The Evolutionary Genetics of Personality[†]

LARS PENKE[1,2]*, JAAP J. A. DENISSEN[3] and GEOFFREY F. MILLER[4]

[1]*Humboldt University, Berlin, Germany*
[2]*International Max Planck Research School LIFE, Berlin, Germany*
[3]*Utrecht University, Utrecht, The Netherlands*
[4]*University of New Mexico, Albuquerque, NM, USA*

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

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:

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

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):

$$h^2 = V_A/V_P \qquad (2)$$

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:

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

or, equivalently,

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

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
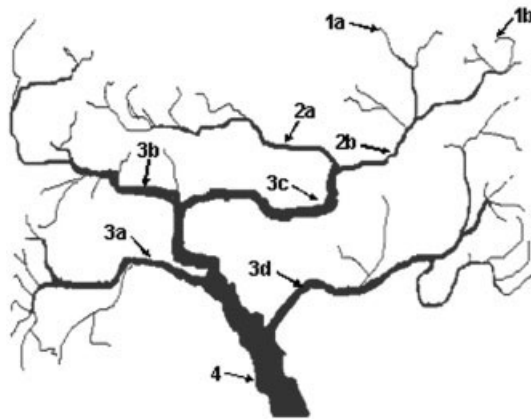
Figure 1.   The watershed model of genetic variation. Mutations at specific loci (1a, 1b) disrupt narrowly defined mechanisms such as dopaminergic regulation in the prefrontal cortex (2b). This and other narrowly defined mechanisms contribute noise to more broadly defined mechanisms, such as working 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
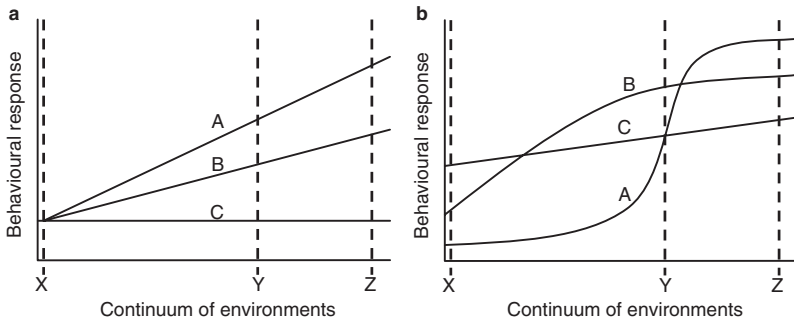
Figure 2.   Two examples for individual reaction norms. Both figures show the individual reaction norms of three genotypes (A–C) along a continuous environmental dimension. The trait in (a) has simple reaction norms, where all genotypes react linearly to environmental changes and differ only in their 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
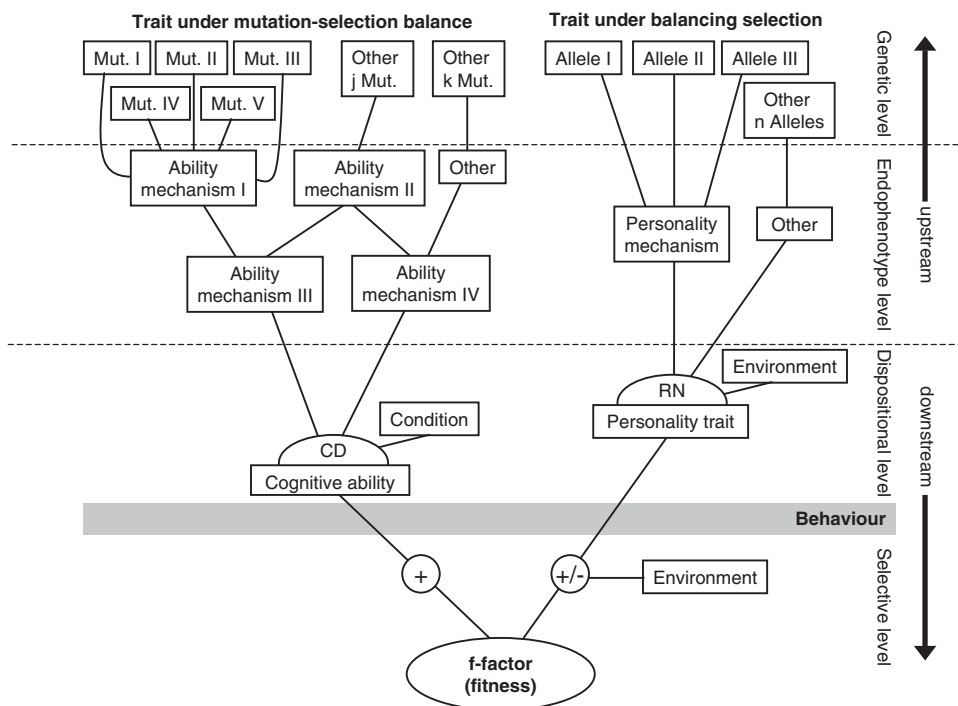
Figure 3.   An integrative model of the evolutionary genetics of personality. *Note*: Mut., mutation; CD, condition-dependency; RN, reaction norm.

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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Discussion on 'The Evolutionary Genetics of Personality' by Penke, Denissen and Miller

OPEN PEER COMMENTARY

Out of the Armchair

TIMOTHY C. BATES

Department of Psychology, University of Edinburgh, UK
tim.bates@ed.ac.uk

Abstract

Penke et al. (this issue) attempt to explain personality and cognition from theory rather than empirical study. This overstates the constraints on evolution, while underestimating the power of cross-species HapMap data to directly identify our evolutionary history. Independent of armchair-speculation, information benefiting human understanding, health and well-being is flowing from exactly the research the target suggests should not be pursued. Copyright © 2007 John Wiley & Sons, Ltd.

The target begins, as does much of evolutionary psychology, from an assumption that human evolution is 'finished': that we are in an evolutionary steady-state, with directional selection and its associated linkage-disequilibrium and departures from Hardy–Weinberg equilibrium long since washed away. This leads directly to models of residual heritability for ability as an irreducible legacy of IQ being distributed across thousands of mutable genes and personality as a simpler system with variation retained due to correlations between genotype and survival being variable to the point of reversing in sign. This commentary focuses on the ability model and conclusions for research.

The target paper ignores evidence from the HapMap (Wang, Kodama, Baldi, & Moyzis, 2006) and from candidate genes (Evans, Mekel-Bobrov, Vallender, Hudson, & Lahn, 2006) suggesting that much of human evolution has a recent history, not just over the last 500 000 years when brain size doubled, but including the period since some humans left Africa and importantly, the 5–10 000 years since the invention of agriculture. These data showing recent and even current selection affecting neuronal function, as well as reproductive and immune function, protein and DNA metabolism and cell-cycle control violate the assumptions on which the target paper is based.

Despite there having been only one small genome-wide linkage scan for cognition, one pooled association study (with density an order of magnitude lower than believed adequate) and exactly zero dense genome-wide association studies for cognition, the authors conclude that searching for genes for cognition is futile: too many to find, too small to be of use and too variable to be easily marked. I suggest that, such data as are available support conclusions exactly opposite to those proposed.

The search for genes is already reaping rewards. The sole linkage scan report found three regions related to IQ (Posthuma et al., 2005), the pooled association study 6 regions (Butcher et al., 2005) (now replicated) and OMIM contains over 1000 major-effect genes for cognition helping elucidate pathways to normal ability. Multiple polymorphism combinations suggest substantial normal single-gene effects on ability (Dick et al., 2007). Paradoxically, for the personality model of the target paper, the success of cognition research greatly exceeds that of the search for personality genes (Willis-Owen & Flint, 2007).

The target makes many additional far-ranging claims about human evolution. They note that cognitive differences may be almost identical to total mutation load. However, while the phenotypic correlation between ability and developmental stability is robust, the sole (as yet unpublished) study on the genetic correlation between developmental stability and IQ found a genetic correlation of 0! Theory predicts it should be close to 1.0, and this represents a massive challenge to the genomic fitness-IQ model, suggesting that the FA-IQ correlation may be environmentally mediated.

Even if we accepted that variance in cognition reflects an inability to remove mutation, much else is left unexplained: for instance, why is mean IQ not much lower or much higher, despite exemplar groups differing by 1 or even 2 *SD* on mean IQ? This is empirical proof that far from being bound by an upper limit imposed by mutation, evolution can move human IQ over massive ranges. Tangentially, this raises the use of Houle's effect-size measure (∂^2/mean). This standardisation was designed to highlight additive variance overshadowed by environmental and non-additive noise. In the case of intelligence, additive phenotypic effect-sizes are already clear, but it is also unclear that linear division by mean trait value is appropriate for ability. Ratio-scaled indices of cognition such as brain mass scale with body mass: should this not be first subtracted out? And frontal-lobes scale as a power function, invalidating linear transforms such as division.

The authors emphasise the *possible* reliance of cognition on many thousands of gene effects. However, gene count *per se* is largely irrelevant for selection. Mutation is important in edge case such as traits outwith selective pressure, where we will expect equal accumulate rates for synonymous and non-synonymous mutations, and traits like human aging where the phenotype appears at too great a distance from reproduction to be selected on. However, even if intelligence is distributed across the entire genome, its heritability leaves it highly modifiable, as the coefficient of selection remains dependent on selective pressure and selectability (i.e. heritability). Indeed, Stoltenberg (1997) suggested replacing h^2 with 'selectability' to highlight its meaning. It is worth noting too that the proposed pleiotropy of cognition with health and physical-fitness (Rae, Digney, McEwan, & Bates, 2003) simply enhances selection for cognition by selecting for higher IQ whenever strength or health has a positive effect on survival. This might even explain the paradoxical 'excess' of human intelligence, despite strong reproductive selection against ability over the last century.

Finally, the claim that small average effects of single genes will hinder gene discovery is false. Rare alleles with major effects in these genes are excellent candidates for pedigree-based analysis and already researchers have discovered some 1000 brain function loci in this way (see OMIM). Similarly, the number of genes currently determining expression of a cognition does not limit the size of increases effected by single-gene changes. Indeed, the massive increase in human brain size over the last 500 000 years is probably due to just a handful of genes such as ASPM and MCPH1 (Zhang, 2003). Some of these show selection even in the last 5000 years (Evans, Vallender, & Lahn, 2006), perhaps related to cognitive functions such as reading ability, language impairment and/or social function, each of which is highly variable and heritable and every bit as dependent on the basic cellular material of the brain as is general ability. But each has shown highly significant linkage and association: Dyslexia appears to be controlled by a dozen or so genes (Bates, Luciano, Castles, Coltheart, Wright, & Martin, 2007), which are rapidly being understood at the level of neuronal development (Luciano, Lind, Wright, Martin, & Bates, in press). Human cognitive-genetics seems redolent with linkage-disequilibrium signals associated with recent evolution, as is most of the human genome.

In summary, to understand how, when and why cognition evolved requires hard empirical work detecting signals of selection, tracking genes over time and establishing biochemical pathways... evolutionary theory *per se* is of limited utility.

Personality: Does Selection See It?

ANNE CAMPBELL

Psychology Department, Durham University, UK
a.c.campbell@durham.ac.uk

Abstract

Selective neutrality offers a parsimonious explanation for personality variation. Bodily variations which do not compromise function (e.g. differences in intestine route) require no special explanation. Variations of the mind are not in principal different from those of the body. A plausible explanation for such neutrality exists which does not require speculative stories about the circumstances of balancing selection. Copyright © 2007 John Wiley & Sons, Ltd.

Occam's razor has been loosely translated as 'All things being equal, the simplest solution tends to be the best one'. Principally for this reason, I have more sympathy than the authors with the selective neutrality of personality. As they observe, the precise route taken by the intestines may vary widely between people. This is an appealing example of neutrality because of the invisibility of intestines and their irrelevance to our social world. Personality traits seem different. We are struck on a daily basis by the differences between people. They form such a central part of social discrimination in our brief lifetimes that it is intuitively hard to accept that this wondrous human variety may be of no special

evolutionary relevance. But would any biologist seriously consider devoting years to the study of individual differences in intestine route?

Personality differences may be no more than 'spandrels' of selection for pathogen resistance (Tooby, 1982). The evolutionary advantage of sexual reproduction is that genetic recombination gives us an edge in the human-pathogen arms race (Hamilton, Axelrod, & Tanese, 1990). The uniqueness of each individual—with respect to those polymorphisms that have no impact on the overall functioning of the organism—offers a moving target to fast-reproducing pathogens. 'Pathogens select for protein diversity introducing the maximum tolerable quantitative variation and noise into the human system . . . protein variation gives rise to a wealth of quantitative variation in nearly ever manifest feature of the psyche; tastes, reflexes, perceptual abilities, talents, deficits, thresholds of activation . . . ' (Tooby & Cosmides, 1990, p. 49). But such variation will not survive if it compromises the integratative functioning of the component parts and so threatens the complex evolved monomorphic design. In short, sustained variability points strongly to functional irrelevance. And if pathogens can explain the evolution of sexual reproduction in terms of the creation of genetic diversity, why should that diversity not be expressed as much through the mind (personality) as the body (intestines)? Biology does not respect any dividing line between them. Penke et al.'s scepticism about neutrality rests on 'strong evidence that personality differences have direct effects upon fitness'. Yet the most striking aspect of Figueredo, Sefcek, Vasquez, Brumbach, King, and Jacobs's (2005) review is the absence of unanimity about relationships between personality and fitness. For example, 'cheerful' adults have fewer health problems but cheerful children have a higher mortality risk across their lifespan. Without a stronger theoretical rationale for trait choice, we risk a fishing expedition in which chance associations will be found due to the sheer number of computed correlations. Even if some traits can be shown to have 'pervasive effects on social, sexual and familial life', such contemporary proximal effects may not translate into different long-term inclusive fitness outcomes.

If personality differences reflect adaptations then we would expect to find a multimodal distribution. Anisogamy evolved because there was an equal advantage in producing numerous small, cheap gametes or fewer large, expensive gametes. Once this cleavage began, there was no advantage in producing intermediate-sized ones. Disruptive selection should apply equally to individual differences as adaptations. We should expect to see a number of human 'types' rather than a continuous normal distribution. (Indeed the picture is even more complicated since humans vary not just on one trait but on five simultaneously, creating a near infinite range of individual differences.) The normal distribution of personality variation does not suggest 'types' but a 'continuum' resulting from allelic variation over a number of genetic loci. Personality variations are expected to be polygenic in origin and, under selective neutrality, 'genetic variation can be expected to be mainly additive'. If 10 coins (gene loci) are each flipped simultaneously the likely outcome is a normal distribution—the probability of 10 heads (extreme introversion) or 10 tails (extreme extraversion) is extremely low. (True, a similar distribution might be seen as a 'snapshot' under balancing selection. But that snapshot would have to be taken at precisely the time or place at which the forces favouring the two strategies were momentarily in perfect balance.)

If there is to be a search for function, I agree with the authors that we have been uncritical in taking the Big Five as the compass. These traits emerged from people rating themselves in terms nominated by another set of people (psychologists). The extent to which such traits are significant for molecular genetics or evolution—as opposed to

human social perception—is debatable. Instead, the authors suggest that the search for adaptive significance might begin by identifying endophenotypes (specific biopsychological processes). Korte, Koolhaas, Wingfield, and McEwen's (2005) work provides a recent example of this approach. Across a range of species, they have identified two responses to stressors. 'Doves' show a strong HPA response but a lower SAM response while Hawks show the reverse pattern. These differences have been linked 'upstream' to genetic polymorphisms and neurotransmitter activity, and 'downstream' to manifest behaviours (fear, aggression, sensitivity to environmental threats).

