The Evolutionary Genetics of Personality[†]

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Abstract

Genetic influences on personality differences are ubiquitous, but their nature is not well understood. A theoretical framework might help, and can be provided by evolutionary genetics. We assess three evolutionary genetic mechanisms that could explain genetic variance in personality differences: selective neutrality, mutation-selection balance, and balancing selection. Based on evolutionary genetic theory and empirical results from behaviour genetics and personality psychology, we conclude that selective neutrality is largely irrelevant, that mutation-selection balance seems best at explaining genetic variance in intelligence, and that balancing selection by environmental heterogeneity seems best at explaining genetic variance in personality traits. We propose a general model of heritable personality differences that conceptualises intelligence as fitness components and personality traits as individual reaction norms of genotypes across environments, with different fitness consequences in different environmental niches. We also discuss the place of mental health in the model. This evolutionary genetic framework highlights the role of gene-environment interactions in the study of personality, yields new insight into the person-situation-debate and the structure of personality, and has practical implications for both quantitative and molecular genetic studies of personality. Copyright © 2007 John Wiley & Sons, Ltd.

Evolutionary thinking has a long history in psychology (James, 1890; McDougall, 1908; Thorndike, 1909). However, the new wave of evolutionary psychology (Buss, 1995; Tooby & Cosmides, 2005) has focused almost exclusively on human universals—the complex

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psychological adaptations that became genetically fixed throughout our species due to natural selection (Andrews, Gangestad & Matthews, 2002) and that should therefore show zero genetic variation and zero heritability (Tooby & Cosmides, 1990). In sharp contrast, one of personality psychology's most important findings in the last three decades has been that virtually every aspect of personality is heritable (Plomin, DeFries, McClearn & Mc Guffin, 2001). This fact is now so well established that Turkheimer (2000; Turkheimer & Gottesman, 1991) even called it a law. The mismatch between evolutionary psychology's adaptationist focus on human universals and the omnipresence of heritable variance in human personality might explain why early approaches towards an evolutionary personality psychology (Buss, 1991; MacDonald, 1995, 1998; Tooby & Cosmides, 1990) remained rather unsatisfactory (Miller, 2000a; Nettle, 2006a). On the other hand, traditional behaviour genetics did not explain the evolutionary origins and persistence of genetic variation in personality, and sometimes even viewed genetic variation in traits as evidence of their evolutionary irrelevance. Thus, the evolutionary psychology of human universals and the behaviour genetics of personality differences share a biological metatheory, but had almost no influence on each other (Plomin et al., 2001; Tooby & Cosmides, 1990, 2005).

We believe that this mutual neglect has been unfortunate for both fields, and has especially harmed the development of an integrative evolutionary personality psychology. Evolutionary studies of species-typical universals and individual differences were already successfully merged during the 'Modern Synthesis' in the 1930s, when Sir Ronald A. Fisher, Sewell Wright, J. B. S. Haldane, and others united the branches of biology that were founded by the cousins Charles Darwin (the father of adaptationism) and Sir Francis Galton (the father of psychometrics and behaviour genetics) (Mayr, 1993). These 1930s biologists created what is now known as 'evolutionary genetics', which deals with the origins, maintenance, and implications of natural genetic variation in traits across individuals and species. Evolutionary genetics mathematically models the effects of mutation, selection, migration, and drift on the genetic basis of traits in populations (Maynard Smith, 1998; Roff, 1997). In the following, we will argue that personality psychology needs an evolutionary genetic perspective in order to draw maximal benefits from behaviour genetic findings and the evolutionary metatheory. This is important, since understanding the evolutionary behaviour genetics of personality is fundamental to the future development of a more unified personality psychology (McAdams & Pals, 2006).

OVERVIEW

The central topic of this review is how evolutionary genetics can inform our theoretical understanding of heritable personality differences and their genetic foundations. We use 'personality differences' in the broad European sense of encompassing individual differences in both cognitive abilities and personality traits (e.g. Eysenck & Eysenck, 1985). Cognitive abilities reflect an individual's maximal performance in solving cognitive tasks. It is well-established that a single continuum of general intelligence (g), ranging from mild mental retardation to giftedness, explains a large proportion of the individual differences in cognitive abilities across domains (Jensen, 1998), especially on genetic level (Plomin & Spinath, 2004). Our discussion on cognitive abilities will be focused on this general intelligence dimension. Personality traits reflect an individual's set of typical behavioural tendencies exhibited in situations that leave room for diverse adaptive responses. The myriad of personality trait dimensions are usually organised in structural

models. Broad personality trait domains, as in the five factor model of personality (FFM), are generally regarded as stable and temperamental in nature (John & Srivastava, 1999). They are what we mean by 'personality traits'.

We argue that the classical distinction between cognitive abilities and personality traits is much more than just a historical convention or a methodological matter of different measurement approaches (Cronbach, 1949), and instead reflects different kinds of selection pressures that have shaped distinctive genetic architectures for these two classes of personality differences. In order to make this argument, we will first give a brief introduction to the nature of genetic variation and the major mechanisms that contemporary evolutionary genetics proposes for its maintenance in populations. After this, we will critically review earlier evolutionary approaches to personality and clarify the role of environmental influences within this approach. This will culminate in an integrative model of the evolutionary genetics of personality differences, including new, theory-based definitions of cognitive abilities and personality traits, as well as a discussion of how common psychopathologies (such as schizophrenia and psychopathy) may fit into an evolutionary genetic model of personality differences. Finally, we will discuss this model's implications for an integrated evolutionary personality psychology grounded in both behaviour genetics and evolutionary genetics.

WHAT IS GENETIC VARIATION?

Most personality psychologists now accept Turkheimer's (2000) first law of behaviour genetics ('everything is heritable'). Yet how does systematic genetic variation in personality traits arise? A complete understanding of the insights offered by evolutionary genetics requires a brief review of some of the basics of genetics and evolutionary theory, which we provide in the following.

The human genome

The human genome consists of about 3.2 billion base pairs that are unequally spread across 24 distinct chromosomes. Only about 75 million (2.3%) of these base pairs are organised in roughly 25 000 genes (i.e. regions or 'loci' translated into actual protein structures); the rest (traditionally called 'junk DNA') do not code for proteins, but may play important roles in gene regulation and expression (Shapiro & von Sternberg, 2005). On average, any two same-sex individuals randomly drawn from the total human population are 99.9% identical with regard to their base pairs (Human Genome Project, 2001), even though genomic identity is somewhat further attenuated by copy-number variations (CNVs, individual differences in the repetitions of DNA segments) (Redon et al., 2006). This species-typical genome contains the universal human heritage that ensures the highly reliable ontogenetic reoccurrence of the complex functional human design across generations ('design reincarnation', Barrett, 2006; Tooby, Cosmides, & Barrett, 2005). Adaptationistic evolutionary approaches usually care only about this universal part of the genome and its species-typical phenotypic products (Andrews et al., 2002; Tooby & Cosmides, 2005).

Mutation

During an individual lifespan, the genome is passed from mother cells to daughter cells by self-replication, and if this results in a germline (sperm or egg) cell, half of the genome

eventually ends up combining with an opposite-sex germline cell during sexual reproduction, and is thus passed from parent to offspring. While genomic self-replication is astonishingly precise, it is not perfect. Replication errors can occur in the form of point mutations (substituting one of the four possible nucleotides in a base pair for another one, also referred to as single nucleotide polymorphisms (SNPs)), CNVs (duplications or deletions of base pair sequences), or rearrangements of larger chromosomal regions (e.g. translocations, inversions). All of these copying errors are referred to as *mutations*, and they are ultimately the only possible source of genetic variation between individuals. Recent scans of whole human genotypes reported 9.2 million candidate SNPs (International HapMap Consortium, 2005) and 1447 candidate CNV regions (Redon et al., 2006).

Sexual reproduction endows an individual with a unique mixture of their parents' genotypes. In the short term, this process of sexual recombination is the major cause of genetic individuality. In the evolutionary long-term, however, sexual recombination is less important, since it just reshuffles the parental genetic variation that was once caused by mutation. By convention, mutations that continue to be passed on to subsequent generations and that reach an arbitrary threshold of more than 1% prevalence in a population are called '*alleles*'. Since all alleles are mutations, we regard this distinction as hardly helpful. In contrast, '*polymorphism*' is a more neutral term for genetic variants that can be at any prevalence. In order to highlight the evolutionary genetic perspective, we will use the terms 'mutation' and 'polymorphism' interchangeably.

Some mutations are phenotypically neutral, often because they do not affect protein structure or gene regulation. Most mutations in protein-coding and genomic regulatory regions, however, tend to be harmful to the organism because they randomly disrupt the evolved genetic information, thereby eroding the complex phenotypic functional design (Ridley, 2000; Tooby & Cosmides, 1990). Only very rarely does a random mutation improve the functional efficiency of an existing adaptation in relation to its environment, which is more likely if the environment has changed since the adaptation evolved (Brcic-Kostic, 2005). Deletions, insertions, and larger rearrangements of base pair sequences tend to have quite strong disruptive effects on the phenotype, often leading to prenatal death or severe birth defects. Point mutations (SNPs) and duplication-type CNVs (Hurles, 2004), on the other hand, can have phenotypic effects of any strength, including quite mild effects, and it is likely that they are the most common source of genetic variation between individuals.

Behaviour genetics

Quantitative traits, such as intelligence and personality traits, are polygenic—they are affected by many mutations at many genetic loci, each of which is called a quantitative trait locus (QTL) (Plomin, Owen & McGuffin, 1994). Quantitative behaviour genetics basically compares trait similarities across individuals that systemically differ in the genetic or environmental influences they have in common (e.g. identical vs. fraternal twins, adoptive vs. biological children), to decompose the variation of quantitative traits, and their covariances with other traits, into genetic and environmental (co)variance components. It also tries to estimate how much of the genetic (co)variance is due to 'additive effects' of QTLs (which allow traits to 'breed true' from parents to offspring) versus interactions between alleles at the same genetic locus (dominance effects) or across different genetic loci (epistatic effects). Dominance and epistatic effects lead to non-additive genetic variance (V_{NA}) between individuals, as opposed to the additive genetic variance (V_A)

caused by additive effects. Together with the environmental variance (V_E) and gene-environment (GxE) interactions, these components determine the phenotypic variance (V_P) that we can observe in personality differences. In contrast to quantitative behaviour genetics, molecular behaviour genetics uses so-called 'linkage' and 'association' methods to directly analyse human DNA variation in relation to personality variation, to identify the specific QTLs that influence particular trait (co)variations (Plomin et al., 2001).

Natural selection

Mutations in functional regions of the genome provide half of the basic ingredients for biological evolution. The other half is natural selection, which is the differential reproduction of the resulting phenotypes (Darwin, 1859). Any mutation that affects the phenotype is potentially visible to natural selection, though to varying degrees. Of course, those rare mutations that actually increase fitness will tend to spread through the population, driving adaptive evolution. Selection is most obvious against mutations that lead to premature death or sterility. Such mutations are eliminated from the population within one generation, and can only be reintroduced by new mutations at the same genetic loci. Mutations with less severe effects tend to persist in the population for some time; they are selected out of the population more quickly when their additive effect reduces the *fitness* of the genotype (i.e. its statistical propensity for successful reproduction) more severely. This relationship between the additive phenotypic effect of a genetic variant and its likely persistence in a population is described by the *fundamental theorem of natural selection* (Fisher, 1930).

