PART III

Evolutionary Genetics of Personality

9 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;

ED: Pls check if the edited RH is ok?

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?

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—mere 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 β_2-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 & Miller, 2006; Thornhill & 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, 2007). 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 & Møller, 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

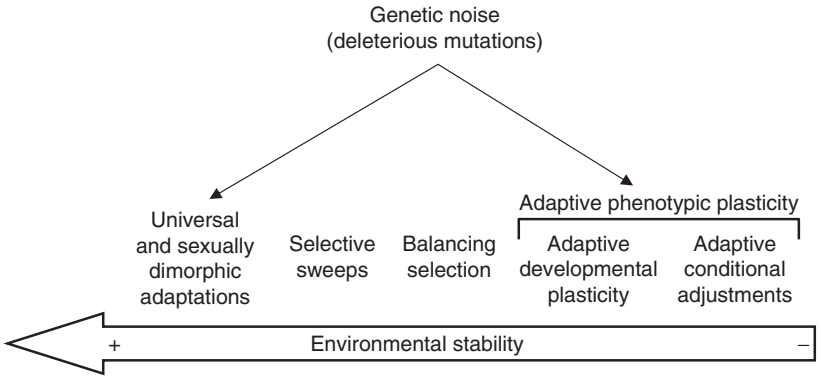

Figure 9.1

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 inter-individual 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.

Acknowledgements

I am funded by the UK Medical Research Council (Grant No. 82800). This work was undertaken within The University of Edinburgh Centre for Cognitive Ageing and Cognitive Epidemiology, part of the cross council

Lifelong Health and Wellbeing Initiative. Funding from the BBSRC, EPSRC, ESRC and MRC is gratefully acknowledged. I thank David Buss, Patricia Hawley, Wendy Johnson, and Geoffrey Miller for helpful comments.

References

Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: on the threshold of a new neurobiology. *Nature Reviews Genetics*, *9*, 341–355.

Aguilera, A., & Gomez-Gonzalez, B. (2008). Genome instability: A mechanistic view of its causes and consequences. *Nature Reviews Genetics*, *9*, 204–217.

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.

Bamshad, M., & Wooding, S. P. (2003). Signatures of natural selection in the human genome. *Nature Reviews Genetics*, *4*, 99–111.

Bates, T. C., Luciano, M., Lind, P. A., Wright, M. J., Montgomery, G. W., & Martin, N. G. (2008). Recently-derived variants of brain-size genes ASPM, MCPH1, CDK5RAP and BRCA1 not associated with general cognition, reading or language. *Intelligence*, *36*, 689–693.

Batty, G. D., Deary, I. J., & Gottfredson, L. S. (2007). Premorbid (early life) IQ and later mortality risk: Systematic review. *Annals of Epidemiology*, *17*, 278–288.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*, 647–670.

Bentley, D. R., Balasubramanian, S., Swerdlow, H. P., Smith, G. P., Milton, J., Brown, C. G., et al. (2008). Accurate whole human genome sequencing using reversible terminator chemistry. *Nature*, *456*, 53–59.

Bernard, L. C., Mills, M., Swenson, L., & Walsh, R. P. (2005). An evolutionary theory of human motivation. *Genetic, Social, and General Psychology Monographs*, *131*, 129–184.

Bielby, J., Mace, G. M., Bininda-Emonds, O. R. P., Cardillo, M., Gittleman, J. L. et al. (2007). The fast-slow continuum in mammalian life history: An empirical reevaluation. *American Naturalist*, *169*, 748–757.

Bouchard, T. J., jr., Lykken, D. T., Tellegen, A., & McGue, M. (1996). Genes, drives, environment, and experience: EPD theory revised. In C. Benbow & D. J. Lubinski (Eds.), *Intellectual talent: Psychometric and social issues* (pp. 5–43). Baltimore: John Hopkins University Press.

Boyd, R., Gintis, H., Bowles, S., & Richerson, P. (2003). The evolution of altruistic punishment. *Proceedings of the National Academy of Sciences USA, 100*, 3531–3535.

