005).

Temporally varying selection is particularly common in antagonistically co-evolving systems (e.g., Rice & Holland, 1998). Interspecific antagonistic coevolution occurs when individuals of different species co-evolve. Prime examples are predator-prey and host-parasite coevolution. In the latter case, hosts evolve defenses against pathogens, to which pathogens may respond by
evolving means to evade host defenses. In turn, hosts evolve new defenses to counter those newly evolved evasive strategies—which sets the stage for newly effective pathogen tactics, then novel host defenses, and so on. Over time, both hosts and pathogens may evolve elaborate tactical arsenals bearing on their conflicts. Some tactics that were once favored, however, may be lost due to subsequent ineffectiveness.

Intraspecific antagonistic coevolution exists when individuals of the same species coevolve. Several obvious instances occur in humans. Sexually antagonistic coevolution exists due to conflicts of interest between the two sexes. The conditions that benefit female mating, for instance, need not be the conditions that benefit men. (E.g., the within-pair mating rate that benefits males may exceed that which benefits females; the circumstances in which a female will mate with a male may exclude a particular male from consideration against his interests. See Rice & Holland, 1998.) Parent-offspring coevolution exists due to conflicts of interest over the amount of investment that parents offer offspring or the extent to which an offspring engages in conflict with siblings. Mothers and fetuses, for instance, each appear to have evolved tactics aimed at restricting or enhancing, respectively, the rate of flow of nutrients to the fetus (Haig, 1993). Antagonistic coevolution may also operate on individuals engaged in cooperative interactions. Cooperation implies shared interests, but cooperators typically do not fully share interests. Cooperators may be tempted to cheat, which sets the stage for the evolution of tactics to avoid getting cheated upon, to cheat in more subtle ways, to prevent those subtle forms of cheating, and so on.

Each of these forms of antagonistic coevolution may lead to genetic variation underlying tactics. For instance, some females may possess genes that evolved in response to the “latest” male tactics, whereas other females lack them because selection has not operated long enough for loci to be fixed.

Crespi (2008) has argued that antagonistic coevolution is a cause of psychological features associated with forms of psychopathology. According to the maternal-fetal conflict theory (see Haig, 2004, for a review), maternal-fetal coevolution has given rise to imprinting, a process whereby expression of a gene is silenced or suppressed as a function of its parent-of-origin. Imprinting of genes expressed during fetal development has been selected, according to this theory, because paternally-derived fetal genes have greater average conflicts of interests with maternal genes than do maternally-derived fetal genes. A number of fetal genes that foster fetal growth are indeed suppressed when maternally derived. A number of fetal genes that inhibit growth are suppressed when paternally derived. Crespi (2008) argues that maladaptive imprinting contributes to psychotic spectrum and autistic spectrum disorders. Certain syndromes with known etiologies that have psychotic-like symptoms (e.g., Prader-Willi, Klinefelter) are associated with known or suspected overexpression of maternal genes at imprinted loci and resultant undergrowth of the fetus. By contrast, a number of syndromes that have autistic-like symptoms (e.g., Angelman, Beckwith-Wiedemann, Turner) are associated with known or suspected overexpression of paternal genes at imprinted loci and resultant overgrowth of the fetus (see also Crespi & Badcock, 2008). These particular syndromes clearly involve genomic maladaptation (a major chromosomal deletion, duplication, or aneuploidy [e.g., full extra copy of a chromosome]). Some other variations associated with psychotic spectrum features (e.g., schizotypy, anhedonia) or
overgrowth and/or autistic spectrum features (e.g., Asperger’s, social skill deficits) may be affected by many genetic variants and fall toward the extremes of continua. Might some variations be attributable to temporally shifting selection in the context of maternal-fetal conflict? Possibly, though it should be noted that the variations may also reflect nothing more than the effect of deleterious mutations, including at loci that affect imprinting (as implied by Crespi, 2008; but see Crespi et al., 2007). As noted earlier, schizophrenia and autism appear related to rare CNVs. Less pathological variations may relate to less deleterious CNVs or other mutations.

**Selection Varies as a Function of Allele Frequency**

Another form of selection that can maintain genetic variation is negative frequency-dependent selection—selection varying as a function of allele frequencies. To maintain variation, selection must not favor just one allele over all others at the locus across the entire range of possible frequencies. Rather, at least two different alleles must be favored over others when each is rare. Targets of negative frequency-dependent selection probably include major histocompatibility complex (MHC) genes. Class I MHC genes present antigens to leukocytes; if the cell is infected and hence the antigens foreign, leukocytes attack the infected cell. Pathogens can go unnoticed if they evolve peptide structures presentable by the host’s MHC molecules that aren’t distinct from the host’s own cells. As an MHC allele becomes common in the population, pathogens encounter it more frequently and may adapt to that allele. Particular MHC loci (e.g., A and B within Class I) are highly polymorphic, with many different alleles represented in human populations and no one allele typically accounting for more than about 20% of all alleles. Frequency-dependent selection is generally accepted as an explanation of MHC polymorphism (though heterozygote superiority—see below—may also play a role; see Hedrick, 2002).

Frequency-dependent selection can also explain variations in social strategy (or niche selection) and, indeed, there appear to be some fairly well-established illustrations in non-human species (see Nettle, 2006, for a review; see also Wilson, 1998). Partly inspired by non-human examples, a number of scholars have proposed that variations in human personality have been maintained by frequency-dependent selection. Gangestad and Simpson (1990) proposed that variations in women’s sociosexual orientation have been maintained by such selection. Fundamentally, we claimed, women vary in mate preferences, with some women particularly preferring men who possess indicators of “good genes” and others particularly valuing men’s paternal investment, each partly trading off the other to obtain mates providing their most desired features. Women who favor men with good genes indicators, we claimed, are more willing to have sex in absence of commitment with those men, whereas women who value paternal investment tend to seek signs of commitment prior to sex. We reported a variety of evidence consistent with women’s sociosexual orientation being interpretable as tactical variations. Perhaps most interestingly, for instance, we found that women’s sociosexual orientation and associated personality measures (extraversion and lack of constraint or conscientiousness) vary with the sex-ratio of their offspring, with women willing to have uncommitted sex (and preferring particularly attractive mates) having more sons (Gangestad & Simpson, 1990). We did not, however, report compelling evidence that variation has been maintained by frequency-dependent selection. Later, we favored an alternative view—that women’s preferences and sociosexual orientation (men’s too) are partly tactically contingent on personal circumstances and quality (Gangestad & Simpson, 2000).
Other prominent examples exist. Figueredo et al. (2005) proposed that life history variations (with personality correlates discussed earlier) may have been maintained by frequency-dependent selection. (For a general argument that life history trade-offs favor personality variation in species, see Wolf et al., 2007.) Mealey (1995) argued that a subset of psychopathic individuals persist in small numbers in human population as habitual “cheaters,” favored when rare amongst others who benefit through cooperation. McNamara et al. (2009) offered an interesting model demonstrating how variations in trust, trustworthiness, and social awareness can be maintained within a population through frequency-dependent selection (see also Dall et al., 2004).

Tooby and Cosmides (1990) explained personality variation with a different form of frequency-dependent selection. The evolution of vast polymorphism in the MHC system, once again, has partly been maintained by frequency-dependent selection pressure imposed by pathogens. Tooby and Cosmides argued that this form of frequency-dependent selection operates very widely on peptide-encoding genes, as they constitute the environment and available resources to which pathogens adapt. Variations in protein structures maintained by pathogen-driven frequency-dependent selection, however, have small effects on other phenotypic features, including personality. In this view, personality variations represent byproducts of variants selected to enhance disease resistance. At the extreme ends of personality distributions, these byproducts could be selected against (i.e., stabilizing selection could operate). But in this model variation is maintained by frequency-dependent selection, not mutations.

**Recent Broad Claims in Favor of Variable Selection Models**

Recently, the view that variable selection has played major roles in maintaining the genetic variability underlying personality has had strong advocates. In light of high profile papers by Nettle (2006) and Penke et al. (2007a), this view may well be the dominant one in the latest literature. (See also earlier influential papers by Buss & Greiling, 1999; MacDonald, 1995; Wilson, 1994).

In a systematic fashion, Nettle (2008, this volume) argued that each of the Big Five personality traits may have been subject to frequency-dependent as well as spatially and temporally variable selection. He couched his general argument in terms of trade-offs. As one moves along a personality dimension from one end to the other, certain forms of benefits increase, while other forms decrease. (As noted earlier, for instance, caution pays up to a point due to survival advantages, but costs in the currency of lost opportunities increase.) The optimal way of trading off costs and benefits is not invariant across circumstances. Moreover, strategies may sometimes do best when rare. Hence, selection varying across time, space, and strategy frequencies may maintain genetic variation in personality. Penke et al. (2007a) echoed these claims, and offered additional evidence for them.

**Nettle’s Arguments in Favor of Variable Selection**

Nettle’s (2006) theories of context-dependent selection offer predictions about particular benefits and costs associated with each personality dimension (and, at least with respect to extraversion, evidence supports one such prediction; Nettle, 2005). With respect to neuroticism, for instance, those scoring high on the dimension may perform better on some tasks, worse on others. If the relative value of those tasks varies across environments or as a function of relative frequencies of individuals good at them, variable selection could maintain genetic variation. These predictions about benefits and costs are, generally speaking, eminently reasonable and worth studying (see also Penke et al., 2007a). The trade-offs, however, are not uniquely predicted by theories of
variable selection. As noted earlier, conventional understandings of traits subject to stabilizing selection argue that the mean represents an optimum level in the face of trade-offs in energy allocation or other currencies. Such a model too expects that individuals distributed across

the personality dimension make trade-offs differently. The difference is that a pure mutation-selection balance model interprets variations away from the mean as non-optimal trade-offs, whereas variable selection models argue that they have been historically better at some times, in some places, or in some frequencies.

One might argue that optimal trade-offs affected by personality traits likely vary across settings and circumstances. This reasonable observation, however, does not by itself constitute strong evidence that genetic variability has been maintained by variable selection. Adaptation to changing optima can be achieved through contingent alterations based on circumstances (whether heritable or not).

**Purported Signatures of Balancing Selection**

Penke et al. (2007a) presented a number of arguments for why variable selection of various forms (collectively, “balancing” selection), and not mutation-selection balance, has maintained genetic variation in personality. (In fact, their comparison did not explicitly consider stabilizing selection; they considered mutation-selection balance only with respect to traits under directional selection.) Specifically, they argued, variation under balancing selection should often involve polymorphic genes with intermediate frequencies, each with moderate effects. By contrast, mutation-selection balance implies the effects of relatively many genetic loci that are only rarely highly polymorphic, each with weak effects (see also Mitchell-Olds et al., 2007). Penke et al. (2007) pointed to specific polymorphisms, notably the DRD4 (dopamine receptor D4) gene’s effects on novelty-seeking or impulsivity, as evidence that, indeed, personality is affected by variation on genes with intermediate allele frequencies. In addition, they argued, variable selection is more likely to leave substantial residual non-additive genetic effects than is mutation-selection balance. These arguments deserve closer scrutiny.

*Are there major single-gene effects on personality?* In fact, probably not. Candidate gene studies yield few reliable associations with personality traits, and associations that are reliable are, in all likelihood, exceptionally small. Hence, for instance, a meta-analysis yielded no robust associations between 16 candidate genes and major personality dimensions once controls were instituted (Munafò et al., 2003). Later meta-analyses on two relatively promising candidates revealed little evidence of robust associations of 5-HTT (LPR genotype) with anxiety-related traits (Munafò et al., 2009) and evidence of, at best, weak associations between DRD4 (C-521T polymorphism) and novelty-seeking and impulsivity (accounting for, at best, 3% of the phenotypic variance on these traits; Munafò et al., 2008).

Recent genome-wide scans offer no greater promises. Gillespie et al. (2008) detected no significant linkages with the four Eysenck dimensions. And perhaps most impressively, Shifman et al. (2007), utilizing an extreme-group design on a very large sample, failed to find any loci accounting for >1% of the variance in neuroticism, despite having about 50% power to detect an effect accounting for 5–1% of the variance. They concluded,
“Since we failed to find any loci accounting for more than 1% of the variance, the heritability of neuroticism probably arises from loci each explaining much less than 1%” (p. 302, emphasis added).

This situation appears little different from the morphological trait of height. Many robust associations have been found between specific markers and height, given that extremely large sample sizes are available (e.g., Perola et al., 2007), but the top 20 loci identified collectively account for less than 3% of the variance (Martin, 2009). In sum, the available evidence strongly suggests that genetic variance in personality is due to cumulative weak effects of many loci and, hence, does not speak in favor of a substantial role for balancing selection.

Are polymorphic genes with effects on personality common? Examples of polymorphism exist: For instance, the 7-repeat allele of DRD4 associated with ADHD has a relative frequency of about 20% in European populations (e.g., Hattori et al., 2009). But are they common? In fact, it’s hard to say; as noted above, studies have not generally identified single genes with large effects on personality. In a genome-wide search designed to assess whether there are regions in the human genome that are highly polymorphic, aside from MHC and ABO blood group regions, Bubb et al. (2006) found no more additional one than expected under a neutral model—that is, no convincing evidence that balancing selection with deep historical roots has maintained high levels of polymorphism in genomic regions, suggesting that “long-term balancing selection may simply be rare in humans” (p. 2175). Obviously, polymorphisms at functional sites do exist (e.g., Baysal et al., 2007; Fumagalli et al., 2009; Cagliani et al., 2008). In light of Bubb et al.’s analysis, most of these instances may be due to recent balancing selection, incomplete selective sweeps, or near-neutrality. As might be expected, recently discovered ones generally pertain to disease resistance and may be isolated examples (e.g., Hurst, 2009). By yielding no evidence for widespread regions of polymorphic genes in the human genome, however, Bubb et al.’s data also weighs against Tooby and Cosmides’s (1990) pathogen-driven frequency-dependent selection model of personality, which expects widespread levels of polymorphism.

Does the level of non-additive genetic variance in personality traits favor balancing selection? Penke et al. (2007a) argue that the mixture of additive and non-additive variance is pertinent to deciding between mutation-selection balance and balancing selection models. There are two forms of non-additive effects. Dominance variation is due to statistical interactions between alleles at the same locus (such that the effect of one allele depends on what other allele it is paired with). Epistatic effects are due to interactions between allelic variations at different loci. If variation is due to mutations at many loci, each at low frequency, there is little opportunity for variation to be dominance variation, as individuals homozygous for mutations at a locus (a subset whose presence is necessary for dominance effects at the locus) are extremely rare. And simple two-locus additive × additive epistatic effects will be largely measured as additive variance as well when one allele at either locus is rare (Hill et al., 2008). Mutation-selection balance, then, predicts relatively little non-additive variance in a trait. Balancing selection that maintains polymorphisms can, in theory, produce substantial amounts of non-additive genetic variation. Personality traits do appear to possess non-negligible amounts of non-additive genetic variance. For instance, in a sample of over 45,000 individuals, Lake et al. (2000) estimated that, of the total genetic variance in neuroticism (53% and 45% for women and men, respectively), about 30% is non-additive; similarly, Rhee and Waldman (2002) estimated that about 20% of the genetic variance in antisocial behavior is non-additive. Eaves et al. (1998, 1999), Keller et al. (2005), and
Rettew et al. (2008) similarly found evidence for non-additive variance in some personality traits (notably, extraversion and neuroticism), probably largely due to epistasis rather than dominance. 4

4 In fact, precise estimates of additive and non-additive genetic variance are not possible, as different forms of non-additive effects (e.g., epistasis varying in complexity) have different effects on phenotypic similarity of family members. As well, some small amount of correlation between MZ twins could, in principle, be due to shared non-genetic epigenetic effects (e.g., shared effects of developmental noise), which could lead non-additive effects to be slightly overestimated. See, e.g., Bouchard & Loehlin (2001).

Are these non-additive variance estimates inconsistent with mutation-selection balance? In a commentary on Penke et al. (2007a), Keller (2007) argued that given our current state of knowledge, presence or absence of modest levels of non-additive variance are not sufficient to argue that they are a signature of any particular form of historical selection; in response, Penke et al. (2007b) acquiesced. Though Hill et al. (2004) argue that loci with effects due to rare alleles (e.g., mutations) yield mostly additive variance, they acknowledged that they can produce modest non-additive effects.

At this time, then, the signatures of variable selection that Penke et al. (2007a) offer as evidence that it has maintained variation in personality appear to be largely lacking. Of course, lack of evidence for a process does not imply presence of strong evidence against it. The hypotheses put forward by Nettle (2006), Penke et al. (2007a), and others are important and worthy of future efforts to identify and test predictions that follow from them.

GENETIC EFFECTS ON FITNESS IS NON-ADDITIVE

Dominance and Epistatic Effects on Fitness

Selection removes additive genetic variance associated with main effects of alleles on fitness. Interactive effects on fitness are not typically removed by selection. As selection removes additive genetic variance, it leaves a residual of non-additive variance, leading to the expectation that fitness itself will have substantial non-additive variance (e.g., Merila & Sheldon, 1999). As noted above, when effects on fitness are largely due to the cumulative effects of rare mutations, much variance in fitness will be additive. Fitness traits (e.g., longevity, lifetime fertility rate) in animals typically have a good deal of additive genetic variance, even when non-additive genetic variance is present (e.g., Houle, 1992).

Non-additive effects on fitness, however, need not be non-additive effects on other phenotypic traits. Suppose that allelic variation at a locus has purely additive effects on a personality dimension (such that unit increases in number of copies of one particular allele—0 to 1, 1 to 2—have equal effects on the trait). If the trait is under pure stabilizing selection (with mean values optimal), then the effects of these allele changes on fitness are partly non-additive: The effect of an increase from, say, 1 to 2 alleles will depend on other genes the individual carries. If the individual carries many alleles at other loci that predispose low values on the trait, possessing alleles at the target locus that increase trait values (moving the individual toward the mean) increases fitness. By contrast, if the individual carries many alleles at other loci that predispose high values on the trait, possessing those same alleles at the target locus that increase trait values (moving the individual further from the mean) decreases fitness. Effects on the trait are additive; effects on fitness are partly epistatic. Selection against alleles at loci affecting fitness via effects on single traits under stabilizing selection can be very weak partly for this reason (e.g., Crow, 1986). A weakly deleterious allele can
become fairly frequent through random drift, a possible explanation for why some genes with (weak) effects on personality (e.g., the DRD4 tandem repeat polymorphism) are polymorphic (Hattori et al., 2009).

Non-additivity no doubt plays some role in the maintenance of genetic variation in traits. In light of the lack of widespread polymorphism in the human genome, however, it likely does not explain the bulk of variation, leaving substantial roles for mutation-selection balance or variable selection.

Selection Varies Across the Sexes

As already discussed, selection removes variations if it is consistent across time, space, and allele frequencies; if selection is not consistent, variation may be maintained. Yet another way selection can be inconsistent is if one allele at a locus is favored when the carrier is a male and another allele at the locus when the carrier is a female. This condition is referred to as intralocus sexually antagonistic selection.

If selection is stronger in one sex than the other, it will ultimately drive out one allele. But selection can be very weak when selection operates differently for the sexes. Moreover, in some circumstances selection may move allele frequencies to the point at which selection is equal in the two sexes (see Rice & Chippindale, 2001). And, while selection should favor genetic modifiers that suppress expression of an allele disfavored in sex (yielding sex-limiting effects), the evolution of such modifiers can be slow. Studies on a number of species have demonstrated that genetic variations in male and female fitness (or fitness components) are negatively correlated (e.g., Chippindale et al., 2001; Foerster et al., 2007; Brommer et al., 2007; Delcourt et al., 2009; see review by Bonduriansky & Chenoweth, 2009).

At a phenotypic level, sexually antagonistic selection can prevent each sex from reaching its optimum on a trait. Individuals are a mosaic of what has been selected in its own sex and what has been selected for in the other sex (Rice & Chippindale, 2001). And because genetic variation within each sex persists, many individuals possess trait values biased toward what is better suited for the opposite sex.

Sexually antagonistic selection can be thought of as a special case of non-additive effects on fitness. In this instance, the effect of an allele on fitness depends on the sex of the individual. In placental mammals, sex in itself is a function of a particular gene on the Y chromosome, SRY. Hence, sexually antagonistic selection results from interaction effects on fitness of a particular gene and the SRY gene.

Little work has been done on examining the effects of sexually antagonistic selection in humans. In one seminal study, Garver-Apgar (2008) found evidence for sexually antagonistic selection on sexual attractiveness. Across within-sex male and female sibling pairs, sexual attractiveness covaries positively. But in cross-sex sibling pairs, attractiveness of individuals of one sex fails to predict attractiveness of individuals of the other sex. (For possible effects on human disease, see Ober et al., 2008.)

Sexually antagonistic selection could maintain genetic variation in personality traits that are sexually dimorphic, suggestive of different optima (ancestrally) for the two sexes. Manning et al. (2000) propose that within-sex variations in the 2nd to 4th digit ratio (possibly effects of prenatal androgens) may be maintained by intralocus sexually antagonistic selection. Recently, Loehlin et
al. (2009) reported that sexually dimorphic digit ratios correlate (albeit very weakly) with a measure associated with aggressivity and impulsivity (Eysenck’s Psychoticism Scale) in both sexes. Possibly, some within-sex variation on these traits is due to selection acting in opposite directions in the two sexes. As this factor is a major component of Figueredo et al.’s (2005) life history factor (see above), intralocus sexually antagonistic coevolution offers an explanation for some genetic variance along this dimension.

**Many Ideas, Few Empirical Tests**

**Do Empirical Data Support Any One Scenario Over Others?**

Evolutionary biology offers a wide range of possible explanations for genetic variance. Scholars have drawn on this literature to propose a plethora of explanations for the existence of genetic variation in personality. But what data, if any, support any particular view over others?