   Nonetheless, I find the case for balancing selection suspicious on two counts. First, as the authors note, the chief source of selection operating on humans has been conspecifics. While environments may show rapid and fluctuating alterations over time and space, this hardly seems to characterise human interactions. Why would there be sexual selection for anxiety or introversion at one point in time or history, but preference for the opposite qualities at another? Why would anyone at any time or place want an ally that was unreliable and duplicitous? Such questions bring me to my second point, the ubiquitous just-so story. The costs and benefits of extraversion, while providing a lively topic for speculation, will not be solved by 'much more research' in so far as we lack access to the variable social and environmental niches which putatively supported them. In place of stories, what Penke et al. (this issue) have very usefully provided is a profile, linking behaviour genetic to population genetic parameters, which can guide our search for the evolutionary relevance—or irrelevance—of personality.

An Evolutionary Ecologist's View of How to Study the Persistence of Genetic Variation in Personality

NIELS J. DINGEMANSE

Centre for Behaviour and Neurosciences, University of Groningen, The Netherlands
n.j.dingemanse@rug.nl

Abstract

Personality is commonly regarded to involve either 'correlations among behavioural traits' or 'consistent individual differences in behaviour across contexts'. Any evolutionary explanation for the existence of genetic variation in personality must therefore not only address why genetic variation in single behavioural traits is maintained but also why behavioural traits are correlated, and why individuals show limited behavioural plasticity. Copyright © 2007 John Wiley & Sons, Ltd.

Penke et al. (this issue) propose a framework for studying genetic variation in personality. Their framework is important because it outlines why genetic variation in behaviour—a key characteristic of personality—might exist, but is, yet, incomplete. In this commentary I outline why.

   Although many definitions of personality exist (see Réale, Reader, Sol, McDougall, & Dingemanse, 2007, for a recent overview), it is commonly agreed that personality involves either 'genetic correlations among behavioural traits expressed in different environments' (when viewed from a 'character state' perspective; Via & Lande 1985; Via, Gomulkiewicz,

de Jong, Scheiner, Schlichting, & van Tienderen, 1995), or 'consistent individual differences in behaviour across contexts' (when viewed from a 'reaction norm' perspective; de Jong, 1995; Via et al., 1995). Viewed from a character state perspective (which is not explicitly discussed by Penke et al., this issue), genetic variation in personality therefore does not exist if genetic correlations among behavioural traits are all very weak or absent (Figure 1a) but does exist if genetic correlations are tight (Figure 1b). Viewed from a reaction norm perspective, genetic variation in personality does not exist when both the gene × environment interaction (G × E) between a behaviour expressed in different environments is very strong (Figure 1c) *and* the cross-environment genetic correlation is weak (as illustrated in Figure 1a) but does exist if a trait is both heritable in different environments *and* exhibits no (or very weak) G × E (Figure 1d; resulting in a tight cross-environment genetic correlation as shown in Figure 1b). Consequently,
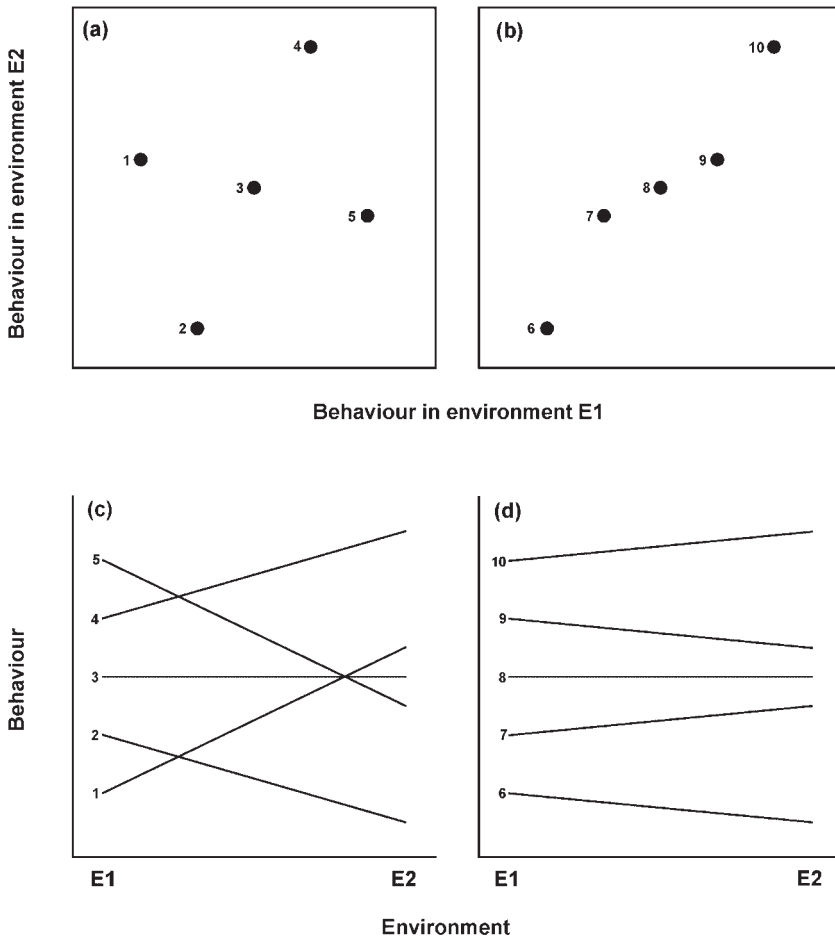

Figure 1. Graphical illustration of genetic variation in personality as viewed from (a–b) a character state approach (both panels plot the breeding values of two behaviours) or (c–d) a reaction norm approach (both panels give breeding values of the same behaviour expressed in two different environments). Note that the two approaches are essentially two sides of the same coin: (a) and (c) depict the same fictional data as do (b) and (d), where each genotype (number) is given either as a dot (a, b) or a line (c, d). Personality does not exist in (a) and (c) but does exist in (b) and (d). Note that the correlation between the breeding values for the behaviours plotted in (a) and (b) represents their additive genetic correlation (Lynch & Walsh 1998).

understanding why genetic variation in personality exists requires insight in evolutionary mechanisms that either (i) simultaneously promote persistence of genetic variation in single behaviours *and* genetic covariation between behavioural traits (Figure 1b) and/or (ii) simultaneously promote persistence of genetic variation in a single behaviour *and* the existence of limited plasticity of the behaviour across contexts (Figure 1d). Penke et al.'s framework addresses mechanisms explaining genetic variation in a single trait; it does not address adaptive explanations for why traits might be correlated or why individuals show limited plasticity.

An evolutionary ecologist's research agenda for studying genetic variation in personality would, depending on the chosen approach, thus include the following topics. If one adopts the character state approach, a fruitful agenda would start by (i) measuring multiple behaviours on individuals with known pedigree relationships, (ii) revealing the genetic structure of personality by estimating additive genetic variances (V_A) and covariances (so-called **G**-matrix) from these data (see Lynch & Walsh, 1998), (iii) measuring the fitness consequences of personality where selection pressures favouring correlations among traits should explicitly be examined (Dingemanse & Réale, 2005; this crucial step is missing from Penke et al.'s framework) and finally (iv) predicting how the **G**-matrix might evolve in response to selection (Steppan, Phillips, & Houle, 2002)—instead of using solely verbal arguments. Such data would reveal whether a combination of balancing and correlational selection does indeed maintain genetic variation in personality.

If one adopts the reaction norm approach, the research agenda would start by explicitly considering that reaction norms are characterised by slopes and intercepts that might both evolve (de Jong, 1990). In contrast, Penke et al. seem to regard personality as a collection of fixed reaction norms that cannot evolve. A fruitful approach would continue by (i) measuring behaviour of the same individuals over multiple contexts (using a set of individuals with known pedigree relationships), (ii) obtaining estimates of intercepts and slopes for each individual that would then be used to estimate V_A in both parameters (Lynch & Walsh, 1998), (iii) measuring how the intercept and slope of an individual (and potentially their interaction) affect fitness (Scheiner & Berrigan, 1998; van Tienderen, 1991) and finally (iv) assessing whether the observed selective pressures would indeed maintain genetic variation in personality. Evidence for disruptive and/or fluctuating selection on intercepts in combination with stabilising selection on slopes would provide evidence in favour of Penke et al.'s balancing-selection hypothesis.

Penke et al. simply invoke constraints on plasticity as an explanation for consistency of behaviour over contexts. Recent studies, however, show that genetic correlations (like those that cause personality) are rarely fixed and can easily change sign across populations or environments within populations (Sgro & Hoffmann, 2004). Penke et al.'s constraints view might thus prove invalid. Furthermore, even genetic correlations that are highly preserved (i.e. exist in many taxa) can often easily be broken by means of artificial selection (Beldade, Koops, & Brakefield, 2002), suggesting that genetic correlations (like those that cause personality) might instead have evolved because natural selection favoured associations between traits (Bell, 2005; Dingemanse & Réale, 2005). The fact that individual variation in behaviour exists in a wide range of taxa (Gosling, 2001; Réale et al., 2007) should therefore not necessarily be viewed as evidence in favour of the view that constraints on behavioural organisation hamper adaptive evolution of behaviour. Instead, natural selection may have favoured the evolution of limited behavioural plasticity while simultaneously maintaining individual variation (Dall, Houston, & McNamara, 2004; McElreath & Strimling, 2006).

Consilience is Needed, and Consilience Needs Bipartisan Expertise

HARALD A. EULER

Institute of Psychology, Department of Economics, University of Kassel, Germany
euler@uni-kassel.de

Abstract

Despite a common overarching home of biology, evolutionary psychology and behaviour genetics have not fostered mutual exchange. The paper combines expertise in evolutionary genetics and personality theory with didactic skill and makes a strong argument for two mechanisms of evolutionary genetics to explain the persistence of genetic variation in intelligence and personality, thus contributing considerably to inter-disciplinary consilience. Copyright © 2007 John Wiley & Sons, Ltd.

A few years ago the late Mealey (2001) likened evolutionary psychology and behaviour genetics to two sisters of about the same age. Both occupy two different niches within the family with different interests and optimal resource extraction: What is chaff to one sister is wheat to the other. Evolutionary psychology tells stories about human universals and trashes individual differences, whereas behaviour genetics cherishes just these differences. Sister behaviour genetics has exuberantly been telling an old aunt called personality psychology exciting new findings, like that genes are important and that the magic of family influence is just an urban legend. The aunt dislikes genes and considers it improper to talk about such infamous things in front of others. But the other sister also lacks good manners because she retells the kind of stories which the aunt had overheard in her childhood from old relatives called Charles and Herbert, and these stories were considered off-limits as she had learned when she got a bit older. As all three women vie for outside attention to their good looks there is less than complete harmony despite the thick-blooded family ties. Godfather Edward Wilson, a big-name salesman for a cure-all called consilience, occasionally drops in and recommends his remedy.

The authors of the target paper offer a remedy, one with several active ingredients. There is brief but excellent to-the-point primer of genetic variation, optimal for the reader interested in personality theories but not an updated expert in evolutionary genetics. Second, the paper reviews the unsatisfactory previous attempts to reconcile Fisher's dictum that selection winnows out alleles with highest fitness, thus removing all genetic variation in the long run, with the observation of heritabilities galore. The previous conclusion by Tooby and Cosmides (1990) that heritable variation signals a lack of adaptive significance has been indigestible for most evolutionary psychologists because it

tried to entice us to ignore individual differences and thus forget about personality as a worthwhile subject from an evolutionary perspective.

Third and most important, the authors delve into the evolutionary genetics of personality and argue skilfully and persuasively why, of the various possible genetic mechanisms, mutation-selection balance is the prime candidate to explain genetic variance in general intelligence, and balancing selection by environmental heterogeneity the prime candidate to explain variance in personality traits. To bolster these arguments, predictions are derived from the theory of evolutionary biology and evolutionary genetics, currently available data are mustered and clear judgements are offered. Suddenly, several loose ends in our theorising might become connectable: different heritability estimates and different proportions of non-additive genetic variance for general intelligence and personality dimensions; different impact of shared environment on intelligence and on personality dimensions; inbreeding depression and outbreeding elevation for intelligence but not for personality; generally higher heritability for sexually selected than for naturally selected traits.

Most helpful to evaluate systematically the possible genetic mechanisms in genetic variation is Table 1 in Penke et al. (this issue). Admittedly, the entries are ordinal at best and vague at worst, but they suffice to navigate the reader through the sometimes demanding subject matter and provide a different vantage point, and they suffice to evaluate by comparative evidence. The watershed model has its own charm and merits, not the least because it may help to reconcile approaches in evolutionary anthropology with those in evolutionary psychology. The former insists on fitness measures and settles as far downstream as possible. The latter, unless they commit betrayal of their discipline, have to find their niches upstream along tributaries. The model makes salient that both approaches are working with the same body of water, in fact with the same water.

In the last decade evolutionary approaches and adaptionistic theorising have finally gained increased acceptance within the psychologies of continental Europe (Euler & Voland, 2001). The target paper exemplifies for personality psychology how promising and gainful an inter-disciplinary approach with bipartisan expertise can be and how much it can contribute to consilience of estranged disciplines.

Genetic Variance and Strategic Pluralism

AURELIO JOSÉ FIGUEREDO and PAUL GLADDEN

Department of Psychology, University of Arizona, Tucson, USA
ajf@u.arizona.edu, pgladden@email.arizona.edu

Abstract

Penke et al. (this issue) have written a provocative paper on the evolutionary genetics of personality, ascribing the maintenance of genetic variation in personality to balancing selection and in cognitive abilities to a balance between mutation pressure and directional selection. Some of the theory and evidence presented appear supportive, but both the theoretical predictions and the supporting empirical evidence remain tentative. Copyright © 2007 John Wiley & Sons, Ltd.

Penke et al. (this issue) (PDM) have written a provocative paper on the evolutionary genetics of personality. The ideas presented are extremely exciting and worth further

research, but we have certain reservations about some of the conclusions drawn from the existing body of theory and evidence.

After making and evaluating differential predictions about the expected structure of the genetic variability in traits that would be maintained by neutral selection, balancing selection and mutation-selection balance, respectively, PDM draw the following three major conclusions: (1) that genetic variability in personality traits is maintained by balancing selection, (2) that genetic variability in cognitive abilities is maintained through mutation-selection balance and (3) that neutral selection does not adequately explain the observed genetic variability in either personality or cognitive ability. While we are inclined to agree with them on all three major points, although perhaps for different reasons, we found that some of the logical inferences made in PDM's argument were difficult to follow and require further clarification. The problem stems, in part, from ambiguities and incomplete equivalences in the terminology used by PDM and in relation to the original sources cited.

PDM argue that there are high absolute values of additive genetic variance in traits closely related to fitness (termed 'fitness traits' by Merilä & Sheldon, 1999) because fitness and life-history traits are potentially affected by mutations at a large number of genetic loci. Therefore, even though fitness traits might be under strong directional selection, a large absolute value of additive genetic variance can be maintained by the opposing action of mutation pressure. Thus far, we agree with them. However, PDM also assume that fitness-relevant traits are necessarily and exclusively subjected to directional selection as opposed to balancing selection. In contrast, we argue that any traits under balancing selection must also be closely connected to resultant fitness. For example, as PDM note, balancing selection has been proposed by ourselves and others as an explanation for the maintenance of genetic variability in life-history traits. Although life-history traits are definitely relevant to fitness, alternative reproductive strategies might nonetheless have equal fitness payoffs, especially within complex social ecologies.