To summarise, any genetic variation in any human trait is ultimately the result of mutational change in functional regions of the species-typical genome. Natural selection counteracts disruptive changes by eliminating harmful mutations from the population, at a rate proportional to the mutation's additive genetic reduction in fitness. Only mutations that affect the organism's fitness in a positive or neutral way can spread in the population and will reach the 1% prevalence of an 'allele'. Most psychological traits, including personality differences, are complex in design and continuously variable across individuals, indicating that many polymorphisms at many loci are responsible for their genetic variation.

WHY IS THERE GENETIC VARIATION IN PERSONALITY?

Also else being equal, it seems plausible that natural selection should favour an invariant, species-typical genome that codes for a single optimal phenotype with optimal fitness. In other words, evolution should eliminate genetic variation in all traits, including all aspects of personality. So how can personality differences still be heritable (i.e. genetically variable) after all these generations of evolution? To answer this fundamental question, an evolutionary genetic approach to personality is needed.

With the growing acceptance of evolution as a metatheory for psychology, more and more personality psychologists are trying to conceptualise personality in an evolutionary framework. Unfortunately, these good intentions seldom lead to more than an affirmation that certain heritable dimensions are part of our evolved human nature (Ashton & Lee, 2001; McCrae & Costa, 1996; McAdams & Pals, 2006). Even worse, some conceptualisations of human cognitive abilities ignore genetic variation completely and

discuss these heritable, variable traits as if they were invariant adaptations (Cosmides & Tooby, 2002; Kanazawa, 2004). Other authors (Buss, 1990; Ellis, Simpson & Campbell, 2002; Goldberg, 1981; Hogan, 1996) take genetic variation in personality differences for granted, and try to understand evolved features of our 'person perception system' that explain why we categorise others along these dimensions. Few have attempted an evolutionary genetic approach to explain the persistence of heritable variation in personality itself.

Evolutionary genetics offers a variety of mechanisms that could explain persistent genetic variation in personality differences. These mechanisms include selective neutrality (where mutations are invisible to selection), *mutation-selection balance* (where selection counteracts mutations, but is unable to eliminate all of them), and *balancing selection* (where selection itself maintains genetic variation). Recent theoretical developments make it possible to predict how each of these mechanisms would influence certain genetic and phenotypic features of traits (Table 1). Conversely, if these features are known for a given trait, it is possible to identify which evolutionary processes likely maintained the genetic variants that underlie its heritability. We will now review existing attempts to explain personality differences from an evolutionary perspective, and evaluate them in the light of modern evolutionary genetics.

CAN SELECTIVE NEUTRALITY EXPLAIN GENETIC VARIANCE IN PERSONALITY?

Tooby and Cosmides (1990) developed an early and highly influential perspective on the evolutionary genetics of personality. They reviewed the state of evolutionary genetics at that time, but, as major advocates of an adaptationistic evolutionary psychology, they focused on species-typical psychological adaptations and downplayed genetic variation as minor evolutionary noise. In their view, one plausible mechanism that could maintain genetic variation in psychological differences is selective neutrality (Kimura, 1983). This occurs when fitness-neutral mutations (that have no net effect on survival or reproductive success, averaged across all relevant environments) accumulate to increase genetic variance in a trait. For example, the exact route that the small intestine takes within one's abdomen may have little influence on digestive efficiency, so neutral genetic variation that influences patterns of gut-packing could easily accumulate. In the evolutionary short-term, selective neutrality allows genetic variance in traits to increase.

However, what happens in the evolutionary long-term to selectively neutral traits? Since neutral mutations are, by definition, unaffected by natural selection, the only evolutionary force that can affect neutral genetic variation is genetic drift—and drift always tends to decrease genetic variance. Drift is basically the fixation (to 100% prevalence) or elimination (to 0% prevalence) of a polymorphism by chance. There is only one factor that is known to be important for the efficacy of drift: it is stronger when the 'effective population size' (N_e) (the average number of reproductively active individuals in a population) is smaller (Lynch & Hill, 1986). What is really critical for the effect of genetic drift is the minimum N_e during occasional harsh conditions (e.g. ice ages, disease pandemics) that created 'genetic bottlenecks' (especially small effective population sizes). In humans, 10 000 seems to be a good estimate for the minimum N_e (Cargill et al., 1999). Mathematical models show that, with such a relatively large N_e, drift is fairly weak and

Table 1. A comparison of evolutionary genetic mechanisms for the maintenance of genetic variation and empirical predictions for affected traits

	Selective neutrality	Mutation-selection balance	Balancing selection
Genetic variation is due to...	...mutations that are not affected by selection because their phenotypic effect is unrelated to fitness in any environment	...an accumulation of many old and new, mildly harmful mutations that selection has not yet wiped out of the population	...polymorphisms that are maintained by selection because the fitness pay-off of their phenotypic effects varies across environments
Predictions for an affected trait			
Number of genetic loci (mutational target size)	No prediction	Very large	Medium
Number of polymorphic loci (QTLs)	Likely small	Large	Small
Average gene effect on trait	No prediction	Small	Medium
Prevalence of polymorphisms	Intermediate	Rare	Mostly intermediate
Relation to fitness	Neutral	Unidirectional	Contingent on environment
Average fitness across environments	Equal	Unequal	Approximately equal
Additive genetic variance (V_A)	No prediction	Large	Medium
Ratio non-additive to total genetic variance (D_α)	Small	Medium	High
Environmental variance (V_E)	No prediction	Large	Medium
Expression dependent on overall condition	No	Yes	No
Inbreeding depression/heterosis effects	Weak or none	Strong	Weak
Average social evaluation/sexual attractiveness	Neutral	Strong unidirectional favouritism	Weaker, conditional favouritism

selective neutrality could, in principle, account for almost all genetic variance in any human trait (Lynch & Hill, 1986).

So far, so good: perhaps most genetic variation in human personality is due to selective neutrality—maybe there is no average net fitness cost or benefit to being extraverted versus introverted, or agreeable versus egoistic. However, the critical assumption for selective neutrality is that genetic drift is more important than natural selection in affecting a trait's genetic variance. This is only the case if the selection coefficient *s* is less than about $1/4N_e$ (Keller & Miller, 2006a). Thus, the larger the effective population size, the harder it is for a trait to be selectively neutral. Given the reasonably large estimate of minimum human N_e from above (10 000), a typical human trait is selectively neutral only if the average net fitness of individuals with a certain polymorphism is between 99.997 and 100.003% of the average fitness of individuals without that polymorphism (Keller & Miller, 2006a). For example, an allele that influences extraversion would be truly neutral only if extraverts had, not just the same number of 1st-generation offspring as introverts, but (almost) exactly the same average number of 15th generation descendants (great[13] grandchildren). In addition, this finely-balanced neutrality must hold across all relevant environments: if there are some environments in which outgoing, risk-seeking extraverts do better, and other environments in which shy, risk-averse introverts do better (a GxE interaction), then extraversion would be under balancing selection (see below), not selective neutrality.

This makes selective neutrality an implausible explanation for heritable personality differences, because human personality traits influence outcomes in all areas of life (Ozer & Benet-Martinez, 2006), including such obviously fitness-relevant aspects as health (Neeleman, Sytema & Wadsworth, 2002), life expectancy (Friedman et al., 1995), mating strategies (Nettle, 2005), and reproductive success (Eaves, Martin, Heath, Hewitt, & Neale, 1990). Indeed, similar non-neutral relationships between personality and fitness have been observed in various other species (Dingemanse & Réale, 2005). The relation between cognitive abilities and fitness components has also been impressively demonstrated by Gottfredson (2004, in press), Deary (Deary & Der, 2005; Deary, Whiteman, Starr, Whalley, & Fox, 2004), and Miller (2000b; Prokosch, Yeo & Miller, 2005).

How could we tell if a heritable individual difference was the outcome of selective neutrality? Typically, selective neutrality leads to a distinct structure of genetic variation in quantitative traits (such as personality differences). If a mutation affects the phenotypic expression of a trait, it will first of all have a main effect, which means it will contribute to the additive genetic variance (V_A) of the trait. Only if the mutation happens to interact with other polymorphisms (at the same or other loci, through dominance or epistasis, respectively), will it contributes to the non-additive genetic variance (V_{NA}) of the trait. This is exactly the same logic that holds for any statistical analysis: ceteris paribus, main effects are much more likely than interaction effects. Since all else is equal under selective neutrality by definition, we can expect low absolute values of V_{NA} for any selectively neutral trait (Lynch & Hill, 1986; Merilä & Sheldon, 1999), and a very small proportion of non-additive genetic variance (D_α), defined by Crnokrak and Roff (1995) as:

$$D_\alpha = V_{NA}/(V_{NA} + V_A) \qquad (1)$$

Traits with a recent history of selection, by contrast, should show a significant absolute and proportional amount of V_{NA} (Crnokrak & Roff, 1995; Merilä & Sheldon, 1999; Stirling, Réale & Roff, 2002). This follows from Fisher's (1930) fundamental theorem of

natural selection: since V_A is passed directly from parents to offspring, it will be reduced very quickly by natural selection for any non-neutral trait. V_{NA}, on the other hand, is affected much more weakly by selection, since the interacting genetic components that constitute the V_{NA} are continuously broken apart by sexual recombination and thus not passed from parents to offspring. As a result, a high proportion of V_{NA} in a trait would argue against the trait's selective neutrality. There is now strong evidence that personality traits show substantial V_{NA} (Eaves, Heath, Neale, Hewitt, & Martin, 1998; Keller, Coventry, Heath, & Martin, 1998)—including some initial molecular evidence for epistatic interactions (Strobel, Lesch, Jatzke, Paetzold, & Brocke, 2003)—which suggests they are not selectively neutral. In contrast, cognitive abilities seem to show less V_{NA} (Chipuer, Rovine & Plomin, 1990), a point we consider later.

As summarised in Table 1, genetic variation persists in populations through selective neutrality only if its phenotypic consequences are (almost) completely unrelated to fitness in any environment. This genetic variation can be expected to be mainly additive. While it is possible that this holds for some relatively trivial traits (e.g. gut-packing design), it is highly implausible for major personality differences, given their pervasive effects on social, sexual, and familial life.

CAN MUTATION-SELECTION BALANCE EXPLAIN GENETIC VARIANCE IN PERSONALITY?

Mutation rates and mutation load

As stated previously, a truly neutral trait has to show a close-to-null relationship to any fitness component in any environment. All traits that do not fulfil this very strict requirement are subject to natural selection. As long as the direction of selection is relatively constant, Fisher's (1930) fundamental theorem predicts that the additive genetic variance of the trait will be reduced to the point where one genetic variant becomes fixed as a universal, species-typical adaptation. The rate of reduction in a trait's genetic variance is influenced by two factors with opposing effects: the mutation rate (which increases genetic variance) and the strength of selection (which decreases genetic variance). The mutation rate tells us how fast new mutations are introduced into functional parts of the genome (i.e. protein-coding genes and their regulatory regions). Comparative molecular genetic studies suggest that humans have a comparatively high mutation rate (Eyre-Walker & Keightley, 1999), with the best available estimate being an average of about 1.67 new mutations per individual per generation (Keightley & Gaffney, 2003). Given reasonable assumptions about mutations arising in a Poisson frequency distribution, one can calculate that the probability of a human being born without any new mutations is slightly lower than one in five (Keller, in press). Importantly, this estimate includes only non-neutral mutations (polymorphisms that are visible to selection). As argued above, almost all non-neutral mutations tend to be harmful, and selection is stronger against more harmful mutations. For example, a mutation that reduces number of surviving offspring by 1% will persist for an average of 10 generations in a large population, passing through the genotypes of about 100 individuals during that time. A mutation with a weaker 0.1% fitness reduction (which is still 10 times stronger than selective neutrality in humans) will persist for four generations longer, afflicting about 1000 individuals (Garcia-Dorado, Caballero & Crow, 2003). Because harmful

mutations with dominant effects are an easier target for selection, only recessive mutations are likely to persist for a longer time (Zhang & Hill, 2005).