At this point, there perhaps exist no particularly strong arguments for one particular view as the primary process responsible for the genetic variance in human personality. Certain personality variations associated with psychopathological syndromes (schizophrenia, autism) are associated with deleterious mutations (e.g., CNVs), favoring mutation-selection balance as at least one potent reason for these variations (see also Keller & Miller, 2006). But “normal” personality variations have substantial genetic variance too. What processes explain that variance? And, granting that a number of different processes probably operate to some degree, what are their relative impacts?

As discussed, Penke et al. (2007a) argued that the genetic architecture of normal personality variations contains signatures of historical variable selection: moderate-sized effects of single loci, effects by genes with relatively high levels of polymorphism, and significant amounts of non-additive genetic variance. As also noted, data on the polymorphism of relevant loci is largely lacking, data on non-additive variance lack clear implications, and data on moderate effects of single loci are generally contrary to what Penke et al. (2007a) claim should be expected from variable selection.

Resolution of these issues demands new discriminating tests of the possibilities.

**Associations Between Developmental Instability and Personality**

Recently, Jeff Simpson, Randy Thornhill, and I have attempted to test one set of predictions derived from one form of mutation-selection balance. Low-level mutations may have, as byproducts, effects on developmental instability—the imprecise expression of developmental design. Fluctuating asymmetry (or FA: absolute right-left asymmetry on bilateral traits that are symmetrical at the population level) is the most widely used measure of developmental instability (e.g., Møller & Swaddle, 1997). Consistent with the idea that mutations affect developmental instability, Carter et al. (2009) found that inbred lines of fruit flies had greater FA of wing dimensions than outbred ones. Inbreeding increases mutational effects in offspring by substantially increasing the number of loci that are homozygous for mutations. A “double-dose” of a mutation at a particular site (the mutation in a homozygous state) typically has deleterious effects on fitness much greater than double the deleterious effect of a “single dose” of the mutation (the mutation in a heterozygous state). Human developmental instability appears to be moderately heritable (e.g., Johnson et al., 2008).

If a trait partly reflects the effects of low-frequency mutations, then, one might expect it to covary with FA. This line of reasoning led researchers to examine associations between FA (measured as
a composite of multiple traits’ unsigned asymmetries) and psychometric intelligence, \( g \). Several studies have found that FA negatively predicts \( g \) in Western samples (Furlow et al., 1997; Prokosch et al., 2005; Luxen & Buunk, 2006; Bates, 2007), though others report mixed (Rahman et al., 2004) or no (Johnson et al., 2007) evidence for an association (see also Euler et al., 2008). (On average, \( r \) is .2 to .3.) More studies are needed, but if the association is robust, this effect may reveal that \( g \) is partly a function of rare mutations, and that genetic variance in \( g \) is due to mutation-selection balance of variants affecting a fitness trait. A number of proximate processes are consistent with this scenario: \( g \) may be directly diminished by instability of brain development (a DI-mediated model), developmental instability may be a byproduct of rare mutations that directly affect gene expression in the brain and compromise its performance (a direct causal model), or investment in brain growth and maintenance underlying \( g \) may be contingent on condition, as broadly affected by mutations (a condition-dependent model).

If personality variations similarly represent (ancestral) fitness traits on which variation is maintained by mutation-selection balance, personality traits too might be associated with FA. Several studies have examined associations between FA of facial features and personality. One replicable finding emerges: A negative correlation between extraversion and FA (Fink et al., 2005; Pound et al., 2007; Shackelford & Larsen, 1997, found mixed results, but their sample size was small), consistent with extraversion being a fitness trait ancestrally, its variance partly maintained by the effect of mutations on trait levels. By contrast, Luxen and Buunk (2006) detected no robust associations between body FA (asymmetry of ears, ankles, wrists, fingers, elbows) and Big Five traits, and in unpublished data neither did we (see below). Future work might examine more closely specific variations in social potency or vigor rather than the broader trait of extraversion.

But what if personality traits have been under stabilizing, not directional, selection, with variance due to mutation-selection balance? No linear association can be expected, but a related approach can be applied. If individuals at the extremes of a trait dimension tend to have rare mutations, which have as byproducts effects on developmental instability, then FA should relate to the quadratic component of trait variance, with trait extremes being associated with high FA. (Such an association exists with 2nd to 4th digit ratios; extreme ratios relate to high FA [Manning et al., 2006].) Simpson, Thornhill, and I examined these associations using measures of Big Five traits in three samples of men, with a total \( N \) over 250. This study has not yet undergone critical peer review. I present our results as merely illustrative of findings that, if robust, are consistent with mutational variation subject to stabilizing selection.

Within each sample, we calculated partial correlations between each trait and the quadratic component of the trait (trait values squared), with linear effects of trait values controlled. For each trait, we estimated the mean effect across samples. Averaged across all traits, they were positive: Individuals at the extremes of Big Five measures possessed greater FA than did individuals close to mean values. But effect sizes varied widely across traits: highly robust for agreeableness (for which the mean partial \( r \) was about .3), significant as well for conscientiousness and openness (with effects roughly half that for agreeableness), weakly and non-significantly positive for neuroticism, and near-zero for extraversion. We detected no consistent linear associations between Big Five measures and FA.

If these findings replicate, they are consistent with the idea that at least some personality variations have been subject to stabilizing selection, maintained by mutation-selection balance.
One possibility is that developmental instability itself results in variable personality outcomes, sometimes leading to extreme levels. Consistent with this idea, Rose et al. (1987) and Bogel et al. (1994) found that identical twins with greater developmental instability are less similar in personality than those with lower developmental instability. Alternatively, rare mutations both affect personality outcomes and have, as byproducts, effects on developmental instability. These processes are not mutually exclusive.

**Summary**

What do we know about the evolutionary processes that account for the existence of genetic variations expressed in human personality?

1. Psychologists have drawn upon a rich (though still evolving) literature in evolutionary genetics to generate a variety of plausible possible explanations for these genetic variations.

2. Currently, however, few data permit firm conclusions about which processes have been particularly important. In all likelihood, personality traits have been subject to stabilizing selection against extremes. But is the variability within intermediate regions of distributions due to ancestral mutation-selection balance or ancestral variability in selection? If the latter, what forms of variability? Recent genome-wide scans reviewed might weigh against widespread effects of variable selection. Though cultural differences (e.g., as a function of geography) exist, they are weak. Together, these data might suggest that balance between mutation and stabilizing selection has likely been a major reason personality variation persists. But firm conclusions are premature. Moreover, different models may apply to different traits. (E.g., sexually antagonistic selection may be important to sexually dimorphic traits.)

3. Novel tests are needed. As illustration, I presented one attempt to assess a particular mutation-selection model. As our ability to measure specific genetic variants in the genome more widely increases, our ability to perform novel tests will undoubtedly increase. Behavior genetics has become increasingly molecular, and technological capabilities to assess the genome will understandably push research further in that direction—but, as Bouchard (1994) noted, behavior genetics will become an explanatory discipline when it brings evolutionary reasoning to bear on its findings. Indeed, perhaps the same could be said of personality psychology.

**References**


Johnson, W., Segal, N. L. & Bouchard, T. J., Jr. (2007). Fluctuating asymmetry and general intelligence: No phenotypic or genetic association. *Intelligence*. 368


13 Are Pleiotropic Mutations and Holocene Selective Sweeps the Only Evolutionary-genetic Processes Left for Explaining Heritable Variation in Human Psychological Traits?

Geoffrey F. Miller

We evolutionary psychologists pride ourselves on applying the latest evolutionary biology to illuminate human nature. Yet most of us have not kept up with the last decade’s astounding progress in human evolutionary genetics. We’re still focused on kin selection, reciprocity, sexual selection, and costly signaling as ways to explain the psychological adaptations that (supposedly) don’t vary much across people. But when it comes to explaining individual differences, we have not yet discerned how 21st century evolutionary genetics clarifies heritable variation in cognitive abilities, personality traits, or psychopathologies. Those of us over age 35 especially need the humility to acknowledge that genetics Ph.D. students typically know more than we do about the state of the art in multivariate behavioral-genetic modeling, how to run genome-wide association studies using DNA chips, or how to make inferences about ancestral selection pressures from molecular-genetic data.

I’m no exception. Until this sabbatical year when I started trying to catch up, I had no idea what I was missing. I assumed, like many evolutionary psychologists, that vague memories of out-dated undergraduate biology classes, plus some acquaintance with genetic correlations, life-history trade-offs, and frequency-dependent selection, would suffice to understand individual differences. Now I think we need to do better. We have been blind-sided by new genomic technologies, databases, and theories. These are only somewhat relevant to explaining universal psychological adaptations, but they are crucial to explaining heritable variation in psychological traits.

Here’s a little test—a few basic questions that might appear on a typical graduate course exam in human evolutionary genetics (e.g. one based on the excellent textbook by Jobling, Hurles, & Tyler-Smith, 2004). Consider how many you can answer coherently.

1. Explain the evolutionary importance of the different types of mutations, including CpG transitions and transversions, indels, microsatellites, L1 and Alu retrotransposons, and segmental duplications.

2. Explain the effects of gene conversion and genetic admixture on linkage disequilibrium.

3. Explain how increased male reproductive variance affects the effective population sizes and genetic drift rates of X, Y, autosomal, and mitochondrial genes.

4. Explain the five main measures of selective neutrality: the McDonald-Kreitman test, Tajima’s D, the HKA test, $O_0$, and H.

5. Explain how ‘wombling’ can help detect genetic boundaries in phylogeography.

If you scored only 3 out of 5, that’s 60%, a D-. Yet this is the sort of material that we evolutionary psychologists need to master—and to teach to our own students. We can’t rely anymore on the view that heritable individual differences are just genetic noise arising as a side-effect from host-parasite coevolution (Tooby & Cosmides, 1990). This re-tooling will be tough,
but it’s our job as the self-appointed disseminators of Darwinian theory in the behavioral sciences. If evolutionary psychologists don’t make the connections between current evolutionary genetics and individual differences research, who will? So far, the other likely candidates—behavioral genetics, psychiatric genetics, and clinical neurogenetics—have not been filling the gap.

This is our disciplinary challenge, and once we face it, we immediately confront a daunting puzzle: Most human psychological traits show far more heritable variation than would be expected if trait variation depended on just a few genes of major effect, and if evolution imposed stabilizing selection favoring a single optimal value of the trait (Carey, 2002; Pagel & Pomiankowski, 2007; Plomin, DeFries, McClearn, & McGuffin, 2008). This is true for all three main classes of psychological traits that are stable, heritable, widely predictive, and cross-culturally universal:

- **Personality traits** such as the Big Five—openness to experience, conscientiousness, extraversion, agreeableness, and emotional stability (Bouchard & Loehlin, 2001; John, Robins, & Pervin, 2008; Matthews, Deary, & Whiteman, 2004; McCrae, Terracciano, et al. 2005; Miller, 2009)—and more specific traits such as sexual promiscuity (Gangestad & Simpson, 2000) and political engagement (Fowler & Schreiber, 2008);

- **Psychopathology traits**, including the general dimensions of internalizing and externalizing (Krueger & Markon, 2006), and more specific dimensions such as the schizophrenia spectrum (Shaner, Miller, & Mintz, 2004; Sullivan, Kendler, & Neale, 2003), autism spectrum (Shaner, Miller, & Mintz, 2008; Veenstra-VanderWeele, Christian, & Cook, 2004), and psychopathy spectrum (Markon & Krueger, 2005; Moffitt, 2005);

- **Cognitive traits** such as general intelligence (in the sense of the g factor arising from the all-positive correlations among mental abilities—Deary, Whalley, & Starr, 2008; Jensen, 1998), and its subordinate factors such as verbal ability, spatial ability, creativity (Kaufman, Kozbelt, Bromley, & Miller, 2007), social intelligence (Emery, Clayton, & Frith, 2008), emotional intelligence (Matthews, Zeidner, & Roberts, 2004), and mating intelligence (Geher & Miller, 2007).

A central question for any Darwinian analysis of such a trait is: what evolutionary processes have maintained the trait’s surprisingly high heritable variation? The simplest answers require an equilibrium assumption—that all of the alleles underlying the trait’s current genetic variation have been at some sort of evolutionary equilibrium for at least the last several hundred generations (such that the trait’s current heritability, genetic correlations, and other quantitative features perfectly reflect their pre-Neolithic values). Assuming equilibrium, then there are just three key possibilities: The trait’s genetic variation is fitness-neutral, or adaptive, or maladaptive (Keller & Miller, 2006; Mitchell-Olds, Willis, & Goldstein, 2007; Penke, Denissen, & Miller, 2007). Each possible explanation is discussed in turn below; after that, we’ll see what happens if we relax the equilibrium assumption.
Perfect Neutrality: Implausible For Psychological Traits That Predict Anything Interesting

The perfect neutrality model for any given trait posits that the trait’s variation is exactly fitness-neutral. This means that trait has had no significant fitness consequences in any domain of life (survival, growth, mate attraction, fertility, parenting, or socializing) across recent generations. In principle, mutations are free to accumulate in fitness-neutral traits, potentially yielding the heritable variation that we see today. In practice, traits are only fitness-neutral if they are subject to a selection coefficient smaller than $1/N_e$, where $N_e$ is the effective population size that represents the effects of genetic drift (Jobling, Hurles, & Tyler-Smith, 2004). This $N_e$ is estimated to be about 10,000 for ancestral hominids (Eyre-Walker & Keightley, 2007), so any trait that decreases reproductive success by even 0.01% (1/10,000) would not have been neutral. It would have been eliminated by selection.

Such a perfect degree of fitness-neutrality is implausible for all human traits that psychologists care about. This is because traits that don’t predict behavioral outcomes in any domain of life are not considered to have any predictive validity, so don’t attract any scientific attention. The traits that we want to understand—personality, psychopathology, and cognitive traits—are studied precisely because they do predict success, failure, or variation in some important life-domains (Buss & Greiling, 1999; Nettle, 2006), as shown by a formidable range of empirical research (Deary, Whalley, & Starr, 2008; Jensen, 1998; Krueger & Markon, 2006; Matthews, Deary, & Whiteman, 2004).

Balancing Selection: Three Empirical Problems That Arose In The Last Few Years

The balancing selection model posits that the trait’s variation is adaptive. According to this model, the trait had fitness consequences and was under selection, but the optimal trait value varied across space, time, ecology, population, age, sex, health, social status, mate value, and/or some other contextual variable. If each observed trait value had exactly equal average fitness payoffs under different circumstances, selection could have maintained a polymorphic mixture of alleles underlying trait variation (Gangestad & Yeo, 1997). Special cases of this phenomenon include frequency-dependent selection (different alleles are favored depending on their commonality versus rarity), host-parasite coevolution (different alleles help defend the organism against different fast-evolving parasites), sexually antagonistic co-evolution (different alleles are favored in males versus females), and speciation (different alleles are favored in different breeding populations, such that separate species form).

Balancing selection is an ideologically attractive way for liberal academics to explain individual differences: It suggests equal evolutionary adaptiveness across psychological variants, so it seems to validate the full range of human psycho-diversity. Inspired by evolutionary game theory models of alternative stable strategies (Vincent & Brown, 2005), evolutionary psychologists have often used balancing selection to explain heritable variation in human psychopathology (Mealey, 1995), human personality (Nettle, 2005), and animal personality (Nettle, 2006). In an earlier paper (Penke, Denissen, & Miller, 2007)
my co-authors and I suggested that balancing selection may explain heritable variation in the Big Five personality traits. However, I’m not so confident any longer, since balancing selection has three key empirical problems that have only become apparent in the last couple of years.

**Problem 1: The Failure of Genome-wide Association Studies (So Far....)**

In evolutionary theory, balancing selection can maintain only a small number of genes with moderate to strong effects, such that most of the genetic variation is concentrated on a small number of loci (Kopp & Hermisson, 2006). Functionally, we might expect that traits maintained by balancing selection should evolve to be controlled by one or a few major polymorphic loci (Penke, Denissen, & Miller, 2007), and these key loci should evolve to act as master developmental switches. For example, the sex ratio is maintained by balancing selection, so sexual differentiation in mammals evolved to be controlled by just one master gene, the SRY gene on the Y chromosome. Also, the biochemical variation underlying immune system defenses are under (frequency-dependent) balancing selection against fast-evolving pathogens, so these variants have evolved to be controlled by a localized cluster of about 140 ‘major histocompatibility’ (MHC) genes on chromosome 6, spanning about 3.6 Mb between the flanking markers MOG and COL11A2 (Piertney & Olivier, 2006). Consistent with balancing selection, MHC diversity is higher in human populations exposed to higher pathogen loads (Prugnolle, Manica, Charpentier, Guegan, Guernier, & Balloux, 2005). In general, balancing selection creates an ‘allelic spectrum’ biased towards a few high-frequency alleles at each of one or a few major loci (Reich & Lander, 2001).

We might expect similar outcomes for any psychological traits under balancing selection: Genes of major effect should be easy to find in linkage and association studies, especially in the genome-wide association studies (GWASs) that seemed so promising in the early 2000s (Stoughton, 2005). GWASs of complex human traits are becoming ever more successful in identifying a few loci per trait that might lead to useful biomedical investigations of diseases associated with that trait. However, GWASs of complex human traits have been very disappointing so far in the proportion of genetic variance that the identified loci explain—typically less than 2% (Maher, 2008; Weiss, 2008). The Affymetrix Genome-Wide Human SNP Array 6.0, a widely-used DNA chip for GWASs, can identify 1.8 million genetic markers for each individual’s genotype, including about 900,000 single nucleotide polymorphisms (SNPs) and about 950,000 copy number variants (www.affymetrix.com).

So far, despite intense GWAS efforts in the last four years, even the most enthusiastic reviews (e.g. Altshuler, Daly, & Lander, 2008) note that only about 150 out of these million-odd SNPs have shown any reliable associations with any human trait or disease. For example, a high-profile *Nature* paper with over 150 co-authors, which claimed to represent “a thorough validation of the GWA approach” and which has been cited more than 600 times in the 18 months since publication, actually found only 24 SNPs (out of 500,000 sampled) that showed any statistically significant associations with any of 7 major mental and physical diseases (Burton, Clayton, Cardon, Craddock, Deloukas, Duncanson, et al. 2007). The few replicated alleles that have been found in GWASs account for only a tiny percentage of trait variance, even when they are aggregated. This is true for the morphological trait of height (Visscher, Macgregor, Benyamin, Zhu, Gordon, Medland, et al, 2007), and for the psychological traits of intelligence (Butcher, Davis, Craig, & Plomin, 2008), and the Big Five personality traits (Gillespie, Zhu, Evans, Medland, Wright, & Martin, 2008; Terracciano, Sanna, Uda, Deiana, Usala, Busonero, et al.,
2008; Wray, Middeldorp, Birley, Gordon, Sullivan, Visscher, et al. 2008). Such elusive alleles are not what we would expect from traits under balancing selection. More direct genetic methods have also found very few loci outside the MHC complex that seem to have been under balancing selection (Bubb, Bovee, Buckley, Haugen, Kibukawa, Paddock, et al. 2006; Hendrick, 2006).

The GWAS revolution is still very much underway, and some replicable genetic variants will be found sooner or later that, in aggregate, might explain 5% to 10% of the heritable variance in some psychological traits. Yet even the most ardent GWAS researchers recognize that there is a big problem of ‘the missing heritability’ (Maher, 2008): If most psychological traits are at least moderately heritable, why is it proving so hard to find the specific genes that account for their heritability?

**Problem 2: Pervasive Inter-correlations and Fitness-related Correlations**

Traits maintained by balancing selection should not correlate very much with each other, if they were shaped by disparate selection pressures favoring different polymorphic strategies in distinct domains of survival and reproduction. For example, if extraversion variance reflects a balanced trade-off between sexual benefits and accident risks (as suggested by Nettle, 2005), but openness variance reflects a balanced trade-off between out-group social interaction benefits and out-group pathogen-infection dangers (as suggested by Schaller & Murray, 2008), and if those four factors (sexual benefits, accident risks, social benefits, pathogen dangers) did not reliably co-vary under ancestral conditions, then extraversion should not be correlated with openness.

Yet recent evidence suggests that all psychological traits show at least modest inter-correlations (ranging from less than r = .1 for single test items to r = .3 or so for higher-level aggregate scales). These correlations all seem to be positive if traits are measured on a worse-to-better scale of quality, whether indexed by social attractiveness, sexual attractiveness, social status, academic grades, economic success, or reproductive success (at least in natural fertility populations). For instance, the all-positive inter-correlations among cognitive abilities give rise to a general intelligence (g) factor (Jensen, 1998). Similar hierarchical factor models also seem necessary for both personality traits and psychopathology traits. If the Big Five personality traits are not forced into an orthogonal factor rotation, they show weak but generally positive correlations, and these can be best represented by two higher-order factors of Stability (spanning conscientiousness, agreeableness, and emotional stability) and Plasticity (spanning openness and extraversion) (DeYoung, 2006; Digman, 1997), which themselves are positively correlated, yielding a single General Factor of Personality (GFP) (Figueroedo, Vasquez, Brumbach, & Schneider, 2007; Musek, 2007; Rushton, Bons, & Hur, 2008; Rushton & Irwing 2008, in press). However, this GFP is clearly not as strong as the g factor: It doesn’t explain nearly as high a proportion of variation in the Big Five as g does for cognitive traits, at either the phenotypic or genetic levels (Yamagata, Suzuki, Ando, Ono, Kijima, Yoshimura et al., 2006). Also, debate continues about whether this GFP is an artifact of socially desirable responding or a genuine superordinate trait with predictive validity (McCrae, Yamagata, Jang, Riemann, Ando, Ono et al., 2008), and whether this GFP correlates with intelligence (Gladden, Figueredo, & Jacobs, 2008).