Bürger, R. (2000). *The mathematical theory of selection, recombination, and mutation*. West Sussex: Wiley.

Bürger, R. (2005). A multilocus analysis of intraspecific competition and stabilizing selection on a quantitative trait. *Journal of Mathematical Biology, 50*, 355–396.

Buss, D. M. (2003). *The evolution of desire* (4th ed.). Basic Books.

Buss, D. M. (2009). How can evolutionary psychology successfully explain personality and individual differences? *Perspectives on Psychological Science, 4*, 359–366.

Carroll, J. B. (1993). *Human cognitive abilities*. Cambridge: Cambridge University Press.

Cochran, G., & Harpending, H. (2009). *The 10,000 year explosion: How civilization accelerated human evolution*. New York: Basic Books.

Crespi, B., & Badcock, C. (2008). Psychosis and autism as diametrical disorders of the social brain. *Behavioral and Brain Sciences, 31*, 241–261.

Deary, I. J. (2000). *Looking down on human intelligence: From psychometrics to the brain*. Oxford: Oxford University Press.

Deary, I. J., Penke, L., & Johnson, W. (2010). The neuroscience of human intelligence differences. *Nature Reviews Neuroscience, 11*, 201–211.

Deary, I. J., Whalley, L. J., & Starr, J. M. (2008). *A lifetime of intelligence: Follow-up studies of the Scottish Mental Surveys of 1932 and 1947*. Arlington, VA: American Psychological Association.

Del Giudice, M. (2009). Sex, attachment, and the development of reproductive strategies. *Behavioral and Brain Sciences, 32*, 1–67.

Denissen, J. J. A., & Penke, L. (2008a). Individual reaction norms underlying the Five Factor Model of personality: First steps towards a theory-based conceptual framework. *Journal of Research in Personality, 42*, 1285–1302.

Denissen, J. J. A., & Penke, L. (2008b). Neuroticism predicts reactions to cues of social inclusion. *European Journal of Personality, 22*, 497–517.

Denissen, J. J. A., Penke, L., Schmitt, D. P., & van Aken, M. A. G. (2008). Self-esteem reactions to social interactions: Evidence for sociometer mechanisms across days, people, and nations. *Journal of Personality and Social Psychology, 95*, 181–196.

DeYoung, C. G., Peterson, J. B., & Higgins, D. M. (2005). Sources of openness/intellect: Cognitive and neuropsychological correlates of the fifth factor of personality. *Journal of Personality, 73*, 825–858.

Eaves, L. J., Kirk, K. M., Martin, N. G., & Russell, R. J. (1999). Some implications of chaos theory for the genetic analysis of human development and variation. *Twin Research, 2*, 43–48.

Ebstein, R. P. (2006). The molecular genetic architecture of human personality: Beyond self-report questionnaires. *Molecular Psychiatry*, *11*, 427–445.

Ellis, B. J. (2004). Timing of pubertal maturation in girls: An integrated life history approach. *Psychological Bulletin*, *130*, 920–958.

Evans, P. D., Gilbert, S. L., Mekel-Bobrov, N., Vallender, E. J., Anderson, J. R., Vaez-Azizi, L. M., et al. (2005). Microcephalin, a gene regulating brain size, continues to evolve adaptively in humans. *Science*, *309*, 1717–1720.

Eyre-Walker, A., & Keightley, P. D. (2007). The distribution of fitness effects of new mutations. *Nature Reviews Genetics*, *8*, 610–618.

Fay, J. C., Wyckoff, G. J., & Wu, C. I. (2001). Positive and negative selection on the human genome. *Genetics*, *158*, 1227–1234.

Figueredo, A. J., Sefcek, J. A., Vasquez, G., Brumbach, B. H., King, J. E., & Jacobs, W. J. (2005). Evolutionary personality psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 851–877). Hoboken: Wiley.

Finch, C. E. & Kirkwood, T. B. L. (2000). *Chance, development, and aging.* Oxford: University Press.

Fink, B., & Penton-Voak, I. (2002). Evolutionary psychology of facial attractiveness. *Current Directions in Psychological Science*, *11*, 154–158.