It follows that there is a *mutation load* of older, mildly harmful, and mostly recessive mutations in any individual at any point in time. This mutation load is mostly inherited from parents to offspring, 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. According to very conservative estimates, the average number of mildly harmful mutations carried by humans is about 500 (Fay, Wyckoff & Wu, 2001; Sunyaev et al., 2001) and the standard deviation is 22 (or higher, given assortative mating, as we discuss below) (Keller & Miller, 2006a). This mutation load may account for a substantial portion of genetic variance in many fitness-related traits—perhaps including personality differences.

Mutational target size

For a long time, Fisher's fundamental theorem was thought to imply that traits that affect fitness more strongly should show less V_A (Falconer, 1981). In the early 1990s, however, Price and Schluter (1991) and Houle (1992) showed that the reverse is true: more fitness-related traits actually tend to have *higher* V_A. The reason that this could remain unnoticed for more than half a century was that evolutionary geneticists used to standardise additive genetic variance (V_A) by the total phenotypic variance (V_P) of the trait, yielding its narrow-sense heritability (h^2):

$$h^2 = V_A/V_P \tag{2}$$

Insofar as heritability was taken as a rough proxy for additive genetic variance, this gives profoundly misleading results, because V_P contains both the non-additive genetic (V_{NA}) and the environmental variance (V_E). Even if V_A is large, h^2 can be small when V_{NA} and/or V_E are even larger. Since V_E is especially population- and trait-specific, h^2 is not very informative for comparing genetic variances. Houle (1992) instead proposed to use the 'coefficient of additive genetic variation' (CV_A) for comparisons across traits, populations, and species. It is defined as:

$$CV_A = [\text{sqrt}(V_A)/M] * 100 \tag{3}$$

or, equivalently,

$$CV_A = [\text{sqrt}(V_P * h^2)/M] * 100 \tag{4}$$

with M being the phenotypic trait mean and 100 a conventional scaling-factor. The CV_A thus standardises V_A by the mean of the trait, whereas h^2 standardises V_A by its total phenotypic variance. As long as all traits are measured on a ratio scale and some basic scaling effects are taken into account (Stirling et al., 2002), CV_As are directly comparable across traits and species, which does not hold for h^2s. For many traits across many species, it turned out that V_A increases with the fitness-relevance of a trait (Houle, 1992; Pomiankowski & Møller, 1995; Stirling et al., 2002). Because very high residual variances ($V_{NA} + V_E$) often overshadow substantial V_As, low h^2 values often fail to reflect this pattern (Merilä & Sheldon, 1999; Rowe & Houle, 1996; Stirling et al., 2002).

But how could the traits under strongest selection show the highest V_As? The key seems to be the number of genetic loci that could potentially disrupt the trait by mutating, which is

called the *mutational target-size* of a trait (Houle, 1998). Since mutations occur with random probability at any genetic locus, the number of mutations that affect a trait (i.e. its mutation load) increases linearly with the number of genetic loci that affect the trait. Note that we are referring to the total number of genetic loci that could potentially affect the trait if they became polymorphic due to mutation, not the number of loci that are actually polymorphic at a given point in time (i.e. the QTLs), which are only about 10% of the potential loci (Pritchard, 2001; Rudan et al., 2003). Fisher's (1930) fundamental theorem works best for traits that are affected by only one genetic locus (Price, 1972; Ewens, 1989). The more genetic loci affect a trait, the greater the probability that any of these loci will be hit by a mutation, the more mutations will accumulate in the trait, and the harder it will be for selection to deplete the V_A of this trait. Instead of reaching genetic uniformity, non-neutral traits with large mutational target sizes will therefore be stuck in a balanced state of mutation and selection.

The trait with the largest mutational target-size is, of course, fitness itself: it is influenced by all selectively non-neutral parts of the genome (Houle et al., 1994). Fitness should therefore have a very large CV_A, which is in fact the case (Burt, 1995). Similarly, other traits closely related to fitness (e.g. so-called life history traits, such as longevity or total offspring number) are usually complex compounds of various heritable traits, leading to high mutational target sizes. For example, longevity is potentially influenced by disruptions in any organ system—circulatory, nervous, endocrine, skeletal, etc.—so its mutational target size includes the mutational target sizes of all these organ systems. Consistent with this, very high CV_As have been reported for life-history traits in various species (Houle, 1992), including humans (Hughes & Burleson, 2000; Miller & Penke, in press). In contrast, low CV_As can be found in genetically simpler traits less related to fitness, such as some morphological traits (e.g. bristle number in fruit flies or height in humans—Miller & Penke, in press; Pomiankowski & Møller, 1995).

The watershed model

Cannon and Keller (2005; see also Keller & Miller, 2006a) introduced the *watershed model* (Figure 1) as an analogy to illustrate the relation between genetic variation and the mutational target size of traits. Its basic point is that 'downstream' traits, which are closely related to overall fitness, require the adaptive functioning of virtually the whole organism—the integrated functioning of many subsidiary 'upstream' mechanisms—behavioural, physiological, and morphological. Just as many small creeks join to become a stream, and several streams join to become a river, many genetic and neurophysiological micro-processes (e.g. the regulation of neural migration, axonal *myelinzation*, and neurotransmitter levels) might interact to become a specific personality trait. These personality traits will interact to influence success in survival, socialising, attracting mates, and raising offspring—which in turn determines overall fitness. The upstream micro-processes, such as the regulation of a particular neurotransmitter, may be influenced by only a few genes. The broader middle-level processes, such as reactivity to social stress, are influenced by all genes that affect the corresponding upstream processes. The same holds true for even broader (i.e. more downstream) domains of organismic functioning—which are equivalent to broad components of fitness itself (e.g. sexual attractiveness, social status, foraging efficiency)—these depend on all of the genes that affect all of their upstream processes. A similar argument holds for environmental influences, which, when affecting upstream
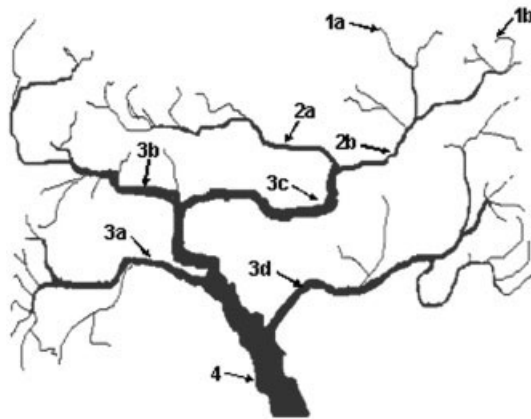
Figure 1.   The watershed model of genetic variation. Mutations at specific loci (1a, 1b) disrupt narrowly defined mechanisms such as dopaminergic regulation in the prefrontal cortex (2b). This and other narrowly defined mechanisms contribute noise to more broadly defined mechanisms, such as working memory (3c). Working memory in conjunction with several other mechanisms (3a, 3b, 3d) affect phenotypically observable phenotypes, such as cognitive ability (4). If enough noise is present in the upstream processes, specific behavioural distortions may arise, such as mild mental retardation. All tributaries eventually flow into fitness (Reproduced from Cannon & Keller, 2005, with permission from www.annualreviews.org).

processes, accumulate in downstream traits. But because selection is much less effective in reducing V_E, the V_E of fitness components tends to be large, which reduces their heritability. Merilä and Sheldon (1999) argued that V_{NA} is as robust against selection as V_E, which would imply a high D_α for traits under mutation-selection balance. However, more recent evidence questions the robustness of V_{NA} to selection in downstream traits (Stirling et al., 2002). The exact expected size of D_α for traits under mutation-selection balance must thus be regarded an unresolved issue, though it is likely in the medium range.

Developmental stability and the *f*-factor

As an addition to the watershed model, developmental stability theory (Polak, 2003) explains how mutations that are spread across the genome influence fitness. It argues that organisms often fail to develop according to the evolved blueprint in their genome, since either the blueprint itself or the relevant environmental factors are disrupted. In such a case, the evolved fit between genome and environment is disrupted. Whereas the genomic blueprint is disrupted by mutations, the organism's developmental environment can be disrupted by factors such as pathogens and toxins. From a fitness perspective, the exact combination of disruptive factors doesn't matter: what counts is the total reduction in phenotypic functionality due to developmental instability. Similarly, only the total mutational damage in the genome is what counts for natural selection. Which genetic sequences the mutations disrupt are largely unimportant—and likely different for each human being.

   An established measure of developmental stability is the bilateral symmetry of body parts that show perfect symmetry at the average population level (e.g. ankle breadth or ear

length), usually aggregated across many body parts. Even though this only taps into morphological developmental stability, body symmetry shows relations to all kinds of fitness components in various species (Møller, 1997), including humans (Gangestad & Simpson, 2000; Gangestad & Yeo, 1997). One well-replicated correlate of body symmetry is general intelligence (Bates, 2007; Luxen & Buunk, 2006; Prokosch et al., 2005). Thus, some genetic and environmental disruptions can apparently impair both cognitive and morphological development. The watershed metaphor breaks down a bit at this point, because it fails to reflect the fact that most mutations are pleiotropic in their effects (Marcus, 2004): each mutation will tend to disrupt several downstream traits. Those harmful effects will be positively intercorrelated in the affected downstream traits (not because the effects are positive, but because they are consistently negative). Therefore, pleiotropic mutations should lead to a 'positive manifold' of intercorrelations among the efficiencies of mid-level processes and of fitness components. In addition, intercorrelations between various processes may arise through developmental interdependence (van der Maas et al., 2006). According to Miller (2000c), this should allow the extraction of a 'general fitness factor' or '*f*-factor' that reflects (inverse) overall mutation load. Just as the *g*-factor of general intelligence (Jensen, 1998) is at the top of a multi-level hierarchy of intercorrelated cognitive abilities, *f* is at the top of a similar hierarchy of genetically intercorrelated upstream traits and processes. In fact, Miller and colleagues (Miller, 2000c; Prokosch et al., 2005) argued that *g* is an important subfactor of *f*, reflecting the integrative functioning of the cognitive system. The V_A of *g* may therefore reflect the aggregate harmful effects of mutations at any of the thousands of genetic loci that affect our brain development and functioning, each of which decreases our cognitive abilities a tiny bit.