Similarly, the widespread comorbidities across categorical psychopathologies can best be represented by hierarchical dimensional models in which personality disorders reflect extremes of the Big Five personality traits (Markon, Krueger, & Watson, 2005; Widiger & Trull, 2007), and
other disorders reflect high values on ‘externalizing’ and ‘internalizing’ dimensions, which are themselves positively correlated (Krueger & Markon, 2006).

Even across the domains of personality, psychopathology, and intelligence, phenotypic correlations are ubiquitous. For example, general intelligence correlates positively with openness to experience at both the phenotypic and genetic levels (Wainwright, Wright, Luciano, Geffen, & Martin, 2008); neuroticism (reverse-scaled emotional stability) correlates positively with the internalizing (or ‘negative affectivity’) dimension of psychopathology at both the phenotypic and genetic levels (Clark, 2005; Hettema, Neale, Myers, Prescott, & Kendler, 2006); and the externalizing dimension seems to reflect a combination of low intelligence, low conscientiousness, and low agreeableness (Lynam & Widiger, 2007; Saulsman & Page, 2004; Vitacco, Neumann, & Jackson, 2005).

So, some of the emerging evidence suggests that all major dimensions of human individual differences may fit into a unified hierarchical factor model, in which a general ‘fitness factor’ (representing general genetic quality) is superordinate to all three factors of \( g \), the GFP, and mental health (Keller & Miller, 2006; Miller, 2007). This general fitness factor also seems to be superordinate to developmental stability as manifest in body symmetry (Prokosch, Yeo, & Miller, 2005) and physical attractiveness (Zebrowitz & Rhodes, 2004), and to general physical health as manifest in longevity (Deary et al., 2008) and fertility (Arden, Gottfredson, Miller, & Pierce, 2009). However, debate continues about relationships among \( g \), the GFP, mental health, physical health, developmental stability, and sexual attractiveness. Nonetheless, without positing a hierarchical fitness model that represents a substantial portion of psychological and physical variance, it is very hard to explain apparent ‘good genes’ mate choice for mental traits among humans, since a single ‘goodness’ dimension implies a general fitness factor in psychometric analysis, and a general ‘mate value’ factor in subjective judgment (Miller & Todd, 1998; Neff & Pitcher, 2005).

**Problem 3: Adaptive Flexibility of Behavior Is Often Better Than Hard-wired Variation**

For clever, big-brained primates like us, balancing selection seems especially weak at explaining heritable variation in psychological traits. Suppose there is some ecological or social domain that favors different strategies under different conditions. The alternative strategies could evolve as ‘hardwired’ genetic polymorphisms, or as ‘softwired’ developmental trajectories sensitive to early environmental cues during some sensitive period, or as flexible behavioral tactics that remain sensitive to current environmental cues throughout life (Figueredo, Vasquez, Brumbach, Schneider, Sefcek, Tal, et al., 2006). Hardwired polymorphisms make sense for alternative strategies that require a very early developmental commitment to growing a particular phenotype, such as a male or female body, but they make less sense for behavior. A key function of big, clever brains is to register a huge array of current environmental cues when deciding what to do (Tooby & Cosmides, 2005).

Humans are clearly flexible enough to do so with regard to most classic dimensions of psychological variation across individuals. When cognitive challenges increase, we can think longer, harder, and more sombrely, increasing our effective intelligence (Andrews, Aggen, Miller, Radi, Dencoff, & Neale, 2007); when over-learned habits will suffice, we can afford to act
automatically and unconsciously. Likewise, when a new situation calls for a different personality, we enter altered states of consciousness called emotions that instantiate the required temperament: Angry (disagreeable) if fighting, friendly (extraverted) if socializing, free-spirited (open and impulsive) if courting. If we already have emotions that are adaptively flexible and environmentally contingent; why do we need personality traits, which can be seen as genetic biases in the relative frequency of experiencing different emotions and having different act propensities (Buss & Craik, 1983; Fleeson, 2001)? The flexibility of emotional states argues against the adaptive utility of heritable personality traits. More extreme situations can even evoke temporary states that resemble psychopathologies: short-term depression after failures, short-term psychosis during trances, or short-term obsessiveness during hunting, gathering, or grocery-shopping.

In some sense, the highest-fitness humans may not have stable cognitive, personality, or psychopathology traits—only an exquisitely adaptive flexibility in matching one’s current response mode to one’s current environmental challenges. The question then becomes: What evolutionary benefit could arise from hardwiring such behavioral flexibility at the level of a genetic polymorphism? Any situation that would impose balancing selection on the behavioral strategies of a small-brained insect should, among big-brained primates, simply favor the evolution of new psychological adaptations—new ways of being sensitive to environmental variables. Obviously, short-term adaptive flexibility cannot explain heritable variation in personality traits, but it casts doubt on facile arguments about balancing selection for psychological traits.

**Pleiotropic Mutations: The Last Equilibrium Model Left Standing?**

The pleiotropic mutation model posits that the trait’s variation is maladaptive. The trait has fitness consequences, and selection favors a certain optimal value or range on the trait, but the trait is so polygenic (depends on so many alleles at so many genetic loci) that harmful mutations are constantly eroding genetic quality, and creating deviations from the optimal trait value (Zhang & Hill, 2005). The few mutations with strongly harmful effects and high penetrance (e.g. dominance)—that is, major Mendelian disorders—are quickly eliminated by selection. However, the many mutations with very slightly harmful effects and weaker penetrance (e.g. that are partially or fully recessive) are under much weaker purifying selection, so persist much longer in the population (Eyre-Walker & Keightley, 2007). The result is that every human carries at least several hundred old, mildly harmful mutations inherited from previous generations, plus at least a few new mutations, mostly due to spermatogenesis errors in the father (Ellegren, 2007). These mutations are surprisingly common in humans, and range in size from single nucleotide polymorphisms (SNPs) such as insertions, deletions, and changes in single DNA base-pairs (Boyko, Williamson, Indap, Degenhardt, Hernandez, Lohmueller, et al. 2008; Gorlov, Gorlova, Sunyaev, Spitz, & Amos, 2008), through tandem repeats, segmental duplications, and copy number variants for longer stretches of DNA, and larger inversions and translocations of chromosomal segments, up to whole-chromosome aneuploidies (e.g. trisomy-21) and uniparental disomies (UPDs) (Feuk, Carson, & Scherer, 2006). Traits may be affected by mutations not only in classic protein-coding regions of the genome (‘exons’), but also in regulatory and promoter regions before and after genes (Keightley, Lercher, & Eyre-Walker, 2005; Wray 2007), and in regions that code for various non-coding RNAs that
coordinate gene regulation (Amarall, Dinger, Mercer, & Mattick, 2008; Bartel, 2004). Traits subject to pleiotropic mutation should be influenced by rare, evolutionarily transient variants across a very large number of loci, which should produce mostly additive genetic effects in aggregate (Hill, Goddard, & Visscher, 2008), and which should prove very difficult to find in GWAS studies (Gorlov et al., 2008; Keller & Miller, 2006; Weiss, 2008). Thus, the pleiotropic mutation model can more easily explain the hierarchical structure of fitness and fitness components (Keller & Miller, 2006; Miller, 2007).

**Evolution Out Of Equilibrium**

A big problem with all three equilibrium models—perfect neutrality, balancing selection, and pleiotropic mutation—is that human traits are unlikely to have been at evolutionary equilibrium for the past several hundred generations. New genetic evidence shows that human evolution did not stop in the Pleistocene, but has accelerated throughout the Upper Paleolithic (50k to 10k years ago) and the Holocene (the last 10k years) (Hawks, Wang, Cochran, Harpending, & Moyzis, 2007; Kelley & Swanson, 2008; Meisenberg, 2008; Neilsen, Hellmann, Hubisz, Bustamante, & Clark, 2007; Nettle & Pollet, 2008). Selection pressures cannot have remained the same after the human dispersals out of Africa and the Upper Paleolithic revolution, especially since the rise of agriculture, domestication, money, and institutionalized monogamy. These changes may not have had enough time to produce complex, new, cross-culturally universal psychological adaptations (Andrews, Gangestad, & Matthews, 2002; Tooby & Cosmides, 2005), but they could have had dramatic effects on the patterns of genetic variance underlying human personality, psychopathology, and cognitive traits.

**Cryptic Genetic Variation Uncovered by Modern Environmental Complexity?**

The emergence of cities, complex cultures, stratified societies, and divisions of labor probably created many new social and sexual niches in which new and more diverse psychological traits could thrive. The proliferation of these new niches may have uncovered huge amounts of cryptic genetic variation (Gibson & Dworkin 2004) in psychological traits—variation that was not manifest phenotypically under ancestral conditions, but that is manifest under more diverse modern conditions. For example, polymorphic alleles that now create heritable variation in openness to experience may have been hidden away in the Pleistocene, when there simply weren’t many new ideas, values, or experiences towards which one could be open or closed. The degree of openness manifest in prehistory may have been tightly constrained by the relative simplicity, conservatism, and insularity of small-scale hunter-gatherer societies (Marlowe, 2005). In effect, openness could have been developmentally canalized under ancestral conditions (Flatt, 2005). With no variation manifest in actual openness, any genetic loci that influenced potential openness in prehistory may have been invisible to selection, and polymorphisms could have accumulated at those loci (Schlichting, 2008). The result can be described with a ‘capacitor model’ (Le Rouzic & Carlb, 2008; Moczek, 2007): The latent trait of openness becomes ‘genetically charged’ with heritable variation under Pleistocene conditions, and this variation can become manifest and ‘discharged’ in novel environments, as when human cultures became more complex, dynamic, and permeable in the Holocene.

However, there are two serious problems with this cryptic variation model for explaining major human psychological traits. First, it is really a special case of the perfect neutrality argument, which doesn’t work for traits that we care about. Cryptic variation could accumulate in prehistory only if it had no fitness consequences in any domain of life. For example, prehistoric propensities
towards higher openness would not have been fitness-neutral if they had any effect on the
likelihoods of migrating to new habitats, mating with out-group members, learning new survival
or courtship skills, or trying new foods or drugs (cf. McCrae, 1996). If heritable variation in the
trait influenced development, survival, mating, or parenting in any way, it could not have
accumulated as truly cryptic variation. Second, even if cryptic variation became apparent only
since the rise of Holocene civilizations a few thousand years ago, there would have been plenty
of time for selection since then to shape current patterns of variation. Assuming an average 25-
year generation time, five thousand years is about 200 generations. Previously cryptic alleles that
turn out to impose even a 1% decrement in lifetime reproductive

success in the Holocene will be reduced from 50% prevalence to 5% prevalence in 100
generations, and will virtually vanish within 200 generations (see Keller & Miller, 2006, Figure
2). Overall, cryptic genetic variation in important psychological traits is unlikely to have
accumulated in a fitness-neutral way in prehistory, and even if it did, it is unlikely to have persisted under historical selection pressures.

Cryptic variation may have played one important role in recent human evolution—it could, in
principle, have provided a vast reserve of potentially adaptive variants that could prove their
fitness under evolutionarily novel conditions (Barrett & Schluter, 2008). When our ancestors
faced the new challenges of Holocene living, they did not have to wait around for new mutations
to accumulate. Cryptic variation, decanalized by new environmental conditions, could have
offered a great head-start for genetic adaptation to civilized life. It just would have been subject
to intense new natural and sexual selection pressures from that point onwards.

**Ongoing Selective Sweeps Since the Pleistocene**

The last 50,000 years of human evolution are likely to have been driven by massive changes in
selection pressures (Meisenberg, 2008), increased genetic admixture across populations
(Klimentidis, Miller, & Shriver, in press), and an increased number of potentially beneficial
mutations per generation due to dramatically increased population sizes (Hawks, Wang, Cochran,
Harpending, & Moyzis, 2007). These changes would have affected not only the human
populations that launched the Upper Paleolithic revolution and the rise of civilizations, but all
populations that experienced any gene flow from those populations—including contemporary
hunter-gatherers.

An obvious example concerns the possibility of genetic admixture between expanding human
populations and indigenous archaic hominid populations such as Asian Homo erectus (Cox,
Mendez, Karafet, Pilkington, Kingan, Destro-Bisol, et al., 2008), European Neanderthals
(Weaver & Roseman, 2008), or indigenous African ‘Rhode-sioids’ (Brauer, 2008). Genetic
models suggest that our human ancestors could easily have ‘poached’ any useful genes that had
independently evolved among other hominid lineages, and any such genes that gave even a small
selective advantage could have swept through not only local human populations, but all human
populations, assuming moderate rates of migration and gene flow (Garrigan & Kingdan, 2007;
Hawks, Cochran, Harpending, & Lahn, 2008). The genetic and fossil evidence concerning
Neanderthal admixture is mixed (pro: Hawks et al., 2008; Trinkaus, 2007; con: Hodgson &
Disotell, 2008), but completion of the Neanderthal Genome Project should clarify the issue.

More recent selective sweeps probably accompanied both the Upper Paleolithic revolution (c.
30,000 years ago in Europe) and the Holocene emergence of complex civilizations in the last
10,000 years. These cultural revolutions produced rapid expansions of human population, spatial range, ecological diversity, and social complexity, which created thousands of new ecological, economic, social, and sexual niches. These in turn must have imposed strong, dynamic, and local selection pressures that affected human gene frequencies in different populations.

For example, with the invention of money as a form of ‘liquid fitness’ came the possibility of sexual selection for wealth—and for any psychological traits that facilitated its acquisition, investment, arbitrage, and defense (Clark, 2007; Ellis, 2001; Nettle & Pollet, 2008). Ruling-class males in some populations attained unprecedented reproductive success through bride-capture, harems, slaves, prostitutes, and mistresses, perhaps driving rapid selective sweeps for certain traits associated with leadership, belligerence, oppressiveness, rapaciousness, and risk-taking (Earle, 2002; Summers, 2005). Very recent increases in the anonymity and mobility of some societies may have created more reproductively rewarding niches for psychopaths (Mealey, 1995; Moffitt, 2005). Conversely, religiously imposed monogamy in some populations may have increased selection for fidelity, conscientiousness, agreeableness, and parental investment (Miller, 2007), and may have greatly increased the strength of assortative mating for personality and cognitive traits, thereby amplifying genetic variance in those traits (Hooper & Miller, 2008).

With each Holocene selective sweep in favor of some new higher-fitness alleles, other phenotypic side effects and linked loci would be carried along to higher population frequencies, which may explain genetic variation in apparently unrelated traits.

New mutations that happen to have beneficial effects on one trait are likely to have pleiotropic effects on other traits that reduce their net fitness benefits by a factor of two, on average (Otto, 2004). For example, if selection in favor of general intelligence suddenly became more intense in some populations, this could have favored the spread of new IQ-boosting alleles even if those alleles have a range of harmful side-effects on physical or mental health that would have been counter-selected under Pleistocene conditions (Cochran, Hardy, & Harpending, 2006). Hypothetically, the same alleles that increase intelligence through modified sphingolipid metabolism and lipid storage in the brain could also disrupt a variety of bodily organs and tissues, perhaps creating higher risks of Tay-Sachs, Canavan disease, Niemann-Pick disease, torsion dystonia, and mucolipidosis type IV. The result would be increased heritable variation in these side effect traits while the selective sweep is underway. Medical geneticists focused on the neurological disorder of torsion dystonia (painful muscle contractions resulting in uncontrollable distortions of posture and limb position) might then wonder how the genetic risk factors for this disease could have persisted ancestrally, and might speculate about hidden adaptive benefits to weird postures—without realizing that the disease is an evolutionarily transient side effect of strong recent selection for intelligence.

Also, given one new mutation favored by selection, any nearby loci on the same chromosome would be carried along to higher frequencies due to linkage disequilibrium. This genetic hitchhiking would spread not just a single allele, but a whole haplotype (set of alleles in linkage disequilibrium, which is broken down only slowly by genetic recombination), and the linked genes could increase the observed variance in physical or psychological traits that are functionally unrelated to the favored trait. This linked-loci effect is absolutely fundamental to the entire HapMap project, which traces the structure of haplotypes across human populations (International HapMap Consortium, 2007). For example, an ongoing selective sweep in favor of
an openness allele could, in principle, increase the frequency of a schizotypy allele that is a few million base pairs away on the same chromosome. The result would be a phenotypic and genetic correlation between openness and schizotypy (e.g. Miller & Tal, 2007), which could easily lead the unwary to posit a balancing selection model in which the fitness benefits of openness balance out the fitness costs of schizophrenia. Indeed, any such haplotypes during a selective sweep would tend to show a complex mix of fitness costs and benefits that could easily be mistaken for the outcome of balancing selection.

Both of these effects—linked loci that create haplotype divergence across and within populations, and harmful pleiotropy that reduces fitness even in traits that are genetically correlated with favored traits—can produce substantial maladaptive genetic variance. The stronger selection has been in recent human evolution, the stronger these maladaptive side-effects are likely to have been, potentially explaining a lot of imperfection in human bodies and minds.

Genetic variance created by recent selective sweeps would, like that created by pleiotropic mutations, show an elusive molecular-genetic basis. Any alleles with very strong positive fitness effects would sweep to fixation rapidly in any given population, so would not leave behind much genetic variance. The alleles that do still explain genetic variance because they are still undergoing a sweep are likely to have milder fitness effects, so will show weaker—or perhaps undetectable—effect sizes in GWAS studies. Moreover, the specific alleles favored in Holocene sweeps may differ across populations, so any GWAS associations that do emerge may not replicate across populations. Finally, any haplotypes undergoing a sweep may contain thousands of genetic variants that might be detected in GWASs, but most of those will have no functional relationship to the trait being favored; they will simply be in linkage disequilibrium with the functionally relevant alleles. Here again, without a good understanding of the evolutionary genetics, GWAS studies of human intelligence, personality, and psychopathology may continue to yield results that are rare, weak, and unreplicable, and that explain only a small percentage of genetic variance.

**Conclusion**

If we take seriously the notion that human evolution has continued and accelerated in the Holocene, then we have to rethink two foundational assumptions of traditional evolutionary psychology: (1) the Pleistocene ‘environment of evolutionary adaptedness’ (EEA) as the most recent relevant evolutionary environment for understanding individual differences, and (2) evolutionary equilibrium models (perfect neutrality, balanced selection, and pleiotropic mutation) as the most relevant theories for explaining heritable variation in human traits. Our most important psychological traits are likely to have been subject to strong selective sweeps (rapid increases in the frequency of new fitness-increasing alleles) due to increased population density (Hawks, Wang, Cochran, Harpending, & Moyzis, 2007), more intense social and sexual competition (Summers, 2005), more virulent pathogens (Schaller & Murray, 2008), more technology-associated hazards (Gottfredson, 2007), and more selective and assortative mating (Miller, 2000). Evolutionary theorists don’t yet know how to combine the genetic models that assume evolutionary equilibrium, and the genetic models relevant to human populations that have been evolving faster than ever in the last few hundred generations. In the next few years, the main challenge in evolutionary personality psychology will be to develop the relevant evolutionary
genetic models that accept the importance of ongoing post-Pleistocene human evolution, and to understand and test their distinctive empirical predictions.

If pleiotropic mutations and Holocene selective sweeps do turn out to explain most individual differences in psychological traits, this would have implications for future research at several levels. At the molecular-genetic level, we might expect that most variation in intelligence, personality, and mental health traits will arise from differences in overall mutation load (Keller & Miller, 2006), rather than from allelic variants at just a few loci (as expected from balancing selection models, and as assumed by most current gene-hunting methods, including GWAS searches for SNPs and copy number variants). At the neurogenetic level, we might expect overall mutation load to influence general neurodevelopmental stability (Prokosch, Yeo, & Miller, 2005), as assessed by overall brain size, anatomical typicality, neurochemical typicality, precision of neuronal growth patterns, and/or neural conduction velocities or signal-to-noise ratios, rather than expecting particular alleles to have clear effects on specific cortical areas, neurotransmitter systems, or fiber tracts. At the psychometric level, we might expect all reliably measurable psychological traits to have some position within a hierarchical structure that spans intelligence, personality, and psychopathology, and that encompasses aspects of psychological functioning that were previously pigeon-holed as ‘cognition,’ ‘emotion,’ ‘motivation,’ and ‘consciousness.’ Finally, at the sociological level, a renewed appreciation that mutation load and recent selection are important in explaining human psychodiversity might lead researchers to reconsider a range of issues in moral philosophy, socio-political ideology, and bioethics. We may need to find radical new ways to reconcile the empirical facts about human diversity with our classical liberal values of equality, meritocracy, democracy, and multiculturalism. In short, evolutionary genetics is undermining the old anthropological assumption about ‘the psychic unity of mankind,’ and we better learn the scientific and moral sophistication to deal constructively with the consequences.

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References


Personality differs considerably among individuals within a population and affects many facets of an animal’s life, including how it solicits resources from its parents, explores and selects habitats for foraging and breeding, socializes with conspecifics, finds and guards mates, and raises its offspring (Gosling, 2001; Réale et al., 2007; Réale & Dingemanse, 2010). Depending on their personalities, humans too are subject to different experiences throughout their lives for similar broad situations (Nettle, 2006; Buss, 2009b). One puzzle for both behavioral ecologists and evolutionary psychologists is to understand why we observe the preponderance of consistent individual differences in behavioral responses to such a wide variety of challenges (Gosling, 2001; Penke et al., 2007; Réale et al., 2007; Buss, 2009b).