Flinn, M. V., & Alexander, R. D. (2007). Runaway social selection. In S. W. Gangestad & J. A. Simpson (Eds.), *The evolution of mind* (pp. 249–255). New York: Guilford Press.

Frazer, K. A., Murray, S. S., Schork, N. J., & Topol, E. J. (2009). Human genetic variation and its contribution to complex traits. *Nature Review Genetics*, *10*, 241–251.

Gangestad, S. W., Haselton, M. G., & Buss, D. M. (2006). Evolutionary foundations of cultural variation: Evoked culture and mate preferences. *Psychological Inquiry*, *17*, 75–95.

Gangestad, S. W., & Simpson, J. A. (2000). The evolution of human mating: Trade-offs and strategic pluralism. *Behavioral and Brain Sciences*, *23*, 573–644.

Gangestad, S. W., & Yeo, R. A. (1997). Behavioral genetic variation, adaptation and maladaptation: an evolutionary perspective. *Trends in Cognitive Sciences*, *1*, 103–108.

Garcia-Dorado, A., Caballero, A., & Crow, J. F. (2003). On the persistence and pervasiveness of a new mutation. *Evolution*, *57*, 2644–2646.

Geher, G., & Miller, G. F. (Eds.). (2007). *Mating intelligence: Sex, relationships, and the mind's reproductive system*. Mahwah: Erlbaum.

Gibson, G., & Dworkin, I. (2004). Uncovering cryptic genetic variation. *Nature Reviews Genetics*, *5*, 681–U611.

Gottfredson, L. S. (2004). Intelligence: Is it the epidemiologists' elusive "fundamental cause" of social class inequalities in health? *Journal of Personality and Social Psychology*, *86*, 174–199.

Gottfredson, L. S. (2007). Innovation, fatal accidents, and the evolution of general intelligence. In M. J. Roberts (Ed.), *Integrating the mind: Domain general versus domain specific processes in higher cognition* (pp. 387–425). Hove: Psychology Press.

Hammerstein, P. (Ed.) (2003). *Genetic and cultural evolution of cooperation*. Cambridge: MIT Press.

Harris, E. E. (2008). Searching the genome for our adaptations. *Evolutionary Anthropology*, *17*, 146–157.

Hawks, J., Wang, E. T., Cochran, G. M., Harpending, H. C., & Moyzis, R. K. (2007). Recent acceleration of human adaptive evolution. *Proceedings of the National Academy of Sciences USA*, *104*, 20753–20758.

Heath, S. C., Gut, I. G., Brennan, P., McKay, J. D., Bencko, V., Fabianova, E., et al. (2008). Investigation of the fine structure of European populations with applications to disease association studies. *European Journal of Human Genetics*, *16*, 1413–1429.

Hedrick, P. W. (1986). Genetic polymorphism in heterogeneous environments: A decade later. *Annual Review of Ecology and Systematics*, *17*, 535–566.

Hedrick, P. W. (1990). Genotypic-specific habitat selection: A new model and its application. *Heredity*, *65*, 145–149.

Hedrick, P. W. (2006). Genetic polymorphism in heterogeneous environments: The age of genomics. *Annual Review of Ecology Evolution and Systematics*, *37*, 67–93.

Hoffmann, A. A., & Willi, Y. (2008). Detecting genetic responses to environmental change. *Nature Reviews Genetics*, *9*, 421–432.

Houle, D., Hughes, K. A., Hoffmaster, D. K., Ihara, J., Assimacopoulos, S., Canada, D., & Charlesworth, B. (1994). The effects of spontaneous mutation on quantitative traits. I. Variances and covariances of life history traits. *Genetics*, *138*, 773–785.

Houle, D. (1998). How should we explain variation in the genetic variance of traits? *Genetica*, *102/103*, 241–253.

Hyde, J. S. (2005). The gender similarities hypothesis. *American Psychologist*, *60*, 581–592.

Jensen, A. R. (1998). *The g factor: The science of mental ability*. Westport, CT: Praeger.