Further predictions

Every trait under mutation-selection balance has to be a downstream trait, with mutations occurring randomly across all of the loci that contribute to its 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 it (Turelli & Barton, 2004). The mutations that cause the V_A of more complex downstream traits will thus be numerous, but individually rare, evolutionarily transient, and phenotypically mild in their effects. As a consequence, they will be extremely hard to detect using standard molecular genetic methods (linkage and association studies), and they will be very unlikely to replicate across populations (because different evolutionarily transient mutations tend to affect different populations). Furthermore, since the sheer number of involved loci will impede selection's ability to deplete V_A, the magnitude of D_α for downstream traits will likely be in the medium range (Stirling et al., 2002). These predictions (Table 1) are consistent with what is currently known about the genetic structure of *g* (Plomin, Kennedy, & Craig, 2006; Plomin & Spinath, 2004). Enormous efforts to identify single genes of major effect underlying intelligence led to meagre success at best, and to the conclusion that a huge number of pleiotropic polymorphisms must be responsible for its genetic variation (Kovas & Plomin, 2006). The situation is different for personality traits, however, since good candidates for underlying polymorphisms have been identified (Ebstein, 2006), and most of these have intermediate prevalence rates (Kidd, 2006). In addition, the amount of V_{NA} found in personality traits is often as high as the V_A component (Eaves et al., 1998; Keller et al., 2005), indicating a large D_α of 0.50 or higher. These characteristics of personality traits cannot be explained by mutation-selection balance.

Since traits with a large mutational target size tend to be most affected by mutations that are both rare and recessive, the probability that two copies of the same mutation come together in a single individual and unleash their full deleterious potential is much higher when both parents are genetically related. This is called inbreeding depression. Its counterpart is called heterosis or outbreeding elevation, and occurs when pairings of recessive, deleterious mutations are broken up by sexual recombination in offspring of highly unrelated parents (e.g. parents from different ethnic groups). Due to the predicted genetic structure of traits under mutation-selection balance, we can expect them to show both inbreeding depression and heterosis effects (DeRose & Roff, 1999; Lynch & Walsh, 1998). Such evidence exists for intelligence (reviewed in Jensen, 1998), but is, to the best of our knowledge, absent for personality traits. For example, the offspring of cousin marriages tend to be less intelligent, but we do not know of any evidence that they tend to be more or less extraverted, conscientious, or agreeable than average.

Finally, the typically harmful effects of mutations lead to a clear prediction about the social perception of their phenotypic effects. Since a high mutation load disrupts an organism's functional integrity and ultimately fitness, it should lead to a less favourable social evaluation by those who are looking for a good sexual partner, friend, or ally. The mating context is most important here, because about half of a sexual partner's mutation load will be passed along to one's offspring (Keller, in press). Indeed, virtually all modern evolutionary theories of mate choice argue that any phenotypic trait that reliably signals that a potential mate has a low mutation load will be sexually attractive (Keller, in press; Kokko, Brooks, Jennions & Morley, 2003; Miller, 2000b, c). In an influential paper, Rowe and Houle (1996) argued that sexual selection would drive the evolution of any sexually attractive trait towards higher reliability by making its expression more *condition-dependent*, that is more dependent upon (and revealing of) the overall phenotypic condition (e.g. health, vigour) of the organism. Condition is a trait with very large mutational target size, near the downstream end of the watershed model (Figure 1), and very closely related to fitness (Tomkins, Radwan, Kotiaho & Tregenza, 2004). A condition-dependent trait is thus affected by larger parts of the genome—it will actually 'move downstream', insofar as it becomes sensitive to the efficiency of a larger number of upstream processes. This can explain why, across species, morphological traits that are preferred in mate choice (e.g. the plumage of finches) tend to have much higher CV_A than morphological traits that are irrelevant for mate choice (e.g. bristle number in fruitflies) (Pomiankowski & Møller, 1995), and almost as high as extreme downstream traits such as longevity and fertility.

Since traits that reliably reveal genetic quality (low mutation load) and general phenotypic condition tend to be highly variable within each sex and highly attractive to the other sex, mating markets in socially monogamous species (such as humans) tend to be competitive. Each individual tries to attract the highest-quality mate who will reciprocate his or her interest. Given a period of mutual search in such a competitive mating market, socially monogamous couples tend to form that are closely matched on the average attractiveness level of their sexually attractive traits (Penke, Todd, Lenton, & Fasolo, in press). This phenomenon, called assortative mating (Vandenberg, 1972), is a typical population-level outcome for traits that are under mutation-selection balance, but it is much less likely for traits that are less related to fitness. Mate preferences for higher intelligence, and assortative mating with respect to intelligence, are well-established phenomena in humans, as is the condition-dependent expression of intelligence (Miller, 2000c; Miller & Penke, in press). In contrast, mate preferences for personality traits tend to be modest in size and variable across individuals (Figueredo, Sefcek, & Jones, 2006). In

addition, there is almost no assortative mating for personality traits (Eaves et al., 1999; Lykken & Tellegen, 1993; Vandenberg, 1972). Thus, mate preferences for personality traits show quite a different pattern than mate preferences for universally sought traits, such as intelligence, mental health, and physical attractiveness—which are all presumably condition-dependent and under mutation-selection balance.[1]

To summarise, mutation-selection balance is a very plausible mechanism for maintaining genetic variation in traits that reflect the overall functional integrity of the organism, including general intelligence and general health. This is reflected in the following features: high additive genetic variation, an elusive molecular genetic basis, condition-dependence, inbreeding and outbreeding effects, strong mate preferences, and assortative mating (Table 1). Personality traits do not match these features nearly as well, suggesting that mutation-selection balance may not account for much genetic variance in personality traits.

CAN BALANCING SELECTION EXPLAIN GENETIC VARIANCE IN PERSONALITY?

In both selective neutrality and mutation-selection balance, genetic variation is maintained because selection is unable to deplete it—either because the variation is selectively neutral, or because too much new variation is continually reintroduced. A quite different mechanism is the maintenance of genetic variation by selection itself. This only works if the selective forces that act on a trait are balanced, which occurs when both extremes of the same trait dimension are favoured by selection to the same degree under different conditions. Such balancing selection can happen in a variety of ways.

Variants of balancing selection

One form of balancing selection is *overdominance* (also called heterozygous advantage), which occurs when individuals with different alleles at the same genetic locus have a higher fitness than individuals with two identical copies. Sickle-cell anaemia is a famous textbook case of overdominance, but other examples have rarely been found in nature (Endler, 1986) or in animal experiments (Maynard Smith, 1998). Also, it is now widely believed that overdominance is evolutionary unstable and thus an unlikely candidate for maintaining genetic variation, especially in the long-term (Bürger, 2000; Keller & Miller, 2006a; Roff, 1997).

Another form of balancing selection is *antagonistic pleiotropy*, which occurs when polymorphisms have a positive effect on one fitness-related trait and a negative effect on another (Hedrick, 1999; Roff, 1997). A special case is sexually antagonistic co-evolution, where genetic variants are under opposing selection pressures in men and women (Rice &

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[1]Another domain of heritable personality differences for which strong assortative mating exists are some social attitudes, like conservatism or religiosity (Eaves et al., 1999; Lykken & Tellegen, 1993). However, unlike the basic personality traits and abilities we treat in this article, these attitudes must be regarded as complex developmental outcomes of GxE interactions (Eaves et al., 1999, pp. 77–78). Another noteworthy difference between attitudes and fitness-related traits like intelligence and attractiveness is that there seems to be no universal consensus in either sex on the desired attitudes of an ideal mate. It is thus implausible that competitive mating market dynamics cause assortative mating for attitudes in a similar way as they do for fitness components. Instead, social homogamy (i.e. mate search within the own peer group that tends to share similar attitudes) and later dyadic assimilation appear to be more promising explanations.

Chippindale, 2001). Since selection will usually fix the polymorphism with the least total fitness cost, antagonistic pleiotropy could only maintain genetic variation if the fitness costs of all alleles at such a locus are exactly equal (averaged across environments). In addition, all heterozygous allele combinations have to provide all phenotypic fitness benefits that would be provided by both corresponding homozygous combinations (reversal of dominance, Hedrick, 1999; Curtisinger, Service & Prout, 1994). Furthermore, independent of the number of genetic loci that affect a quantitative trait, antagonistic pleiotropy can maintain genetic variation only at one genetic locus (or two in the case of sexually antagonistic co-evolution) per trait (Turelli & Barton, 2004). Due to these highly restrictive conditions, it is very unlikely that antagonistic pleiotropy plays a major role in maintaining genetic variation (Hedrick, 1999)—although the special case of sexually antagonistic co-evolution might contribute to sex differences in personality and some within-sex personality variation (Keller & Miller, 2006b).

A more likely variant of balancing selection is *environmental heterogeneity*. When a trait's effect on fitness varies across space or time, significant genetic variation can be maintained in populations (Roff, 1997), even in quantitative traits (Bürger, 2000; Turelli & Barton, 2004). A necessary requirement for this to happen is that spatial or temporal fluctuations in selection pressures must occur such that the trait's net fitness effects are nearly neutral when averaged across all relevant spatio-temporal environments. It is not enough for a trait to be neutral in some environments or during some periods, because selection is very efficient at favouring polymorphisms with higher average fitness outcomes across all relevant environments. Only a fully balanced effect of different alleles across space and time will work to maintain genetic variation.

A related type of balancing selection is called *frequency-dependent selection*. In this case, the spatio-temporal fluctuations in selection pressures usually occur in the social environment of the species, rather than the external physical environment. Frequency-dependent selection can only maintain genetic variations if it is negative, favouring traits as long as they are rare in frequency (Maynard Smith, 1998). (Positive frequency-dependence will drive polymorphisms to fixation through a runaway, winner-take-all effect.) The 'social environment' is used in a very broad sense here, and can include the ratio of cooperative partners to cheaters (Mealey, 1995), the ratio of males to females (Fisher, 1930), the distribution of intra- and inter-specific competitors for limited resources in ecological niches, or even parasite-host relationships (which occurs when viruses, bacteria or other pathogenes are best adapted to exploit the most common host phenotypes—Garrigan & Hedrick, 2003). Mathematical models have shown that negative frequency-dependent selection in any of these ways is a viable way to maintain genetic variance (Bürger, 2005; Schneider, 2006).

Thus, environmental heterogeneity and negative frequency-dependent selection are good candidates for maintaining genetic variance by balancing selection, whereas overdominance and antagonistic pleiotropy can work only in rare cases that meet very restrictive conditions. The bottom line is that balancing selection requires a set of varying selection pressures that favour different phenotypes under different conditions. These fluctuating selection pressures must be stronger than any other unidirectional selection pressures on the same trait that consistently favour a certain optimal trait level in every environment (Turelli & Barton, 2004). If this condition is met, balancing selection leads to two or more different phenotypes (or a continuum of phenotypes) with identical average fitness across environments. Since these phenotypes cannot be further optimised by selection, they are called *evolutionary stable strategies (ESSs)* (Maynard Smith, 1982).

Predictions

Balancing selection leads to some distinctive genetic patterns. Reoccurring periods of selection in different directions tend to deplete the V_A of affected traits and result in higher D_α than found for selectively neutral traits (Roff, 1997). D_α will also be higher for traits under balancing selection than for traits under mutation-selection balance, since the former maintains polymorphisms at fewer genetic loci than the latter (Kopp & Hermisson, 2006), and selection is more effective in depleting the V_A from fewer genetic loci (Stirling et al., 2002; van Oers, de Jong, van Noordwijk, Kempenaers, & Drent, 2005). Furthermore, balancing selection can maintain alleles in a population at intermediate prevalences, while mutation-selection balance cannot (Turelli & Barton, 2004). These characteristics (as summarised in Table 1) make balancing selection a likely candidate for maintaining genetic variation in personality traits, although it is unlikely to explain persistent genetic variance in cognitive abilities.