PDM equate 'downstream' traits with 'fitness' traits. Because PDM argue that fitness-relevant downstream traits are subjected to a balance between mutation pressure and directional selection, they go further to imply that downstream/fitness traits are also less likely to be subject to balancing selection, as indicated by their high levels additive genetic variance. We do not understand why this must necessarily be so. The concept of a downstream trait with high fitness relevance does not seem useful to us for distinguishing between directional and balancing selection. The foundation upon which to make strong differential predictions about the structure of genetic variability between mutation-selection balance and balancing selection therefore seems fragile. Similarly, it is unclear to us why additive genetic variance should tend to be depleted in traits under balancing selection.

PDM's multiple conflation of downstream traits, fitness traits and life-history traits with strong and exclusively directional selection is troubling because human life-history strategy has been shown to be significantly related to personality traits and could therefore be partially under the control of balancing selection, as PDM acknowledge (Figueredo, Sefcek et al., 2005; Figueredo, Vásquez et al., 2005). They cite us as observing that a 'fortuitous side-effect' of variation in life-history strategy and personality 'is that such variation reduces within-group and between-group competition by allowing individuals and groups to fill different socio-environmental niches'. In fact, the predictions that we made were stronger and more specific: (1) that selection for variation in life-history strategy may ultimately be the principal driving force behind selection for variation in personality and (2) that partial release from intraspecific competition within social groups is the evolved adaptive function of this variation, not merely a 'fortuitous side-effect'. In a

separate twin study (Figueredo, Vásquez, Brumbach, & Schneider, 2004; Figueredo et al., 2006), we have also shown a substantial genetic correlation ($r_g = .78$) between a higher-order personality factor and a multivariate composite of a wide array of cognitive and behavioural indicators of life-history strategy. Furthermore, we have reported a substantial broad-sense heritability ($h^2 = .65$) for this general life-history (K) factor. Unfortunately, the twin study did not contain associated data from other (non-twin) siblings, so we were not able to estimate the relative proportions of additive and non-additive genetic variance.

PDM state that significant absolute and proportional levels of non-additive genetic variance indicate that a given trait has had a recent history of selection. We are unclear as to what type of selection is meant here, but we suspect that directional selection is implied. PDM also state that high levels of non-additive genetic variance (specifically dominance variance) are observed in personality traits and that this variability is only explainable by balancing selection because dominance variance levels are expected to be in the middle range for traits under mutation-selection balance, but higher under balancing selection. Since traits with a recent history of selection and traits under balancing selection are both predicted to have significant levels of non-additive genetic variance, we are unclear what, if any, differential predictions there are about the levels of non-additive variance in traits under directional versus balancing selection.

In sum, although we sympathise with their final position, we are skeptical about the apparent certainty with which PDM present their differential predictions as purportedly reliable criteria for discriminating between the alternative mechanisms for maintaining genetic variability. In the literature cited by PDM (e.g. Crnokrak & Roff, 1995; Merilä & Sheldon, 1999; Stirling, Réale, & Roff, 2002), these are treated more tentatively as working hypotheses, for which the evidence is often equivocal, than as empirically well-substantiated observations. In their response to these commentaries, PDM should therefore: (1) better elucidate the inferential steps they made in reaching their conclusions regarding the ultimate causes underlying the maintenance of genetic variability in personality and cognitive abilities and (2) specify the empirical evidence supporting these conclusions, explicitly distinguishing empirical data from theoretical conjecture.

Beyond Just-so Stories Towards a Psychology of Situations: Evolutionary Accounts of Individual Differences Require Independent Assessment of Personality and Situational Variables

DAVID C. FUNDER

Department of Psychology, University of California, Riverside, USA
funder@ucr.edu

Abstract

Evolutionary theory is perhaps better used as a brake on theory than as a source of 'just-so' stories of the origin of characteristics. The target paper admirably employs evolutionary theory to test competing models of the maintenance of individual differences. Areas needing further development include separating personality from situational variables, rather than confounding them, and developing a psychology of situations. Copyright © 2007 John Wiley & Sons, Ltd.

Many personality and social psychologists are skeptical about the relevance of evolutionary theorising to psychology. Why? It is not because they doubt the general truth of evolutionary theory. Rather, the skepticism stems from the proliferation within evolutionary psychology, especially in its early days, of 'just-so' stories reminiscent of the tales by Rudyard Kipling that explained how the whale got its throat, how the camel got its hump and so forth. Kipling invented these stories by observing interesting aspects of nature and then letting his imagination run wild. Evolutionary psychologists have sometimes proceeded the same way, with the result that they seemed ready to explain *anything* from preference to salty foods to spousal murder. While nearly all the evolutionary stories were interesting, and an (unknown) number of them may even be true, their sheer number and variety can feed rather than repel skepticism, and help to fuel wide ranging critiques (e.g. Gould & Lewontin, 1979). The basic problem with these stories, besides their number, is their origin in a strategy of beginning with a known phenomenon and reasoning backwards to a cause—not unlike Kipling's.

But there is another way to use evolutionary theory. Rather than as a source of limitless explanatory theories for the origin of anything, evolutionary psychology can profitably be used as a *brake* on theorising. If one accepts that the diversity of life, including human psychological life, is a product of evolutionary processes, then certain other theoretical positions become less tenable. For example, some versions of psychoanalysis posit the existence of a built-in drive in all persons towards death and destruction, including self-destruction. Is this idea plausible from an evolutionary perspective? For a very different example, some psychologists who study thinking and problem-solving argue that the human mind is fraught with basic design flaws. The many experiments demonstrating how people may systematically and grossly distort certain kinds of information are clever and sometimes entertaining, but is the idea of an information processing system flawed at the level of its basic design evolutionarily plausible (see Funder, 1987, 2000; Gigerenzer, Todd, & the ABC Research Group, 1999)?

The target paper follows this second approach, evaluating three models of the maintenance of individual differences in psychological attributes according to evolutionary plausibility. This approach leads the authors to several interesting conclusions, including a compelling description of the basic difference between attributes of ability and personality, a distinction that has been difficult to make on other grounds. Of particular interest is their explanation of how individual differences in personality can be maintained through the simultaneous existence of environments in which different levels of different traits are most adaptive. For example, exuberant extraversion might be adaptive in an environment that is abundant and relatively risk-free, whereas a more restrained introversion might promote survival under impoverished or dangerous circumstances. While on the whole their analysis is compelling, further development is needed in two respects.

One is the authors' touting of 'individual reaction norms' as preferable to main-effect personality traits. Individual reaction norms are described as similar to Mischel and Shoda's (1995) CAPS model in which each individual's personality is described in terms of his or her if-then connections between situational stimuli and behavioural responses. This model has several shortcomings, including its startling resemblance to Watsonian (pre-skinner) S-R behaviourism, the general statistical weakness of interactions compared to more robust main effects (which the target paper mentions) and the dilemma the model presents between characterising individuals in terms of idiographic patterns (one for every living person) or boiling them down into a relatively small number of 'types'—a problematical approach at best (see Asendorpf, 2002; also Funder, 2006, in press).

For present purposes the most important difficulty with individual reaction norms, as defined, is that they may contradict the purpose for which they are advocated. The authors persuasively argue that personality traits can be differentially adaptive under different circumstances. Thus, to repeat their most simple example, an extraverted person is well-suited to take advantage of a safe environment while an introvert may survive better in a dangerous one. But notice how this example—and others presented in the paper—assumes a *main effect* of extraversion–introversion, not an interaction with safety-dangerousness. An individual's degree of extraversion–introversion is a general or average tendency and individuals at both ends of the dimension continue to exist because each style is adaptive in different environments. But if instead traits are conceived as built-in interactions, why not just evolve a tendency to be extraverted if the situation is safe and introverted if dangers are afoot? The explanation of the survival of individual differences in personality traits as a result of their varying adaptive implications in different environments only works when the traits are thought of as main effects rather than interactions. More generally, the concept of a person-environment interaction is clearer and more analytically tractable when the two constituent terms are kept separate (Funder, in press; Reis, 2007).

A second and related observation is that further research on the interactions between traits and/or genotypes on the one hand, and environmental properties on the other, is at present sorely handicapped by the lack of means for conceptualising and measuring environments. Situations as presented in expositions of the CAPS model, for example, are almost (but not quite) always described hypothetically, as for example, 'Situations 1–6' (Mischel & Shoda, 1995, p. 247). This kind of labelling is presumably promissory to someday providing concrete descriptions, and dimensions for description, of situations. The description of psychological environments (or situations) is perhaps even more important to fulfil the potential of the analysis in the present paper, to describe the circumstances under which different traits, or even aspects of incipient psychopathology, are more and less adaptive. So far we have a small number of very interesting examples, some of which are hypothetical. What we need next are data, and means to gather those data. We need new measuring tools, and a psychology of situations (Wagerman & Funder, 2006).

Life-History Theory and Evolutionary Genetics

STEVEN W. GANGESTAD

Department of Psychology, University of New Mexico, Albuquerque, USA
sgangest@unm.edu

Abstract

Penke et al. (this issue) argue that evolutionary genetics offers important insights into the fundamental nature of personality—how people adaptively adjust to their life circumstances in particular ways, as well as failures to adapt. I strongly endorse this enterprise. It is particularly promising, I suggest, when embedded within life history theory (LHT), a broad evolutionary framework to understand selection on organisms. Copyright © 2007 John Wiley & Sons, Ltd.

Almost a century ago, Fisher (1918) famously showed how, given Mendelian inheritance, quantitative variation can be partitioned into forms of genetic and environmental variance, thereby laying foundations for both quantitative genetics (e.g. heritability estimation) and evolutionary genetics. Whereas, within biology, these topics are intertwined (e.g. Crow, 1986), most quantitative behaviour geneticists and personality psychologists have shown little interest in evolutionary genetics. I applaud Penke et al.'s efforts to remedy this neglect.

Many biologists (e.g. Houle, 1991) contextualise evolutionary genetics in a broad view of selection on organisms, life history theory (LHT). LHT has deep roots in evolutionary biology (for an overview, see Kaplan & Gangestad, 2005) and now pervades adaptationist theoretical analysis (e.g. of sexual selection: e.g. Kokko, Brooks, Jennions, & Morley, 2003; biological signals: Getty, 2006; immune function: McDade, 2003; patterns of aging: e.g. Kirkwood, 1990). My commentary touches on how, jointly, LHT and evolutionary genetics can shed light on the adaptive and maladaptive nature of personality variants.

Life-history allocations. Organisms are designed by selection to harvest energy and convert it into fitness-enhancing activities. Within lineages and their niches, designs that do so most proficiently are selected (e.g. Charnov, 1993). A problem that designs must solve is how to efficiently allocate resources to the development and operation of the organism's many fitness-enhancing features. At optimum performance, the marginal value of allocation (effectively, the effect on fitness of the last unit of allocation) to each feature should be equal. (Otherwise, reallocation could increase fitness.) Optimal allocation changes across the lifecourse and with conditions (e.g. skeletal growth and brain development may be particularly important early in life, allocation to reproductive traits may anticipate the end of growth, optimal allocation to immune function increases with infestation). Selection accordingly shapes organisms' characteristic life histories.

Implications for directional and stabilising selection. Virtually no feature comes for free; a feature's development and maintenance entails opportunity costs. Hence, one can overspend even on 'good' traits (e.g. brain function supporting IQ, immune function, DNA repair). Energy-rich diets in modern cultures don't overcome the problem, as metabolic and developmental processes evolved in leaner conditions impose constraints on proficient allocation of resources in real developmental time. Hence, most traits are (at least partly) under stabilising selection; intermediate trait values are favoured, whereas extremes are disfavoured. Consider height. Extreme tallness or shortness may be selected against (see Nettle, 2002), partly because really tall people may have overallocated to growth, whereas short people may have underallocated to it.

Mutations typically diminish fitness because they reduce the proficiency with which organisms garner and allocate resources, and in at least a couple of ways. Mutations may produce inefficiencies in processes that build fitness-enhancing traits. They can also yield non-optimal allocation. Some extreme variants on traits under stabilising selection (e.g. extreme tallness and extreme shortness) reflect mutation-induced non-optimal allocation. Some mutations lead to overallocation to the trait, others to underallocation (e.g. Houle, 1991).

Mutation-selection balance, then, doesn't only apply to traits under directional selection (see Penke et al., this issue); it can also explain genetic variation on traits under stabilising selection (e.g. Crow, 1986). The latter tend to have *low* additive genetic coefficients of variation (CVAs), despite high h^2 (Houle, 1992; Pomiankowski & Møller, 1995). The

CVA of height is generally less than CVAs of fitness traits (e.g. Miller & Penke, 2007). Brain size too possesses a low CVA (Miller & Penke, 2007). And some personality variation may be maintained by mutation-selection balance under stabilising selection.

For some traits under (partial) stabilising selection, however, the optimum value may be higher than the mean because, once again, some mutations (and other fitness-reducing events, including environmental ones) may reduce ability to develop fitness-enhancing traits. A classic example is clutch size in birds: Although both small and very large clutch sizes are disfavoured (the latter because they overstretch parents' abilities to care for offspring), the fittest parents can produce clutches larger than average (see Parker & Begon, 1986). Similarly, optimal height may be greater than average (Nettle, 2002). PDM imply that IQ has ancestrally been linearly related to fitness, but the low CVA of brain size (partly reflecting IQ) suggests it may be like avian clutch size: partly under stabilising selection, with the optimum greater than the mean, but less than the high end of the range in IQ.

Reactive heritability. Selection may design organisms to adjust their developmental and behavioural strategies based on their particular circumstances, should those circumstances affect the payoffs of strategies. Selection accordingly shapes phenotypic plasticity and norms of reaction (Houston & McNamara, 1992). Plasticity explains, however, not only to environmental variation in traits. If circumstances themselves reflect genetic variation (e.g. compromises in condition due to mutations), so too do outcomes of strategy adjustment. PDM allude to this phenomenon, albeit implicitly, when they discuss the idea that costly, sexually attractive signals evolve to reflect genetic variance in condition. When allocating optimally, individuals in best condition allocate more resources to these traits than do individuals in poorer condition (Rowe & Houle, 1996).

More generally, in long-lived species such as humans, individuals in poor condition may invest proportionately more in survival and less in reproductive traits that entail costs on immediate survival (e.g. Ellison, 2003). Accordingly, heritable variation in condition may translate, through adaptive adjustment, in differences in patterns of a range of phenotypic traits. For example, one life-history view of the endocrine systems in which female oestrogen and male testosterone are involved is that they have been shaped to adaptively modulate allocation to reproductive traits (e.g. female oestrogen promoting current fertility and allocation to gynoid fat deposition; male testosterone promoting traits ancestrally important in mating competition, e.g. muscularity; see Ellison, 2001, 2003). Some variation in traits affected by reproductive hormones, then, may reflect condition-dependent strategy choice, not allelic variation in genes directly affecting hormone production or receptor densities. (Perhaps relevant is the recent finding that prepubertal boys of average IQ tend to have higher testosterone levels than boys of *either* very low *or* very high IQ; Ostatníková et al., 2007.)

Sum. By itself, heritability estimation reveals little about core personality, 'psycho-physical systems that determine (an individual's) unique adjustment to his environments' (Allport; cited by Penke et al., this issue). As these authors make clear, identifying the evolutionary forces responsible for variation *can* yield insights into the nature of adaptation and maladaptations represented by personality variants. The enterprise may be particularly promising when embedded within a life-history framework.