Evolutionary psychologists have traditionally looked at human behavior from an adaptive point of view, by considering the adaptive function of certain behaviors as well as their underlying psychological mechanisms, and by assuming unlimited adaptive plasticity 1

\[ 1 \text{ Unlimited means here that an individual has the potential to express the whole phenotypic variation of a trait in its population.} \]

...to cope with challenges of any type of environmental situation (see Wilson, 1994; Buss, 2009a). Behavioral ecologists have used a similar approach to study the behavior of animals (Dall et al., 2004; Réale & Dingemanse, 2010), an approach that could be referred to as the adaptationist approach. An important difference between behavioral ecologists and evolutionary psychologists, however, is that the latter group assumes that current human behavior and its underlying psychological mechanisms only make sense in the light of particular ancestral environmental conditions (so called “Environment of Evolutionary Adaptedness” (EEA); Symons, 1992; Crawford, 1998). In contrast, behavioral ecologists assume that both past and current selection pressures have shaped current behaviors (Krebs & Davies, 1998). To behavioral ecologists, the EEA is thought to correspond to general conditions that, over a large number of generations (i.e., thousands), have favored the evolution of a particular adaptation that has therefore now been conserved (i.e., the canalization effect of selection: Gibson & Wagner, 2000). As a result, humans would share common psychological mechanisms that enable them to respond to a particular situation in a unique way (Tooby & Cosmides, 1992). Behavioral ecologists have also used an optimality (i.e., adaptationist) approach and considered similar assumptions in their research on animals and humans, even though they do not explicitly mention the EEA as a reference. More specifically, behavioral ecologists normally assume that a population is at equilibrium (a set of conditions that lead to the absence of evolutionary change and result in stable allele frequencies in populations). Under this assumption, they can test their adaptive hypotheses by means of experimental manipulation in current populations; an animal should change its behavior following a change in the environment or in its state, in a way predicted by the adaptive hypothesis. In contrast, it seems that the notion of evolutionary equilibrium is not an important condition for the evolutionary psychology framework.
Although the EEA approach has some advantages when studying human adaptive invariants (or “strategies”), it does not provide any explanation for the existence of consistent individual differences and heritable variation in personality traits (van Oers et al., 2005; Penke et al., 2007; Réale et al., 2007). Such an adaptationist explanation thus misses an important feature of many human behavioral traits in general, and personality traits in particular: The existence of substantial heritable variation despite the presumed effect of a history of erosion due to past selection pressures (Wilson, 1994, 2002; Penke et al., 2007; Buss, 2009b).  

It should be noted that most types of traits have been shown to present some consistent individual differences and heritable variation.

It is thus necessary to reconsider the adaptationist approach to explain the existence of phenotypic variation and the notion of optimality.

To answer this question of why individual variation might persist over evolutionary time (i.e., thousands of generations), despite homogenizing selective forces, theoretical evolutionary biologists have recently started to model situations where selection pressures maintain—instead of erode—individual and genetic variation in behavior (Dall et al., 2004; Wolf et al., 2007, 2008). These models are thereby starting to provide empirical biologists with theory to be tested using empirical data (Réale, 2007). One of the most fruitful and popular approaches in current evolutionary biology (Endler, 1986; Brodie et al., 1995; Kingsolver et al., 2001; Blows, 2007) to testing how selection pressures maintain the genetic variance of personality traits is to use what is called the “selectionist approach” (also known as multivariate selection analysis; Lande, 1979).

In this paper we discuss the potential benefit of using the selectionist approach to study human and animal personality traits. We will first briefly introduce the selectionist approach, describe the methods proposed to run multivariate selection analyses, and follow with a review of the most recent results showing how animal personality scientists are beginning to apply this method to test the fit of evolutionary scenarios for the maintenance of variation in personality traits. Finally, we will review the evidence from recent studies on human personality that suggest that selection can indeed affect personality dimensions. Not every scenario will apply equally to humans and animals. However, we assume that humans and many animals broadly face the same types of ecological challenges that they must cope with. Therefore, studies on both human and non-human animals are likely to provide a general explanation for the maintenance of individual variance in personality traits.

The selectionist approach

The selectionist approach aims to reveal the strength and the shape of selection acting on phenotypic traits, the main process responsible for adaptive evolutionary changes. The process generally consists of estimating the covariance between a series of phenotypic traits and fitness within a single generation (Brodie et al., 1995). As such, this approach has the advantage of embracing the potential complexity of how selection acts on multi-trait organisms, which is exactly the approach necessary for evaluating evolutionary hypotheses.

The approach sometimes turns out to be disadvantageous because of methodological limitations associated with the study of multiple traits and the constant development of more and more complex statistical techniques (e.g., Blows, 2007).
We intentionally use the term selection in its broadest sense in order to include both natural (i.e., measured from individual differences in survival) and sexual (i.e., measured from individual differences in mating/reproductive success) selection pressures potentially affecting the traits of interest.

An individual’s personality is generally composed of multiple traits or factors (Réale et al., 2007). We will thus focus on methods enabling researchers to evaluate how selection acts on a set of traits. Selection happens within one generation, through differential survival and/or reproduction among individuals of a population. A phenotypic selection study essentially evaluates the shape and the strength of direct and indirect selection acting on traits during a single episode of selection (Arnold & Wade, 1984a,b; Brodie et al., 1995; Endler, 1986; Hersch & Philips, 2004; Lande & Arnold, 1983). Phenotypic traits are generally correlated to fitness to estimate the strength of selection acting on the traits. Lifetime reproductive success (LRS), the contribution of an individual (in terms of offspring) to the next generation, is often used as an index of fitness (Clutton-Brock, 1987). However, LRS is the measure of the overall contribution of an individual at the end of its lifetime. LRS thus only provides a general outcome of all the actions of selection on one trait over the lifetime of an organism. Selection can, however, act in an antagonistic way on a given trait at different life stages or on two related traits. One might therefore consider different fitness indices representative of successive life stages, such as survival to reproductive maturity, age at sexual maturity, fecundity, reproductive longevity, post-reproductive longevity, or mating success (Arnold & Wade, 1984b).

Directional selection is shown by a linear relationship between phenotypic variation of a trait and fitness. This relationship can also be curvilinear and show that selection favors intermediate phenotypic values of the trait at the expense of the extremes, in which case selection is said to be “stabilizing.” Inversely, selection can favor the extremes of the distribution over the average of the population (i.e., disruptive selection), or favor combinations of traits (i.e., correlational selection). Indices of selection are needed to predict the evolutionary response of a set of traits, which is typically done using Lande’s equation: \( \Delta z = G b \), where \( \Delta z \) represents the vector of changes in the average value of a set of traits from one generation to the next, \( G \) is the matrix of additive genetic variance/covariance for those traits (i.e., the additive genetic variance of a trait represents the additive genetic effects of multiple genes, each gene being assumed to have a small effect, Lynch & Walsh 1998), and \( b \) represents the vector of selection gradients acting on each of these traits (Lande, 1979). Additive genetic variance is the only genetic variance responsible for phenotypic changes in a population over time (Lynch & Walsh, 1998) and is therefore the only one considered in \( G \). This equation shows that evolutionary changes (i.e., \( \Delta z \)) and selection (i.e., \( b \)) represent two different processes (although the former depends on the latter) and that, in the absence of additive genetic variance (i.e., \( G = 0 \)), selection will not induce an evolutionary response (though we should note that most personality traits in humans and animals appear to be heritable and therefore to show non zero additive genetic variance; van Oers et al., 2005; Réale et al., 2007; but see Dingemanse et al., 2009 for an example of environment-specific heritable variation in personality traits). More generally, the characteristics of \( G \) capture any constraints, caused by pleiotropic effects (i.e., the effect of one gene on several phenotypic traits) or by linkage disequilibrium (i.e., the non-random association of alleles from two or more genes, potentially generating genetic correlation between two phenotypic traits) on
the potential evolution of the traits under study (Blows & Hoffmann, 2005). A large number of methods are available to estimate $\mathbf{G}$ in animals or humans, and we invite the readers who would like to get more information on these methods to consult the following studies: Roff (2000) and Steppan et al. (2002). In this paper we are mainly interested in the estimation of $b$.

Directional selection gradients $b_i$ of a trait $i$ are estimated with partial regression coefficients in a multiple regression of fitness, $w$, as a function of a set of traits measured on individuals in a population. A directional selection gradient reflects the change in the mean phenotypic value of a trait resulting from direct selection on this trait, while holding the effects of other traits constant (Arnold & Wade, 1984b; Lande & Arnold, 1983). One can obtain standardized selection gradients that enable comparison of the strength of selection between different traits or for the same trait between years, environmental conditions, or populations, when phenotypes are standardized (expressed in units of standard deviation) and the fitness of individuals is expressed in relative terms (by dividing individual fitness values by the fitness of the average individual in the population; i.e. Arnold & Wade, 1984b; Lande & Arnold, 1983). It is also possible to estimate the disruptive/stabilizing selection gradient, $g_{ii}$, of a trait $i$ as twice the quadratic regression coefficient of that trait (Lande & Arnold, 1983; Fairbairn & Reeve, 1999; Stinchcombe et al., 2008). In the same way, fitting the interaction between two traits $i$ and $j$ allows estimation of the correlational selection gradient, $g_{ij}$ (Arnold & Wade, 1984a, b; Lande & Arnold, 1983). Correlational selection gradients can reveal cryptic evolution; that is, cases where selection cannot be detected when focusing on a single trait because selection acts directly on the correlation between two traits (Blows, 2007).

The phenotypic selection approach proposed by Lande and Arnold (1983) can become a real challenge when one has a large set of traits to analyze. Alternatives are available such as the diagonalization of $\mathbf{G}$ or $g$ matrices (Blows 2007) or the use of structural equation modeling. The latter method is well known in psychology and permits the estimation of any type of selection shape while decreasing effects of potential environmental bias (see Scheiner et al., 2000, 2002).

Finally, selection gradients estimated on the phenotypic values can be biased by environmental effects. If, for example, the environment is affecting both fitness and the expression of a trait (i.e., via phenotypic plasticity) in the same direction, spurious correlations between a trait and fitness would result that would not reflect selection in action (Hadfield et al., 2010).

**Potential benefits of a selectionist approach to the study of personality**

One might ask why it is important to study selection on human personality traits. Our goal here is not to impose a new system of reference to evolutionary psychology, but rather to propose useful tools and concepts that could broaden our ability to understand human and animal personality variation. There are several aspects of potential interest in this context borrowed from the selectionist approach (see Table 14.1).

First, selection is the foundation for the theory of evolution (Darwin, 1859) and it is thus necessary to study selection acting on different traits, in different species, populations, and conditions, if we are to understand evolutionary processes. If current adaptations partly result from past selection, future adaptations must be partly shaped by current selection pressures. The selectionist approach offers a formal way of testing evolutionary hypotheses (Fairbairn & Reeves, 1999), and in particular several selectionist scenarios
Table 14.1. Reasons for applying the phenotypic selection approach to human and animal personality traits.

1. Provides standardised measures of the strength and the shape of selection acting on traits that can be compared across traits and studies.

2. Allows comparison of direct and indirect selection pressures acting on different (correlated) traits, between the sexes at different life stages, periods or locations, or in different populations.

3. Allows a formal way of testing adaptive scenarios to explain the maintenance of personality traits.

4. Provides a way of testing for the permanence of selection pressures in current populations.

5. Provides a way of estimating the relative importance of gene flow versus selection on the evolution of local adaptations when analysed jointly with molecular genetics data.

6. Enlightens certain phenomena in modern societies that are related to public health issues related to the maintenance of variation in personality traits (Penke et al., 2007; Réale et al., 2007). For instance, frequency-dependent selection, fluctuating selection due to spatial or temporal heterogeneity in the environment, life history trade-offs, and selection acting on trait correlations have all been proposed as evolutionary reasons for the maintenance of the great diversity in personality found in animal and human populations (Wilson et al., 1994; Ellis et al., 2006; Penke et al., 2007; Réale et al., 2007; Wolf et al., 2007; see details in section IV). Furthermore, some studies have suggested that the maintenance of variance in personality traits might be a by-product of selection on other traits (Nettle 2006; Penke et al., 2007). Importantly, formal tests of alternative scenarios for how selection acts to maintain this variation are still very rare in animals (summarized in section VI) and almost absent in humans (summarized in section VII).

Second, the potential antagonistic effects of local selection and gene flow due to immigration has been one of the main explanations for the maintenance of genetic variation in local populations, and the absence of evolutionary equilibrium in many animal populations (Slatkin, 1985; King & Lawson, 1995; Storfer & Sih, 1998; Postma & van Noordwijk, 2005; Moore et al., 2007). Understanding the link between personality traits, dispersal, and local selection pressures in humans may help us better understand the dynamics of changes in personality traits (Dingemanse et al., 2003; Cote & Clobert, 2007). Furthermore, by using phenotypic selection analyses on human personality in different cultural contexts, it might be possible to reveal certain invariant aspects of selection acting on human personality as well as local adaptations that jointly shape similarities and differences in personality observed among different cultures (section VII). For example, Schaller and Murray (2008) analyzed differences in personality between countries and showed that variation in mean extraversion and openness scores were negatively related to the prevalence of infectious diseases, suggesting that infectious diseases represent a strong selective factor on personality dimensions. We should thus expect selection against extraversion and openness to be strong in countries with high prevalence of infectious diseases, but negligible in countries less affected by these diseases.

Third, evolutionary psychology generally assumes that the EEA does not extend to very recent evolutionary times (i.e., the last few generations). As a consequence, evolutionary psychologists have advocated that the study of the adaptive function of human behavior should be considered
within the framework of the EEA, and that studying the effect of behavior on fitness in current societies does not provide useful information on the adaptive value of behavior (Symons, 1992; Tooby & Cosmides, 2005; for a review of different approaches on human behavior see Smith, 2000). Two views are worth considering in this context, and the selectionist approach can offer interesting solutions to tests hypotheses in both cases. One hypothesis is that for many human behavioral adaptations, the current environment may not be that different than the EEA (Crawford, 1998; Winterhalder & Smith, 2000). The selectionist approach would be a very appropriate way to test this hypothesis (see Nettle & Pollet, 2009, for an empirical test of the question). Alternatively, selection pressures in the EEA may differ from those of the current environment (Tooby & Cosmides, 2005). If so, humans might be subject to so-called “evolutionary traps,” like animal species that currently inhabit urbanized environments (Schlaepfer et al., 2002). In such environments, populations that have been shaped by selection according to past environmental conditions do not fit with the environmental conditions they currently inhabit; thus strong selection should exist, and the selective approach will provide information on the current selection pressures acting on human traits. More generally, with the accumulation of estimates of selection (e.g., on different personality traits in different populations) it will be possible to test for the degree of invariance in the selection pressures acting on different types of traits and to investigate the prevalence in the permanence of selection pressures in current populations.

Finally, selection studies on personality traits in humans are valuable because they might reveal any potential mismatch between the personality of the average individual and the current environmental conditions. Selection being generally measured in terms of survival and reproductive abilities, the selectionist approach could enlighten some phenomena in modern societies that are related to public health issues. Selection only improves fitness of an organism, not its welfare, and as a result an adaptive behavioral strategy can cause distress or put individuals in dangerous situations. For example, too much impulsivity can drive a person to take inconsiderate risks, which in our current societies can lead to extremely violent and deleterious behavior (Daly & Wilson, 2005).

Selection and the maintenance of variance in personality traits

We can use the phenotypic selection approach to formally test alternative hypotheses proposed to explain the maintenance of genetic variance of personality traits (see for example Penke et al., 2007; Réale et al., 2007; Buss, 2009b). Here we provide a brief overview of the way to evaluate the fit of these hypotheses. For all methods outlined below, we assume that some information on both personality and fitness of individuals is available in the population, for example, through the long-term survey of marked or recognizable individuals.

Fluctuating selection. This type of selection occurs when heterogeneity (e.g., temporal or spatial, including frequency-dependent selection) in the environment creates antagonistic selection pressures on a trait within a generation or between successive generations. There has been some controversy about the potential effect of fluctuating selection on the maintenance of genetic variance on the long term. However, theoreticians have argued that fluctuating selection can help maintain genetic variance in species with overlapping generations (e.g., Sasaki et al., 1997). If fluctuating selection exists, we would expect selection gradients to vary between geographic areas or years (Fig. 14.1A), or between different (social or non-social) micro-environments.
within the same population at the same time (for examples see section on evidence for selection on personality in wild animals). Ideally, differences in selection pressures should be linked to fluctuations in specific environmental characteristics. For example, in great tits (Parus major) and red squirrels (Tamiasciurus hudsonicus) it seems that selection favors active-aggressive females when food resources are abundant, but disfavor them under low food abundance (Dingemanse et al., 2004; Boon et al., 2007). This relationship between food resources abundance and personality is assumed to result from the positive link between metabolic rate and activity-aggressiveness and from how individuals with different energy metabolisms cope with the abundance or the rarity of resources (Boon et al., 2008; Careau et al., 2008). Importantly, if enough estimates across subpopulations or over time were available, we might expect the total action of selection to average out (i.e., a flat fitness surface implying no net selection).

**Frequency-dependent selection.** Frequency-dependent selection, a special case of heterogeneous selection, happens when the fitness outcome of a phenotype or strategy depends on its frequency in the population relative to other phenotypes or strategies. Negative frequency-dependent selection refers to a situation where individuals with relatively rare phenotypes show the highest fitness. In order to test for the presence of frequency-dependent selection, one would ideally measure selection by experimentally modifying the proportion of individuals of different phenotypes (in case of discrete phenotypes, or the proportion of artificial subclasses in case of continuous traits) in different experimental populations, and then estimate the covariance between fitness and the trait in the different populations. Such an experiment has been performed with blotch-sided lizard (Uta stansburiana) color morphs by Sinervo and Lively (1996). We acknowledge that this will be impossible to realize in human populations. However, descriptive data may also be used where the proportion of individuals with a certain personality type varies naturally either within or between countries or across a number of years (e.g., Camperio-Ciani this volume). If negative frequency-dependent selection exists, we would expect to observe a negative relationship between selection gradient.
Figure 14.1. Different scenarios for how selection acts on personality traits: A) fluctuating selection. The different regression lines represent the covariation between a personality trait Z and relative fitness w in a given population over successive years or for different subpopulations characterized by different environmental conditions. The selection gradient (i.e., the measure of the strength of selection acting on trait Z) differs according to the year or the subpopulation, maintaining the variance in Z over the long term. B) Frequency-dependent selection. Selection gradient b on a personality trait is measured in several social environments (open circles) differing in the proportion of individuals expressing a certain phenotype A for the trait Z. In this situation frequency-dependent selection should be detected by a negative relationship between b and the proportion of individuals in each environment that expresses the phenotype A. A positive b indicates that selection favors individuals showing A, whereas a negative b indicates that selection disfavors them. Note that for continuous traits the proportion of individuals expressing A can be replaced by the average value of Z in the population in each social environment. C) Life-history trade-offs. The direction of selection on Z changes with life-history stage (i.e. early or late) at which it is measured. D) Antagonistic selection between the sexes. Selection favoring high values of Z in one sex simultaneously favors low values of Z in the other sex.

on trait Z and the proportion of individuals of a specific phenotype of trait Z in the population (Fig 14.1B).

Life-history trade-offs and the maintenance of variation in personality. Very recently it has been shown theoretically (Wolf et al., 2007) and empirically (Biro & Stamps 2008; Réale et al., 2009)
that personality differences can be construed as facets of different life-history strategies. Such an idea has also been pursued in the field of evolutionary psychology (Figueroedo et al., 2005; Penke et al., 2007; Buss, 2009). For example, aggression and boldness might be associated with fast growth, early maturation and reproduction, and short lifespan, whereas less aggressive, shy individuals might enjoy highest fitness when they extend their developmental period, delay their reproduction, and extend their life spans. Other traits such as metabolic rate are assumed to have coevolved with personality and life-history strategies (Careau et al., 2008; Biro & Stamps, 2008). More generally, personality differences are more and more considered to represent alternative adaptive strategies coexisting within the same population (Figueroedo et al., 2005; Nettle, 2006; Buss, 2009; Réale & Dingemanse, 2010). In the case of an association between personality traits and life-history strategies, we should expect antagonistic selection pressures on personality traits at different life stages. For example, selection favoring high value for a trait early in life should favor low value of that trait later in life (Fig. 14.1C). We also should expect that environmental or cultural conditions favoring the evolution of a given population towards a particular life-history strategy (e.g., delayed reproduction) should also lead to a parallel evolution of personality. This expectation should be reflected by the covariation between personality and life history among populations.

**Antagonistic selection between the sexes.** Antagonistic selection can help maintain variation in traits when the same genes affect the traits of males and females (i.e., traits are genetically correlated between the sexes) but selection acts differently across sexes (e.g., favoring high values in males but low values in females). This hypothesis has been supported for some traits in wild populations (Foerster et al., 2008). In this case, we should expect that selection in favor of males with a high phenotypic value for a given personality trait would favor females with a low phenotypic value for that same trait (Fig. 14.1D). Importantly, testing the validity of this hypothesis involves both the estimation of sex-specific selection pressures as well as the cross-sex genetic correlation of the trait of interest, for example using pedigree analysis (Brommer et al., 2007).