Balancing selection and personality traits

When Tooby and Cosmides (1990) argued that heritable personality differences are basically evolutionary noise, they suggested that parasite-host co-evolution (Garrigan & Hedrick, 2003), a form of negative frequency-dependent selection, might explain the striking amount of evolutionary 'noise' in human behavioural traits better than selective neutrality. Nonetheless, the central message was the same for both evolutionary processes: since the heritable aspects of personality are random by-products of functionally superficial biochemical differences that exist—at best—to prevent our lives from parasites, studying personality differences from an evolutionary perspective is a big waste of time. However, as argued above, there is strong evidence that personality differences have direct effects on fitness. In addition, Keller and Miller (2006a) noted that, for parasite-host co-evolution to explain personality variation as a by-product, there would have to be a very high degree of overlap between genetic loci that affect immune system function and genetic loci that affect personality differences—which seems unlikely.

MacDonald (1995, 1998) made an important step away from Tooby and Cosmides' 'neutral personality assumption' by proposing that five independent behavioural systems under balancing selection explain the dimensions of the FFM of personality. While he regarded both extremes of each dimension as maladaptive, with stabilising selection working against them, he assumed that the relatively broad middle range of each personality dimension reflects equally viable behavioural strategies (i.e. ESSs). MacDonald (1998) also argued that the viability of these strategies should vary across environmental niches. Following MacDonald (1995, 1998), Nettle (2006a) developed more specific hypotheses about the potential fitness costs and benefits associated with each of the FFM dimensions. If these evolutionary cost-benefit trade-offs were exactly the same in every environment, they could maintain genetic variance only through antagonistic pleiotropy, which tends to be evolutionary unstable. However, if the relevant selection pressures fluctuate across time or space, favouring different optima on the cost-benefit curves, they could maintain the range of viable personality trait levels. For example, Nettle (2006a) argued that the high extraversion yields fitness benefits by promoting mating success, social alliance formation, and environmental exploration, but at the cost of increased physical risks and decreased romantic relationship stability. When environments are physically riskier to oneself and one's offspring (who benefit from relationship

stability), high extraversion may be a net fitness cost; but when conditions are safer, high extraversion may yield a net fitness benefit. Environmental fluctuations would thus maintain genetic variation in extraversion.

The challenge in any such balancing selection argument is to identify the specific costs and benefits relevant to each personality trait across different environments. Originally, Nettle (2005) also hypothesised that extraverts might conserve energy by investing less parental effort in offspring, but failed to find supportive evidence. In fact, Nettle's list of extraversion costs and benefits might still be too long, with some proving to be fitness-irrelevant by-products. On the other hand, these are only some of the plausible costs and benefits. Different ones can be suggested for this and other personality traits (Denissen & Penke, 2006). Even if balancing selection proves a good general account of heritable personality traits, much more research would be needed to identify each personality trait's relevant fitness costs and benefits across different environments.

Environmental niches for personality traits

Recently, Camperio Ciani and colleagues (Camperio Ciani, Veronese, Capiluppi & Sartori, 2007) reported an interesting natural experiment that indirectly supports a role for balancing selection by environmental heterogeneity in sustaining the genetic variance of personality traits. They studied average personality differences on the FFM dimensions of Italian coast-dwellers compared to Italians living off the coast on three small island groups. After matching populations for cultural, historical and linguistic background, and controlling for age, sex and education, they found that individuals from families that have lived on small islands for at least 20 generations were lower in extraversion and openness to experience than both mainlanders and more recent immigrants to the island. This pattern makes cultural or developmental explanations for the population differences unlikely—it suggests change on the genetic level. Even though individual fitness consequences of these traits were not measured directly, the apparent recent evolution of genetic differences between populations in these two traits suggests that the fitness payoffs of these two personality traits were historically distinct in these different environments.

In non-human species, recent studies suggest that environmental heterogeneity does impose varying selection pressures on personality traits. Dingemanse, Both, Drent and Tinbergen (2004) could directly measure the fitness payoffs of personality differences (on a carefully assessed shyness-boldness dimension) in the great tit (*parus major*), which varied with food availability across breeding seasons. Similar evidence of environmental heterogeneity favouring personalities exists for some other species (reviewed in Dingemanse & Réale, 2005).

More direct evidence for the importance of environmental heterogeneity in the evolutionary genetics of human personality comes from studies of the global distribution of polymorphisms at the DRD4 locus. This gene regulates dopamine receptors in the brain and has been associated with personality traits such as novelty seeking and extraversion (Ebstein, 2006). The prevalences of different DRD4 alleles differ dramatically across world regions. The evolutionarily newer 7R allele, which is more common in risk-prone, response-ready, extraverted novelty seekers, is much more prevalent in European and American populations than in Asian populations (Chang, Kidd, Livak, Pakstis, & Kidd, 1996). This allele appears to be favoured by selection (1) when benefits can be gained from migrating to new environments (Chen, Burton, Greenberger, & Dmitrieva, 1999; Ding et al., 2002), and (2) under resource-rich environmental conditions (Wang et al., 2004).

Referring to these findings, Harpending and Cochran (2002) noted that under conditions of environmental harshness and resource scarcity (as is common in hunter-gatherer societies), intensive cooperation, strong family ties, stable pair bonds, and biparental investment are necessary for survival and successful reproduction. These ancestrally typical conditions would maintain the more risk-averse, ancestral form of the DRD4 gene. But under more luxuriant environmental conditions, when children can survive without so much paternal support (as in most agricultural and modern societies), the more risk-seeking 7R allele should be favoured by selection, as it leads to a personality more prone to sexual promiscuity and intrasexual competition (Gangestad & Simpson, 2000; Schmitt, 2005).

Arguments for frequency-dependent selection

The role of competition demands some more attention here. Competition, whether for mates, food, or other limited resources, is often a zero-sum game: the winner gains a benefit, but the loser usually pays a cost, at least in the form of wasted time and effort. As the competition within a niche becomes more intense, selection may eventually favour less competitive individuals who refrain from seeking these benefits to avoid the associated costs. This is the logic of the so-called 'hawk-dove game', the classic example of negative frequency-dependent selection (Maynard Smith, 1982). In fact, some evolutionary geneticists have argued that most environmental niches are actually social in nature, because the fluctuating selection regimes caused by environmental heterogeneity are almost always mediated by within-species competition that often takes the form of negative frequency-dependent selection (Bürger, 2005; Kassen, 2002). It is interesting in this regard that personality differences have been found almost exclusively in social species (Figueredo et al., 2005a) and that they tend to have stronger effects on fitness over social than non-social paths in most species (Smith & Blumstein, 2007). Personality appears to be fundamentally social, perhaps reflecting the diversity of social and sexual strategies that can prosper in socially variegated groups that confront fluctuating, heterogeneous environments. This might be especially true for human personality after our species achieved 'ecological dominance' (i.e. reliable mastery of food acquisition and protection from predators and other hazards), which somewhat buffered our ancestors from spatio-temporal variation in the non-social environment (Alexander, 1989). Explicit arguments that negative frequency-dependent selection could maintain genetic variance in specific personality traits have been proposed by Gangestad & Simpson (1990) for female sociosexuality (i.e. promiscuity) and by Mealey (1995) for psychopathy.

Another application of negative frequency-dependent selection to explain personality has been proposed by Rushton (1985) and extended by Figueredo et al. (2005a, b). They argue that virtually all human individual differences, including broad personality factors, intelligence, attachment styles, reproductive strategies, growth, longevity, and fecundity, may reflect a single underlying 'life-history' dimension of variation in the organism's allocation of investment in growth versus survival versus reproduction across the life-course. Drawing a parallel to a similar, well-established dimension of between-species differences in evolutionary ecology, they suggest that this life-history dimension is maintained by negative frequency-dependent selection within and across human groups. A fortuitous side effect is that such variation reduces within-group and between-group competition by allowing individuals and groups to fill different socio-environmental niches. Figueredo et al. (2005a, b) hypothesised that if a broad set of physical and psychology traits (e.g. intelligence, personality traits, sociosexuality, longevity) are subject

to hierarchical factor analysis, a superordinate '*K*-factor' will emerge that reflects variation on this life history dimension (note that this hypothesised *K*-factor is distinct from the *f*-factor discussed above).

A critical point from an evolutionary genetic perspective is that frequency-dependent selection (like any form of balancing selection) is only able to maintain polymorphisms at a few major loci (Kopp & Hermisson, 2006; Turelli & Barton, 2004). As a consequence, frequency-dependent selection on the *K*-factor would only be possible if a few polymorphisms would function as 'switches' that could simultaneously alter the development and expression of all those many traits the *K*-factor aims to explain, including some of the most important emergent traits at the downstream end of the watershed model (Figure 1), such as longevity, growth, intelligence, and fecundity. As long as there is no evidence that these 'polymorphisms for almost everything' exist, future research on life history variation should distinguish more carefully between (1) mutation-selection balance for downstream traits like longevity, growth, intelligence, and fecundity, (2) the condition- and environment-dependent adjustment of reproductive strategies (Gangestad & Simpson, 2000; Penke & Denissen, 2007), and (3) balancing selection for various independent personality traits at a more upstream level of genetic complexity.

To summarise, balancing selection by environmental heterogeneity, often mediated by negative frequency-dependent selection, seems the most plausible mechanism for maintaining genetic variation in personality traits. In contrast, balancing selection is implausible for maintaining genetic variation in downstream fitness-related traits, such as intelligence.

THE ROLE OF THE ENVIRONMENT IN EVOLUTIONARY GENETICS

Evolutionary adaptationism is often misunderstood as overemphasising genetic influences and neglecting environmental influences on behaviour. In fact, the opposite is generally true: evolutionary theory is fundamentally environmentalistic (Crawford & Anderson, 1989), because it is about the adaptive fit of an organism to its environment—a GxE interaction.

Phenotypic plasticity

One form of this interaction—selection—has already been discussed. Selection acts only upon the complete phenotype, which is at the most downstream end of the watershed model (Figure 1), at the level of overall fitness. But GxE interactions take place all the way upstream, up to the molecular level, where transcribed genes can only produce specific proteins if the required amino acids are present (ultimately a nutritional issue). From this perspective, it is hardly surprising that identical genotypes can produce very distinct phenotypes. This phenomenon is called phenotypic plasticity, and it is probably ubiquitous in nature (West-Eberhard, 2003). The environment thus has two distinct roles in evolutionary genetics: It interacts with the genotype in the ontological development of the phenotype, and then, as a selective regime, determines the phenotype's fitness and decides its fate.