Behaviour Genetics' Neglected Twin: Gene-Environment Correlation

KERRY L. JANG

Department of Psychiatry, University of British Columbia, Vancouver, Canada
kjang@interchange.ubc.ca

Abstract

The target paper posits that the driving force behind balancing selection is gene-environment interaction (G × E) that describes environmental control of genes. It is argued that G × E is insufficient to maintain genetic variability and that the concept of gene-environment correlation or genetic control of the environment leads to different conclusions regarding mental illness and hierarchical personality models. Copyright © 2007 John Wiley & Sons, Ltd.

Penke et al. (this issue) make a strong, logical argument that observed individual differences in personality are a reflection of genetic variability caused by balancing selection. Their argument relies heavily on the behavioural genetic concept of gene-environment interaction (G × E) and recent empirical research that has shown to exert a major influence in personality and psychopathology. G × E occurs when genotypes are differentially expressed when exposed to varying environmental conditions. It is argued that such genetic variability is maintained in a population because it confers fitness advantages by allowing organisms to adaptively react to different environmental conditions or—to use their term—niches.

However, the role of G × E as the primary mechanism for balancing selection is insufficient to explain genetic variability. This becomes clear when their arguments are used to try to explain mental illness and the genetic basis underlying the hierarchical structure of personality. Beginning with mental illness, they argue that mental illness is a consequence of genetic variants no longer fitting environmental conditions so that ' . . . modern societies produce mismatches between heritable temperaments and available niches'.

Explaining Mental Illness. It follows that mental illness exists simply because humans cannot reproduce fast enough to keep up with environmental change and these variants survive because they have not had a chance to be selected out of the population. However, it can also be argued that such genotypes are maintained due to improvements to health care and because attitudes towards the mentally ill ensure these individuals survive to reproduce. This is a form of gene-environment interplay called gene-environment correlation (r_{GE}).

Gene-environment correlation refers to the process in which underlying genetic factors influence the probability of exposure to specific events—simply put, the genetic control of exposure to the environment. Plomin, DeFries, and Loehlin (1977) discussed three general types: passive, active and reactive. Passive genotype-environment correlation occurs because children share heredity and environments with members of their family and can thus passively inherit environments correlated with their genetic propensities. Reactive genotype-environment correlation refers to experiences of the child derived from reactions of other people to the child's genetic propensities. Active genotype-environment

correlation is known as 'niche building' or 'niche picking' (Plomin, DeFries, & McClearn, 1990, p. 251) and refers to individuals actively selecting or creating environments commensurate with their underlying genetic propensities.

Assuming that some form of r_{GE} exists, its operation maintains genetic variability because these genes are operating in an active, passive or reactive manner to create all of the varied environments required for expression. This also helps to clarify some evolutionary psychological theorising on mental illness that attempts to identify fitness advantages for mental illness. Under this model, mental illness has no fitness advantages and exists as a true pathology. In short, r_{GE} creates 'stably unstable' environments that would maintain genetic variability for psychopathology. It should also be noted that for normal personality and behaviour, r_{GE} provides a powerful alternative explanation for genetic variability underlying this range of behaviour.

Hierarchical Structure of Personality. What influence does $G \times E$ have on the covariance of traits, and by extension, hierarchical models of personality? The authors suggest that the context-dependent nature of two traits can be used to determine if they are in a pleiotrophic relationship—indexed by a positive genetic correlation (r_G)—that results when both respond within the same general reaction range when exposed to the same environments. If they do not share a common genetic basis, then the two traits can react in opposite ways—resulting in a negative genetic correlation. Thus, the absence of sign or valence changes across environments is a necessary condition for the existence of superordinate personality domains.

This is problematical for two reasons. First, what is important to estimating pleiotrophy—that the authors consider the central basis of superordinate traits—is not the change in valence but rather the magnitude of r_G. A zero r_G is far more informative regarding the presence of shared genes than the change in valence. Moreover, demonstrating no change in the valence of r_G across environments as a necessary requirement for pleiotrophy is really an artificial and ecologically invalid consequence of hypothesised reaction ranges whose breadths are not broad enough to encompass zero as the midpoint.

Second, basing decisions on which traits are included as part of a domain (a version of the classic factor definition problem) based on reaction ranges may lead to erroneous conclusions for the reasons outlined above and because of potentially unaccounted for r_{GE} effects that can be misread as $G \times E$ (see Purcell, 2002). Finally, the authors' theory assumes that personality hierarchy is imposed by the action of genes shared across traits. Through the mechanism of r_{GE}, however, environments conducive to maintaining a particular hierarchy also play a role.

Recognising the interdependence of genes and the environment (see Rutter, 2007) and the ability to specify mechanisms such as $G \times E$ as a driver of balancing selection is a major step forward. However, there are other effects, such as r_{GE}, that need to be incorporated into the theory that balancing selection maintains the genetic variability that we observe as individual differences in behaviour.

Don't Count on Structural Pleiotropy

WENDY JOHNSON[1,2]

[1]*Department of Psychology, University of Edinburgh, UK*
[2]*Department of Psychology, University of Minnesota—Twin Cities, USA*
wendy.johnson@ed.ac.uk

Abstract

Penke et al. (this issue) address the evolution of personality, articulating many insightful and provocative ideas. They do not, however, give enough attention to the role of G-E correlation in the processes they outline. Thus they underestimate the difficulty of establishing the existence of structural pleiotropy and overestimate its ability to help us in understanding the development of personality. Copyright © 2007 John Wiley & Sons, Ltd.

In their insightful and provocative paper, Penke et al. (this issue) use the term 'G × E interaction' to refer to the adaptive fit of an organism to its environment. They describe this adaptive process as being comprised of natural selection, or relative reproductive success, and phenotypic plasticity, or the potential for a given genotype to produce different phenotypes in different environments. They note that phenotypic plasticity is not complete, even for behavioural traits: the organism cannot adapt perfectly and instantly to all environmental demands because the cues to optimal adaptation provided by the environment are too unreliable. They point out that, to the extent that environmental cues are reliable, natural selection acts over time to limit phenotypic plasticity, and suggest that what phenotypic plasticity remains largely reflects genotypic differences that persist in the population. This is, of course, possible, but the very unreliability of environmental cues makes it unnecessary. The same genotype could respond differently to different environmental circumstances simply because there are enough ways in which the environment varies that natural selection cannot operate to remove the phenotypic plasticity.

In population genetics, the term 'G × E interaction' has a specific technical definition as genetic control of sensitivity to different environments, or, equivalently, environmental control of expression of genetic influences (Kendler & Eaves, 1986). The adaptive fit of an animal (human or otherwise) to its environment is always more than this: the animal has some choice of exactly what environment it faces. This is captured by another population genetics term, 'G-E correlation', which refers to genetic control of exposure to different environments, or, equivalently, the environmental control of gene frequency (Kendler & Eaves, 1986). For example, when food is scarce in one area, animals will expand the range over which they search for it. There may be genetically influenced individual differences in the extent to which this is true, but the animal that wanders furthest in search of food may have the same reproductive success as one that does not wander as far but has the metabolic efficiency to survive better on less food. Because adaptation involves both G × E interaction and G-E correlation, it would be helpful to use a term that encompasses both. 'G-E transaction' is one such term.

G-E correlation can be completely passive, as when parents transmit both genetic influences and environmental circumstances to their offspring. But often G-E correlation is active: the individual either directly seeks an environment or behaves in a way that elicits

certain kinds of environmental responses. As with phenotypic plasticity, individuals cannot select their environments completely at will. Still, the facts that particular genotypes can produce more than one phenotype and that individuals can select their environments to some degree mean that G × E interaction and G-E correlation are often closely inter-related. This relation takes place because proper measurement of the environment often involves recognition of individual differences in response to that environment, individual differences that generally show genetic influence.

For example, measurement of the environment when food is scarce would mean recognising that some animals are more affected by the relative lack of food than others, perhaps by measuring individual levels of caloric deprivation. But animals with relatively lower levels of metabolic efficiency will be more motivated to expand the range over which they search for food, creating at least statistical if not genotypic pleiotropy between metabolic efficiency and food-seeking range among those animals. Natural selection will tend to have its greatest effects on those who have both low levels of metabolic efficiency and low tendencies to explore in search of food. Genetic influences on food-seeking range will be expressed most strongly among those with low metabolic efficiency, a G × E interaction. The G-E correlation will also be greatest among these animals, because of the selection process involved in food-seeking range. The ways in which G-E transactions are related are discussed in detail in Johnson (2007).

Penke et al. (this issue) correctly point out that phenotypic plasticity is limited because the environment does not reliably signal the most adaptive behavioural strategy. It is this unreliability of environmental cues in the presence of phenotypic plasticity that implies that genetic influences on a trait do not necessarily mean genotypic differences at particular loci. This is because, for any one gene in a genotype, the other genes function as part of the environment. In combination with the ability of an animal to select its environment, this has important implications for the norm of reaction model Penke et al. (this issue) articulate. The norm of reaction concept was developed with organisms under controlled breeding and environmental conditions, and in naturalistic settings the concept breaks down in important ways. For example, in the simplified terms of Penke et al.'s Figure 2b, people with genotype A may avoid environment Z completely, and people with genotype B may be over-represented there. This implies that genetic correlations observed across the environmental range may not reflect similarities and differences in genotype in any predetermined, formulaic way even when the correlations do not change sign.

Penke et al. (this issue) suggest that structural pleiotropy or functional, physiological or developmental links between genetic influences on different traits that constrain independent phenotypic expression of the traits in all environments, may help us to understand personality development. The ability to select our own environments makes it likely that structural pleiotropy is rare for personality traits, and that it is very hard to be sure that we have observed it even when it does exist. This may explain the relative weakness of the structural hierarchy of personality traits that depends on structural pleiotropy, as indicated by the genetic correlations between factors of the Five-Factor Model (FFM) that are theoretically independent (Jang, Livesley, Angleitner, Riemann, & Vernon, 2002; Jang et al., 2001), problems that show up in the phenotypic models of the hierarchy as well (e.g. Roberts, Bogg, Walton, Chernyshenko, & Stark, 2004). Though it would be nice if we could rely on structural pleiotropy to understand personality and its evolution, it seems likely that we will have to make do largely without it.

Standards of Evidence in the Nascent Field of Evolutionary Behavioural Genetics

MATTHEW C. KELLER

Virginia Institute for Psychiatric and Behavioural Genetics, Virginia Commonwealth University, USA
matthew.c.keller@gmail.com

Abstract

Penke et al. (this issue) argue that the genetic variation underlying cognitive abilities is probably due to evolutionarily recurrent, deleterious mutations at the thousands of loci that could potentially affect cognitive development, whereas the genetic variation underlying personality is probably due to balancing selection. This may well be correct, but I argue that some of the standards of evidence they forward are not well supported by evolutionary genetics theory. It is important at this early stage of evolutionary behavioural genetics to critically debate the standards of evidence that will help us distinguish between alternative hypotheses. Copyright © 2007 John Wiley & Sons, Ltd.

I applaud Penke et al.'s (PDM) attempt to understand the evolutionary processes that explain the genetic and environmental causes of variation in personality and cognitive abilities. Their paper is the most recent in a growing movement to use evolutionary genetics to bridge the gaps between behavioural genetics and evolutionary psychology (Gangestad & Yeo, 1997; Keller & Miller, 2006; Macdonald, 1995; Mealey, 1995; Miller, 2000b; Yeo & Gangestad, 1993; Yeo, Gangestad, Edgar, & Thoma, 1999)—an endeavor that can be termed 'evolutionary behavioural genetics'. In particular, PDM's framework is largely consonant with one that Miller and I recently forwarded regarding the evolutionary persistence of genetic variation underlying mental disorders (Keller & Miller, 2006), and so it is not surprising that I should mostly agree with their viewpoint. However, expounding upon our agreements would be a disservice to the type of critical debate that is important to scientific progress; this principle applies doubly to young scientific movements such as evolutionary behavioural genetics. Therefore, in this commentary, I endeavour to point out concerns I have with PDM's interpretation of data or theory, and forward alternative explanations that I do not feel have necessarily been laid to rest. Nevertheless, my approach should not obscure the fact that, overall, my agreements with this paper far outweigh my concerns.

PDM's thesis is that cognitive abilities have been under directional (and probably sexual) selection over evolutionary time, and that recurrent mutations at a large number of loci account for the genetic variation underlying these abilities. They argue that personality, on the other hand, is more likely to have been under some type of balancing selection (and, in particular, probably frequency-dependent selection), and so differences in personality have had fitness costs and benefits that cancel each other out over evolutionary time. This conclusion may very well be correct, but I do not think that some of the evidence marshalled in favour of this hypothesis is quite as clear-cut as PDM seem to imply. In particular, I am unconvinced that the genetic architecture of traits tells us much about the evolutionary mechanisms responsible for their variation.

PDM state that mutation selection predicts greater additive genetic variation than balancing selection, and that the degree of non-additive genetic variation is highest for balancing selection, moderate for mutation selection and lowest for neutrality (PDM, Table 1). At the same time, many measures of personality appear to demonstrate high levels of non-additive genetic variation (Eaves, Heath, Neale, Hewitt, & Martin, 1998; Keller, Coventry, Heath, & Martin, 2005; Lake, Eaves, Maes, Heath, & Martin, 2000) whereas the genetic variation underlying cognitive abilities appears to be mostly additive in nature (e.g. Rijsdijk, Vernon, & Boomsma, 2002; but see also Pedersen, Plomin, Nesselroade, & McClearn, 1992). Do such findings lend support to the hypothesis that balancing selection accounts for the variation in personality whereas mutation selection accounts for the variation in cognitive abilities? I do not think they do.

Several studies on non-human animals have found that traits most related to fitness tend to have high levels of additive genetic variation (as measured using CVAs) (Houle, 1992; Price & Schluter, 1991) but even higher levels of non-additive genetic variation, resulting in low narrow-sense heritabilities of such traits (Crnokrak & Roff, 1995; Falconer, 1989; Roff, 1997). There is also convincing data that mutation selection accounts for much of the genetic variation underlying such fitness-related traits (Charlesworth & Hughes, 1999; Houle, 1992, 1998). Therefore, the evidence does not seem to support PDM's blanket assertion that mutation selection predicts higher levels of additive than non-additive genetic variation—indeed, the opposite is probably true. That said, I should add that there is some, albeit imperfect, evidence that sexually selected traits in particular show higher levels of additive genetic variation compared to other fitness-related traits (Pomiankowski & Møller, 1995), a finding consistent with Miller's (2000a) and PDM's hypothesis that cognitive abilities have been under sexual selection. This may occur because selection favours mating signals that reveal as much additive genetic variation as possible (Pomiankowski & Møller, 1995).

I am also unconvinced that balancing selection generally leads to high levels of non-additive genetic variation (PDM's Table 1). Certainly some forms of it do—over-dominance for fitness for example. But other forms of it—frequency-dependent selection and temporal/spatial variability in the fitness landscapes, for instance—predict high levels of additive genetic variation. Thus, I would argue that the ratio of additive to non-additive genetic variation tells us little about the relative merits of mutation selection versus balancing selection.

Finally, in keeping with the critical spirit of my commentary, I feel impelled to backtrack on an assertion that I made previously and one cited by PDM. Contra Keller et al. (2005), I am no longer convinced that observations of non-additive genetic variation necessarily make neutral explanations unlikely. It is true that traits that are closer to neutral evolutionarily (e.g. morphological traits) tend to show higher ratios of additive to non-additive genetic variation (Crnokrak & Roff, 1995; Mousseau & Roff, 1987) whereas traits under more intense selection tend to show lower ratios (Crnokrak & Roff, 1995; Falconer, 1989; Roff, 1997), but the rule is not hard and fast. The reason is that the detection of non-additive genetic variation is highly sensitive to scale—it depends on how the trait is measured. For example, twin studies find evidence for high levels of non-additive genetic variation underlying absolute skin conductance, whereas the genetic variation of 'range corrected' skin conductance (a mere change in scale) appears to be purely additive in nature (Lykken, 2006). Along these lines, how are we to know the true scale along which psychological constructs, such as personality, are actually measured, or whether the micro-traits (or endophenotypes) underlying psychological constructs combine additively or multiplicatively?