**Correlational selection.** This cryptic form of selection occurs when individuals with particular combinations of trait values do best. Correlational selection can lead to a fitness “ridge” where a particular set of multivariate phenotypes have the same fitness. For instance, aggressive/bold or non-aggressive/shy individuals might live longer and/or reproduce more than both aggressive/shy and non-aggressive/bold ones, with the former two categories performing equally well. Such a scenario would produce a “flat” ridge of high fitness which could allow genetic variation in both traits to persist. Brodie (1993), for instance, documented such a fitness ridge when estimating selection acting on antipredator behavior and color patterns in the garter snakes (*Thamnophis ordinoides*). Correlational selection might not only act on two traits expressed in the same individual (as outlined above), but might also act on two traits expressed in related individuals (Wilson & Réale, 2006; Smiseth et al., 2008). This latter case can happen for example when one trait is expressed in the parent (e.g., maternal performance) and the other is expressed in the offspring (e.g., solicitation) (Mousseau et al., 2000). Although correlational selection is straightforward to estimate (i.e., the interaction term between two traits in a multiple linear model; see above) it represents a particular statistical challenge in terms of sample size and power (Kingsolver et al., 2001).
Selection, evolutionary response and the limits of the selectionist approach

Changes in the environment can lead to rapid evolutionary responses in a population, re-shaping the adaptive value of alternative behavioral phenotypes. Estimates of selection gradients coupled with estimates of the genetic variances in a population in principle enable one to predict the potential direction of evolution (see earlier section on the selectionist approach). Selection affects the characteristics of a population by changing the frequency of alleles involved in the variation of a trait, provided that the trait under selection is heritable. Changes in the mean or the variance of a trait in a population across successive generations may reflect changes in allele frequency associated with this trait. The impact of selection at the genetic level should in principle be effective across at least two successive generations. However, the ability to detect such an evolutionary change depends on the power of our analyses in relation to the size of the sample analyzed. Furthermore, genetic constraints could limit the independent evolution of traits, whilst low genetic diversity can hamper response to selection (Blows & Hoffmann, 2005). Low genetic variation for a trait under selection can reduce the fitness of individuals and increase the risk of extinction of the population. In such a case, current selection might be largely irrelevant for understanding past adaptations. Cryptic evolutionary response may also occur as a result of the antagonistic effect of concurrent environmental changes (Merilä et al., 2001). In such a case changes at the genetic level (e.g., in the frequencies of genes influencing a trait) may be hidden by stronger and opposite changes in the plastic response of a trait following environmental changes. As a result, changes in the trait at the phenotypic level over time may not reflect its changes at the genetic level. This particular mechanism indicates that selection could happen with or without visible evolutionary response and that showing selection does not provide a demonstration for the evolutionary response of a trait to selection (Lande, 1979).

At the same time, the effects of selection at the phenotypic level may not be obvious, although recent studies have shown that less than ten generations can sometimes be enough to detect visible evolutionary changes (Grant & Grant, 1995; Hendry & Kinnison, 1999; Réale et al., 2003). Such findings contradict the common idea that it is impossible to study evolutionary consequences of selection within an individual’s lifetime. Notwithstanding, such changes represent minor evolutionary changes representing the first steps of adaptation to local conditions, and differ by far from evolutionary novelties or speciation.

A further limit of the multivariate selection approach is its restricted power to detect weak selection pressures. Very low—albeit invariant—selection pressures may not be detected as such by a phenotypic selection analysis, but still produce evolutionary adaptations. Studying selection over several generations in a population may thus not be enough to detect long-term adaptive effects (i.e., Grant & Grant, 2007). Further analyses of selection using long-term data sets or multiple sites are necessary to analyze which of the two processes of weak selection pressures acting uniformly or of strong rare events of selection is prevalent on the evolutionary process of populations. Studies on human populations, where large numbers of individuals can be analyzed in different countries with contrasted environmental conditions, could greatly contribute to this question.

Evidence for selection on personality in wild animals

Few studies have documented selection acting on animal personality traits in natural populations (Dingemanse & Réale, 2005; Réale et al., 2007; Smith & Blumstein, 2008), though it should be
noted that this topic is receiving increasing interest in the current behavioral ecology literature. At the time of this writing, a total number of 14 studies on a wide range of taxa (birds, insects, lizards, mammals, and snakes) have both documented selection acting on personality in natural populations and simultaneously provided analyses that can be used to test the adaptive hypotheses for the maintenance of personality variation outlined in the earlier section on potential benefits of a selectionist approach to the study of personality (heterogeneous forms of selection, life-history tradeoffs, antagonistic and correlational selection).

Heterogeneous forms of selection. Heterogeneous forms of selection acting on personality traits appear to be the norm rather than the exception in wild animal populations: nine out of ten studies (90%) that have tested for the occurrence of heterogeneous selection have detected it (Boon et al., 2007; Both et al., 2005; Cade & Cade, 1992; Cote et al., 2008; Dingemanse et al., 2004; Duckworth, 2008; Kontiainen et al., 2009; Quinn et al., 2009; Réale & Festa-Bianchet, 2003; van Oers et al., 2008). Temporal variation in selection on avian personality has been documented in a Dutch (Dingemanse et al., 2004) and a British (Quinn et al., 2009) population of great tits, where directional forms of survival selection acting on exploratory behavior changed between years in the former study and nonlinear forms of female fecundity selection were year-specific in the latter study. However, a twelve-year study on Ural owls (Strix arulensis) detected directional selection favoring high aggressiveness in mothers in the majority of years, but no temporal patterns in selection (Kontiainen et al., 2009). In mammals, year-to-year variation in selection on personality has been detected in red squirrels’ activity and aggressiveness as a result of fluctuation in food resources abundance (Boon et al., 2007) and on boldness and docility in bighorn sheep (Ovis canadensis) experiencing contrasted predation regimes (Réale & Festa-Bianchet, 2003).

Spatial variation in selection, thought to be an important form of heterogeneous selection that maintains genetic variation (Mousseau et al., 2000) has received relatively little attention. The hypothesis of spatial variation in selection has been supported by two personality studies in birds. First, in the British great tits discussed above, fast-exploring individuals performed best in low-density habitats—though only in one of four years (Quinn et al., 2009). Second, in western bluebirds (Sialia Mexicana), experimental field research revealed that selection favored aggressive individuals in newly settled populations, but disfavored aggression in older populations (Duckworth & Badyaev, 2007; Duckworth, 2008). Manipulation of the environmental conditions has confirmed the occurrence of fluctuating selection acting on animal personality. In male Texas field crickets (Gryllus texensis), heritable variation exists in mate-attraction behavior. Phenotypic selection analysis revealed that “calling” is favored among males in low but not in high-density treatments (Cade & Cade, 1992). Furthermore, experimental manipulation of density revealed that sociable common lizards Lacerta vivipara are favored under high densities whereas less sociable ones survived better under low densities (Cote et al., 2008).

Variation in the social environment can also induce heterogeneous selection. In the Dutch great tits, both slow- and fast-exploring females produced offspring of high quality only when paired with a male of the same phenotype (Both et al., 2005). Assorted pairs also produced most recruits in certain years (Dingemanse et al., 2004).
Interestingly, males paired assortatively for personality also suffered most from extra-pair paternity in their nest (van Oers et al., 2008). In this population, the pattern of fluctuating selection has been hypothesized to result in overall stabilizing selection favoring birds of intermediate exploration phenotype (Dingemanse et al., 2004). Females paired assortatively might therefore benefit from seeking offspring produced from extra-pair matings, particularly with mates with dissimilar phenotype. The complex pattern of extra-pair paternity recorded by van Oers and colleagues (2008) might thus reflect an example of adaptive mating behavior. In summary, the few studies that have evaluated heterogeneous selection on personality strongly suggest that this form of selection is very common in nature.

*Other evolutionary mechanisms.* The occurrence of other evolutionary mechanisms maintaining personality variation (like life-history tradeoffs, sexual antagonistic selection, and correlational selection) have received even less attention than fluctuating selection, though evidence exists in the literature for each one of them. For instance, the role of personality in life-history tradeoffs appears to have been studied only in bighorn sheep, where support for this idea was found (Réale et al., 2009). Bold rams survived better than shy rams, but shy rams enjoyed greater reproductive success early in life. The same pattern was found for docility. Furthermore, docility and boldness were negatively genetically correlated (bold individuals were also less docile), which may therefore result in a relatively flat fitness landscape. One study is consistent with the occurrence of sexual antagonistic selection: in the Dutch great tits, survival selection acting on exploratory behavior was always directional, but opposite in direction for males versus females (Dingemanse et al., 2004). Moreover, artificial selection experiments in the laboratory were unable to show sex-dependent expression (van Oers et al., 2004) for this behavioral trait, suggesting that the sexual antagonistic survival selection might have occurred at the genetic level, thereby helping to maintain genetic variation in avian personality.

Finally, correlational selection gradients have received very little attention. They have been estimated, but not detected, in the red squirrel (Boon et al., 2007), where the correlation between aggression and activity did not appear to be under survival selection (though it should be noted that this form of selection is extremely difficult to detect when sample sizes are low; Kingsolver et al., 2001). To date there is only one study that documented correlational selection involving a heritable behavior. In garter snakes *Thamnophis ordinoidis*, color and antipredator behavior have long been presumed to function interactively. Reversals are stereotypic changes in direction during flight, an evasive maneuver allowing snakes to employ crypsis after initial detection by a predator. An experimental field study indeed confirmed that survival selection favors a correlation between color and flight behavior, thereby helping maintain genetic variation in both (Brodie, 1992).

*Value of the phenotypic selection approach: an example.* Very few of the studies cited above have used the phenotypic selection approach (but see Brodie, 1992; Cade & Cade, 1992; Duckworth & Badyaev, 2007; Quinn et al., 2009; Réale & Festa-Bianchet, 2003). Animal personality traits exist because differences between individuals are correlated across time or across behaviors (Réale et al., 2007; Dingemanse et al., 2010). Therefore, it appears to us that evolutionary trajectories of personality traits cannot be understood without considering in detail how selection acts on suites of correlated traits (e.g., Boon et al., 2007), and which components are selected for directly versus indirectly. The study on Texas field crickets illustrates what can be learned from applying multivariate selection analyses. In this species, individuals differed both
in the time spent calling during the night and in the time spent actively searching for mates. These two traits were positively correlated across individuals in the low-density but not in the high-density treatment (Cade & Cade, 1992). In the former treatment, both traits were under selection because selection acted directly on time spent calling, thereby causing indirect selection on searching behavior. In contrast, no direct selection was detected in either trait in the high-density treatment. Importantly, had selection acted directly on one of the traits in the high-density treatment, it would not have triggered an indirect response in the other because they were not correlated. This example thereby illustrates the necessity of understanding both the association between different behaviors that define an individual’s personality and the selection pressures acting on either component.

**Evidence of natural selection on human personality**

As we can see above, phenotypic selection has rarely been used in personality studies in animals as of yet. In humans, even fewer studies have proposed such an approach (Eaves et al., 1990; Nettle & Pollet, 2009; Jokela, 2009). Nevertheless, in addition to genomic evidence that human populations have experienced accelerated selection over the last 40,000 years (Hawks et al., 2007), phenotypic selection studies have reported evidence for selection on other traits in humans. For example, Schluter (1988) reanalyzed the data of Karn and Penrose (1951) and showed evidence of stabilizing selection on birth mass. Kirk and colleagues (2001) have shown that age at first reproduction was the main determinant of fitness in a post-industrial population. Pettay and colleagues (2007) found that wealth affected selection on life-history traits in women of a Finnish pre-industrial population. In poor families, age at first reproduction was under stronger selection, whereas in wealthier families selection acted mainly on age at last reproduction. More recently, Nettle and Pollet (2009) compared the strength of selection on male wealth and similar traits, such as land ownership, livestock, and hunting ability. They reported systematically positive selection gradients varying between 0.10 and 0.68 depending on the society and the type of trait. Interestingly, they also showed some negative selection on female wealth, suggesting the possibility for antagonistic selection between the sexes in current societies. Other studies showed negative or positive selection on female height (cited in Pollet & Nettle, 2008), suggesting the possibility for environment-dependent fluctuating selection.

Yet none have used the full potential of phenotypic selection analysis and formally tested the evolutionary hypotheses on personality traits presented above (see section on selection, evolutionary response and the limits of the selectionist approach). Despite this lack of studies, some evidence for selection on human personality traits exists in the psychology literature. Eaves and colleagues (1990) provided the first and only formal phenotypic selection analysis on personality. Using personality dimensions in a sample of 1101 Australian women, they showed negative correlational selection on neuroticism and extraversion, with the highest fitness for both the high-extravert/low-neurotic and low-extravert/high neurotic females, intermediate fitness for females that had intermediate scores on both axes, and lowest fitness for low-extravert/low neurotic and high-extravert/high-neurotic females.

Other studies reveal selection on personality traits, even though they do not use a selectionist approach per se. For example, in their review on personality as a predictor of important life outcomes, Roberts and colleagues (2007) showed that mortality is generally associated with high neuroticism, low conscientiousness, low extraversion, and low agreeableness. In their recent meta-analysis, Kern and Friedman (2008) found a positive influence of conscientiousness on
longevity. Personality also affects the age at first sexual intercourse (Atkins & Hart, 2008), which may have consequences on fitness. On one hand, early sexual activity may affect age at first birth, which has been shown to be an important determinant of fitness (see above; fitness increases directly with age at first birth). On the other hand, those individuals with such a personality have a higher chance to experience risky situations—such as an increase risk of sexually transmitted diseases—with consequences on survival (Atkins & Hart, 2008). Other facets of life history are dependant on personality dimensions. Leadership, a component of type-A personality, increases the probability of having children by the age of 39 (Jokela & Keltikangas-Järvinen, 2008). High extraversion, high openness, and low conscientiousness affect the probability of infidelity (Orzeck & Lung, 2005), which may have some consequences on extra-pair reproduction and in fine fitness. This non-exhaustive list indicates that there are good reasons to think personality is subject to selection. We have not found any results indicating the presence of non-linear selection on human personality, but there was no mention that they had been considered in the studies. However, further standardized studies would be necessary to test for the hypotheses explaining how selection can help maintain variation in personality. Studies may also report systematically the presence (or absence) of significant non-linear relationship between personality traits and fitness.

Conclusion

With this chapter we hope to emphasize the importance of the study of selection pressures on human and animal personality traits in both the evolutionary psychology and behavioral ecology. Scientists working in these domains should aim at finding the general principles explaining how selection acts on personality traits and how it can be at the origin of the variance in behavioral responses in wild populations. The next two steps for researchers interested in the field will be: 1) to find a general model that can satisfactorily describe the differences in personality in both human and non-human animals, and 2) to accumulate empirical evidence for the different possible selective scenarios on personality traits described in this chapter.

It traditionally has been considered that the adaptionist and the selectionist approach would lead to totally incompatible conclusions on biological phenomena (but see: Moore & Boake, 1993; Roff, 1994; Gomulkiewicz, 1998). However, these two approaches are not incompatible, but focus on different facets of the evolutionary process: while the former is looking at how selection has shaped adaptations and is thus centered around the product of past, invariant selection pressures (or to be more precise, the product of selection pressures over a long period of time) and very broad characteristics of living organisms (e.g., how does an animal deal with the potential compromise between foraging and predator detection, or, what is the best compromise between offspring number and quality?), the latter is mainly interested in understanding how selection processes can work on current phenotypic and genetic variation to modify characteristics in a population (Smiseth et al., 2008). Both can provide similar conclusions if the shape of selection pressures has been maintained constant through evolutionary times and if selection is strong enough to be detected at the time scale of one generation.
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Most researchers now agree that personality in our species is also influenced by genetic processes, and that genetic differences contribute to a variety of personality profiles and response norms to the various situations life poses. A growing number of molecular, twin and pedigree studies has suggested heritability in personality traits. At present, the variability in personality traits among individuals is well-established (McCrae & Costa, 1987) and has a (30% to 60%) heritable component in various population studied (Benjamin et al., 1996; Bergeman et al., 1988; Cloninger et al., 1993; Ebstein et al., 1996; Plomin et al., 1994; Tellegen et al., 1988). These studies suggest that there are specific loci with various alleles influencing different (usually opposing) response norms to similar situations (Buss, 1991; Lohelin et al., 1998; Plomin and Nesselroade, 1990; Plomin & Caspi, 1999). In this chapter we will not ignore or dismiss the environmental effect of personality variation, but we will especially focus on the genetic aspect of it, and try to answer the following question: Why are there a multitude of different alleles in different loci that influence differently the expression of personality traits?

Selection is generally understood to favor the most successful traits and eliminate all others given enough time, thus transmitting the single most successful phenotype to the whole population at the expenses (extinction) of all less successful ones (Fisher, 1930). Yet, personality traits are highly variable in all populations studied. Most researchers agree that personality traits vary not only because they fail to coalesce into a single successful profile, but also because there is an inherent permanent presence of a variety of alleles influencing all personality traits in different ways (Lohelin & Rowe, 1992; Turkheimer, 1998). This state of affairs gives rise to a crucial question: Why have millions of years of evolution not eliminated genetic variation in all personality traits, favoring a unique, most adapted one?

Penke and colleagues (2007) examined three possible alternative hypotheses to account for the persistence of a large amount of variation in personality profiles: 1) Neutral selection, 2) Mutation-selection equilibrium, and 3) Balanced selection. Accordingly, these authors evaluated the evolutionary dynamics and requirements of all of the hypotheses within a behavioral genetics perspective, and suggested that balanced selection and some of its variants more adequately explained the persistence of a large number of alleles influencing personality traits. At a theoretical level, their arguments are convincing. What is yet missing, however, is an empirical test of competing predictions that arise variously from the three contrasting hypotheses.

Selective hypothesis promoting variability in personality traits

Neutral Selection

A first possibility discussed by Penke et al. (2007) is that the genetic differences vary continuously, but have no selective value (Kimura, 1983). That is, individual differences are neutral or a by-product of selection for other behavioral or physical traits (Tooby & Cosmides, 1990).
Consequently, personality profiles do not contribute to differential reproductive success per se. Portinga et al. (2002) have argued that a genetic explanation for group differences in personality traits is unlikely, because there is no compelling reason why certain traits would be differentially selected in the different groups.

However, even if there are various physical traits that can be variable and yet selectively neutral (Lynch & Hill, 1986), it seems unlikely that individual differences in personality, known to be of paramount importance in social situations for deciding with whom to cooperate or mate (e.g., Ozer & Bennett Martínez, 2006), would be selectively neutral. Hence, it seems highly unlikely that such variable traits should not influence reproductive success, which goes against the neutral selection hypothesis.

**Mutation Selection Equilibrium**

The logic of the *mutation selection equilibrium* hypothesis is that genetic variability is maintained in the population because selection progressively eliminates fitness-reducing alleles, but random mutations continuously generate new alleles. Most mutations are generally less favorable than established variants (Tooby & Cosmides, 1990), and, given enough time, they tend to be eliminated by selection.

Even if humans have a higher within-species variability than other species, nevertheless the mutation rate is relatively low (1.67 new mutations per individual per generation; Keyghtley & Gaffney, 2003) and can affect any part of the genome. If many loci are involved in the control of a trait (e.g., a relatively large portion of the genome), each mutation has a greater chance of appearing in one of them (Penke et al., 2007). If the disadvantage produced by the single mutated allele is relatively low, then this mutation would be eliminated in the population relatively slowly. Hence, mutation selection equilibrium might produce considerable genetic variability at any given time. Genetic variability composed not of neutral variants but of a number of new relatively disadvantageous mutations not yet eliminated by selection. Mutation selection equilibrium has been advocated for very important general traits in humans such as general intelligence (g factor), longevity, attractiveness, fitness itself, and fecundity (Houle, et al., 1994; Miller & Penke, 2007). One prediction of the mutation selection equilibrium hypothesis is that it should be very difficult to find specific loci controlling the trait variation due to the large number of loci involved. Indeed, each allele controls for such a little portion of variance that its role is virtually undetectable. This has been found true or suggested for all those very general traits possibly influenced by a great multitude of loci, each with small additive effect. For these general traits, despite many efforts, nobody has ever found specific genes significantly involved in their expression (Kovas & Plomin, 2006), except for a general mental disability. This confirms that many genetic factors are involved and it is impossible to determine the individual role of a limited number of them (Penke et al., 2007). However, personality appears to operate differently. After relative little effort, molecular biologists have identified specific loci and alleles that influence personality traits; and not in small additive ways. As an example, the MAOA-LPR low activity allele was associated with alcoholism, particularly antisocial alcoholism, only among sexually abused subjects (Ducci et al., 2008).

The short allele 5HTTLPR polymorphism is partly responsible for a higher predisposition to impulsive and violent antisocial behavior in association with drugs and alcohol abuse (Sakai et al., 2006). The STin2 polymorphism
regarding the intron2 of the serotonin transporter has the allele with 12 repetitions of the key sequence, which enhance the transcription efficiency of the gene (SCL6A4) and has been associated with higher chances of schizophrenia and with obsessive compulsive behavior (Baca-Garcia et al., 2007) and anxious behavior (Ohara et al., 1999). A simple substitution of Val amino acid, in position 158, with Met one, in the gene COMT significantly reduces catecolamine inactivation, producing higher aggressive behavior both in animal models and psychiatric patients (Volavska et al., 2004) and induces higher anxiety (Olsson et al., 2007). In the polymorphism MAOA-uVNTR involved in catecholamine catabolism (Volavska et al., 2004), the alleles with 3 and 5 replications of a key sequence have been associated with a aggressive and violent behavior (Volavska et al., 2004, Craig, 2007), especially if provoked (McDermott et al., 2009). Finally, DRD4 polymorphism, in which the 7 replications or more of the key sequence in the exon 3 of the gene is reported with hyperactive behavior (DeYoung, 2006), and continuous search for external stimuli (novelty seeking) (Ebstein et al., 1996). The association of DRD4 7 allelr with novelty seeking is a notable example (Ebstein et al., 1996; Ebstein et al., 1997; Ekelund et al., 1999). Personality traits, according to these studies, appear to be influenced by a limited number of loci with a limited number of variants (alleles). This fact disfavors the possibility that genetic personality differences are maintained by a mutation selection hypothesis.

**Balanced Selection**

There are some very well known processes that alter alleles’ frequencies in a population, such as: Directional selection, where one extreme of a trait is favored in a determined environment. Also very common is stabilizing selection, which favors the average expression of a trait and disfavors both of the extremes. Less known is divergent selection, which favors both extremes over the intermediate form. This form of selection is less cited but it is responsible for the evolution of sex differences and anisogamy. Anisogamy was produced by two opposing selecting forces on the same structure: the gamets. The final consequence was that two sexes evolved due to divergent selection with all the beauty and troubles that we face as behavioral sex differences, alternative mating strategies, and personality differences between sexes (Buss, 1991). A particular form of divergent selection can occur between sexes— called sexually antagonistic selection—where one sex is selected against the other, as in the case of sexual orientation (Camperio & Ciani et al., 2008).