John, O. P., Naumann, L. P., & Soto, C. J. (2008). Paradigm shift to the integrative Big-Five trait taxonomy: History, measurement, and conceptual issues. In O. P. John, R. W. Robins & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (pp. 114–158). New York: Guilford Press.

Johnson, W., & Bouchard, T. J. (2007). Sex differences in mental abilities: g masks the dimensions on which they lie. *Intelligence*, *35*, 23–39.

Johnson, W., Jung, R. E., Colom, R., & Haier, R. J. (2008). Cognitive abilities independent of IQ correlate with regional brain structure. *Intelligence*, *36*, 18–28.

Johnson, W., Penke, L., & Spinath, F. M. (2010). *Heritability in the era of molecular genetics: Some thoughts for understanding genetic influences on behavior*. Manuscript submitted for publication, The University of Edinburgh.

Jokela, M., Elovainio, M., Kivimaki, M., & Keltikangas-Jarvinen, L. (2008). Temperament and Migration Patterns in Finland. *Psychological Science*, *19*, 831–837.

Jonason, P. K. Li, N. P., Webster, G. W., & Schmitt, D. P. (2009). The Dark Triad: Facilitating short-term mating in men. *European Journal of Personality*, *23*, 5–18.

Jung, R. E., & Haier, R. J. (2007). The Parieto-Frontal Integration Theory (P-FIT) of intelligence: converging neuroimaging evidence. *Behavioral and Brain Sciences*, *30*, 135–154.

Kanazawa, S. (2004). General intelligence as a domain-specific adaptation. *Psychological Review*, *111*, 512–523.

Kaplan, H. S., & Gangestad, S. W. (2005). Life history theory and evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 68–95). Hoboken: Wiley.

Kaplan, H. S., Lancaster, J. B., & Robson, A. (2003). Embodied capital and the evolutionary economics of the human lifespan. In J.R. Carey & S. Tuljapakur (Eds.), *Lifespan: Evolutionary, Ecology and demographic Perspectives* (pp. 152–182). New York: Population Council.

Keightley, P. D., & Gaffney, D. J. (2003). Functional constraints and frequency of deleterious mutations in noncoding DNA of rodents. *Proceedings of the National Academy of Sciences USA*, *100*, 13402–13406.

Keller, M. C. (2007). The role of mutations in human mating. In G. Geher & G. Miller (Eds.), *Mating intelligence: Theoretical, experimental, and differential perspectives*. Lawrence Erlbaum: Mahwah, NJ.

Keller, M. C., & Miller, G. F. (2006). Resolving the paradox of common, harmful, heritable mental disorders: Which evolutionary genetic models work best? *Behavioral and Brain Sciences*, *29*, 385–452.

Keller, M. C., & Nesse, R. M. (2006). The evolutionary significance of depressive symptoms: Different adverse situations lead to different depressive symptom patterns. *Journal of Personality and Social Psychology*, *91*, 316–330.

Kidd, K. K. (2009). *ALFRED—the ALlele FREquency Database*. Retrieved February 7, 2009, from http://alfred.med.yale.edu.

Kirkwood, T. B. L., Feder, M., Finch, C. E., Franceschi, C., Globerson, A., Klingenberg, C. P., et al. (2005). What accounts for the wide variation in life span of genetically identical organisms reared in a constant environment? *Mechanisms of Ageing and Development*, *126*, 439–443.

Klein, R. G. (2008). Out of Africa and the evolution of human behaviour. *Evolutionary Anthropology*, *17*, 267–281.

Kokko, H., Brooks, R., Jennions, M. D., & Morley, J. (2003). The evolution of mate choice and mating biases. *Proceedings of the Royal Society of London B*, *270*, 653–664.

Kumar, R. A., KaraMohamed, S., Sudi, J., Conrad, D. F., Brune, C., Badner, J. A., et al. (2008). Recurrent 16p11.2 microdeletions in autism. *Human Molecular Genetics*, *17*, 628–638.

Laland, K. N., & Brown, G. R. (2006). Niche construction, human behavior, and the adaptive-lag hypothesis. *Evolutionary Anthropology*, *15*, 95–104.