I do not think that the genetic architecture of traits provides a very reliable clue as to the mechanism explaining their genetic variation. Fortunately, other pieces of evidence can better help us understand the mechanisms responsible for the genetic variation underlying a trait. Several of these are described in PDM (see also Keller & Miller, 2006): the numbers and allelic spectrums of loci affecting the trait, whether the trait shows inbreeding depression (although in addition to mutation selection, overdominance for fitness can also cause inbreeding depression), the degree of assortative mating that occurs on the trait (although assortative mating on deviations from the mean should also be considered if the trait could have been under stabilising selection) and whether its expression depends on overall condition. The effects of paternal age, radiation and trauma on the trait, all consistent with mutation selection, provide additional clues. Furthermore, once an allele that affects trait variation has been identified using, for example, association methods, its base-pair sequence can provide important information regarding the relative merits of ancestral neutrality, mutation selection and balancing selection (Bamshad & Wooding, 2003; Otto, 2000).

PDM's paper is insightful and offers us plenty to consider. I find the argument that cognitive abilities have been under ancestral sexual selection quite compelling, but remain as yet unconvinced by, but open to, PDM's argument regarding the genetic variation in personality. In particular, I find Tooby and Cosmides (1990) hypothesis (personality variation is in part a byproduct of genetic variation that exists for reasons unrelated to personality), MacDonalds's (1995) hypothesis (personality is under weak stabilising selection, such that fitness differences within its normal ranges are trivial) and Buss' (2006) hypothesis (personality is under weak directional selection, and its variation is a byproduct of mutational noise) all to be viable alternatives. My main disagreement with PDM is not in their broad conclusions, however, but rather in some of the standards of evidence they bring to bear on the issue.

The field of evolutionary behavioural genetics is young, and our first steps should be made with the circumspection befitting its fledgling nature. Much wasted time and effort can be averted if, at this stage, we remain wary of groupthink (Janis, 1972). For the sake of our nascent field, it is important to critically debate the standards of evidence that will help us distinguish between alternative hypotheses, and to refrain from forming consensus on major issues too readily.

Humans in Evolutionary Transition?

JAMES J. LEE

Department of Psychology, Harvard University, USA
jameslee@wjh.harvard.edu

Abstract

One shortcoming in this otherwise excellent paper is a neglect of additional hypotheses as to the high heritability of behavioural traits that may have been exposed to directional selection. I point to some evidence that humans are in the midst of an evolutionary transition that may account for the genetic variation in such traits. Copyright © 2007 John Wiley & Sons, Ltd.

The target paper urges bringing the power of modern evolutionary biology to bear on the variation observed in human behavioural traits. As the inauguration of this ambitious undertaking is long overdue, the target paper should prove to be an indispensable reference for some time. The authors' treatment of non-cognitive behavioural traits is particularly cogent. I devote my allotted space to pointing out what I feel are misplaced emphases and premature judgements in their treatment of traits that plausibly have been under directional selection in our evolutionary past.

Citing Fisher's (1930) fundamental theorem of natural selection for the proposition that directional selection should deplete genetic variation, the authors then argue that a special explanation is required for the abundance of genetic variation that is observed in some behavioural traits. Their own special explanation bears some resemblance to the infinitesimal model: the loci underlying fitness-relevant traits are posited to be so numerous and small in effect that selection against deleterious mutants is extremely weak and thus ineffective in removing the additive genetic variance. I have two related quibbles with this hypothesis. First, the fundamental theorem does not concern itself with the ultimate genetic architecture of a trait at all. What the theorem actually says is that the change in mean fitness at any time ascribable solely to natural selection acting on allele frequencies is equal to the additive genetic variance in fitness at that time. To infer from this statement that directional selection should extinguish genetic variation is an extrapolation not entailed by the theorem itself. Readers interested in this point are advised to consult Crow (2002), Edwards (1994), Frank and Slatkin (1992) and Grafen (2003). Second, regardless of the authority cited for it, the extrapolation does not necessarily follow. There are 'sufficiently plausible' reasons for any given failure of directional selection to deplete the additive genetic variance other than the one given by the authors (e.g. Hill & Keightley, 1987). I now provide a partial account.

On the basis of their model, the authors predict the absence or rarity of deleterious alleles at intermediate frequency. However, this assertion that the enhancing alleles for fitness-affecting traits are ancestral and nearly fixed seems to be empirically contradicted by the large number of selective sweeps detected by recent genome-wide surveys. In their scan for long, high-frequency, derived haplotypes in the human genome, Wang et al. (2006) found 1800 sites showing signals of strong and recent selection in or near known coding genes. One of the biological categories enriched for such signals is neuronal function. As their survey failed to detect selection at some loci where single-gene studies have documented selection with a high degree of confidence (e.g. Evans et al., 2005), these signals probably fail to capture the full extent to which selection has been acting in our species.

This extraordinarily large number of selective sweeps in progress reveals that humans are in the midst of an evolutionary transition. Given the absence of selective equilibrium, substantial genetic variation in any trait (including fitness) becomes compatible with several possible genetic architectures and evolutionary histories other than the one envisioned by the authors. This is because such parameters as the additive genetic variance depend on the initial distribution of allelic effects and frequencies. As the variance of a dichotomous random variable is maximised at $p = 0.5$, an architecture biased towards initially uncommon enhancing variants may show an *increase* in the genetic variance under directional selection. The large number of fitness-enhancing variants at intermediate frequencies in the human genome is certainly consistent with a bias of this kind. Such a bias may even be traceable to known developments in human evolutionary history. For example, Evans, Mekel-Bobrov, Vallender, Hudson, and Lahn (2006) have

provided persuasive evidence that an adaptive variant of the brain development gene *MCPH1* was introgressed into the human gene pool from an archaic *Homo* lineage. Hawks and Cochran (2006) argue that such introgressive events have contributed substantially to the evolution of our species, as interbreeding can introduce many more adaptive variants within a given time span than mutation alone.

The authors urge a greater focus in association studies of cognitive abilities on still-rare deleterious mutations, perhaps present in a single population. This commentary sets forth reasons to doubt that loci harbouring variants of this kind account for nearly the entire observed genetic variance in these traits. Resisting the authors' proposal of an ancestral genome encoding a Platonic ideal of human adaptation that is inevitably disrupted by new and deleterious mutations of small effect (where variability in how much of this 'mutational noise' is inherited accounts for individual differences in *g* and other ability factors), I suggest in its place a genome undergoing massive recent turnover in response to selection pressures that are as yet incompletely characterised. The kinds of variants that follow from the authors' proposal are no doubt numerous. But given the tumultuous picture of human adaptive changes that emerges from recent work, a more interesting goal with respect to the illumination of our evolutionary history may be to look for novel enhancing variants across the entire spectrum of frequencies in all populations. The few genes linked to IQ in family-based designs robust against the potentially confounding effects of population substructure all match one or more aspects of this pattern: enhanced IQ associated with derived variants, signs of selection or intermediate frequencies in one or more populations (Blasi, Palmerio, Aiello, Rocchi, Malaspina, & Novelletto, 2006; Comings et al., 2003; Gosso, de Geus et al., 2006; Gosso, van Belzen et al., 2006; Plomin et al., 2004). Given the many ways in which genotype–phenotype association studies can fail, I do not take this relative paucity of results to be evidence of absence. In fact, I am optimistic that forthcoming empirical evidence will help resolve the main issue discussed in this commentary.

Personality Traits and Adaptive Mechanisms

W. JOHN LIVESLEY

Department of Psychiatry, University of British Columbia, Vancouver, Canada
livesley@interchange.ubc.ca

Abstract

The issues addressed in this paper are basic to the foundation of a science of personality. The integration of behavioural genetic and evolutionary psychology perspectives on personality has the potential to contribute to the integrated conceptual foundation that the field needs. The task that the authors seek to explicate—the factors contributing to genetic variability of personality traits—is an important component of this integration although only part of an evolution-informed model of personality. Copyright © 2007 John Wiley & Sons, Ltd.

In focusing on selective neutrality, mutation-selection balance and balancing selection as explanations of genetic variability, the authors give short shrift to earlier explanations. Genetically based variability is a feature of most biological systems and structures. As

Tooby and Cosmides (1990) pointed out in a seminal contribution, this variability does not appear to disrupt the functioning of these adaptive mechanisms. The genetic variability of 'mental mechanisms' including traits does not at first glance appear different from that of other biological systems. Tooby and Cosmides hypothesise that this variation is due to variability at the protein level that does not affect the mechanism's function but does contribute to defence against pathogens. This argument is dismissed largely on the grounds that the alleles associated with the immune system are very different from those associated with personality systems. However, Tooby and Cosmedes argument is more subtle. The argument is not that genetic variability enhances the immune system responses but rather that protein variability creates an ever-changing substrate or micro-environment that makes it more difficult for pathogens to be successful or evolve around host defences. In a sense, sexual recombination creates minor 'lesions' that produce variation independently of function. This parsimonious hypothesis views genetic variability in personality as part of overall variability in adaptive mechanisms. In this sense, genetic differences in sensation seeking or anxiousness do not differ greatly from genetically based differences in the size of a limb or other organ. The authors reject this idea asserting simply that the number of alleles involved in personality variation is far greater although it is unclear that this is the case with complex anatomical structures and physiological systems.

Penke et al. reject the pathogen-defence hypothesis as part of their rejection of selective neutrality as the mechanism maintaining variability. The pathogen-defence mechanism requires that variability is adaptive with regards to the host's resistance but that the normal range of the personality phenotypes is equally adaptive so that no selection pressures occur at this level. They argue that the latter is unlikely because of non-neutral relationships between personality and fitness although the evidence cited refers to the contemporary not ancestral environment. They also maintain that the occurrence of a high degree of non-additive genetic variance argues against the selective neutrality of a trait. The evidence on this point is mixed and the non-additive effects seem to vary across measures. Examination of MZ and DZ correlations from a twin study of personality disorder traits, for example, showed modest evidence of non-additivity: these effects were noted in 3 of 18 primary traits and 25 of 69 sub-traits.

The authors argue to the most plausible mechanism for maintaining genetic variation in personality traits is balancing selection. It is difficult to refute their arguments on the significance of this process. It is useful to note, however, that not all psychological mechanisms or structures are necessarily adaptations. Given the complexity of personality and the many different structures and processes involved, this may not be a one mechanism fits all situation.

Although an evolutionary model of personality would potentially shed light on the origins and function of personality structures and processes, it is not clear that the level of analysis adopted by the authors is optimal for this purpose. Like other accounts of the evolution of personality (Buss, 1991, 1997; Figueredo, Sefcek, Vasquez, Brumbach, King, & Jacobs, 2005) discussion focuses on the higher-order domains of the FFM. However, these domains may be too broad to serve as the basis for formulating hypotheses about the adaptive origins of personality. Although innate mechanisms are complex in design, they are usually specific in function with the different components functioning in an integrated way. Evolutionary psychologists argue that the mental apparatus comprises a relatively large number of these domain-specific mechanisms (Simpson, Carruthers, Laurence, & Stich, 2005). It is not clear that the secondary domains of the FFM have this specificity. Instead, each domain is complex not just in the sense that any psychological adaptation

such as mate selection is complex, but also in the sense that they are multi-dimensional, each consisting of multiple functionally diverse behaviours and potential adaptive mechanisms. Neuroticism, for example, encompasses anxiety and stress management, dependency and submissiveness, impulsivity and impulse control and so on.

A more suitable level of analysis would be the primary traits (or facet traits) that form the secondary domains. As the authors note, behavioural genetic research reveals that many primary traits are etiologically distinct entities, each being associated with substantial genetic variance specific to that trait (Jang, McCrae, Angleitner, Riemann, & Livesley, 1998; Livesley, Jang, Vernon, 1998). The genetic architecture to personality appears to be complex and highly specific and primary traits appear to be the fundamental building blocks. These studies also furnish evidence of substantial pleiotropic influences raising the possibility that secondary domains like neuroticism are merely the downstream consequences of pleiotropy. Under these circumstances the search for adaptive mechanisms associated with personality traits and analyses of reaction ranges and 'personality signatures' are likely to be more productive if focused on more specific constructs.

Personality Theory Evolves: Breeding Genetics and Cognitive Science

GERALD MATTHEWS

Department of Psychology, University of Cincinnati, USA
matthegd@email.uc.edu

Abstract

Penke et al.'s (this issue) paper makes an important contribution to personality theory, with ramifications beyond genetic studies. It may significantly enhance prediction of behavioural expressions of personality traits from a psychobiological standpoint. Some theoretical challenges remain, including the complex nature of both traits and environmental modulators. The evolutionary genetic model may usefully complement the cognitive-adaptive personality theory developed by Matthews. Copyright © 2007 John Wiley & Sons, Ltd.

This is an important paper that should be read by the whole community of personality psychologists, and not just geneticists. Penke et al. (this issue) offer innovative strategies for linking genetic models directly to behavioural expressions of traits. In this commentary, I will focus on the strengths of the authors' approach, some challenging issues and its convergence with my own cognitive-adaptive model of personality, a theory based on cognitive science rather than genetics (Matthews, in press).

The foundation for contemporary personality trait theory is the evidence that traits predict consequential outcomes (Ozer & Benet-Martinez, 2006). Complementary evidence comes from controlled laboratory studies on the behavioural expressions of traits (Matthews, Deary & Whiteman, 2003). To date, psychobiological models have proved frustratingly limited in their abilities to predict individual differences in behaviour to any degree of precision (Matthews & Gilliland, 1999). Much remains to be done to

develop the Penke et al. model to the point that it makes detailed predictions of behaviour. However, it may be uniquely promising for the following reasons:

Focus on individual differences. Penke et al. rightly indicate both the neglect of systematic individual differences in personality within current evolutionary psychology, and the limitations of traditional behaviour genetic studies. It is encouraging that genetic models have advanced to the point that differing evolutionary explanations for personality variation can be tested against empirical data—this approach has legs.

Solving the isomorphism problem. Zuckerman (1991) pointed out that traits do not map isomorphically onto individual brain systems; instead, traits appears as higher-order emergent properties of multiple systems. The 'watershed' metaphor offers a principled account of why this should be so.

Traits as biosocial constructs. Penke et al. correctly emphasise individual differences in social problem-solving strategies as a key basis for traits. Handling social threats provides adaptive challenges that are much different to those of the spiders, snakes and saber-tooth tigers that provide the prototypical threats in many psychobiological accounts of anxiety (Matthews, 2004). The complexities of handling the subtle challenges of social competition—often in parallel with cooperation, as in sibling rivalry—require more attention.

The evolutionary genetic model has much promise, but there are some potential obstacles to further development of the theory.

Imaging over-enthusiasm. The identification of narrowly defined 'endophenotypes' potentially provides the essential link between polymorphisms and specific, measurable behaviours. However, linking specific polymorphisms to individual differences in brain activation patterns is of limited explanatory power; most studies fail to demonstrate any functional significance to brain activation. Coupled with the somewhat elusive nature of the molecular genetics of personality (e.g. Munafo, Clark, Moore, Payne, Walton, & Flint, 2003), modern brain-imaging studies may recapitulate the limitations of traditional psychophysiology as a means for identifying mediating mechanisms that directly govern behaviour (see Matthews & Gilliland, 1999). Brain-imaging is invaluable for discriminating component processes, but behavioural studies are requisite for tracing the adaptive implications, if any, of the process concerned.