Ideally, organisms would fare best if they could fit themselves perfectly and instantly to the environmental demands in every situation—morphologically, physiologically and behaviourally. Of course, developmental constraints render such an unlimited degree of phenotypic plasticity implausible for physical traits (e.g. no drowning mammal can suddenly develop gills, no matter how advantageous such a transformation would be). In

contrast, unlimited behavioural plasticity has been an attractive scientific vision for a long time, both in psychology (i.e. radical behaviourism) and biology (i.e. traditional behavioural ecology; Krebs & Davies, 1997). But even in the case of behaviour, unlimited plasticity is impossible to achieve adaptively, because the environment does not reliably signal the likely fitness payoffs of all possible behavioural strategies (see Miller, in press). In a complex world, environmental cues that can guide adaptive behaviour are inherently noisy, often contradictory, and unpredictably variable (Brunswik, 1956; Gigerenzer, Todd, & the ABC Research Group, 1999). The unreliability of environmental cues means that any behavioural plasticity based on trial-and-error learning must take time, because it must depend upon a decent sample of action-payoff pairings. Thus, given the complexities of real-world environments, organisms cannot instantly discern and implement the optimal behavioural strategy, so fitness-maximising by unlimited behavioural plasticity is an impossible ideal.

Universal constraints on phenotypic plasticity

Fortunately, evolution constrains behavioural plasticity in adaptive directions, just as it constrains physical development. As long as environmental features are sufficiently stable and fitness-relevant (e.g. women get pregnant but men don't, rotten food is toxic, children demand more care and protection than adults), natural selection will fixate psychological mechanisms such as emotions, preferences, and learning preparednesses that adaptively bias our reactions to the environment over ontogenetic development. This relieves us from the impossible task of learning our most basic behavioural dispositions de novo every generation (Barrett, 2006; Figueredo et al., 2006; Tooby et al., 2005). These kinds of GxE interactions—interactions between inherited psychological adaptations and ancestral adaptive challenges—are the central subject of adaptationistic evolutionary psychology. Cervone (2000) argued that they also constitute interesting building blocks for personality theories. However, adaptationistic evolutionary psychology deals principally with interactions between the universal genetic make-up of our species and fitness-relevant aspects of the environment that reoccurred over evolutionary time. Such interactions might explain the non-genetic variation in some personality domains (e.g. attachment styles—Buss & Greiling, 1999), but are largely uninformative about heritable personality differences.

Individual constraints on phenotypic plasticity

When selection cannot deplete all genetic variation (for any of those reasons discussed above), different genotypes persist simultaneously in the population. Genotypes might differ in their response to the environment, leading to the statistical effect that behaviour geneticists refer to as a GxE interaction (Moffitt, Caspi & Rutter, 2006). In humans, such interactions have been found, for example, between the MAOA polymorphism and childhood maltreatment in the development of conduct behaviour (Caspi et al., 2002), and between the 5-HTT polymorphism and stressful life events in the development of depressiveness (Caspi et al., 2003). By systematically varying both the genotypes and the environments, evolutionary geneticists studying non-human species can determine a typical response function for each individual genotype, a so-called *reaction norm* (Via et al., 1995) (Figure 2). While a GxE interaction is a population statistic, an individual reaction norm can be regarded as a characteristic of an individual genotype (Pigliucci, 2005). Reaction norms were originally used to study the developmental plasticity of
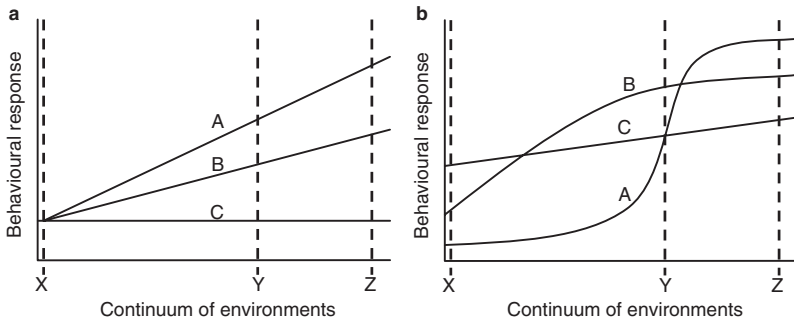
Figure 2.   Two examples for individual reaction norms. Both figures show the individual reaction norms of three genotypes (A–C) along a continuous environmental dimension. The trait in (a) has simple reaction norms, where all genotypes react linearly to environmental changes and differ only in their slope. The trait in (b) has complex reaction norms, where genotype C reacts linearly and genotypes A and B react non-linearly in different ways. This leads to different rank orders of reaction strength at point X, Y, and Z on the environmental dimension, implying the absence of structural pleiotropy (Figure 2b is redrawn after van Oers et al., 2005).

morphological or life-history traits, but when behavioural ecologists realised the systematic limits of behavioural flexibility, they began to view heritable response styles—known to psychologists as personality traits—as behavioural reaction norms. (Sih, Bell, Johnson, & Ziemba, 2004; van Oers et al., 2005).

While behavioural ecologists discovered animal personality only recently (Sih et al., 2004), their immediate equation of personality traits with individual reaction norms helped them to circumvent the 'person-situation debate' in personality psychology (Mischel, 2004). Instead of looking for personalities that reliably predict behaviour across all possible situations, or situations that reliably predict behaviour across all possible personalities, behavioural ecologists quickly adopted a reaction-norm view of personality that neatly resembles the personality signatures view of Mischel and Shoda (1995). Personality signatures describe stable patterns of contingent (if-then) relationships between personalities, situations, and behaviours—just as reaction norms describe stable contingencies between genotypes, environments, and phenotypic outcomes. These person-situation contingency profiles turn out to show reasonable consistency (Borkenau, Riemann, Spinath & Angleitner, 2006; Mischel & Shoda, 1995), but it is a different type of consistency than the well-known rank-order stabilities of personality traits across situations (Mischel, 2004). However, unlike individual reaction norms, personality signatures describe environment-behaviour functions for persons, not for genotypes. Although Mischel and Shoda (1995) acknowledge the possibility that genes influence personality signatures, their Cognitive-Affective Personality Systems model emphasises the importance of learned beliefs, appraisals, expectancies, and goals, organised in cognitive-affective units. However, personality signatures show substantial heritabilities (Borkenau et al., 2006), so these cognitive-affective units are apparently influenced by genetic variation, and a genotype-oriented reaction-norm view may be appropriate.

To describe an individual reaction norm does not require a mechanistic model of the psychological processes that mediate between environmental contingencies and behaviours. Reaction norms simply relate dimensional variations in genotypes and environments to variations in behavioural outcomes. Thus, the shapes of individual reaction norms are what can be equated with personality traits (van Oers et al., 2005). While reaction norm shapes can be simple (e.g. linear) when relating polymorphisms at a single gene locus to the environment (as for example in Caspi et al., 2003), they can

become more complex when polygenic genotypes (as in the case of personality traits) are related to the environment (de Jong, 1990). Furthermore, while the studies by Caspi et al. (2002, 2003) provide examples of reaction norms in personality development (i.e. GxE interactions during childhood predict adolescent personality), the concept of individual reaction norms is not limited to a developmental time frame. Reaction norms can also describe GxE interactions in the production of ongoing behaviour, analogous to Mischel and Shoda's (1995) personality signatures.

Note that reaction norms can be determined for any phenotypic trait, including cognitive abilities. However, we believe that reaction norms are much more informative for personality traits than for cognitive abilities. Reaction norms provide an elegant tool to disentangle the twofold role of the environment for personality traits as both a source of phenotypic plasticity within a generation and of fluctuating selection pressures across generations. This more nuanced view of environmental influences on behaviour is unnecessary for fitness components such as cognitive abilities that are more likely under mutation-selection balance, in which case selection pressures push traits in roughly the same direction (minimum genetic mutation load, maximum phenotypic efficiency) across all kinds of environments. In addition, the phenotypic plasticity of general intelligence apparently reflects simple condition-dependency, as g declines with adverse environmental influences (e.g. starvation, dehydration, sickness) that decrease general condition (Miller & Penke, in press). Since the genetic variation in g accounts for almost all genetic variation in cognitive abilities (Plomin & Spinath, 2004), the reaction-norm view seems less helpful for cognitive abilities than for personality traits.

INDIVIDUAL REACTION NORMS AND THE HIERARCHICAL STRUCTURE OF PERSONALITY TRAITS

The hypothesised existence of complex individual reaction norms has an interesting implication for the hierarchical structure of personality traits. We illustrate this with an example modified from van Oers et al. (2005) (Figure 2): Let two personality traits (say, depressiveness and anxiousness) be described by reaction norms to a continuum of environmental stress. For depressiveness, we assume the simple reaction norm found by Caspi et al. (2003) (Figure 2a): Genotype A shows high depressiveness in highly stressful environments (i.e. point Z), medium depressiveness in the less stressful environment Y, and no depressiveness in the calm environment X. Genotype B shows the same reaction on a lower level (i.e. B's individual reaction norm has a smaller slope), while C is resilient in all environments. Let us now assume a hypothetical, more complex reaction norm for anxiousness based on the same three genotypes and environments (Figure 2b). In environment Z, the rank order of the anxious reactions is the same as for depressive reactions for the three genotypes (A > B > C), implying a positive genetic correlation between the two traits in this environment. (Note that the reaction norm model assumes that all relevant environmental influences are captured either in the environmental dimension or in confidence intervals around the reaction norm functions, so that we can speak of genetic correlations here.) The critical effect of complex reaction norms is revealed at the other two points of the environmental dimension: In environment Y, genotypes A and C react with an identical degree of anxiety, and genotype B reacts only slightly more strongly. The genetic correlation between anxiety and depressiveness in this environment would therefore be close to zero. Finally, in environment X, the rank order of the anxious reactions for the three

genotypes is the inverse of their rank order for depressive reactions in the same environment, leading to an apparent negative genetic correlation. In this purely hypothetical example, subsuming both traits in a higher order factor (here neuroticism) would not be warranted, since their relationship is highly context-dependent. More generally, delineating hierarchical personality structures would be impeded by sign changes in the genetic correlations among personality traits measured across environments. Therefore, van Oers et al. (2005) regard the absence of sign changes in genetic correlations of related facet traits across environments as a necessary condition for the existence of superordinate personality domains. This leads us to specific requirements concerning how personality-related genes must affect multiple personality traits.

Structural pleiotropy

Except for some rare and evolutionarily unstable cases (called linkage disequilibria), genetic correlations are always caused by pleiotropy, the effect of polymorphisms on multiple traits (Roff, 1997). Pleiotropy has been shown for the hierarchical structure of the FFM in twin studies (Jang, Livesley, Angleitner, Riemann, & Vernon, 2002; Jang, McCrae, Angleitner, Riemann, & Livesley, 1998; McCrae et al., 2001; Yamagata et al., 2006). But as in our hypothetical example, pleiotropy in itself does not prevent sign changes in genetic correlations between traits across environments. Sign changes can only be prevented by functional, physiological, or developmental links between the effects of polymorphisms on one trait and their effects on another trait. Such a condition, called *structural pleiotropy*, poses a developmental constraint on the independent phenotypic expression of both traits in all environments (de Jong, 1990). To be sure, structural pleiotropy does not mean that complex reaction norms, such as those depicted in Figure 2b, are theoretically implausible. Instead, the central point is that, for two traits to be facets of the same higher-order factor, the rank order of the phenotypic effects produced by different genotypes must not reverse across environments. The traits in Figure 2a and b cannot belong to the same higher-order factors, but both can, together with other traits, belong to different factors.