Langlois, J. H., Kalakanis, L., Rubenstein, A. J., Larson, A., Hallam, M., & Smoot, M. (2000). Maxims or myths of beauty? A meta-analytic and theoretical review. *Psychological Bulletin*, *126*, 390–423.

Lao, O., Lu, T. T., Nothnagel, M., Junge, O., Freitag-Wolf, S., Caliebe, A., et al. (2008). Correlation between genetic and geographic structure in Europe. *Current Biology*, *18*, 1241–1248.

Leary, M. R., & Baumeister, R. F. (2000). The nature and function of self-esteem: Sociometer theory. In M. P. Zanna (Ed.), *Advances in Experimental Social Psychology* (Vol. 32, pp. 1–61). San Diego: Academic Press.

Lenton, A. P., Penke, L., Todd, P. M., & Fasolo, B. (in press). The heart has its reasons: Social rationality in mate choice. In R. Hertwig, U. Hoffrage, & the ABC Research Group (Eds.), *Social rationality*. New York: Oxford University Press.

MacDonald, K. (1995). Evolution, the Five-Factor Model, and levels of personality. *Journal of Personality*, *63*, 525–567.

MacDonald, K. (2005). Personality, evolution, and development. In R. L. Burgess & K. MacDonald (Eds.), *Evolutionary perspectives on human development* (2nd ed.). (pp. 207–242). Thousand Oaks: Sage.

Maher, B. (2008). Personal genomes: The case of the missing heritability. *Nature*, *456*, 18–21.

Maynard Smith, J. (1982). *Evolution and the theory of games*. Cambridge: University Press.

McCarthy, M. I., Abecasis, G. R., Cardon, L. R., Goldstein, D. B., Little, J., Ioannidis, J. P. A., et al. (2008). Genome-wide association studies for complex traits: consensus, uncertainty and challenges. *Nature Reviews Genetics*, *9*, 356–369.

McCrae, R. R., & Allik, J. (Eds.). (2002). *The five-factor model of personality across cultures*. Berlin: Springer.

McCrae, R. R., Yamagata, S., Jang, K. L., Riemann, R., Ando, J., Ono, Y., et al. (2008). Substance and artifact in the higher-order factors of the Big Five. *Journal of Personality and Social Psychology*, *95*, 442–455.

McEvoy, B. P., Montgomery, G. W., McRae, A. F., Ripatti, S., Perola, M., Spector, T. D., et al. (2009). Geographical structure and differential natural selection amongst North European populations. *Genome Research*, *19*, 804–814.

Mealey, L. (1995). The sociobiology of sociopathy: An integrated evolutionary model. *Behavioral and Brain Sciences*, *18*, 523–599.

Mealey, L. (2000). *Sex differences: Development and evolutionary strategies*. Academic Press.

Mekel-Bobrov, N., Posthuma, D., Gilbert, S. L., Lind, P., Gosso, M. F., Luciano, M., et al. (2007). The ongoing adaptive evolution of ASPM and Microcephalin is not explained by increased intelligence. *Human Molecular Genetics*, *16*, 600–608.

Mendle, J., Turkheimer, E., D'Onofrio, B. M., Lynch, S. K., Emery, R. E., Slutske, W. S., et al. (2006). Family structure and age at menarche: A children-of-twins approach. *Developmental Psychology*, *42*, 533–542.

Miller, G. F. (2000). Sexual selection for indicators of intelligence. In G. Bock, J. Goode & K.Webb (Eds.), The nature of intelligence (pp. 260–275). New York: John Wiley.

Miller, G. F. (2007). Reconciling evolutionary psychology and ecological psychology: How to perceive fitness affordances. *Acta Psycholigica Sinica*, 39, 546–555.

Mitchell-Olds, T., Willis, J. H., & Goldstein, D. B. (2007). Which evolutionary processes influence natural genetic variation for phenotypic traits? *Nature Reviews Genetics*, *8*, 845–856.

Miyake, A., & Shah P. (Eds.) (1999). *Models of working memory: Mechanisms of active maintenance and executive control*. New York: Cambridge University Press.