The perennial problem of the environment. Making something of the 'individual reaction norm' concept requires specification of the environmental factors that control gene expression. Interactionism is the dominant framework for contemporary personality research, but there is a consensus on the difficulties of coding the key environmental modulators of personality. I appreciate the argument is illustrative, but the authors' example of 'environmental stress' is a case in point. There are multitude of environmental stressors that provoke a variety of behavioural responses which are often moderated by cognitions and context (Matthews, Davies, Westerman, & Stammers, 2000). Interaction of anxiety and stress factors depends critically on the person's appraisals of the stressor, blurring the necessary distinction between the individual and the environment.

The distributed nature of personality. The problem in equating traits with individual reaction norms is that the major traits pervade so many distinct adaptive processes. Neuroticism can be readily related to selective attention, executive processing, metacognition, emotion expression, compensatory effort as well as to simple emotionality (e.g. Eysenck, Derakshan, Santos, & Calvo, 2007). We can generate (possibly large) sets of reaction norms to describe the trait, but the coherence and unity of the trait may be lost in the process. However—similar to Mischel's behavioural signatures—empirical investigation of reaction norms may be a useful descriptive strategy.

Genetics and the cognitive-adaptive theory of personality. I was struck by the authors' identification of balancing-selection mechanisms as pivotal for understanding personality. Their analysis converges closely with the cognitive-adaptive theory of personality (Matthews, 1999, 2000, in press; Matthews & Zeidner, 2004). In brief, the theory proposes that traits correspond to adaptive specialisations to some of the more marginal environments that are universal to human societies; e.g., extraversion corresponds to social overload, introversion to underload. Each person (consciously or not) must develop a strategy for handling social threat. High neurotic persons favour anticipation (requiring worry) and avoidance, whereas low neurotics delay response until the threat may be more directly confronted.

Similar to Penke et al.'s model, cognitive-adaptive theory assumes traits confer adaptive gains and costs within specific environments, but are adaptively neutral overall. Cognitive-adaptive theory also states that traits are built on a platform of genetically influenced basic components of the neural and cognitive architectures, which is modified developmentally by socio-cultural learning and autonomous, self-directed shaping of personality. Penke et al.'s theory may add powerfully to understanding the role of genetic antecedents.

Conversely, cognitive-adaptive theory may help to tackle some of the issues facing the evolutionary genetic model. The theory places acquired skills at the forefront of adaptation (cf., Feltovich, Prietula, & Ericsson, 2006); skill acquisition is biased but not directly determined by heritable component processes (corresponding to endophenotypes). The theory also explicitly conceptualises traits as distributed across multiple mechanisms and processes, understood at different levels of abstraction from neural processes (cf. the classical theory of cognitive science: Matthews, 2000). The trait gains unity not from any specific process but from the common functionality of multiple processes in supporting a specific adaptive strategy. It is critical to explore trait consequences across a range of environments to determine its adaptive significance; perhaps evolutionary personality theory needs a little less Mendel and a little more Darwin.

Do We Know Enough to Infer the Evolutionary Origins of Individual Differences?

ROBERT R. MCCRAE

Gerontology Research Center, National Institute on Aging, USA
mccraej@grc.nih.nih.gov

Abstract

Psychologists do not yet understand the role of non-additive genetic influences on personality traits or the number of quantitative trait loci (QTLs) for individual traits. Traits vary in their desirability in mates and in their assortative mating. Thus, it is premature to conclude that individual differences in all or any personality traits have evolved by balancing selection. Copyright © 2007 John Wiley & Sons, Ltd.

From my sporadic reading of the literature on the evolutionary psychology of personality traits, the target paper appears to represent a notable advance in sophistication. It incorporates new thinking on a number of evolutionary principles and makes an effort to

compare rival hypotheses about the origins of individual differences using quantitative estimates of relevant parameters (such as the number of new mutations per individual). I was struck, however, by the frank admission that one of the classical inferences about the relation between fitness and additive genetic variance had been wrong, and the error remained 'unnoticed for half a century'. There is a moral here, I think: These issues are extremely complex, and it is likely to be some time before we can be fully confident that we understand what is really going on.

The paper compares three models of the origins of individual differences, and attempts to rule out two of them—selective neutrality and mutation-selection balance—with regard to personality traits. I will focus on the mutation-selection balance principle, which the authors believe is applicable to intelligence, but not to personality traits. If we assume that their reasoning is correct, then the conclusion hinges on the factual accuracy of the claims that personality traits fail to show 'high additive genetic variation, an elusive molecular genetic basis, condition-dependence, inbreeding and outbreeding effects, strong mate preferences and assortative mating'. These are empirical assertions, and several of them are questionable.

In behaviour genetic studies, it is customary to compare models that include additive and non-additive genetic effects and shared and non-shared environmental effects. There is consistent evidence that shared environmental effects are negligible, but a good deal of variation in whether non-additive variance is included in the chosen model. For example, twin studies of the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992) in Canada and Japan concluded that all factors and facets could be suitably described by an additive model (Yamagata et al., 2006). In contrast, Keller et al. (2005), using a twin-plus-sibling design, argued that non-additive effects were pervasive in personality measures. Additional evidence for non-additive effects comes from a study of extended family members in Sardinia (Pilia et al., 2006). In that study, broad heritabilities (which include non-additive effects) were much closer in magnitude to the heritabilities seen in twin studies than were narrow heritabilities (additive effects only). As Keller et al. point out, the accurate estimation of non-additive effects is difficult, because additive and non-additive effects are strongly inversely related, introducing problems akin to multicolinearity in regression. The data seem to show that there are non-additive effects for some personality traits, but whether the additive effects should be characterised as 'large' or 'medium' (see Table 1 in the target paper) is unclear.

No one who has followed the field would dispute that, to date, the molecular genetic basis of traits has been elusive. After a promising start (Benjamin, Li, Patterson, Greenberg, Murphy, & Hamer, 1996), attempts to link the D4 domapine receptor gene to personality stalled in a series of failures to replicate (Gebhardt et al., 2000; Vandenbergh, Zonderman, Wang, Uhl, & Costa, 1997). Meta-analyses of the literature on the 5HTTLPR seretonin transporter gene polymorphism (Schinka, Busch, & Robichaux-Keene, 2004) have reached only ambiguous conclusions, with some but not all measures of neuroticism showing associations.

These studies examined candidate genes, and what may have eluded researchers was perhaps only the right candidates. A more comprehensive approach seeks replicable findings from a whole genome scan; such studies are currently underway (e.g. Costa et al., 2007), but have not yet reported findings. It thus remains to be seen whether the number of quantitative trait loci (QTLs) for personality traits is large or small.

Are there strong mate preferences for personality traits? Buss and Barnes (1986) gave respondents a list of 76 characteristics they sought in a mate, including *kind*, *intelligent*,

church-goer, *good cook*, *likes children*, *wealthy* and *healthy*. Personality traits like *considerate*, *honest*, *interesting to talk to* and *affectionate* were among the top 10 desiderata; *early riser*, *tall* and *wealthy* were not considered desirable. It is, of course, possible that people's true preferences differ from what they claim: It is socially undesirable to admit to seeking wealth in a mate. Still, the available evidence suggests that people put a high value on personality traits.

Assortative mating is more complex than the authors appear to realise. There is a widespread perception that assortment for personality traits is negligible (about .10) whereas that for intelligence is notably higher (about .40; see Plomin, 1999). Most studies have involved extraversion and neuroticism, and the .10 value is reasonable for those factors. But higher values (.20–.30) have been reported for openness and conscientiousness (McCrae, 1996), and much higher values for traits related to liberalism/conservatism, which is a facet of openness. One might argue that assortment for liberal attitudes proceeds from social causes that have little to do with evolutionary processes. But one might make that same argument for intelligence: Intelligent people may prefer intelligent mates, not because they are higher in fitness, but because they are more interesting to talk to.

In sum, we do not seem to have sufficient information at present about personality traits to distinguish among the options of mutation-selection balance and balancing selection. Until we have such information, we ought to avoid the assumption that all personality traits share a single mechanism of evolutionary origin. Traits are all roughly equally heritable (e.g. Jang et al., 1998) but we have no way of knowing whether they all have similar numbers of QTLs, and we already know that they differ in assortative mating effects. For the time being, it may be wisest to consider evolution one facet at a time.

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What Do We Really Know About Selection on Personality?

DENIS RÉALE

Département des sciences biologiques, Université du Québec, Montréal, Canada
reale.denis@uqam.ca

Abstract

An evolutionary genetic approach to personality in animals and humans necessarily assumes a link between personality traits and fitness. Evolutionary personality psychologists have mainly focused on an a priori conception of this link to build up evolutionary scenarios. Although this approach has added to our understanding of the variance of personality traits, it needs to be accompanied by an empirical examination of the link between these traits and fitness. Several tools developed by evolutionary biologists could therefore be useful in evolutionary personality studies. Copyright © 2007 John Wiley & Sons, Ltd.

Evolutionary ecologists have become interested in personality traits only very recently (Réale et al., 2007), and many felt that, despite a shared interest for similar traits, personality psychologists did not have much in common with them. Using fitness as the currency for their study traits, evolutionary ecologists have mainly been interested in the adaptive function of personality and the ecological role of personality variation. Personality psychologists, on the other hand, seemed to have focused mainly on the social desirability of personalities and the social implications of extreme expressions of personality traits. Penke et al.'s (this issue) thorough review suggests that an interesting convergence may be occurring between the two fields (see also Ellis, Jackson, & Boyce, 2006; Nettle, 2006). Such convergence will promote new ways of looking at personality traits for members of both fields, and should improve our understanding of heritable personality variation.

This said, several points raised in this review may be subject to debate, while other aspects important for the evolutionary study of personality traits are missing. The authors are a bit too quick to reject the role of some factors on personality variation. For example, in a human metapopulation system (Harding & McVean, 2004) genetic drift probably plays a more important role than expected, whereas antagonistic pleiotropy is still one of the main explanations for the maintenance of variation in life-history traits (Roff, 2002). Neither of these explanations is totally incompatible with the hypothesis of fluctuating selection, and both should be examined more thoroughly prior to being rejected. Rather than giving a detailed listing of such points, I will focus on one major aspect that I think deserves more attention: The link between fitness and personality traits is central to an evolutionary genetic approach to personality, but the way the authors propose to examine this link is somewhat vague. In many instances they mention potential relationships between personality or cognitive abilities and fitness, and the importance of the selection regime for the maintenance of genetic variance, but what do we really know about selection on personality? The study of phenotypic selection, an approach that permits us to examine how quantitative traits are shaped by natural or sexual selection, has experienced strong conceptual and methodological developments since the 1980s (Arnold & Wade, 1984; Brodie, Moore, & Janzen, 1995; Endler, 1986; Hersch & Philips, 2004; Lande & Arnold, 1983). However, these developments have been ignored by Penke et al. Below, I show how they can help the development of evolutionary personality studies.

The phenotypic selection study involves evaluating direct and indirect selection acting on traits during a single episode of selection. A directional selection differential (S) represents the change in the mean phenotypic value of a trait resulting from both direct and indirect selection pressures, and is measured as the covariance between the standardised trait and relative fitness. A directional selection gradient (β, i.e. partial regression coefficient in a multiple regression) reflects the change in the mean phenotypic value of a trait resulting from direct selection on this trait, while holding the effects of other traits constant (Arnold & Wade, 1984; Lande & Arnold, 1983). Quadratic terms and interactions between traits can be added to the model to estimate the strength of stabilising/disruptive selection acting on each trait, and correlational selection, respectively. These statistics can be combined with information on the genetic variance/covariance matrix (**G**) to predict the evolutionary response of the traits to selection. Penke et al. (this issue) assume that cognitive abilities are directly and invariably related to fitness, and that personality traits should be under weaker fluctuating selection. Their assumptions, however, are based on an *a priori* conception of how selection acts on these traits. Selection differentials and gradients are standardised statistics. They therefore permit us to compare the strength of

selection between different traits or for the same trait between years, environmental conditions or populations (Kingsolver et al., 2001). Using this approach it is thus possible to determine whether personality and intelligence are under different selection regimes, or to test for the presence of fluctuating selection in space and time. The authors also discuss the possibility that variance in personality traits is maintained as a by-product of selection on other traits (see also Nettle, 2006), a hypothesis that can be tested with the phenotypic selection approach.

Phenotypic selection has rarely been used in personality studies in animals (but see Dingemanse & Réale, 2005; Réale & Festa-Bianchet, 2003). In humans a few studies have proposed an equivalent approach (Eaves, Martin, Heath, Hewitt, & Neale, 1990; Nettle, 2005), but to my knowledge none have used the full potential of phenotypic selection analysis. Although, in principle, such approach could be applied to humans, its use may be limited by a few constraints that would need to be examined further. First, the low power of selection studies requires large sample sizes to detect significant selection gradients within the range generally observed in wild populations (i.e. several hundred individuals: Hersch & Phillips, 2004; Kingsolver et al., 2001). This is especially important if one is interested in detecting weak and invariant selection pressures. Sample size does not seem to be a constraint in studies on humans (e.g. Eaves et al., 1990; Nettle, 2005) and therefore should not be limiting. Second, estimates of individual fitness have to be chosen carefully. Penke et al. propose the f-factor, a general index of fitness, but never mentioned explicitly how to use this factor. Lifetime reproductive success is generally considered the most appropriate estimate of fitness, although related indices are available (Brommer, Gustafsson, Pietiaïnen, & Merilä, 2004; Coulson et al., 2006). Other fitness components, like survival, fecundity or the number of sexual partners can be used, but should be considered with caution because they are potentially involved in evolutionary trade-offs (Roff, 2002). Although the use of such fitness components can be informative for someone interested in decomposing the links between personality and fitness, it can provide an incomplete portrait of selection acting on a trait. Evidence for selection on personality traits in humans using indices more remotely related to fitness should be evaluated with these potential drawbacks in mind.

The evolutionary genetic approach proposed by Penke et al. will certainly provide new sources of inspiration for personality psychologists and evolutionary ecologists. This, and other recent papers (e.g. Ellis et al., 2006; Nettle, 2006), should generate testable predictions that could benefit from methods commonly used in evolutionary biology. We may therefore be witnessing the first steps towards a more integrated evolutionary study of personality in humans and animals.

Personality: Possible Effects of Inbreeding Depression on Sensation Seeking

IRENE REBOLLO and DORRET I. BOOMSMA

Department of Biological Psychology, Vrije Universiteit, Amsterdam, The Netherlands
i.rebollo@psy.vu.nl, Di.boomsma@psy.vu.nl

Abstract

Penke et al. (this issue) state that there are no studies of inbreeding depression on personality. In this response to their paper, we look at the effect of parents being born in the same geographical region on personality in themselves and in their offspring. Results show that when parents come from the same region, both they and their offspring score lower on sensation seeking than when parents come from different regions. These results may suggest effects of inbreeding depression on personality. Copyright © 2007 John Wiley & Sons, Ltd.

Studies of inbreeding depression on intelligence (Jensen, 1998) show evidence for inbreeding depression, but—as stated by Penke et al. (this issue)—there are no studies of inbreeding depression on personality. However, Camperio Ciani, Capiluppi, Veronese, and Sartori (2007) reported an interesting comparison of personality traits in Italian coast dwellers and Italians from three small island groups. Subjects whose families had lived on the islands for at least 20 generations were lower in extraversion and openness to experience. Penke et al. discuss this finding in the context of 'environmental niches' for personality traits, but an alternative explanation might also be possible: the islanders might form a genetically more related group (a genetic isolate) whose offspring shows an effect of inbreeding depression.