An implication of structural pleiotropy is the existence of underlying neurogenetic mechanisms (e.g. neurotransmitter or endocrinological systems) that are shared by all facets of a higher-order trait. An advantage of viewing personality traits as individual reaction norms is that these mechanisms, which should be closely linked to the genotype, can be explicitly separated from the environmental factors with which they interact. In this way, individual reaction norms come much closer to the original personality trait definition by Allport (1937) as 'psychophysical systems that determine [an individual's] unique adjustment to his environments' (p. 48), than to the purely descriptive, empirically derived factors that are normally posited in personality psychology, and they also avoid the often-criticised circularity of the definition of traits as aggregated instances of behaviour, which are then used to predict...behaviour (Denissen & Penke, 2006).

A developmental perspective

If broad personality domains exist because of shared underlying mechanisms (i.e. because structural pleiotropy preserves the sign of genetic correlations between traits across environments), then personality structure likely develops top-down, from these mechanisms to higher-order personality domains (e.g. neuroticism) to lower-order personality facets (e.g. anxiousness, depressiveness). Over the lifespan, these mechanisms

might modulate the cognitive and affective experiences that individuals acquire through interacting with their environments. Thereby, they might act as forms of 'prepared learning' (Figueredo et al., 2006) for the acquisition of the cognitive-affective units emphasised by Mischel and Shoda (1995), and as 'experience-producing drives' (Borkenau, Riemann, Spinath, & Angleitner, 1996) that motivate active niche selection (Denissen & Penke, 2006). These shared mechanisms would be the ties that bind different facet traits within broader personality domains. Together with the influence of unique genetic variation on the level of lower-order traits (Jang et al., 1998, 2002), this would result in the hierarchical structure of personality traits, down to the level of idiosyncratic habits and behavioural patterns.

The dimensionality of personality

Note that this theoretical argument makes no commitment to any particular number of highest-order mechanisms or their interactions. The prominence of the FFM led evolutionary psychologists (MacDonald, 1995, 1998; Nettle, 2006a), including us (Denissen & Penke, 2006), to hypothesise selection regimes at this hierarchical level. However, some of the FFM dimensions may still share some common mechanisms that render them not entirely orthogonal (Jang et al., in press). For example, Jang et al. (2001) showed a significant amount of genetic overlap between the domains of neuroticism and agreeableness, which was partly explained by the 5-HTTLPR polymorphism. It is also possible that several neurogenetic mechanisms interact to form what we observe as broad personality dimensions. Jang et al. (2002) showed that two independent source of genetic variance were necessary to explain the variation of each of the FFM personality domains. If these independent genetic sources reflect independent neurodevelopmental mechanisms, environments may exist in which they no longer contribute to the same behavioural dispositions (de Jong, 1990), and are no longer under parallel selection pressures (Figure 2b). The bottom line is that the genetic architecture of personality might not reflect the phenotypic structure of established factor-analytic models, though it would be surprising if it was completely different. At any rate, we believe that the reaction norms of structurally independent mechanisms constitute a promising level of analysis for an evolutionary personality psychology.

OPERATIONALISING INDIVIDUAL REACTION NORMS

The natural approach to the study of reaction norms would be to observe the behavioural reactions of different genotypes along a well-quantified environmental continuum. However, the standard methods used by evolutionary geneticists to study non-human species (e.g. inbred strains) are of course not available to human psychologists. Identical twins provide a surrogate (Crawford & Anderson, 1989), though a limited one, since only two copies of each genotype exist and the environment cannot be varied experimentally. One alternative is to relate single polymorphisms to behavioural variations that are contingent on certain environmental variables (as done by Caspi et al., 2002, 2003, see also Moffitt et al., 2006). While this approach will certainly become common in the near future as a consequence of cheaper, faster, and more powerful genotyping methods (e.g. DNA microarrays), such studies might still fail to capture the complex polygenic nature of personality traits in the near future.

Another alternative is to assess individual differences directly at the level of hypothetical underlying mechanisms. Here, an *endophenotype* approach appears highly promising. Endophenotypes are phenotypic structures and processes (e.g. neurotransmitter systems or hormone cascades) that can be quantified directly (e.g. by neuroimaging or blood sampling) and that mediate between genes and more complex or abstract traits (Boomsma, Anokhin & De Geus, 1997; Cannon & Keller, 2005). In the watershed model (Figure 1), currently measurable endophenotypes tend to be located at a very upstream level. In the exemplary case of neuroticism, amygdala reactivity (Hariri et al., 2002, 2005) provides an especially good example of a mediating endophenotype, though there are likely several others. Sih et al. (2004), for example, highlighted the role of hormonal mechanisms in animal personality.

Of course, all of these approaches are much harder work than using classical personality questionnaires, so they will probably remain a minority interest within personality psychology. But even questionnaires can be improved to reflect a view of traits as individual reaction norms, by explicitly assessing behavioural reactions to specific fitness-relevant situations, instead of aggregating across arbitrary modern environments (Denissen & Penke, 2006; Mischel & Shoda, 1995). For example, some people may be socially confident at informal parties but not at public speaking, whereas for others, the opposite may apply. To class them both as 'extraverts' may conflate disparate genotypes that lead to distinct endophenotypes, behavioural strategies, reaction norms, and fitness payoffs. Indeed, the quest to maximise internal consistencies within personality scales (e.g. by homogenising the environmental circumstances of behaviours) may lead personality psychologists to eliminate some of the questionnaire items that are most informative about GxE interactions and individual reaction norms.

AN EVOLUTIONARY GENETIC MODEL OF PERSONALITY

The evolutionary genetics of personality can be summarised in the model depicted in Figure 3.

For natural selection, the structure of individual differences is fairly straightforward and simple: all living organisms vary on one major dimension—fitness—which is their statistical propensity to pass their genes on to future generations to come. Miller's (2000c) *f*-factor represents this dimension at the very top of any evolutionary hierarchy of heritable differences—or at the very downstream end of the watershed model (which is why we put *f* at the bottom in Figure 3). The upstream-downstream dimension is shown on the left. Since virtually all psychological differences studied so far show heritability, the central question for evolutionary personality psychology is: how do psychological differences relate to the *f*-factor?

All heritable psychological differences begin with a set of genes that influence the functioning of neurophysiological mechanisms (detectable as endophenotypes). A simplification of the model is that environmental influences are omitted at the genetic and endophenotype levels. This seems justifiable, since environmental effects are probably smaller (due to developmental canalisation) at the upstream levels than at the downstream levels. One or several of the mechanisms on the endophenotype level result in the behavioural tendencies that we observe as traits and abilities at the dispositional level. In relevant situations, these dispositions influence behaviour, and from this point onward, they
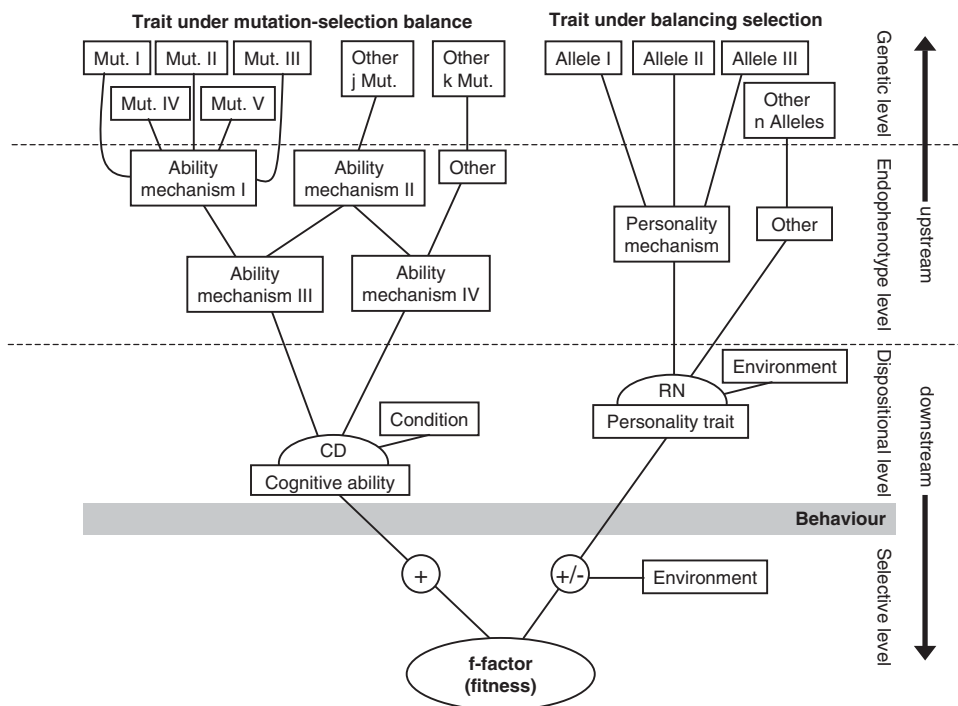
Figure 3. An integrative model of the evolutionary genetics of personality. *Note*: Mut., mutation; CD, condition-dependency; RN, reaction norm.

affect the biological fate of the organism: behaviour influences the organism's adaptive fit to the current environment, and thus influences its overall reproductive success.

Genetic variation in personality differences might be maintained by selective neutrality, mutation-selection balance, or balancing selection—each of which would leave distinctive footprints in a trait's genetic architecture. We have argued that selective neutrality is implausible for most personality differences, given their pervasive effects on fitness-relevant life outcomes. Mutation-selection balance requires that (1) a trait is influenced by enough genes that new mutations disrupt its efficiency at a steady rate, and (2) selection favours trait efficiency strongly enough to eliminate these mutations after some evolutionary time. As a consequence, these traits will be influenced by many interdependent neurogenetic mechanisms on the endophenotypic level, and will show substantial additive genetic variation that affects trait efficiency and thereby influences fitness. Environmental influences on such traits will be mediated mostly by their effects on the organism's overall condition. In line with Miller (2000c; Prokosch et al., 2005), we propose that general intelligence belongs to this category of traits under mutation-selection balance. In this case, the upstream ability mechanisms I and II in Figure 3 could be, for example, the efficiency of cerebral glucose metabolism and the accuracy of prefrontal programmed cell death during adolescence, and the downstream ability mechanisms III and IV could be processing speed and working memory capacity (Jensen, 1998).

An evolutionary genetic conceptualisation of cognitive abilities would thus be: individual differences in the functional integrity of broad systems of the adaptive cognitive apparatus, caused by an individual's load of rare, mildly harmful mutations. In short, cognitive abilities are cognitive fitness components. For such traits, a low mutation load is always beneficial, regardless of the environment.

By contrast, the phenotypic and genetic characteristics that are typically found in studies of personality traits (like those in the FFM) suggest that balancing selection is maintaining the genetic variance in most (if not all) personality traits. Balancing selection can favour different traits in different social or non-social environments. In addition to this role as a varying selection pressure on personality traits, the environment serves a second role earlier on, when interacting with the neurophysiological architecture of the trait (i.e. its personality mechanism or mechanisms) through a reaction norm to form a behavioural tendency. This twofold role may make the environmental influences on personality traits under balancing selection much more numerous, complex, and differentiated than those affecting traits under mutation-selection balance (which may reflect general phenotypic condition rather than specific environmental contingencies). On the other hand, the upstream genes and endophenotypes of personality traits under balancing selection will be fewer than those of cognitive abilities under mutation-selection balance.