Morrow, E. M., Yoo, S. Y., Flavell, S. W., Kim, T. K., Lin, Y. X., Hill, R. S., et al. (2008). Identifying autism loci and genes by tracing recent shared ancestry. *Science*, *321*, 218–223.

Neberich, W., Penke, L., Lehnart, J., & Asendorpf, J. B. (2010). Family of origin, age at menarche, and reproductive strategies: A test of four evolutionary-developmental models. *European Journal of Developmental Psychology*, 7, 153–177.

Nielsen, R., Hellmann, I., Hubisz, M., Bustamante, C., & Clark, A. G. (2007). Recent and ongoing selection in the human genome. *Nature Reviews Genetics*, *8*, 857–868.

Novembre, J., Johnson, T., Bryc, K., Kutalik, Z., Boyko, A. R., Auton, A., et al. (2008). Genes mirror geography within Europe. *Nature*, *456*, 98–101.

Nozawa, M., Kawahara, Y., & Nei, M. (2007). Genomic drift and copy number variation of sensory receptor genes in humans. *Proceedings of the National Academy of Sciences USA*, *104*, 20421–20426.

Ozer, D. J., & Benet-Martinez, V. (2006). Personality and the prediction of consequential outcomes. *Annual Review of Psychology*, *57*, 401–421.

Penke, L. (2009). Adaptive developmental plasticity might not contribute much to the adaptiveness of reproductive strategies. A commentary on M. Del Giudice. *Behavioral and Brain Sciences*, *32*, 38–39.

Penke, L. (2009). An ethological perspective on how to define and study behaviour. A commentary on Furr. *European Journal of Personality*, *23*, 421–423.

Penke, L., & Asendorpf, J. B. (2008). Beyond global sociosexual orientations: A more differentiated look at sociosexuality and its effects on courtship and romantic relationships. *Journal of Personality and Social Psychology*, *95*, 1113–1135.

Penke, L., Denissen, J. J. A., & Miller, G. F. (2007a). The evolutionary genetics of personality. *European Journal of Personality*, *21*, 549–587.

Penke, L., Denissen, J. J. A., & Miller, G. F. (2007b). Evolution, genes, and inter-disciplinary personality research. *European Journal of Personality*, *21*, 639–665.

Penke, L., Muñoz Maniega, S., Houlihan, L. M., Murray, C., Gow, A. J., Clayden, J. D., Bastin, M. E., Wardlaw, J. M., & Deary, I. J. (2010). White matter integrity in the splenium of the corpus callosum is related to successful cognitive aging and partly mediates the protective effect of an ancestral polymorphism in ADRB2. *Behavior Genetics*, *40*, 146–156.

Penke, L., Todd, P. M., Lenton, A., & Fasolo, B. (2007). How self-assessments can guide human mating decisions. In G. Geher & G. F. Miller (Eds.), *Mating intelligence: Sex, relationships, and the mind's reproductive system* (pp. 37–75). Mahwah: Lawrence Erlbaum.

Penton-Voak, I. S., Jacobson, A., & Trivers, R. (2004). Populational differences in attractiveness judgments of male and female faces: Comparing British and Jamaican samples. *Evolution and Human Behavior*, *25*, 355–370.

Pigliucci, M. (2005). Evolution of phenotypic plasticity: Where are we going now? *Trends in Ecology and Evolution*, *20*, 481–486.

Pinker, S. (1994). *The language instinct: How the mind creates language*. New York: Harper Collins.

Pinker, S. (2009). My genome, my self. New York Times, January 11, 2009, p.MM24.http://www.nytimes.com/2009/01/11/magazine/11Genome-t.html?_r=2&hp=&pagewanted=all.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral genetics* (5th ed.). New York: Freeman.

Polak, M. (2003). *Developmental instability: Causes and consequences*. Oxford: University Press.

Pound, N., Penton-Voak, I. S., & Brown, W. M. (2007). Facial symmetry is positively associated with self-reported extraversion. *Personality and Individual Differences*, *43*, 1572–1582.

Ridley, M. (2003). *Evolution* (3rd ed.). Wiley-Blackwell.