To test this hypothesis in an alternative dataset, we took personality data collected in Dutch families consisting of parents and their twin offspring. The families took part in longitudinal survey studies. In 1991 and in 1993 the parents were asked if they had been born in the same geographical region (answers 'yes', 'no' and 'don't know'). We formed two groups of families: those whose parents were born in the same geographical region and those whose parents were born in different regions. Please note that same or different region can be a rural or non-rural part of The Netherlands, the question was only about proximity. We then examined if there were personality differences between the two groups. Personality scores were compared between the two groups in the parental and in the offspring generation. We looked at personality traits related to neuroticism, extraversion and sensation seeking. We hypothesise that if parents were born in the same geographical region, they may genetically be more related than when they come from different areas of the country, and use this test as an indirect way of looking at inbreeding depression (or its opposite 'hybrid vigour').

Participants. This study is part of an ongoing study on personality, health and lifestyle in twin families registered with the Netherlands Twin Register (NTR; Boomsma et al., 2006). Surveys were mailed to twin families every 2 to 3 years. For the present study data from the 1991 and 1993 surveys were used. In total, there were 2905 families. There were 1940

families who took part once (in 1991 or 1993) and 965 who took part at both occasions. Average age of the parents was 46.67 years in 1991 and 47.04 in 1993; average age of their offspring was 17.73 years in 1991 and 20.18 in 1993.

Measures. In both surveys parents of the twins were asked if they had been born in the same region. Data from the two surveys were combined into one yes/no measure. The following 10 personality measures were analysed: neuroticism, extraversion and somatic Anxiety and Test Attitude (ABV; Wilde, 1970); thrill and adventure seeking, boredom susceptibility, disinhibition and experience seeking (Feij & van Zuilen, 1984; Zuckerman, 1971), trait anger and anxiety were measured using the Dutch adaptation of Spielberger's State-trait Anger Scale (STAS; Spielberger, Jacobs, Russell, & Crane, 1983; van der Ploeg, Defares, & Spielberger, 1982) and State-trait Anxiety Inventory (STAI, Spielberger, Gorsuch, & Lushene, 1970). Personality measures were averaged over occasions if subjects participated more than once.

Data analyses. We first looked at personality differences between parents being born in the same geographical region and parents being born in different geographical regions, separately for fathers and mothers. In the offspring generation, the same comparisons were carried out separately for first and second born twins to avoid dependency of observations. Data analyses were carried out with SPSS. We employed MANOVA to study group differences. The use of MANOVA prevents the inflation of overall type I error that derives from the use of multiple univariate tests on a group of correlated variables. In the offspring generation sex was introduced as a covariate.

Results. For fathers there was a significant effect of same region on two Sensation Seeking Scales, i.e. boredom susceptibility and experience seeking. In addition an effect was seen for test attitude. For mothers, experience seeking and test attitude were also significantly different between groups. In mothers, a significant effect was also observed for thrill and adventure seeking, which also is one of the Sensation Seeking Scales, and somatic anxiety (see Table 1). Subjects who were born in the same region as their spouse score higher in test attitude, which assesses the tendency to give socially desirable replies. Subjects who were born in the same region as their spouse score lower on Sensation Seeking Scales. Mothers who were born in the same region as their partner show lower somatic anxiety. The largest effect size was for experience seeking.

Table 1.   Mean values for personality variables that show significant differences in parents of twins

	Father's mean			Mother's mean		
	Same region $N = 1433$	Different region $N = 855$	Effect size	Same region $N = 1557$	Different region $N = 950$	Effect size
Boredom susceptibility	36.44	37.25**	.113	35.43	35.68	
Experience seeking	31.61	33.76**	.289	29.47	31.62**	.282
Thrill and adventure seeking	28.69	29.50		21.75	22.66**	.118
Test attitude	39.45	38.72*	.086	41.28	40.05**	.143
Somatic anxiety	16.47	16.20		18.16	18.67*	.091

Note: p values next to the means correspondent to the F statistic of the between subjects effects of 'same region'. $*p < .05$; $**p < .01$.

Table 2. Mean values for personality variables that show significant differences in both twins

	Twin 1 mean			Twin 2 mean		
	Same region $N = 1581$	Different region $N = 955$	Effect size	Same region $N = 1574$	Different region $N = 954$	Effect size
Boredom susceptibility	38.01	38.56*	.082	37.86	38.77**	.137
Experience seeking	33.95	35.48**	.220	34.12	35.33**	.180
Thrill and adventure seeking	39.09	40.09**	.110	38.83	39.59*	.094
Somatic anxiety	18.66	19.20**	.119	18.66	19.15*	.089

Note: p values next to the means correspondent to the F statistic of the between subjects effects of 'same region' independent of the effect of sex.
*$p < .05$; **$p < .01$.

In the offspring generation there was no effect on test attitude. However, experience seeking, boredom susceptibility, thrill and adventure seeking and somatic anxiety also reached significance in first and second born twins. The direction of the differences was the same as in the parental generation (see Table 2). Experience seeking again shows the largest effect size, and it is the trait that shows significant differences in both parents and both twins.

The reappearance of personality differences between parents who were born in the same region and parents who were born in different regions in the offspring generation suggests the presence of inbreeding depression in personality. This is especially true for sensation seeking traits. These results agree with those of Camperio Ciani et al. (2007) who found that subjects whose families had lived on islands for at least 20 generations were lower in openness to experience. Alternative explanations are also possible, e.g. sensation seekers tend to move around more, and their children inherit their sensation seeking tendencies.

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A Multitude of *Environments* for a Consilient Darwinian Meta-Theory of Personality: The Environment of Evolutionary Adaptedness, Local Niches, the Ontogenetic Environment and Situational Contexts

GAD SAAD

Marketing Department, John Molson School of Business,
Concordia University, Montreal, Canada
gadsaad@jmsb.concordia.ca

Abstract

A consilient and complete evolutionary-based theory of personality must explain the adaptive mechanisms that maintain personality variance at four distinct 'environmental' levels: (1) the environment of evolutionary adaptedness (EEA); (2) the environment as defined by a given local niche; (3) the ontogenetic environment and (4) the situational environment germane to the person-situation debate in personality theory. Copyright © 2007 John Wiley & Sons, Ltd.

I recently completed a project with one of my graduate students (Richard Sejean) wherein we contrasted the decision-making styles of monozygotic and dizygotic twins and found that these possessed a genetic underpinning. The paper by Penke et al. (this issue) (PDM) is *à propos* as it provides us with a parsimonious set of evolutionary mechanisms capable of maintaining genetic variance in decision-making styles. I suppose that the next challenge is to identify the one-to-one 'optimal' mapping between a given decision-making style and a particular environment that would yield such heterogeneity in cognitive proclivities. PDM recognise the importance of this point when they state, 'The challenge . . . is to identify the specific costs and benefits relevant to each personality trait across different environments.' Implicit in addressing this difficult problem is providing an operational definition of the term *environment* in the current context, a point to which I turn to next.

One can speak of the *environment* of evolutionary adaptedness (EEA) that is central to the adaptationist framework. Hence, universal sex differences in sensation seeking and/or risk taking can be construed as sex-specific adaptations shaped by sexual selection. Alternatively, one can talk about the *environment* in the sense of a local niche in which case personality traits that differ recurrently across populations can be interpreted as adaptations to idiosyncratic milieus (as per Camperio Ciani et al., 2007; see also Dall et al., 2004). The ontogenetic *environment* is yet a third type of environment that might shape one's personality via an evolutionary-based mechanism. For example, Sulloway (1995, 1996) has proposed the Darwinian Niche Partitioning Hypothesis as a driver of one's personality. Specifically, he argued that one's birth order yields unique challenges for a given child in its quest to maximise the parental investment that it seeks to receive. Specifically, a child seeks to fill an unoccupied niche as a means of securing maximal parental investment. If a firstborn has already occupied the 'I am the obedient good boy' niche then his younger male sibling must identify alternate niches to fill out. As one goes down the birth order the number of unfilled niches is fewer, which Sulloway argues

drives laterborns' higher scores on openness to experience. Alternatively, in wishing to maintain their privileged position within the sibship, firstborns are much more likely to score high on conscientiousness. Finally, a fourth type of *environment* is the immediate situational one that is central to the person-situation debate in personality research. In this case, one can talk about the malleability of one's personality as a function of situational demands. Personality traits such as self-monitoring or Machiavellianism might be particularly relevant here as they both recognise an individual's ability to adapt to the situation at hand. The malleable nature of one's personality is akin to the inherent plasticity of our immune system. Specifically, the immune system has evolved the species-level adaptation of being adaptable to idiosyncratic challenges faced by any given organism. This is necessary in order for the immune system to maintain a maximal number of degrees of freedom in its ability to mount defenses against as of yet unforeseen and unknowable attacks. Malleable personality traits in a sense are similar in that they recognise that the social environment is the source of a wide range of environmental challenges and as such must allow for situational plasticity. Wilson, Near, and Miller (1996) applied this exact principle in exploring Machiavellianism from an evolutionary perspective as did Saad (2007, Chapter 2). Recent papers by MacDonald (2005) and Michalski and Shackelford (in press) discuss related multi-level taxonomies for understanding the evolutionary forces that can maintain individual variations in personality (see also Bouchard & Loehlin, 2001, for an evolutionary-based behavioural genetic account of personality).

An evolutionary account of personality must explain G × E interactions across all of the relevant multi-layered levels of analyses. This is easier said than done as most scholars including evolutionists oftentimes create rigid binary categories in defining their research approaches, which can lead to epistemological myopia (e.g. adaptationist vs. behavioural ecological approaches; domain-specific vs. domain-general view of the human mind and human universals vs. individual differences). Although most evolutionists recognise the complementarity of these approaches (cf. Laland & Brown, 2002), they seldom conduct research across multiple levels of analyses. This is precisely where I believe the paper by PDM is most insightful namely it posits distinct forms of balancing selection that 'target' several layers of a Darwinian meta-theory of personality. For example, PDM propose that sexually antagonistic co-evolution might be a viable mechanism by which sex differences in personality are maintained whilst arguing that environmental heterogeneity and frequency-dependent selection are likely mechanisms for explaining cross-cultural differences in personality types. This ability to map various sources of personality variance to specific evolutionary mechanisms (at the genetic level) is a necessity if we are to create a truly consilient evolutionary-based theory of personality.

The 'multi-layered' meanings of *environment* as described here are congruent with Universal Selection Theory (UST; cf. Cziko, 1995, 2000), which recognises that evolutionary processes operate across a wide range of levels. For example, while most evolutionists study between-organism selection, UST recognises that Darwinian processes operate within-organisms as well (e.g. Neural Darwinism as per Edelman, 1987; see also Hull, Langman, & Glenn, 2001, for a broad discussion of selection processes). Finally, while I do not wish to rekindle here the individual versus group selection debate, there is evidence to suggest that for some group decision-making tasks, personality heterogeneity of the group members can at times yield superior outcomes (Bowers, Pharmer, & Salas, 2000; Bradley & Hebert, 1997; Mohammed & Angell, 2003). Hence, an intriguing

possibility might be that individual differences in personality are maintained in part because they yield superior group decisions and related outcomes (note that group decision-making is a common decisional context for a social species such as ours).

To conclude, one of the most challenging problems for evolutionary personality theorists will be to identify which form of adaptive process drives a given personality variance, a task tackled admirably by PDM.

Insights From Behavioural Syndromes for the Evolutionary Genetics of Personality

ANDREW SIH[1] and ALISON M. BELL[2]

[1]*Department of Environmental Science and Policy, University of California, Davis, USA*
asih@ucdavis.edu
[2]*Department of Integrative Biology, University of Illinois, Urbana, USA*
alisonmb@life.uiuc.edu

Abstract

Behavioural ecologists have recently begun emphasising behavioural syndromes, an analogue of personality. This new area offers several insights for the evolutionary genetics of human personality. In particular, it suggests that human personality research could benefit from emphasising: the evolution of reaction norms, correlational selection, indirect genetic effects (IGE), G × E correlations, social situation and partner choice and social networks. Copyright © 2007 John Wiley & Sons, Ltd.

We study behavioural syndromes, an analogue of animal personalities (Sih, Bell, & Johnson, 2004; Sih, Bell, Johnson, & Ziemba, 2004). While many studies on animal personalities focus on the Big Five (Gosling, 2001), the emphasis for behavioural syndromes is typically on ecologically important behavioural tendencies that have a long history of study by behavioural ecologists, e.g., boldness or aggressiveness. We ask if these behavioural tendencies carry over across contexts. If they do, we expect that sometimes, these carryovers might result in suboptimal behaviour. For example, is an animal that is more aggressive than others in competitive contests also inappropriately aggressive with mates or offspring? We also ask if different, but intuitively similar tendencies are positively correlated. Are individuals that are more bold with predators also more aggressive with conspecifics? Studies have shown that behavioural types (BTs) can be heritable (van Oers, de Jong, van Noordwijk, Kempenaers, & Drent, 2005), have neuroendocrine correlates (Koolhaas et al., 1999) and affect fitness (Dingemanse & Réale, 2005). Many fundamental questions, however, remain unanswered. Why do BTs (or personalities) exist? If a tendency to be aggressive spills over to cause inappropriate aggressiveness in some contexts (psychopathologies?), why has this spillover not been eliminated by natural selection? What explains the structure of the BS? Why are boldness and aggressiveness sometimes, but not always correlated? When and why are BTs and BS stable over time?

Answering the above questions requires a better understanding of the evolutionary genetics of behavioural syndromes. We were thus quite excited to read Penke et al.'s (this issue) comprehensive review of the evolutionary genetics of human personalities. We applaud, in particular, the authors' enthusiasm for adopting a $G \times E$, reaction norm view on the genetics of personality. Our commentary will focus on areas of excitement in the study of behavioural syndromes that might also prove insightful for building an integrative, evolutionary theory of personality for humans and other animals.

The first challenge is to find a suitable model that can explain the maintenance of genetic variation in personality. Most of the models considered by the authors examine the maintenance of genetic variation in non-plastic traits. Behaviour, however, is by definition, plastic, in that it involves a response to the environment. The most appropriate models should thus be models which consider the maintenance of genetic variation in reaction norms. While the second half of the paper by Penke et al. champions the importance of the reaction norm view, surprisingly, those insights were not applied to the first half of the paper, which reviewed models on the maintenance of genetic variation.

The theoretical literature on the maintenance of genetic variation in reaction norms is small (but see de Jong & Gavrilets, 2000; Zhang, 2005, 2006) but the few models suggest that plasticity can produce some counter-intuitive patterns. For example, in standard models of non-plastic traits, environmental variation and balancing selection tend to facilitate the maintenance of genetic variation (Turelli & Barton, 2004). In contrast, depending on specific scenarios modelled, with reaction norms, greater environmental variation can either increase or decrease the maintenance of genetic variation. The logic on why environmental variation can decrease genetic variation appears to be that with greater environmental variation, plastic genotypes are exposed to stronger overall selection across the range of environments. In any case, the study of both human personality and animal behavioural syndromes could benefit from further development of models on the maintenance of genetic variation in reaction norms.