An evolutionary genetic conceptualisation of personality traits would thus be: individual differences in genetic constraints on behavioural plasticity, which lead to behavioural tendencies that follow individual reaction norms, and produce different fitness consequences in different environments. In short, personality traits are individual reaction norms with environment-contingent fitness consequences.

WHERE DO COMMON PSYCHOPATHOLOGIES FIT INTO THE MODEL?

While this review focuses on personality differences in the normal range, we would like to add some remarks on the place of polygenic psychopathologies in our model. In an extensive discussion of the evolutionary genetics of common psychopathologies, Keller and Miller (2006a, b) argued that mental disorders such as schizophrenia and bipolar disorder are best conceptualised as traits under mutation-selection balance. Indeed, they cite evidence that these disorders possess all the expected characteristics (Table 1). In our model, these disorders are thus fitness components that mark the low end of the f-factor. Some common psychopathologies, however, show clear relationships to personality traits in the normal range, especially to high neuroticism and low agreeableness (Saulsman & Page, 2004). These disorders might be viewed as maladaptive extremes of normal personality traits—rare genotypes that will sometimes occur in polygenic traits due to sexual recombination. For example, extreme extraversion (e.g. impulsive, narcissistic, histrionic, and/or promiscuous behaviour) and extreme introversion (e.g. schizoid, avoidant, hermit-like withdrawal from all social contact) may both be too extreme to yield fitness benefits in any plausible niche (MacDonald, 1995, 1998). But extreme values on normal traits alone are usually insufficient for the occurrence of psychopathologies (Saulsman & Page, 2004), and even high neuroticism and low agreeableness can be adaptive (though not necessarily socially desirable) when the social environment is harsh, risky, and unforgiving, or when it is exploitable and gullible, respectively (Denissen & Penke, 2006; Nettle, 2006a).

An alternative is that modern societies produce mismatches between heritable temperaments and available niches. For example, Harpending and Cochran (2002) argue that the very same 7R-DRD4 allele that predisposes children to attention deficit hyperactivity disorder (ADHD) today may have been adaptive if these individuals lived in a violently competitive, polygamous society. More generally, genetic variation maintained by environmental heterogeneity implies that there are always some individuals for whom an optimal niche does not currently exist. Similarly, negative frequency-dependent selection implies that there are cases in which an individual's usual niche is overcrowded and competitive.

In addition, the pathological nature of personality disorders might also result from a high mutation load, but receive their characteristic symptoms from an interaction of this load with certain personality traits. For example, very high openness to experience might overwhelm individuals whose cognitive abilities are compromised by a high mutation load and consequently lead to a diagnosed schizotypic personality disorder, while it might appear attractive in less mutation-laden individuals, who are able to turn it into exceptional creative outputs (Keller & Miller, 2006b; Nettle, 2006b; Nettle & Clegg, 2006).

PRACTICAL IMPLICATIONS FOR BEHAVIOUR GENETICS

An evolutionary genetic framework for personality psychology has some practical implications for behaviour genetic studies:

(1) Demonstrating that a personality trait is heritable had become scientifically unsurprising by the early 1990s (Turkheimer & Gottesman, 1991), and is not very informative about a trait's nature or etiology, since it confounds information about a trait's evolutionary history, structure, and GxE interactions (Stirling et al., 2002). This is especially true for the broad-sense heritabilities that are estimated in the classical twin design, since they do not distinguish between V_A and V_{NA} (Keller & Coventry, 2005), which is very important in evolutionary genetics (Merilä & Sheldon, 1999). We therefore concur with Keller and Coventry (2005) that more studies using the extended twin-family design (Neale & Cardon, 1992), or other designs that unconfound V_A and V_{NA}, are highly desirable, especially when testing evolutionary genetic hypotheses (Table 1).

(2) Because of the great datasets and twin registries already available, classical twin studies will probably remain the most common type of behaviour genetic publications. However, such studies would be more informative (or less misleading) about the evolutionary genetics of traits if their underlying statistical assumptions were made more explicit. Many personality psychologists seem not to appreciate that classical twin studies can yield a wide range of mathematically equivalent parameter estimates (e.g. for additive genetic vs. dominance vs. epistatic effects) that have very different implications for the evolutionary histories of the traits under investigation (Coventry & Keller, 2005; Keller & Coventry, 2005). We therefore suggest that future publications of classical twin study results make use of the technique developed by Keller and Coventry (2005) and fully disclose the confidence intervals and parameter spaces for their results.

(3) The equation of personality differences with individual reaction norms highlights the fact that GxE interactions are ubiquitous in nature. Similarly, balancing selection on personality traits due to spatio-temporal heterogeneity of selection pressures suggests that GxE correlations are fairly common. Unfortunately, the usual approach in quantitative behaviour genetic studies is additive variance decomposition, which hides both GxE interactions and GxE correlations in apparent main effects (Purcell, 2002). However, the necessary statistical modelling techniques exist to identify such interaction effects (Neale & Maes, 2004; Purcell, 2002), and evolutionary genetics suggests that they should be used more frequently.

(4) For the same reason, the use of personality trait measures (especially self-report questionnaires) that aggregate across situations might have reached its limits in clarifying the genetic architecture of personality (Ebstein, 2006). Both endophenotype approaches and phenotypic measures that aim to keep person and situation separated (Denissen & Penke, 2006; Mischel & Shoda, 1995) provide better alternatives.

(5) Calculating the coefficient of additive genetic variance (CV_A) of a trait, which is very informative about its evolutionary history (Houle, 1992; Stirling et al., 2002), requires a ratio-scale measure (i.e. a measure with a meaningful zero point). Personality questionnaires with rating scales fail to reach this standard. It would be very helpful if valid, ratio-scaled personality measures (e.g. based on quantitative endophenotypes or behaviours measured with regard to their energy output, temporal duration, or act frequency—see Buss & Craik, 1983) could be developed and used in quantitative behaviour genetic studies.

(6) We predict that 'gene hunting' studies will continue to be more successful in revealing the molecular genetic architecture of temperamental personality traits than of general cognitive abilities or polygenic mental disorders (Ebstein, 2006; Keller & Miller, 2006a; Plomin et al., 2006). Evolutionary genetic theory gives a straightforward reason why: while personality traits will be influenced by a limited set of high-prevalence alleles (plus maybe several rare ones, see Kopp & Hermisson, 2006), general intelligence and psychopathologies like schizophrenia will be influenced by rare, recessive, mildly harmful mutations that vary between samples, since they are equally likely to occur at thousands of different, otherwise monomorphic loci, and are removed fairly quickly by selection once they arise. (Note that this goes beyond Kovas & Plomin's (2006) concept of 'generalist genes', which proposes that the same large set of weak-effect polymorphisms underlies cognitive functioning in every individual.) While we do not argue that molecular behavioural geneticists should refrain from studying *g*, common psychopathologies, and other fitness components, we suggest that they take evolutionary genetic predictions of the likely genetic architecture into account when planning studies and interpreting results. A simple first step would be to call the underlying polymorphisms what the empirical evidence suggests they are—rare mutations.

(7) More generally, evolutionary genetics provides a rich theoretical source of hypotheses that should inspire and guide future behaviour genetic studies. For example, factor V (openness to experiences/intellect) is the only domain of the FFM that shows reliable correlations with general intelligence (e.g. DeYoung et al., 2005). From an evolutionary genetics viewpoint, this puts factor V in an ambiguous position: does it reflect an ESS under balancing selection (Denissen & Penke, 2006; Nettle, 2006a), or an important component of the *f*-factor, which should be under mutation-selection balance? If factor V is under balancing selection, its molecular genetic basis should

be much easier to identify—especially if behaviour genetics researchers statistically control for general intelligence when investigating polymorphisms that may influence factor V. Other exemplary evolutionary genetic hypotheses can be found in Miller (2000c) and Keller (in press).

CONCLUSION

Evolutionary psychology has made so much progress in the last 15 years by relying on an evolutionary adaptationist metatheory that guides the identification of ancestral adaptive problems, the likely psychological adaptations that they favoured, and the likely design features of those adaptations that can be investigated empirically (Andrews et al., 2002; Buss, 1995). We have argued that evolutionary genetics can provide a similarly powerful approach to the study of heritable individual differences in personality.

Evolutionary genetics is itself a fast evolving field. While we tried to give an up-to-date overview of evolutionary genetic principles that seemed most relevant for personality psychology, some of those principles will probably be refined, extended, or challenged in the near future. They should thus be viewed as the provisional, current state of the art, not as biological commandments carved in stone. Still, they may help personality psychology enormously by clarifying what is evolutionarily possible and plausible, and what is not. This way, evolutionary genetics can provide personality psychology with new hypotheses, guidance on how to interpret results, and constraints on theory formulation. Ultimately, our grandest hope for evolutionary personality psychology is that, given the enormously rich phenotypic and behaviour genetic datasets on human personality, it might identify new evolutionary genetic principles that also apply to other kinds of traits and other species.

We reviewed the current answers that evolutionary genetics can give to a question that has rarely been asked in psychology: how is the genetic variation that obviously underlies most human differences, including personality differences, maintained in the population? It turned out that only two answers are sufficiently plausible for personality differences: either (1) the trait is dependent on so many genes that a balance between rare, mildly harmful mutations and counteracting selection occurs, or (2) variation in the structure of the physical or social environment leads to spatio-temporally fluctuating selection for different alleles. Both evolutionary genetic mechanisms will lead their affected traits to have certain distinctive characteristics and underlying genetic architectures. We concluded that the first process (mutation-selection balance) probably maintains genetic variance in cognitive abilities, while the second process (balancing selection by environmental heterogeneity) probably maintains genetic variance in most personality traits. Thus, cognitive abilities are best conceptualised as cognitive fitness components, while personality traits reflect individual reaction norms with environment-contingent fitness consequences.

Important tasks for future studies include delineating the hierarchical structure of fitness components (with the *f*–factor on the top) and identifying the exact fitness-related costs and benefits associated with each personality trait, as well as the environmental niches that structure those costs and benefits. Social niches with different degrees and forms of competition are especially good candidates for the latter. A promising road for process-oriented personality psychologists is studying the psychological mechanisms that lead to active niche selection, including adaptive self-assessments (Penke & Denissen,

2007; Penke et al., in press; Tooby & Cosmides, 1990) and experience-producing drives (Bouchard et al., 1996).

Finally, we wish to re-emphasise that most heritable individual differences are not adaptations in their own right. They reflect dimensions in the functional design of a species that tolerate some degree of genetic variation. Mutations at too many non-neutral loci will lead to a breakdown of adaptive design. Likewise, traits under balancing selection will tolerate polymorphism only at a few specific loci, while all others loci (which affect the universal adaptive design of the trait) will be protected from large genetic variation by stabilising selection. Adaptive individual differences exist, but only as conditional strategies that are implemented in universal (i.e. zero-heritability) adaptations and evoked by specific environmental cues (Buss, 1991; Buss & Greiling, 1999; Tooby & Cosmides, 1990). An evolutionary personality psychology based on evolutionary genetics does not contradict this view. Instead, it complements evolutionary psychology by explaining what happens when genetic variation is introduced into systems of interacting adaptations (Miller, 2000a; Gangestad & Yeo, 1997). Since genetic variation is ubiquitous in personality psychology, evolutionary genetics is essential for an evolutionary personality psychology.

ACKNOWLEDGEMENTS

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