Divergent selection can also occur when different environments favor different variants of a trait, and, provided such environments are equally common, the different variants of a trait should be balanced in the population and associated with the different environmental conditions. This kind of selective force is called balanced selection. Balanced selection has been advocated by Penke et al. (2007) to be a good candidate to explain the maintenance of different alleles at different genetic loci which influence personality trait expression. Balanced selection can depend on the difference in environment characteristics (environmental heterogeneity). In this case, the fitness effects of a trait can vary across time and niche, allowing for the maintenance of genetic variation (Roff, 1997). In balanced selection, different alleles do produce dominance effect rather than little additive effect in the expression of a trait, as suggested in the mutation selection equilibrium hypothesis. Balanced selection is apparently found in a number of loci known to influence personality traits. For instance the molecular genetics of extraversion have been partly investigated through its association with novelty seeking (Benjamin et al., 1996). Ebstein et al. (1996) showed that long alleles of polymorphic exon III repeat sequences of the D4DR dopamine
receptor gene on chromosome 11 are associated, together with other behavioral differences, with high levels of a personality trait called novelty seeking, while short ones are at the opposite end of the spectrum and associate with low levels of novelty seeking and high level of harm avoidance (Ebstein et al., 1996; Ebstein et al., 1997; Ekelund et al., 1999; for negative findings see Herbst et al., 2000, but see also Schinka et al., 2002 meta-analysis suggesting partially contradicting results).

There are some reasonable explanations of how the environmental niche could differentially favor personality traits of the most adapted individuals. For instance, the diffusion of large epidemics of lethal disease (Black Death, AIDS, plague, etc.) might have differentially targeted particular individuals with conscientiousness, harm avoidance, novelty avoidance, and closed mentality and introversion. Due to their traits, these individuals had less possibility of contamination relative to others. Because these illnesses were associated with a high mortality rate, avoidance and introversion would have been naturally selected. Perhaps also the depletion of environmental resources that caused individuals to migrate in search of new richer environments might have selected novelty seeking and openness (Chen et al., 1999). Because personality is involved in social relationships (friendship, alliances, sexual partners), we could expect these niches not only to be environmental niches but rather socio-eco niches. The idea of balanced selection is theoretically attractive for personality genetics but has not been tested yet.

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**Empirically Testing evolutionary genetics of personality hypothesis**

If there are environmental contexts that favor certain personality profiles over others, we should find whole populations living in particular environments, all sharing common personality profiles that differ from others not living in such particular eco-socio-niches. In other words, given appropriate difference in environmental characteristics, we should find populations in which the balanced selection favored only certain profiles and not others. This hypothesis is testable.

It has been noted that in large populations, firstly, the effect of culture, geography, and language is difficult to control for (Eysenck, 1982; Lojk et al., 1972, Buss, 1999). Secondly, in large populations with complete sets of socio-eco niches, individuals with different personality traits will actively search for their adequate environment (Barrick and Mount, 1991; Hettema and Kenrick, 1992; Tett et al., 1991). Thus, differences in personality distributions would be averaged out in a large population sampling. This matching of traits to environments explains why alleles influencing these personality traits are rarely eliminated through natural selection and high population variability is thus preserved.

On the contrary, the prediction from balanced selection suggests that selective balance would be disrupted where there is a substantial reduction in eco-socio niches such that certain phenotypes would be favored over others. To test the balanced selection hypothesis, we should then approach small homogeneous populations, under particular conditions: a) populations that are sufficiently ancient to allow selection to act, b) populations that are sufficiently small (selection works better in small populations), c) those with a very limited array of socio-eco niches (to alter the balanced effect), and d) those that are relatively isolated (to avoid the dilution effect of external immigration).
Small Island Populations Can Empirically Test Evolutionary Genetics of Personality Hypothesis

Populations with all the previous characteristics are not easy to find, but could be high mountain isolated communities, desert nomads, or best of all, populations living on small islands. In these sort of populations, the three genetic hypotheses produce mutually exclusive predictions that can be tested: the neutral selection hypothesis predicts that isolated populations would genetically drift in allele frequencies producing all sorts of population-level personality profiles distinct from those of large populations. The mutation selection hypothesis predicts no differences at all in these small populations compared to large ones, other than due to random forces. Finally the balanced selection hypothesis predicts that the personality profile of the small population should differ from the large population due to a substantial reduction of socio-eco niches, according to the predictable adaptive requirements of the remaining socio-eco niches. Hence, in small populations with similar socio-ecological conditions, personality should evolve personalities always in the same direction.

Here, we will show the results of a series of studies focusing on population personality profiles of ten small islands divided between three archipelagos of the west coast of Italy.

Are There Any Personality Differences Between Small Island Populations and Corresponding Mainland Populations?

Populations Under Investigation

The following islands were considered for this study: Giglio in the northern archipelago, Ponza and Ventotene in the central archipelago, and all seven of the Aeolian Islands in the southern archipelago. We compared each population of these archipelagos with those of three small Italian mainland villages facing the three archipelagos: Castiglione della Pescaia (facing the northern archipelago at 25 nautical miles distance), Gaeta (facing the central archipelago at 30 nautical miles distance), and Milazzo (facing the southern archipelago at 20–40 nautical miles distance). We examined 1,784 subjects from the populations living in the three archipelagos and the three corresponding mainland villages (Fig. 15.1). As can be seen in Figure 15.1, each archipelago was sufficiently far from the others to be considered an independent sample. No significant movement of people was reported between the islands of different archipelagos either in the present or in the past, while movement within archipelagos was common due to the relatively short distance between islands (Roani Villani, 1993). By comparing populations within the same identical culture and language (Camperio Ciani et al., 2004), we investigate personality differences in small populations, coming from Italy, all living in small archipelagos made up by several islands, and we compare them with control groups from villages on the mainland facing these same archipelagos.

The islands selected in each archipelago all meet the required conditions of isolation as defined in the hypothesis testing predictions: 1) They are all
Figure 15.1. Geographic location of the three archipelagos examined along the Italian Tyrrhenian Sea. We sampled a total of 1784 subjects equally distributed in all three archipelagos according to ages and sexes and classified as: Mainlanders: Subjects resident and born in the three villages facing each archipelago; Immigrants: Subjects resident in the archipelago with no grandparents born on the islands; Islanders non-ancient origin: Subjects resident in the archipelago with 1 to 3 grandparents born on the islands; Ancient origin islanders: Subjects resident in the archipelago with 4 grandparents born on the islands (for further details see Camperio Ciani et al., 2007).

composed by small islands, between 10 and 40 miles offshore, with only one or two old settlements; 2) the islanders were forced to resettle from mainland villages before the sixteenth century, and for at least 20 generations they have experienced a high degree of isolation from the respective mainland; 3) population growth on each island was limited, in the past, by the small available surface area, and therefore the population experienced constantly high levels of emigration (Camperio Ciani et al., 2007; De Fabrizio, 2000; Roani Villani, 1993).

Furthermore, these islands show a significant reduction of socio-eco niches because the natural environment offers scarce and inaccessible resources on
land and at sea. The social environment is limited by the small size of the communities, which do not interact much with the outside world according to the scarcity of transportation to the mainland.

In the last few decades, however, some immigrants were facilitated by the development of connections with the mainland, and attracted by tourism. The presence of recent immigrants allowed us to test the effect of phenotypic flexibility to environmental requirements on personality traits.

**Assessment of Personality**

In order to assess personality traits, we used an adjective-based Big Five questionnaire (Norman, 1963). According to the Big Five theory of personality, five basic independent dimensions of personality may be identified: *extraversion*, *agreeableness*, *conscientiousness*, *emotional stability*, and *openness to experience* (Costa and McCrae, 1992; Goldberg, 1990; McCrae and Costa, 1987). Research conducted on individuals of various cultures and languages have identified the same five-dimension personality structure in several different cultures (Goldberg, 1993); moreover, personality is shown to be stable over time when assessed within this framework (Costa and McCrae, 1994, 1997); finally, all personality dimensions show inheritable components relatively untouched by life experiences (Buss, 1991; Lohelin et al., 1998; Plomin and Nesselroade, 1990; Plomin & Caspi, 1999). For these reasons, the Big Five personality analysis framework is particularly suitable for an evolutionary personality investigation, like the one conducted here (Buss, 1996).

Subjects were investigated in their original location between 1997 and 2000, by visiting them in their homes. A snowball method was used to cover most of the population (Cicchitelli et al., 1992). All subjects were interviewed by a team of trained interviewers (6 for Giglio, 7 for Ponza and Ventotene, and 12 for the Aeolian Islands) all unaware of the aims of the present study.

We compared a total of 993 islanders (those born on their respective island and having fully answered all of our questionnaires) with their corresponding control group of mainlanders (n=598, after having excluded incomplete questionnaires). Since many studies showed a correlation between personality traits and sex, age, and education level (Costa et al., 2001; Feingold, 1994; Goldberg et al., 1998), we conducted a preliminary stepwise regression analysis (Camperio Ciani et al., 2007).

This analysis showed that living on islands and, as expected, sex, age, and education level had significant effects on personality traits. Therefore, all the following results refer to MANCOVA analysis of variance with sex, age, and education level assumed as covariates.

We found, after eliminating the effect of the above-mentioned three variables, islanders had significantly different scores from those of control mainlanders; namely, islanders showed a higher level of *conscientiousness* and of *emotional stability* and a lower level of *extraversion* and *openness*. No significant difference was found as regards to *agreeableness* (Fig. 15.2). By eliminating the effect of different groups’ composition by age, gender, and education level, the multivariate model reveals a significant difference of openness between emigrated and sedentary islanders, unidentifiable when looking at the group means (47.65 vs. 47.56), and undetected by the effect size univariate statistic (d=.01). In particular, people who emigrated are mostly males (66%), 11 years older, and with lower education levels than sedentary people.
Mean and confidence interval for each personality trait. Personality test scores are given in T-scores. T-scores reported here were corrected for age, gender and education level. Mainlanders=598, Islanders=993.

![Figure 15.2. Big Five scores of Islanders and Mainlanders.](image)

All the three archipelago/mainland population pairs showed a similar pattern of results. No significant difference among the three archipelago scores standardized against mainland scores was found for any personality trait. A preliminary 3x2 MANCOVA, with Location (North vs. Center vs. South) by Island/Mainland as factors, showed no significant difference in Big Five scores due to Location, and no significant interaction of Location by Island/Mainland, for any Big Five dimensions (For means, SD of T-scores, and MANCOVA analysis of variance and effect size, see Camperio Ciani et al., 2007).

These results show that islanders coming from three distinct archipelagos isolated from each other share a consistent, distinctive personality profile confirming the balanced selection hypothesis and disconfirming both the neutral selection and the mutation selection equilibrium hypotheses.

Islanders differ from their respective control mainlanders as they are more conscientious, more emotionally stable, and with a lower level of extraversion and openness. These differences are significant and consistently observed in all the archipelago/mainland population pairs that were investigated. These results are observed after adjustment for differences due to sex, age, and education level. Moreover, these differences could not be due to major educational, social,
cultural, religious, historical, or linguistic differences with the mainland. This sharply differs from other studies, which compared populations that were geographically and ethnically or linguistically far apart (Eysenck, 1982; Lojk et al., 1972). What we found here was not expected according to the neutral selection hypothesis predictions, nor by the genetic drift or founder effect, as we found the same differences and in the same direction in all three populations. This suggests adaptive differences rather than neutral drift. Further, the mutation selection equilibrium hypothesis is obviously disconfirmed because its predictions did not expect any significant difference between the islands and corresponding mainland populations. Therefore, only the balanced selection hypothesis predictions fit our results.

**Are These Genetic Differences or Only Effects of Individual Development in Different Environments?**

The role of the environment could have been important in shaping personality traits in this study (Newcombe et al., 1967). Two opposing hypotheses could be advocated to explain the differences in personality profiles reported in the previous investigation: 1) They could originate either through prolonged exposure to peculiar situations, producing a phenotypic flexibility to environment (environmental hypothesis, e.g., Newcomb et al., 1967),

or 2) they could be progressively selected in the genetic structure of islanders (genetic hypothesis, e.g., Plomin et al., 1994).

When individuals who either emigrated from the mainland early in age or who have lived for an extensive period of time in the archipelagos are compared with islanders with a long-lasting insular ancestry, different predictions arise from the two hypotheses. The environmental hypothesis predicts that immigrants to the islands should adjust their personality traits to those of islanders that have lived there for many generations because they all share the common island environment. In contrast, the genetic hypothesis predicts that immigrants—by sharing their genotype with the mainland population—should maintain the mainlander’s personality phenotype, leaving it relatively untouched by island life experience.

To test these hypotheses, we identified a subset of islanders from our total sample, defined as “ancient origin islanders.” These individuals were not only born on the island but also had all four grandparents born on the island. Since immigration to these islands is a recent phenomenon, individuals with all four grandparents from the island can be confidently considered of long-lasting insular ancestry, as confirmed by Catholic parish registers (over 20 generation or more). Then we compared these ancient origin islanders (n=624) against immigrants—individuals with no grandparents coming from the island—(n=193, mean years since immigration=22.37 years ago; SD=15.50 years) and mainlanders (n=598). The data were analysed with MANCOVA, including age, sex, and education level as covariates.

We found that immigrants and mainlanders did not differ in any of the five personality dimensions (see Figure 15.3). This means that the personality profile of immigrants or kin of immigrants from the mainland to the islands is not different from mainlanders. In contrast, these individuals differed significantly from ancient origin islanders on three personality dimensions. They were more extroverted, more agreeable and more open than islanders. Again this pattern of results was similar in all three archipelago/mainland pairs.

The similarity in personality profiles between immigrants and mainlanders does not confirm the environmental hypothesis, since it shows little flexibility of personality traits in response to environmental changes. On the contrary, this study supports the genetic hypothesis. However, we
cannot rule out a variant of the environmental hypothesis, which suggests that early experience might still influence immigrants. (Camperio Ciani et al., 2007; Camperio Ciani & Capiluppi, 2010; Forgas and Van Heck, 1992). Our data merely show that, once on the island, the immigrants do not acquire the personality traits of the ancient origin islanders, even if they share the same insular environment for over 20 years.

Mean and confidence interval for each personality trait. Personality test scores are given in T-scores. T-scores reported here were corrected for age, gender and education level. Mainlanders=598, Immigrants Islanders=193, Non-ancient origin Islanders=369, Ancient origin Islanders=624.

**Figure 15.3. Big Five cores of Immigrants, Ancient origin Islanders and Mainlanders.**

Scores on extraversion, openness, and agreeableness are higher in immigrant descendants than in ancient origin islanders. These data are again consistent with the genetic hypothesis based on a balanced selection process, which predicts that even if all subjects had been living on the island for an extended period of time, immigrants and immigrant descendants would not acquire the ancient origin islander phenotype, unless an immigrant descendent were the child of inter-breeding.
This model suggests that balanced selection might have a fundamental role as an evolutionary process in producing and maintaining a large array of genetic differences in personality traits of humans. There is, however, a final question: Which selective mechanism allows such genetic differences to evolve in such small isolated populations?

There are two candidate hypotheses: selective mortality, of those less adapted to their socio-econiche. This hypothesis is substantially contrary to the balanced selection model which does not predict mortality. Alternatively, it is in line with the balanced selection hypothesis to abandonment of the niche through emigration (displacement), hence producing genetic differences through gene flow (Lohelin and Rowe, 1992). Chen et al. (1999) have advocated selective mortality to explain personality evolution. These authors investigated a large sample of populations all over the world which had migrated several thousand of miles during human history over hundreds of generations. These authors suggested that people scoring high in novelty seeking survived better in migrating societies where they could explore and exploit environments better, thus accumulating new resources essential to improve survival. Conversely, they argue that sedentary populations obtain resources not by exploring new environments but by developing intensive methods for using limited amounts of land (Netting, 1993). Within these societies, novelty seeking and exploratory behaviors would have serious costs and would be selected against. They hypothesize that the differences between these populations are the product of a slow Darwinian natural selection process of differential mortality in the two differing population types (Chen et al., 1999).

This hypothesis again produces two testable predictions: 1) We should find evidence of particularly high mortality. Even if mortality is not particularly high, personality differences should be minimal and changes in personality should happen through a high number of generations; and 2) There should be no differences in personality between surviving individuals who decide to remain on the island and those who decide to emigrate.

In contrast, we propose that gene flow (rather than selective mortality), caused by a strong emigration, explains the differences in the personality profiles shown in the present research. If adaptation to the environment took place by means of a progressive gene flow through a strong emigration, then we predict that islanders—without presenting a different mortality from mainlanders—would nevertheless rapidly (within a few generations) become less extroverted. Indeed, the extroverted individuals, like novelty-seekers (Benjamin et al., 1996), are expected to be more emigration-prone because they have a more outward attitude and more curiosity for novel environments. Thus, these individuals’ alleles fade away since they leave no descendants on the island. Islanders would also become less open to experience. This is due to the fact that life on small islands with few kilometers of surface and only one or two villages is clearly more repetitive than on the mainland, and individuals with high levels of openness would find less cultural, social, or intellectual stimulation in such a confined insular environment. All this might induce extrovert and open individuals to leave the island.

The gene flow hypothesis produces opposite predictions from the selective mortality one: 1) It does not suppose a higher than normal (for those times) mortality, and 2) it predicts that personality differences should be significant in individuals who emigrate compared to those who
remained on the islands (Camperio Ciani et al., 2007). If selective emigration is consistent and immigration in the past was marginal, personality differences between islanders and mainlanders will arise quite rapidly.

First, through Catholic parish records of birth and death, dating back to the sixteenth century, De Fabrizio could not find any evidence of notably high mortality in these islands (De Fabrizio, 2000; Roani Villani, 1993). Life was hard and resources limited, but these islands, once under pirate control, were rather safe and seldom affected by the frequent conflicts afflicting the corresponding mainland regions. Most mortality occurred in infants, but this is similarly the case throughout rural Italy and it is difficult to imagine that this infant mortality pattern is due to personality differences. On the other hand, by defining an emigrant as one who has an island birth registration, but no death record, this author suggests an average emigration rate per generation to be approximately 30% of the population over the last 400 years (De Fabrizio, 2000).

We compared the scores of personality traits of islanders who were born on the islands and then chose to emigrate (n=209) with those of islanders born on the islands who decided to remain (n=741 non-emigrants). Not all participants had all four grandparents from the island as in the second study. Each subject answered the same previous questionnaires as emigrant or not from the islands. The data were analyzed with MANCOVA including age, sex, and education level as covariates.

Results show that the population of individuals who emigrated from the small archipelagos were significantly more extroverted and open than islanders who did not emigrate (see Figure 15.4). Again, there were no significant differences between the three archipelagos populations.

Our data do not confirm the prediction of the selective mortality hypothesis. Furthermore, a small differential mortality between individuals with different personalities would entail several hundreds of generations (an excessively long time in our case) to show a measurable effect (De Fabrizio, 2000). Wilson (1975) observed that it is theoretically possible to have measurable natural selective changes in as few as 40 generations in humans. However, this is possible only in very small populations (a few dozen individuals) with extremely high mortality—very different conditions than our study.

Means and confidence intervals for each personality trait. Personality test scores are given in T-scores corrected for age, gender, and education level. Emigrants=209, Non-emigrants=741.
Instead, the gene flow hypothesis fully predicts the observed different personality profiles. Such gene flow requires three different conditions: 1) Personality traits which do not change with time or external conditions will make individuals more emigration-prone; 2) environmental conditions induce emigration of individuals who are high in extraversion and openness; and 3) immigration in the population of new individuals is minimal. Indeed, personality traits are proven to be rather stable (McCrae and Costa, 1987), emigrants are significantly more extroverted and open than islanders who do not emigrate, and all historical accounts report that immigration has consistently been just occasional in those islands that are unattractive for making a living (De Fabrizio, 2000; Roani Villani, 1993). Given these conditions, if emigration is strong, gene flow will rapidly drain the set of alleles influencing high extraversion and openness from the island’s gene pool. Consequently, the remaining islander population will become, on average, progressively less open and extraverted. In the present case, emigration and gene flow were strong, as confirmed by the fact that we found almost one-third of the sampled population had emigrated in the last generation and this corroborates historical data (De Fabrizio, 2000). In contrast, immigration—which might have counteracted the effect of this gene flow—historically was virtually absent on these small islands until the last century (De Fabrizio, 2000; Roani & Villani, 1993).
Discussion

We tested the three mutually exclusive predictions derived from the corresponding hypotheses on the genetic underpinnings of personality polymorphisms: Neutral selection, mutation selection equilibrium, and balanced selection. We concluded that only the predictions derived from balanced selection were confirmed and all the others disproved through empirical testing on small population personality differences.

We showed that populations from the island villages were significantly different from those of the mainland villages for four of the Big Five personality dimensions: extraversion and openness to experience and higher in conscientiousness and emotional stability. This result could not be considered a by-product of cultural and language differences since the linguistic roots, ethnic composition, social norms, and cultural environment of islanders are similar to those of the mainlanders used as controls, given that they come from the adjacent coastal area.

We further found that 1) immigrant descendants were higher on extraversion and openness with respect to ancient origin islanders, and 2) islanders who emigrated scored higher on extraversion and openness compared to islanders who had not emigrated. We have suggested that a strong emigration of extroverted and open individuals could have caused a gene flow that drained the set of associated alleles from the island gene pool. We suggest that this gene flow resulted in a personality profile that is adaptive for the geographically and socially restricted island environment (socio-eco niches) in the sense that relatively closed and introverted individuals do not leave the insular environment, while extroverted and open individuals search for new, more suitable environments.

According to evolutionary psychologists, extroverts and those high on openness are known to be more likely to pursue short-term mating (Buss, 2003). It is clear that also for this aspect opportunities for short-term mating are likely to be far fewer on the small islands compared to the mainland. Hence, socio-sexual motivations might further add inducement to emigration of extroverted and open individuals from these small islands, thus promoting gene flow.