Roberts, B. W., Kuncel, N. R., Shiner, R., Caspi, A., & Goldberg, L. R. (2007). The power of personality: The comparative validity of personality traits, socio-economic status, and cognitive ability for predicting important life outcomes. *Perspectives on Psychological Science*, *2*, 313–345.

Roff, D. A. (1992). *Evolution of life histories: Theory and analysis*. New York: Chapman & Hall.

Roff, D. A. (1997). *Evolutionary quantitative genetics*. New York: Chapman & Hall.

Roff, D. A., & Fairbairn, D. J. (2007). The evolution of trade-offs: where are we? *Journal of Evolutionary Biology*, *20*, 433–447.

Rowe, L., & Houle, D. (1996). The lek paradox and the capture of genetic variance by condition dependent traits. *Proceedings of the Royal Society of London B*, *263*, 1415–1421.

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.

Rushton, J. P., Bons, T. A., & Hur, Y. M. (2008). The genetics and evolution of the general factor of personality. *Journal of Research in Personality*, *42*, 1173–1185.

Rushton, J. P., & Irwing, P. (2008). A General Factor of Personality (GFP) from two meta-analyses of the Big Five: Digman (1997) and Mount, Barrick, Scullen, and Rounds (2005). *Personality and Individual Differences*, *45*, 679–683.

Saulsman, L. M., & Page, A. C. (2004). The five-factor model and personality disorder empirical literature: A meta-analytic review. *Clinical Psychology Review*, *23*, 1055–1085.

Schaller, M., & Murray, D. R. (2008). Pathogens, personality, and culture: Disease prevalence predicts worldwide variability in sociosexuality, extraversion, and openness to experience. *Journal of Personality and Social Psychology*, *95*, 212–221.

Schmitt, D. P. (2003). Universal sex differences in the desire for sexual variety: Tests from 52 nations, 6 continents, and 13 islands. *Journal of Personality and Social Psychology*, *85*, 85–104.

Schmitt, D. P. (2004). The Big Five related to risky sexual behaviour across 10 world regions: Differential personality associations of sexual promiscuity and relationship infidelity. *European Journal of Personality*, *18*, 301–319.

Schmitt, D. P. (2005). Sociosexuality from Argentina to Zimbabwe: A 48-nation study of sex, culture, and strategies of human mating. *Behavioral and Brain Sciences*, *28*, 247–275.

Schneider, K. A. (2006). A multilocus-multiallele analysis of frequency-dependent selection induced by intraspecific competition. *Journal of Mathematical Biology*, *52*, 483–523.

Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., et al. (2007). Strong association of de novo copy number mutations with autism. *Science*, *316*, 445–449.

Segal, N. L. (2005). *Indivisible by two: Lives of extraordinary twins*. Cambridge: Harvard University Press.

Sharp, N. P., & Agrawal, A. F. (2008). Mating density and the strength of sexual selection against deleterious alleles in Drosophila melanogaster. *Evolution*, *62*, 857–867.

Simpson, J. A., & Gangestad, S. W. (1991). Individual differences in socio-sexuality: Evidence for convergent and discriminant validity. *Journal of Personality and Social Psychology*, *60*, 870–883.

Stearns, S. (1992). *Evolution of life histories*. Oxford: University Press.

Stefansson, H., Rujescu, D., Cichon, S., Pietilainen, O. P. H., Ingason, A., Steinberg, S., et al. (2008). Large recurrent microdeletions associated with schizophrenia. *Nature*, *455*, 232–U261.

Stone, J. L., O'Donovan, M. C., Gurling, H., Kirov, G. K., Blackwood, D. H. R., Corvin, A., et al. (2008). Rare chromosomal deletions and duplications increase risk of schizophrenia. *Nature*, *455*, 237–241.

Sunyaev, S., Ramensky, R., Koch, I., Lathe, W., Kondrashov, A. S., & Bork, P. (2001). Prediction of deleterious human alleles. *Human Molecular Genetics*, *10*, 591–597.