If confirmed, the present findings might offer new perspectives to evolutionary psychologists who may be puzzled by the existence of large individual differences in personality traits. Our research supports the balanced selection hypothesis and further suggests that gene flow might help in explaining the preservation of a large variety of individual personality differences. The various alleles that produce personality differences seem very efficient at preserving themselves. Only particular environments with a great reduction of alternative socio-eco niches, such as the isolated communities studied here, will induce the abandonment of certain alleles from the gene pool, through emigration (gene flow), leaving only the most adapted alleles, thus highlighting the genetic component of personality differences at the population level.

References


Part IV Practical Applications
One of the major domains of individual differences is the study of psychopathology—mental disorders. In addition to differing on personality traits and mental ability, individuals also differ to what degree they have or are susceptible to develop mental disorders and the form the mental disorder takes. Like psychological research in general, research on psychopathology suffers from the lack of an overarching evolutionary and functional approach that would “carve nature at its joints” (Buss, 1995). The lack of understanding of the nature of psychopathology has resulted in diagnostic manuals such as APA’s DSM and WHO’s ICD, both of which attempt to define categories based on descriptive research in order to free diagnosis from theoretical, historical baggage.

Clinical psychology and psychiatry are important applied disciplines that are responsible for the treatment of the many who suffer from mental disorder and psychological pain. Alas, the field of mental health care is often divided into camps; “mindless” biopsychiatry that reduces almost every symptom to a neurotransmitter or brain disorder, and “brainless” psychotherapy that attempts to keep pet theories and practices alive despite scant empirical support. There is a need for integrative, biopsychosocial, and empirically supported approaches that address both symptoms and the interpersonal, therapeutic relationship. One major problem is that the conditions that are treated are poorly understood. An evolutionary approach might be able to help integrate the many different levels of analysis (Gilbert, 1995, 1998; Kennair, 2003; Nesse, 2002, 2005) and might also help us understand the functions of certain phenotypes or the malfunction of others (Troisi & McGuire, 2002). Unfortunately, in general the broad set of theoretical approaches within current evolutionary psychopathology has been too diverse to assist cumulative theory building.

Evolutionary psychology has already shown a great integrative potential within psychology (Buss, 1995; Kennair, 2002). This integrative power is also evident in bridging biology, the social sciences, and the humanities (Buss, 2005). This is due to the multi-disciplinary relevance of evolutionary theory, the cross-disciplinary approach of evolutionary psychology, as well as the broad relevance of considering the psychological level of analysis (the cognitive mechanisms behind human universal information processing).

The present chapter considers how an evolutionary psychology approach may assist the development of a scientifically based, general definition of psychopathology, as well as help integrate the disparate field of evolutionary psychopathology. The latter goal will require changes to mainstream evolutionary psychological theory so that it will encompass the major phenomena and symptoms of psychopathology. These changes will therefore be discussed. In so doing, I will not address specific disorders per se, but rather focus on psychopathology in general and how an evolutionary psychology approach may provide the theoretical foundation for defining the nature of mental disorders. Throughout the chapter examples from different mental disorders will be used to illustrate general principles.

Evolutionary psychopathology has been subject to a most exciting surge in both original and fruitful theoretical and empirical work the last few decades (see Kennair, 2003, for a review). However, the different, competing approaches, hypotheses and theoretical foundations make this
a heterogeneous field. Scientists with different theoretical orientations within evolutionary psychopathology argue for all the possible positions in discussions of the theoretical underpinnings of evolutionary psychology. Such include: whether adaptations are identified through formal analysis vs. assumed at the outset; whether the adaptations should be the focus of research vs. adaptiveness (i.e., behavior as maladaptive or functional in the current ecology), whether the mind is a set of specific mechanisms or modules vs. a general problem solver; whether selection has designed species-specific adaptations vs. comparative studies are fundamental; and whether human universals are the appropriate unit of analysis vs. individual differences are genetic and adaptive, etc. (see also Buss & Reeve, 2003; Gangestad & Simpson, 2007; Hagen, 2005).

To overcome the lack of cumulative research, theory building, and integration of findings within evolutionary psychopathology, an overarching meta-theory is needed. Evolutionary psychology is the research program of choice, in part due to the focus on cognitive, functional adaptations (Cosmides & Tooby, 1999). The evolutionary psychology research program (Buss, 1995; Tooby & Cosmides, 2000) is therefore able to map both functions and dysfunctions (Baron-Cohen, 1997)—laying the foundation for a harmful dysfunction analysis of pathology (Wakefield, 1999).

**What is psychopathology?**

Providing a general definition of mental disorder is not the only problem. There is neither full consensus on what behaviors, states, or syndromes are the best descriptions of specific mental disorders, nor on what disorders warrant inclusion. Each new edition of the diagnostic manuals may be considered progress as it reflects a continuous attempt at increasing the scientific foundation of diagnosis. However, an overarching understanding of how to carve the nature of psychopathology at its “joints” (i.e. delineate discrete functional units) (Buss, 1995) and what phenotypes are most relevant, is missing from modern mental health nosology. In the meantime, subjective, individual suffering (e.g., depression) is classed together with intersubjective assessment of nuisance (dissocial personality disorder). Also problematic are the many value systems that define the harmfulness of these states (including society, culture, individuals, and scientific theories of varying quality). Some disorders are therefore only reluctantly recognized (e.g., panic disorder), while others are only grudgingly removed from diagnostic manuals (e.g., the dissociative disorders or homosexuality). There is therefore an obvious need for an overarching understanding of the nature of psychopathology, which may be provided by evolutionary theory (e.g., Kennair, 2003; Nesse, 2005). But as Wakefield makes clear, we cannot escape the value factor.

Although there are many attempts to define psychopathology (also from an evolutionary perspective; Cosmides and Tooby, 1999; Troisi and McGuire, 2002), the most influential and debated contribution is Wakefield’s (1999, 2007) concept of the harmful dysfunction, which he (2007, p. 149) calls a hybrid account:

According to the [Harmful Dysfunction] analysis, a disorder is a harmful dysfunction, where “harmful” is a value term, referring to conditions judged negative by sociocultural standards, and “dysfunction” is a scientific factual term, referring to failure of biologically designed functioning. In modern science, “dysfunction” is ultimately anchored in evolutionary biology and refers to failure of an internal mechanism to perform one of its naturally selected functions.
Mental disorder or psychopathology is thus defined as a harmful dysfunction. While critics Fulford and Thornton (2007) claim that values are the most important part of the definition of harmful dysfunction, many evolutionary psychologists might find the value focus problematic. Natural scientists often advocate that a definition of psychopathology ought to be entirely founded on objective (or value free) science. From this perspective one might claim that Wakefield’s definition of dysfunction is all that is needed to define pathology, and that the subjective “harmfulness” criteria could be dropped altogether. This stance would suggest that dysfunctions may be detectable regardless of whether the subject or society experiences “harmfulness.” Further, it suggests that all mechanisms not functioning as they evolved to do will be accepted as “pathological.” But neither of these claims is defendable. I would argue that Wakefield is correct. We need to consider both factors: If we were able to discover such a non-harmful dysfunction, it would not be something we would wish to treat or call pathology. Our values define homosexuality, increased general intelligence of the population over time (that has us processing information in an evolutionary novel manner), the kind of individuality typical of modern Western culture, or lack of violent responses to threats and infidelity out of the domain of psychopathology. Even if to some degree mental mechanisms are not functioning as they evolved to function in the EEA (environment of evolutionary adaptedness, i.e., the past environment in which the adaptation was selected) we do not necessarily wish to class the resulting behavior as pathology. Therefore, we need to include a consideration of harmfulness as well as dysfunction.

However, the major problem is that the harmful dysfunction definition is dependent on future evolutionary mental health research in order to provide a nosology/taxonomy of functions in order to derive dysfunctions. Understanding psychopathology based on an analysis of harmful dysfunction will therefore not be relevant from a practical clinical perspective without an evolutionary psychology of normal functional psychological mechanisms as well as psychopathology—that is, a science of function is necessary to define dysfunction. Whether or not something has an evolved function is currently not a question that may be easily resolved—the mapping of our adaptations has only just begun (Tooby & Cosmides, 2000). Thus, despite Wakefield’s definition having the greatest promise of providing mental health research with a theoretically and scientifically based overarching definition of psychopathology, it currently lacks the research needed to be able to categorize mental states (harmful dysfunction and non-harmful dysfunction, as well as harmful function and non-harmful function) and create a more valid nosology.

It is possible that the combination of Wakefield’s approach to the definition of psychopathology and the necessary evolutionary psychology basic research of mapping both functional as well as dysfunctional mental mechanisms could emerge as a major influence on future diagnostic systems. As such, work on individual differences and human universals involved in both normal and psychopathological mental states are potentially relevant and beneficial from an applied, clinical perspective. As Cosmides & Tooby (1999, p. 463) suggest:

In sum, trying to decide where a condition belongs in an evolutionary taxonomy is not a sterile exercise in categorization. Achieving a genuine understanding of the adaptations that comprise our bodies and minds, how they interact, and how they breakdown, can produce new and
important insights into how to conceptualize and, therefore, treat conditions that cause human suffering.

**Three types of “Psychopathology”**

Adding to the complexity outlined above, “psychopathology” is not a clear and homogenous category. Many forms of disorder may not be defined due to statistical deviancy, suffering, or sequelae (Troisi & McGuire, 2002)—as they are not unusual, do not cause suffering, and there is no identified sequelae.

Kennair (2003, see Figure 1) suggests three types of phenotypes that clinicians define as psychopathology: adaptive psychological pain; socially undesirable effects of adaptations functioning as they were selected in the EEA; and mechanism failure. Of these, only mechanism failure would be considered true pathology (dysfunction) from a strictly biological, functional perspective. But if we were to apply Wakefield’s (1999) definition, this would only be considered pathology if these mechanism failures are also considered harmful. Psychological pain and socially undesirable traits are considered harmful, but need not be due to dysfunctions. Thus, Wakefield’s definition might assist clinicians redefine how they understand psychopathology, while at the same time highlighting the need for research on biological function.

**Adaptive Psychological Pain**

The few mainstream evolutionary psychologists who have looked into what clinicians currently define as mental disorder have usually concluded that the behaviors and mental states actually are adaptive even if they cause discomfort (e.g., Hagen, 1999; Watson & Andrews, 2002) rather than conclude that the mental disorder is due to mechanism failure. Hagen (1999) suggests that postpartum depression is an evolved adaptive behavior that is elicited by the lack of investment from the father, causing him to have to invest more in their common offspring. The notion that something proximally maladaptive (e.g., depression) can be ultimately adaptive is controversial, even among evolutionary psychopathologists (e.g., Paul Gilbert or Randolph Nesse). Nesse & Williams (1996) point out that sometimes pain is an evolved defense. Nesse and Gilbert (e.g., Nesse, 2000; Sloman & Gilbert, 2000) generally suggest that mechanisms involved in depression may be adaptations (e.g., mechanisms to prevent evolutionary maladaptive behavior, by causing discomfort), but that Major Depressive Disorder probably never was evolutionarily adaptive. Consider also Marks’ (1988) explanation of blood phobia—where the patient faints at the sight of blood due to a sudden drop in blood pressure. The phobia-induced drop in blood pressure is atypical of phobias, and the fight or flight mechanism, which is typical of fear activation, but makes sense if the individual is in danger of losing blood. But as such, many states that cause psychological pain (e.g., Hagen’s theory of postpartum depression or Mark’s approach to blood phobia) may not be dysfunctions in the evolutionary sense of the word.

Adaptive psychological pain, including different types of anxiety (e.g., spider phobia) and some types of depression, may therefore not be psychopathology according to the harmful dysfunction definition. Despite not being defined as psychopathology, such phenotypes may be considered *treatable conditions* (Cosmides & Tooby, 1999). Whether such conditions ought to be treated would need to be decided based on an understanding of the function of the evolved defense and possible consequences of blocking this defense (Nesse & Williams, 1996).
Socially Undesirable Behavior

Some conditions listed in current diagnostic manuals may be considered pathology by society at large because they are undesirable or harmful, but are not considered pathology primarily by the subject experiencing them; they are ego-syntonic (congruent with the patient’s value system or desires) or even pleasurable. Such disorders include both substance abuse as well as personality disorders. The previously discussed possibly functional states (i.e., depression, blood phobia) were considered harmful by the individual due to the psychological pain they caused. In contrast, for the present case, the possibly functional states are considered harmful by society.

An understanding of pathology needs to incorporate the idea that the context and understanding of the condition is often what defines a state as a disorder. Cosmides and Tooby (1999) call this “value-condition divergence.” It might be behavior that is generated by a fully functioning evolved adaptation, but this behavior may be considered harmful or undesirable by society. Diagnosing ego-syntonic dysfunction is challenging given our current lack of understanding of function—the mapping of the mind’s normal psychological functions is still fairly limited. Also, many types of conditions may prove to not be treatable, at least not with current evidence-based psychotherapeutic, social, or medical interventions. Mealy (1995) suggests for example that dissocial personality disorder might have increased fitness, as such, despite a lack of empathy, people with dissocial personality are functioning from an evolutionary perspective. Despite this, society in general finds sociopathic on non-empathic behavior undesirable, and would like to provide treatment. So far such treatment is not available.

Thus an unhealthy, exaggerated intake of calories in subjects with no metabolic dysfunction, substance abuse, and several types of dependencies may all be due to fully functioning adaptations in a culture where the availability of calories, alcohol, and other ingestible substances is a consequence of the same adaptations that cause the problem behavior. The same may be the case for violent jealousy—it may have been adaptive in the past by increasing reproductive success, but is not acceptable in official Western culture. These examples would therefore not be pathologies according to the harmful dysfunction definition. Clinicians need to be able to differentiate between pathology and undesirable function (including treatable conditions).

Mechanism Failure

The most obvious and prototypical definition of disorder is that the evolved mental mechanism is malfunctioning—mechanism failure or dysfunction. An adaptation may be calibrated differently due to genes, development, or current or past environment. There is a limit, though, to how much such factors may adjust the reactivity of an adaptation before the mechanism can be considered to be malfunctioning. But malfunction is not as easy to identify as one might think. It is premature to conclude that every disorder in the diagnostic manuals is mainly explained by mechanism failure. There will therefore be many diagnoses that are not covered by the harmful dysfunction definition, and thus might be relevant for consideration as treatable conditions or functionally adaptive states.

There are few mental disorders that are clearly due to mechanism failure; I will discuss some candidates below. Whether the mechanism failure is caused by inadequate environmental stimulation, developmental disturbance, or genetic deficiencies is also difficult to ascertain. Despite this, mechanism failure is still the most typical explanation within mental health
research: Pathology is often claimed to be due to brain disorder, as in the American National Institute for Mental Health’s (NIMH) explanation of depression.

On the other hand, when evolutionary psychology attempts to offer competing theoretical explanations, it is not surprising given the adaptationist program that one typical hypothesis is that the disorder is not due to mechanism failure, but actually is adaptive or due to the workings of adaptations. There is potential in considering both adaptations (states that function, so that one may avoid iatrogenic conditions) as well as dysfunctions (in order to provide Wakefield’s definition with an empirical foundation).

Also, most of the obvious brain malfunctions are grouped apart from mental disorders, and are assessed and handled by neuropsychologists rather than psychiatrists and clinical psychologists. One example of mechanism failure is the case history of the patient with neurological damage that caused a specific impairment of the cheater detection mechanism discovered by Cosmides (Stone et al., 2002). But this kind of explicit mechanism failure is not typically a mental health case.

Among mental disorders, some features of schizophrenia are more typical neuropsychological mechanism failures, or impairments. Further, the mood swings of bipolar disorder are maladaptive in modern society, and seem to be dysfunctional—but we do not know enough to conclude. In general, depression seems to reduce fitness. The anxiety disorders (fear of non-threatening stimuli) reduce function—although the basic emotion fear is adaptive when it protects us against real threats. Yet again, it is hard to conclude that there is mechanism failure. Also, anxiety disorders and obsessive-compulsive disorders respond rapidly to psychological treatment, and thus the mechanism failure model seems unlikely (it seems to be more a case of oversensitive calibration, rather than dysfunction). The personality disorders reduce social adaptation. But even suicidal behavior may increase inclusive fitness (de Catanzaro, 1995). The lack of empathy in psychopathy (Mealy, 1995) may be adaptive for the individual, in general as a social strategy and under specific ecological conditions (e.g., war, famine). Many dysfunctions may exist, but they need to be researched from a functional, adaptationist perspective before we may conclude that they really are dysfunctions. Thus the conclusion is that while we have a number of candidates for true mechanism failure, we are not currently able to conclude.

An Integrative Revised Evolutionary Psychology for The Study of Psychopathology

In order to conceptualize individual differences in psychopathology, mainstream evolutionary psychology needs to integrate theory and findings from evolutionary developmental psychology, behavioral genetics, and research on the etiology of disorders, as well as modern cognitive behavioral therapy research as a basic science of proximate mechanisms. Evolutionary psychology’s integrative potential is largely due to the fact that evolutionary approaches are aware of different levels of analysis, but also that the cognitive mechanism level of analysis makes for a generic theory in all approaches that consider systematic information processing. As such, other research programs within mental health science that focus on mental mechanisms and information processing, such as cognitive behavioral therapy research (Alford & Beck, 1997; Gilbert, 2002; Kennair, 2007), might be of special interest to evolutionary psychologists venturing into this field.

There are therefore two major tasks for an evolutionary psychology of psychopathology:
First, evolutionary psychology must be expanded to include mental disorders. This means continuing evolutionary psychology’s task of predicting and mapping the functional, adapted mind. When considering psychopathological phenomena, this mapping would include describing syndromatic dysfunctions and providing good descriptions of the phenotypes and adaptations that need to be studied. A good understanding of the abnormal demands an understanding of normal phenomena and universals, as well as when and how these phenomena malfunction.

The second challenge is to provide the current disparate evolutionary approaches to psychopathology with an overarching meta-theory. Providing evolutionary psychopathology with a relevant integrative theory will demand theoretical developments to better conceptualize and predict individual differences. This will involve evolutionary psychology becoming fundamentally developmental, incorporating genetic individual differences at a conceptual level, and focusing on predicting environmental cues that may alter or harm the development of adaptations.

The model presented in Kennair (2003) may aid further evolutionary psychology research into psychopathology (see Figure 16.1). The model itself is a synthesis of different approaches within mainstream evolutionary psychology and general evolutionary psychopathology. I also recommend considering Buss and Greiling’s (1990) and Nesse’s (2005) lists of factors that cause individual differences. The Kennair (2003) model highlights a few of the important revisions of evolutionary psychology theory that might be


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**Figure 16.1. The Evolution Psychology of Psychopathology**

necessary when investigating phenomena that are not universal (e.g., either due to genetics or due to age). The following section elaborates on the levels of analysis and phenomena an evolutionary psychology approach will need to encompass based on Figure 16.1.
The Individual’s Genetic Potential

The individual’s genetic potential includes both the human universal adaptations and by-products of those adaptations (Tooby & Cosmides, 1992). Research into adaptive snake phobia (Mineka et al., 1984), the function of blood phobia (Marks, 1988), Watson and Andrews’ (2002) approach to depression in general, Hagen’s (1999) approach to postpartum depression, or de Catanzaro’s (1995) suggestion that suicide may historically have increased fitness, are prime examples of investigations of adaptations that result in mental disorders. Much of the mainstream evolutionary psychology research would naturally focus on adaptations. But the analysis additionally needs to be expanded to cover other phenomena such as by-products (non-selected processes of the mind with no current or previous function, Buss et al., 1998).

Crow’s (2000; Berlim et al., 2003) research on schizophrenia is an example of conceptualizing the disorder as a by-product rather than the selected trait. Crow’s theory attempts to resolve the schizophrenia paradox: How and why a disorder that reduces fitness has not been selected out of the human gene pool, but is equally frequent in almost all ethnic groups. Crow suggests that the speciation event that provided humans with a capacity for language and brain lateralization is the reason why schizophrenia exists. Crow’s approach may be called a by-product approach to mental disorder. The human universal is the capacity for language—the accidental by-product is schizophrenia for a subset of the population due to genes that are universal in the species’ gene pool.

Another possible by-product disorder is panic disorder. Panic disorder is maintained by a misinterpretation of bodily or cognitive sensations (anxiety symptoms). The patient typically believes these are signs that he or she is going to faint, have a heart attack, choke, or go mad. As these are not real threats, it would seem that our capacity to incorrectly interpret our bodily sensations causes the disorder. The treatment of panic disorder is relatively simple—the patient is helped through behavioral experiments to discover that the imagined catastrophes cannot come true (Clark et al., 1999; Kennair, 2007).

This level must also include an analysis of individual genetic differences. Some of these differences may be noise or pathogen defense (Tooby & Cosmides, 1990); others may be recent harmful mutations that are part of the explanation of subpopulations of specific disorder patterns (Ozaki et al., 2002). And some of these may be genetic differences that result in different adaptive morphs within the population (e.g., Caspi et al., 2003), due to either frequency dependent selection (Tooby & Cosmides, 1990) or assortative mating (Rowe, 2002).

Findings from behavioral genetics also need to be integrated with evolutionary psychology research (Segal, 2005; Segal & Hill, 2005). Such integration is necessary for evolutionary psychology to become updated on modern developmental research. Behavioral genetics has provided new understanding of developmental processes, and the effects of genes as well as non-shared environment. Phenotypic plasticity may be an important explanation when considering individual differences—and environmental cues may thus cause individual differences (Buss & Greiling, 1999; Tooby & Cosmides, 1990; West-Eberhard, 2003). At the same time, it is necessary to also consider the possible consequences of genetic differences when one refocuses from human universals to an in-depth and integrative approach to individual differences (see Wilson, 1994). Behavioral genetic research also shows that environmental factors are important.
Sex differences are crucial—and are a specific level of analysis when considering human universals and individual differences. Evolutionary psychologists expect to find sex differences only when males and female have recurrently faced different adaptive problems over human evolutionary history (Buss, 1995)—in domains such as mate choice, parental investment, paternal certainty (e.g., in jealousy, Buss & Haselton, 2005, or mate preferences, Buss et al., 1990). The fact that there are robust cross-cultural sex differences in the susceptibility to develop mental disorders (e.g., Gater et al., 1998) is important theoretically. Most of the relevant genes are shared between the sexes, and both genes and environments cause, e.g., depression (Sullivan, Neale & Kendler, 2000). Evolutionary psychology is already able to handle sex differences and hypothesize about effects of environmental factors. An evolutionary psychology that is more aware of individual genetic differences and development might be a fruitful approach to address, explain, and predict sex differences in mental disorders.