Thornhill, R., & Møller, A. P. (1997). Developmental stability, disease and medicine. *Biological Reviews of the Cambridge Philosophical Society*, *72*, 497–548.

Tomkins, J. L., Radwan, J., Kotiaho, J. S., & Tregenza, T. (2004). Genic capture and resolving the lek paradox. *Trends in Ecology and Evolution*, *19*, 323–328.

Tooby, J., & Cosmides, L. (1990a). On the universality of human nature and the uniqueness of the individual: The role of genetics and adaptation. *Journal of Personality*, *58*, 17–67.

Tooby, J., & Cosmides, L. (1990b). The past explains the present. Emotional adaptations and the structure of ancestral environments. *Ethology and Sociobiology*, *11*, 375–424.

Tooby, J., & Cosmides, L. (2005). Conceptual foundations of evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 5–67). Hoboken: Wiley.

Tooby, J., Cosmides, L., & Barrett, H. C. (2003). The second law of thermodynamics is the first law of psychology: Evolutionary developmental psychology and the theory of tandem, coordinated inheritances: Comment on Lickliter and Honeycutt. *Psychological Bulletin, 129*, 858–865.

Tooby, J., Cosmides, L., & Barrett, H. C. (2005). Resolving the debate on innate ideas: Learnability constraints and the evolved interpenetration of motivational and conceptual functions. In P. Carruthers, S. Laurence & S. Stich (Eds.), *The innate mind: Structure and content*. New York: Oxford University Press.

Trivers, R. L. (1972). Parental investment and sexual selection. In B. Campbell (Ed.), *Sexual selection and the descent of man* (pp. 136–179). Chicago, IL: Aldine-Atherton.

Turelli, M., & Barton, N. H. (2004). Polygenic variation maintained by balancing selection: Pleiotropy, sex-dependent allelic effects and GXE interactions. *Genetics, 166*, 1053–1079.

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science, 9*, 160–164.

Visscher, P. M., Hill, W. G., & Wray, N. R. (2008). Heritability in the genomics era: Concepts and misconceptions. *Nature Reviews Genetics, 9*, 255–266.

Walsh, T., McClellan, J. M., McCarthy, S. E., Addington, A. M., Pierce, S. B., Cooper, G. M., et al. (2008). Rare structural variants disrupt multiple genes in neurodevelopmental pathways in schizophrenia. *Science, 320*, 539–543.

Warner, D. A., & Shine, R. (2008). The adaptive significance of temperature-dependent sex determination in a reptile. *Nature*, 451, 566–568.

Weiss, L. A., Shen, Y. P., Korn, J. M., Arking, D. E., Miller, D. T., Fossdal, R., et al. (2008). Association between microdeletion and microduplication at 16p11.2 and autism. *New England Journal of Medicine, 358*, 667–675.

West-Eberhard, M. J. (2003). *Developmental plasticity and evolution*. Oxford: University Press.

Williamson, S. H., Hubisz, M. J., Clark, A. G., Payseur, B. A., Bustamante, C. D., et al. (2007). Localizing recent adaptive evolution in the human genome. *PLoS Genetics, 3*, e90.

Wilson, D. S. (1994). Adaptive genetic variation and human evolutionary psychology. *Ethology and Sociobiology, 15*, 219–235.

Wilson, E. O. (1975). *Sociobiology: A new synthesis*. Harvard: Harvard University Press.

Woods, R. P., Freimer, N. B., De Young, J. A., Fears, S. C., Sicotte, N. L., Service, S. K., et al. (2006). Normal variants of Microcephalin and ASPM do not

account for brain size variability. *Human Molecular Genetics*, *15*, 2025–2029.

Xu, B., Roos, J. L., Levy, S., Van Rensburg, E. J., Gogos, J. A., & Karayiorgou, M. (2008). Strong association of de novo copy number mutations with sporadic schizophrenia. *Nature Genetics*, *40*, 880–885.

Yeo, R. A., Gangestad, S. W., & Thoma, R. J. (2007). Developmental instability and individual variation in brain development: Implications for the origin of neurodevelopmental disorders. *Current Directions in Psychological Science*, *16*, 245–249.