**Developmental Adaptations**

Mainstream evolutionary psychology has focused primarily on the adult human mind. The reproductive focus of evolutionary theory has made the sexually mature adult the relevant subject of study for most evolutionary researchers. Bjorklund (2003) and the field of developmental evolutionary psychology are critical to this focus. The human animal’s life history is unique: We are born so early relative to our level of maturation at birth that we need more caretaking for a longer period of time than other species. Our complex social systems also demand years of socialization and maturation to navigate (e.g., Flinn, 2005; Flinn & Ward, 2005). Thus, survival at every point of the human development is important for the individual to reach sexual maturity and reproduce. Also, human females live longer in a non-reproductive state than other mammals.

Developmental psychopathology (e.g., Rutter, 2006) is a foundational approach to the study of mental disorder. Most types of pathology—from schizophrenia to social phobia—have developmental precursors or developmental or life history events (e.g., first pregnancy) characterized by large hormonal changes (i.e., puberty) accompanied by a large reorganization of the brain that seem to increase likelihood of disorder onset. This developmental reality suggests that different adaptations may be coming online in theoretically predictable patterns.

Evolutionary developmental psychology (Burgess & MacDonald, 2005; Ellis & Bjorklund, 2005) has experienced a boost of interest the last few years. This work is filling a conceptual hole and a lacking level of analysis within mainstream evolutionary psychology. One of the sets of human universals is our developmental adaptations and species’ life history. There are both differences and similarities between members of our species due to maturational and developmental processes—not only environmental input or genome. Considering these factors may aid the merger of developmental psychology, behavioral genetics, and evolutionary psychology (Kennair, 2005).

Evolutionary developmental psychopathology (e.g., Pitchford, 2001) is therefore a necessary level of analysis to add to any investigation of mental disorder. And while the trend within evolutionary psychology is toward a greater interest in developmental psychology, there has been little empirical research (although consider e.g., Ellis, 2003; Ellis & Essex, 2007; Hawley & Little, 1999). The current model suggests that both normal evolutionary psychology and evolutionary psychological investigation of psychopathology need to consider developmental aspects closely—including life history theory (Kaplan & Gangestad, 2005).
Certain disorders appear at different times through development and are considered differently depending on the age and context of the individual. The same behavior in a five-year-old child might be age typical learning of social rules and rituals, but be considered pathological OCD in a teenager. Some disorders seem to “burn out” with age (e.g., borderline personality disorder), while others may become more pronounced (e.g., depressions). Disorders are often considered more chronic today (e.g., depressions often return), and we are more aware that shy children often turn into socially phobic or avoidant adults. As with the normal range of human personality, many of the same questions about stability and change are relevant. Many disorders (e.g., generalized anxiety disorder) are present throughout life, and hence suggest stability. Moreover, different individuals may have different developmental courses of their disorder. And the same disorder may manifest itself differently at different ages, much as personality traits do.

Etiological research from a behavioral genetics perspective is also an area that one needs to consider. The fact that many disorders have a genetic component is accepted by most researchers today. The surprising findings (Pinker, 2002; Turkheimer, 2000) are the actual effects of environmental factors: Family environments do not shape personalities as our theories have suggested; where family members are similar due to genetics, they are different due to non-shared environment.

**Environmental Influences**

Evolutionary psychology has traditionally explained most phenotypic variance through environmental variance—an evolved universal human nature differentially stimulated by differing contexts (Tooby & Cosmides, 1990). Thus, Buss and Greiling (1999) suggest that the Belsky-Steinberger-Draper hypothesis may be an important developmental and environmental explanation of how an individual’s developmental pathway may be shifted due to specific environmental cues and contexts in interaction with a universal human nature. The Belsky-Draper hypothesis (Belsky, 2000; Belsky, Steinberger & Draper, 1991; Draper & Harpending, 1982; Quinlan, 2003) suggests that girls raised without an investing father mature sexually, partake in sexual activity, and become pregnant earlier than girls raised by an investing father. This is due to the early experience of a father’s investment calibrating the specific adaptations evolved to assess the likelihood of paternal investment in the relevant ecology and generating coordinated psychophysiological behavior and development that solves the statistically likely adaptive problem. Rowe (2000) criticized this research for focusing too much on environmental causes, pointing out that the traits being studied are heritable. Ellis et al. (2003) found further support for the Belsky-Steinberger-Draper hypothesis, while Mendle et al. (2006) provide research that tested both evolutionary and behavioral genetic hypotheses where genetics seemed to explain most of the variance (but see also Tither & Ellis, 2008).

It would seem that an assortative mating model best explains the findings, as both environmental as well as genetic factors explain variance (see Rowe, 2002). One has to combine two perspectives: The importance of heritability, not merely environmental input (Wilson, 1994), and the importance of having a developmental perspective (Kennair, 2005; Pitchford, 2002). The idea that different environments can elicit different sets of adaptations due to specific ecological cues attains importance from both life history and evolutionary developmental perspectives.
Within behavioral genetic research on personality traits, the most surprising finding is the effect of the environment. The fact that genes explain a moderate amount of variance was an important empirical finding, although probably as expected from a genetic perspective. Turkheimer (2000; see also Pinker, 2002) sums up the effects of the environment thus: Genes explain more of the variance than growing up in the same family, but environmental effects—other than families (shared environment)—explain most of the variance. At the group level of analysis, genes make siblings similar, but the environment makes them different. Without resorting to group selection explanations, this does make some sense from an evolutionary perspective. There is limited genetic variance in our species, compared to similar species (Cosmides, Tooby & Kurzban, 2003). Parents and their children, or siblings, are 50% genetically similar—and in many cases in the EEA with small groups (Dunbar, 1993), and thus mating among closer relatives than what is typical today, this number may have been larger. If genes made us similar—which they necessarily would—and parents and siblings made us even more similar, as most theories outside of behavioral genetics suggest, then there would be very little phenotypic variance. If phenotypic variance was beneficial, selection would have increased the likelihood of being influenced by environmental forces other than those one was most genetically similar to, reducing effects of shared environment over lifespan. See also Harris (1995) for an alternative model that invokes the evolutionary importance of peer influences.

These perspectives are relevant for psychopathology. Genes influence the development of disorders. Poulton and Menzies (2002) and Kendler, Prescott and Myers (2002) challenge the mainstream ideas of the etiology of phobias specifically—and psychopathology in general. Poulton and Menzies (2002) challenge the idea that association is the major explanation of how phobias are acquired (see Rachman, 1977). Summing up years of longitudinal research (e.g., Poulton et al., 1998, 1999), they conclude that it would seem that non-associative explanations better explain how phobias develop. Kendler, Myers, and Prescott (2002), moreover, challenged the stress-diathesis model of the etiology of phobia. They conclude that there is little support for the idea that disorders are caused by the interplay between genetic vulnerability and the severity of the traumatic experience or stressor. For many years most clinicians have considered the anxiety disorders to be caused by conditioning experiences—and that these disorders thus had the most obvious pathways. While non-shared environmental factors explain most of the variance of who develops anxiety disorders, we now know that we do not really know what specific factors in the environment explain specific disorders. This is true of all disorders. Evolutionary psychologists interested in how individual differences develop, and how differences in mental mechanisms are maintained, may be able to predict under what circumstances the development of normal mental adaptations will be harmed or changed.

Theories about effects of shared environment lack strong empirical support and the general findings within behavioral genetics challenge most of the existing consensus. Thus mainstream evolutionary psychology’s approach to phenotypic plasticity and effects of environmental cues on developmental pathway shifting mechanisms (e.g., Buss & Greiling, 1999; Ellis et al., 2003) or phenotypic variance—might be an important approach for the study of etiology. Incorporating insights from genetics and developmental studies with evolutionary analysis increases the sophistication of the models and empirical tests. Despite the importance of genes, non-shared environmental factors account for most of the variance (Turkheimer, 2000). And sometimes
common genes, but different environments, present as different phenotypes or psychopathological syndromes (Kendler, 2004).

It is further important to consider possible mismatch (e.g., Nesse & Williams, 1996; Nesse, 2005) between the modern or current environment and the evolved mental adaptations that evolved to function under different environmental conditions. This means that the adaptations may not be receiving the expected and thus adequate environmental input and stimulation, causing them to process information in a non-adaptive manner in the current environment. Alternatively, the adaptations’ development may be disturbed (Buss & Greiling, 1999; Kennair, 2003; Nesse, 2005). There is little that decisively concludes that the modern environment has caused an increase in mental disorder (Nesse, 2005), although there is some evidence that depression is rising (Compton et al., 2006; Klerman & Weissman, 1989), especially among young women. Any rapid change of prevalence suggests that recent changes in western societies may cause increased mismatch, although what these changes are we do not know. Sandseter and Kennair (submitted) suggest that limiting risky play among kindergarten children, a recent environmental change, may cause an increase of anxiety due to less possibility for habituation or learning to cope with one’s environment and normal interaction with one’s ecology.

Social cognition, social development, and social selection have been important for human mental evolution—our species’ most important selection forces have been other hominins. As such we have replicated and conserved our most psychologically relevant environmental features as these consist of the social and relational behavior generated by the human nature (i.e., the behavior) of other people. It is therefore reasonable to assume that most of our relational and social context has been relatively stable since our species evolved and migrated out of Africa. And, as Buss (1996) suggested, when considering the evolution of personality traits, other people’s traits laid the basis for some of our most important and stable adaptive problems, and also through cooperation some of the most available solutions to our adaptive problems. Consider Flinn’s recent approach (2005; Flinn & Ward, 2005) to the evolved developing social mind; it suggests that the very recent breakdown of intergenerational families might have detrimental effects on mental health.

The environment is more than psychosocial; it is also biological and chemical. The environment must be conceptualized as the biopsychosocial context at all levels from genes and chemicals and nutrients to social and political factors. One must consider how these contexts influence the development of the individual’s adaptations. Evolutionary psychology has generally been sensitive to environmental influences—especially social factors. Biochemical factors may need to be given more attention (e.g., Mysterud & Poleszynski, 2003). One may further note how mismatch will not necessarily only have negative effects. Despite the ready availability of sugar in our modern environment causing dental caries, pathological obesity, and diabetes, an increased availability of calories throughout development may cause the population-wide increase in brain development manifesting in an increase in a population’s intelligence (the Flynn effect). As such, it would be interesting to investigate the effects of the evolutionary and culturally novel childrearing practice of letting infants sleep on their own: Does it increase the likelihood of disorder? Does it cause a greater individuality—and thereby better function in Western society? Mismatch that is too detrimental would be selected against (e.g., the child rearing practices of orphanages in Ceau§escu’s Romania; Rutter, 2006).
Limitations and Foundations of the Integrative Revised Model

This model is an attempt to describe a synthesis of recent work within both general evolutionary psychopathology and mainstream evolutionary psychology. The model itself does not make any specific predictions. It aims to make explicit several of the important biopsychosocial factors and levels of analysis that are involved in the study of psychopathology: Human nature and individual genetic differences; age and life history; environmental factors and how one hypothesizes these factors to influence the state or expression of the disorder; and whether the disorder is or was adaptive or due to malfunction. For Wakefield’s definition to be relevant, it is crucial that there exists work that can illuminate the conditions and phenotypes that may be classed as “dysfunctions.”

The mainstream evolutionary psychology research methodology is central to generating predictions based on strategic analysis of the EEA and the computational, functional analysis of information-processing modules of the mind, based on the application of middle level evolutionary theories to generate predictions about the evolved architecture of the modern mind. One must be able to identify function in order to assess dysfunction. It is also based on evolutionary psychology’s modular model of the mind, with a focus on adaptations at all levels of the model; in the investigation of spandrels (as the adaptation is the testable hypothesis) as well as in mechanism malfunction or developmental analysis. What is needed is a focus on the relevant phenotypes within psychopathology, and an increased focus on individual differences, development, and genetics, all while maintaining the focus on multiple adaptations and the effects of environments on adaptive shifts in development or causing phenotypic variance.

Integrating Evolutionary Psychology with Evolutionary Psychopathology

Above I suggested possible revisions to evolutionary psychology theory and methodology for the study of the nature of psychopathology. The main message is the double claim that the general field of evolutionary psychopathology needs an integrative and rigorous metatheory and research program and that the cognitive psychological science of evolutionary psychology is the best candidate. Moreover, mainstream evolutionary psychology must be somewhat modified to become more focused towards individual differences and developmental issues; which is why this volume is important.

Changes through the last few years within mainstream evolutionary psychology have already laid some of the groundwork for a more relevant theory for psychopathology. Much of the work by David Buss and his students on sex differences and individual differences (including personality traits, e.g., Buss, 1996; Buss & Greiling, 1999) have moderated the universal human nature approach. Differences due to age have also become more central (see Burgess & MacDonald, 2005; Ellis & Bjorklund, 2005; Kennair, 2005).

Evolutionary psychopathology needs an integrative metatheory in order to provide cumulative theory building and an overarching research program. The alternative to evolutionary psychology becoming the integrative meta-theory for the study of the nature of mental disorders would seem to be to not have any overarching model and research program.
What are the major hindrances that prevent evolutionary psychology from already being an integrative metatheory? To a large degree, opposition to evolutionary psychology may be due to academic traditions and borders between different areas of research. Clinical psychology and psychiatry have their own strong traditions and models of human nature. Within these disciplines the influence of human ethology is greater than that of evolutionary psychology (e.g., through the work of John Bowlby; Ainsworth & Bowlby, 1991). The divide between social and cognitive research psychology and academic clinical psychology has been great, and there has been remarkably little transmission of information and models between these disciplines. An integrative approach such as evolutionary psychology might be able to improve this situation.

The developmental pathways of psychopathology—the etiology of mental disorders—may be understood better from an evolutionary life history perspective (Kaplan & Gangestad, 2005). Developmental issues and the question of why some people develop disorders are among the most interesting and least understood mysteries of mental health care. For years these quandaries were considered answered by Freudian theory or by mainstream developmental psychology. Today we are aware that even the whys and hows of simple phobias are not as obvious as we once thought (e.g., Kendler, Myers & Prescott, 2002; Poulton & Menzies, 2002). We actually know less about development than we thought we did. Many studies are comparisons of children and parents. Similarities between parents and children usually are more due to common genes than environment (Plomin et al., 2000; Turkheimer, 2000). Thus most developmental research has probably studied effects of heritability more than the effects of environmental factors. We are therefore not aware of many environmental factors that explain the etiology of psychopathology. The evolutionary approach might be able to generate predictions that help identify influential environmental factors.

There are several fruitful consequences to including the study of psychopathology into the mainstream normal psychology research program of evolutionary psychology. Baron-Cohen (1997) pointed out how the malfunction of the mind may inform us of how the mind works. Stone et al. (2002) illustrate how the lack of a specific mental ability in a patient with brain injury is part of the evidence of the existence of a specific module in the fully functioning brain. Considering psychopathology as something apart from normal function is a limited perspective. Mental disorder is to a large degree part of universal human nature. Although most people do not have mental disorders, the percentage of reproductive age population that does is so large that mainstream evolutionary inquiry into how and why is warranted.

Many clinicians within evolutionary psychopathology are skeptical about the contributions of non-clinicians (e.g., the work from non-clinicians on the benefits of depression; Hagen, 1999, 2002, 2003; Watson & Andrews, 2002). This may be due to different causes: A protectionism among clinicians of their own status and position, the clinicians’ preconceptions of the phenomena, or a lack of insight on the part of the non-clinicians on the specific clinical phenomenon due to a lack of training. Increased research on psychopathology within evolutionary psychology will necessarily mean that more non-clinicians will focus on clinical questions.

**Evolutionary Informed Treatments**

While some authors (e.g., Sloman & Atkinson, 2000; Troisi & McGuire, 2002) already have suggested interventions based on evolutionary insights, it is important to note that from a scientific perspective, a treatment must be proven effective through clinical trials before one can...
recommend interventions to patients. Although it is possible to imagine several theoretically predictable interventions, it is far from certain that these work or address the relevant maintaining factors of the disorder. One needs to compare interventions to no-treatment control groups, and compare them with other evidence-based therapies. It is important to not jump to conclusions based on science in such a manner that the applied intervention is not scientific: Science-based interventions must also be evidence-based.

Currently there are few therapies based on evolutionary theory. Even fewer of these are documented through clinical trials. For example, even though Ilardi (2009) has developed a treatment package that combines a broad set of insights from evolutionary studies with cognitive behavior elements, as of this writing this approach still needs to document efficacy.

Additionally, Gilbert and co-workers (Gilbert & Irons, 2005; Gilbert & Mayhew, 2008; Gilbert & Procter, 2006) have developed a treatment method based on insights from ethological work such as Price (1967). Gilbert’s treatment, called Compassionate Mind Training, may be considered an ethological cognitive behavior therapy in that it focuses on the reduction of internalized hierarchical mindsets such as low self esteem, shame, and self criticism, which are generic features of mental disorder. Despite promising results, this approach still needs further work to prove its efficacy with major diagnostic categories.

These beginnings are promising, as is an increased interest in the evolved nature of psychopathology. This might provide us with a broader set of interventions in our clinical work.

Conclusions

Thus far, evolutionary psychopathology has largely been a theoretical, rather than empirical, endeavor, by focusing on how one may theoretically understand and explain the evolution of psychopathology or the nature of mental disorders. As with evolutionary personality psychology (Buss, 1991), theory in evolutionary psychopathology is ahead of the data (see Figueredo et al., 2005). This is probably true of all science, but the gap needs filling. If evolutionary personality psychology shall continue to be relevant, there will need to be a surge of empirical research in the next few years. The same is true for evolutionary psychopathology.

On the other hand, empirical research needs an overarching meta-theory to guide investigation. Accumulation of knowledge is hard to accomplish without such a framework. Empirical investigations will under such circumstances tend toward fragmented data; dustbowl empiricism. Also, without an integrative meta-theory there will be an unfruitful discussion between researchers within the field over theoretical foundations, methods, and whether the research is truly evolutionary or how to best synthesize findings.

The evolutionary psychology research program offers a set of meta-theoretical rigorous principles: To focus on mental adaptations rather than merely on phenotypes or on adaptiveness; to attempt to predict what adaptations may exist based on an analysis of the past environment; and to use established evolutionary middle-level theories in an attempt to formulate hypotheses about modern human nature. A specific meta-theory and research program for the study of the evolution of mental disorders may prove fruitful and integrative, causing greater interest in the study of clinical topics among empirical research psychologists.

The current integrative, synthetic approach might help to bridge the gap between mainstream evolutionary psychology and clinicians and personality psychologists. Mainstream evolutionary psychology is first and foremost the study of human universals (Tooby & Cosmides, 1990)—but
as soon as one considers important topics such as sex differences or personality traits due to
stable selection pressures, one needs to expand the meta-theory. An integrative meta-theory for
all of psychology needs to consider both human universals and individual differences (Scarr,
1995). Topics such as personality or psychopathology demand meta-theory and models that can
handle individual differences.

A broad, integrative biopsychosocial approach may also convince clinicians and theoreticians
outside of evolutionary approaches of what an evolutionary perspective has to offer. In
conclusion, the present chapter argues

that mental mechanisms—which are the major integrative feature of evolutionary psychology—
need to be the focus of future evolutionary psychopathology. At the same time, evolutionary
psychology needs to better understand how these mental mechanisms develop, are influenced by
different environments and genetic differences, and break down or cause social or emotional
maladjustment.

Evolutionary psychology is—after two decades—still a rather young approach, and is still
maturing theoretically and expanding into new research areas. Evolutionary psychology’s forte is
its widespread meta-theory and well-defined research program within psychology. Thus, it offers
a foundation for a more rigorous evolutionary study of human nature and concomitant functional
mental mechanisms. Evolutionary psychopathology, more generally speaking, is an older
approach, including theorists such as Freud, Bowlby, Meyer and Price. Researchers using
different evolutionary approaches to psychopathology cover almost the entire field of mental
disorders, while neither having an integrative research program nor an integrative theory or
model of human nature that collects larger groups of researchers. Thus there is little accumulative
power within the field, and not enough focus on mental mechanisms. Furthermore, within general
psychiatry and clinical psychology, there is no general understanding or agreement of what
constitutes mental disorder. The scientific progress of the field depends in large part on defining
these phenotypes and phenomena.

Being convinced that the human universal adaptations are predictable and fundamental aspects of
our minds, I would claim that the evolutionary psychology research program is the most relevant
first approach when studying individual differences in the form of psychopathology. We need a
predictive, evolutionary mechanism-focused science of function as well as dysfunction.

As argued here, the most promising general definition of psychopathology is Wakefield’s concept
of the harmful dysfunction. Despite clinicians treating treatable conditions (i.e. clinicians in
general have disparate theoretical definitions of pathology, and in general do not consider
function or dysfunction, merely theoretically defined treatable conditions), the harmful
dysfunction might be the best definition of pathology. The practical application of this new
definition is dependent upon the development of an evolutionary psychology of psychopathology
that focuses on the mental mechanism, and that can provide us with an objective nosology of
mechanism failure that enables clinicians to recognize true dysfunctions. This restructuring has
potential novel clinical value. The future definition and understanding of mental disorder may be
decided by researchers that work under the empirical and theoretical auspices of evolutionary
psychology.
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