Another evolutionary process that deserves attention here is correlational selection, where the fitness of one personality trait depends on how it is combined (correlated) with another behavioural trait. Unlike models that examine environmental heterogeneity and balancing selection which typically assume stabilising selection with different optima in different environments, evolution via correlational selection is explicitly combinatorial. As the authors note, very high openness to experience combined with high IQ might result in exceptional creativity whereas very high openness combined with low IQ might be viewed as a schizotypic personality disorder. In stickleback fish, boldness and aggressiveness are positively correlated in high predation regimes, but uncorrelated in low predation regimes (Bell, 2005). Experimental exposure to actual predation showed that this correlation is generated by a combination of selection and behavioural plasticity (Bell & Sih, unpublished work). A greater emphasis on correlational selection should be crucial for both theoretical and empirical analyses of the evolution of personalities.

Evolutionary theory can also contribute to human personality genetics by providing a theoretical framework for studying the genetics of social interactions. Social interactions introduce an exciting twist to evolutionary genetics, the possibility of important indirect genetic effects (IGEs) (Wolf, Brodie, Cheverud, Moore, & Wade, 1998). IGEs occur when an individual's phenotype (e.g. its aggressiveness) depends not just on its genotype but on its social environment (e.g. the aggressiveness of others). Since the behaviour of other individuals has a genetic component, the social environment has a genetic component. This, in effect, decouples the standard genotype–phenotype relationship. The behaviour of

each individual depends not just on its own genotype, but on the genes of all interacting individuals in its social network. IGEs can have major impacts on evolutionary dynamics. To our knowledge, however, the effects of IGEs on the maintenance of genetic variation has not been quantified.

Standard evolutionary models, models of IGEs and game models all start with the assumption that individuals experience available environments and the mix of genotypes in their social environment in proportion to their relative frequency. In fact, individuals often exercise situation choice—habitat choice, social situation choice and partner choice. If different personalities have a genetic tendency to choose different situations, this produces a G × E correlation. In the context of partner choice, different personalities might occupy different positions in the social network (which could be quantified using social network metrics). Unlike habitat choice, social situation and partner choice feature the fascinating complication that individuals cannot independently dictate their own social environment. Social structure and each individual's social partners depend also on the interplay of choices by other individuals. Integrating this reality into evolutionary genetic models should also prove insightful.

Using Newer Behavioural Genetic Models and Evolutionary Considerations to Elucidate Personality Dynamics

SUSAN C. SOUTH and ROBERT F. KRUEGER

Department of Psychology, University of Minnesota—Twin Cities, USA
south088@umn.edu, krueg038@umn.edu

Abstract

We expand on the theme of transactions between persons and situations, and genes and environments. Newer models for twin data can handle genotype-environment transaction effects explicitly, and such models can be used to better articulate the origins of variation in personality. Copyright © 2007 John Wiley & Sons, Ltd.

Penke et al. (this issue) are to be commended on a deep and fascinating contribution to the personality literature. As Penke et al. note, newer techniques in modelling twin data offer ways of more explicitly articulating genotype-environment transactions. We agree with Penke et al. that these newer techniques are central to advancing inquiry in personality genetics, and that interpretation of findings generated by these models will be enhanced by evolutionary thinking.

Traditionally, behaviour genetic inquiry has focused on twins because twins are plentiful and studying them provides a way of cleanly separating the different impacts of genotypes and environments on human individual differences. In particular, behaviour genetic studies of personality traditionally focused on dividing up the variation in personality traits into the contributions of genetic (most often additive genetic, or A

factors), shared or 'common' environmental factors (C, those environments that make people the same because they grew up in the same family) and non-shared environmental factors (E, those environments that make people different in spite of growing up in the same family). Such research consistently finds that A is a substantial proportion of the total variance of a trait (often 40–50%), with the rest of the variation attributable to E (Krueger, Johnson, Plomin, & Caspi, in press). As Penke et al. note, these findings are no longer surprising to many, but they continue to be of central importance for at least two reasons. First, they clearly invalidate models of human individual differences that assume that people are 'blank slates'—models that have been historically influential in academic psychology (e.g. classical behaviourist accounts of personality). Second, these findings continue to confound both theoretical and empirical inquiry in personality psychology. If genes are so important to personality, why are specific genetic polymorphisms connected with personality so hard to find (Ebstein, 2006)? And if the non-shared environment (E) is so important to personality, what are the key environmental factors involved, and why have these also been so hard to identify (Turkheimer & Waldron, 2000)?

We do not have easy answers to these tough questions, but we do believe that some key directions can be drawn out from Penke et al.'s thoughtful section on 'practical implications for behavioural genetics'. As Penke et al. note in point no. 3, models for 'genotype × environment interaction (G × E) and correlation (r_{GE})' have been developed recently, and they should be used more frequently. Characterising these models in terms of G × E and r_{GE} is fine as shorthand, but working with these models also leads us to believe that the concepts of G × E and r_{GE} do not do justice to the transactional phenomena that can be articulated with newer approaches to modelling twin data. Recall that classical behaviour genetic inquiry in personality consists of parsing the variance in personality into ACE effects. The newer models Penke et al. are citing (e.g. Purcell, 2002) continue to involve decomposing a variable of interest (a target variable) into ACE effects, but these effects can now be expressed as contingent on the level of another variable (a moderator variable). Hence, in these models, a moderator variable with its own ACE effects moderates the ACE effects on a target variable. The resulting problem with the language of G × E and r_{GE} is that both the moderator and target variables have ACE components— neither variable is purely 'genetic' nor purely 'environmental'. It is not just that purely genetic factors interact and correlate with purely environmental factors (G × E and r_{GE}). Rather, both genetic and environmental effects on both target and moderator variables transact continuously. We will use some findings from our own research to illustrate this point.

Krueger, South, Johnson, and Iacono (submitted) examined genetic and environmental (ACE) influences on the broad personality traits of negative emotionality, positive emotionality and constraint in adolescents (the 'Big Three' traits, higher in the trait hierarchy than the Big Five traits focused on by Penke et al., this issue; Markon, Krueger, & Watson, 2005). Specifically, Krueger et al. (submitted) examined how ACE effects on those traits varied vary as a function of aspects of the parent–adolescent relationship. Both positive (Parental Regard) and negative (Parental Conflict) aspects of the adolescent's relationship with both parents were partly heritable (South, Krueger, Johnson, & Iacono, submitted), and both moderated the variance components of positive and negative emotionality.

Interestingly, at high levels of conflict, the shared environment had a notable effect on the variance in adolescents' personalities. Indeed, for adolescents with levels of conflict two standard deviations greater than average, the variance in negative emotionality was as

attributable to the shared environment (C) as it was to genetic factors (A). This finding fits well with Penke et al.'s emphasis on how circumstances the organism encounters should affect the origins of personality variation in a dynamic fashion. It is tempting to frame this finding in the language of $G \times E$: the 'environment' of conflict with parents changes the 'genetic' effect on negative emotionality. However, the finding does not fit neatly into the $G \times E$ framework because (a) the 'environment' of conflict is partly heritable, driven in part by genetic characteristics of the adolescent (cf. Rowe, 1994) and (b) it is not just the genetics of negative emotionality that are affected; environmental contributions to negative emotionality also change as a function of conflict.

While this type of transactional modelling is in its infancy, it has exciting applications in studying personality. An evolutionary theory of personality can guide this work by providing hypotheses about circumstances where gene-environment transactions are likely to occur. As Penke et al note, when socio-cultural relations are beyond normal boundaries, the organism needs to adapt to maximise fitness, so these may be circumstances where specific genetic and environmental effects are highlighted. Broadly speaking, evolutionary psychology can guide our thinking about when and where genes and environments matter, and should thereby be able to help us identify the effects of both specific genetic polymorphisms and environmental circumstances on behaviour more reliably.

Neurogenetic Mechanisms Underlying Cognition and Temperament

ALEXANDER STROBEL

Department of Psychology, Johann Wolfgang Goethe University Frankfurt/Main, Germany
alex.strobel@gmx.de

Abstract

This commentary discusses the target paper's sharp distinction between neurogenetic mechanisms underlying cognitive abilities and temperament. Evidence for associations of genetic polymorphisms with both temperament traits and cognitive control functions and for a shared or at least overlapping neuroanatomy and neuromodulation of cognitive control and of temperament traits may imply that we should consider the existence of cognitive reaction norms. Copyright © 2007 John Wiley & Sons, Ltd.

Penke et al. (this issue) (PDM) must be applauded for their thoughtful and stimulating review of the evolutionary genetics of personality. Their model of the genetic, neurobiological and environmental influences on cognitive ability and temperament traits provides a broader view on the factors underlying individual differences than many other contemporary models, and the theoretical and practical implications of their integrative approach for personality research go far beyond behaviour genetics.

This commentary relates to PDM's assertion that the distinction between cognitive abilities and temperament reflects different kinds of selection pressures that have shaped distinct genetic architectures underlying cognitive ability and temperament. Indeed, their model may explain why molecular genetic research has been less successful in discovering genetic variation underlying g, while some progress has been made in identifying molecular genetic influences on temperament traits.

However, PDM's sharp distinction between the neurogenetic mechanisms underlying cognitive abilities versus those mediating temperament differences (see Figure 3 of the target paper) may be challenged if we apprehend cognitive abilities not only as to comprise abilities like reasoning, or verbal, numerical and figural abilities, but as to also encompass basic cognitive functions like cognitive control or working memory. Exemplary evidence for this view comes from a twin study by Posthuma, Mulder, Boomsma, and de Geus (2002), who observed a correlation between psychometric IQ, assessed with the WAIS-III, and cognitive control processes, assessed with the Eriksen Flanker task. Interestingly, this correlation was completely mediated by an underlying set of common genes.

In recent years, numerous studies have reported molecular genetic influences on cognitive control or working memory. Intriguingly, accumulating evidence suggests that genetic variation impacting on cognitive functions is also associated with individual differences in temperament traits. In the following, I will shortly review two examples:

(1) Variation in the transcriptional control region of the gene encoding the brain-expressed isoform of the serotonin-synthesising enzyme tryptophan hydroxylase (TPH2), TPH2 G-703T, which is associated with amygdala reactivity to emotional faces (Brown et al., 2005; Canli, Congdon, Gutknecht, Constable, & Lesch, 2005), was shown to be associated with the temperament trait harm avoidance, with individuals without the −703 T/T genotype exhibiting higher scores in harm avoidance (Reuter, Küpper, & Hennig, 2007). In another study (Reuter, Ott, Vaitl, & Hennig, 2007), this polymorphism was also associated with specific measures of executive control as assessed with the Attention Network Test (ANT, Fan, McCandliss, Sommer, Raz, & Posner, 2001), with individuals without the T/T genotype showing enhanced executive control. Supportive evidence comes from an own study (Strobel et al., in press), where individuals without the TPH2 −703 T allele showed less reaction time variability and committed fewer errors than T allele carriers in a continuous performance task.

(2) A polymorphism in the gene encoding the catecholamine-metabolising enzyme catechol-O methyltransferase, COMT Val158Met, which results in reduced enzyme activity in the presence of the Met allele (Lachman, Papolos, Saito, Yu, Szumlanski, & Weinshilboum, 1996), has been related to higher scores in harm avoidance (Enoch, Xu, Ferro, Harris, & Goldman, 2003) and neuroticism (Eley et al., 2003). On the other hand, the Met variant has been associated with better performance in cognitive tests of prefrontal function including better working memory (Egan et al., 2001; Goldberg et al., 2003) and less perseverative errors in the Wisconsin Card Sorting Test (Egan et al., 2001, Malhotra, Kestler, Mazzanti, Bates, Goldberg, & Goldman, 2002).

Several further examples could be given for such pleiotropic effects, e.g. for polymorphisms in the genes encoding brain-derived neurotrophic factor or the serotonin

transporter. It appears that this evidence provides examples for *antagonistic pleiotropy*, i.e. genetic polymorphisms have a positive effect on one trait and a negative effect on another. However, as PDM convincingly argue, antagonistic pleiotropy tends to be evolutionary unstable. Rather, the mentioned findings may be viewed as examples for *structural pleiotropy* (at least in a broader sense), i.e. polymorphisms influence neurobiological mechanisms that are shared by different traits. Indeed, the brain circuitry assumed to be involved in cognitive control (e.g. Miller & Cohen, 2001) shows considerable overlap with structures suggested to modulate temperament traits (e.g. Depue & Collins, 1999; Gray & McNaughton, 2000). This brain circuitry comprises prefrontal cortex, amygdala, hippocampus, nucleus accumbens, thalamus and other structures, with the information flow within this cortico–subcortico–thalamic network being crucially dependent on neuromodulatory influences exerted by dopamine (see Grace, 2000), but also, among others, serotonin (Robbins, 2005). Hence, genetic variation impacting on dopamine function (e.g. via variation in COMT enzyme activity) or serotonin function (e.g. via TPH2-mediated variation in serotonin availability) is likely to influence a number of behaviours associated with the cortico–subcortico–thalamic circuitry, although neuromodulatory influences and the information flow within this network may differ from one situation (being confronted with emotional stimuli) to another (being challenged by demanding cognitive tasks).

How, then, could the evidence for shared or at least overlapping neurogenetic mechanisms underlying both temperament and cognitive control be reconciled with the model proposed by PDM? Perhaps, we might consider to assume a third category besides—or between—cognitive abilities as fitness components under mutation selection and temperament traits as reaction norms with environment-contingent fitness consequences being under balancing selection. I would suggest this third category to comprise *cognitive reaction norms*. These cognitive reactions norms may also be under balancing selection, because cognitive control functions—albeit being *cognitive* in nature and being recruited when cognitive ability is challenged—are *reaction norms* in the sense that they are to some degree also situation- or environment-contingent: There are situations, or environments, where the ability to shield working memory representations against distracting information enhances fitness, and there are situations, or environments, where flexible updating of representations and rapid switching of goals or of the means to achieve them is more appropriate.

It remains to be determined how exactly associations of genetic polymorphisms with cognitive functions and temperament are mediated by variation in the same versus different brain functions. Nevertheless, the assumption of a category of behavioural differences located between and sharing genetic and/or (endo)phenotypic variance with cognitive ability and temperament could help to resolve the ambiguous nature of temperament traits correlated with general intelligence.

The Relevance of Personality Disorders for an Evolutionary Genetics of Personality

ALFONSO TROISI

Department of Neurosciences, University of Rome, Italy
alfonso.troisi@uniroma2.it

Abstract

The epidemiology of personality disorders confirms the importance of the evolutionary approach to a better understanding of individual differences in personality traits and adds credibility to the evolutionary genetic model. A full appreciation of the potential of the evolutionary genetic framework requires a critical revision of current measures of personality. Copyright © 2007 John Wiley & Sons, Ltd.

Penke et al. (this issue) address the unsolved question of explaining persistent genetic variation in personality differences, examine data for and against three evolutionary genetic mechanisms (i.e. selective neutrality, mutation-selection balance and balancing selection), and conclude that balancing selection by environmental heterogeneity seems best at explaining genetic variance in personality traits. The paper focuses on personality differences in the normal range and limits the discussion of personality disorders to sketching some hypotheses that could explain their origin. However, a detailed examination of the epidemiology of personality disorders confirms the importance of the evolutionary approach to a better understanding of individual differences in personality traits and adds credibility to the evolutionary genetic model proposed by Penke et al.

The National Comorbidity Survey Replication (NCS-R) study has recently reported data on the prevalence and correlates of DSM-IV personality disorders in the general population of the United States (Lenzenweger, Lane, Loranger, & Kessler, in press). Two unexpected findings were that personality disorder is a relatively common form of psychopathology (point prevalence: 9.1%) and that a diagnosis of personality disorder not comorbid with Axis I syndromes has only modest effects on functional impairment. Taken together, these findings cast doubt on the traditional view of personality disorders as dysfunctional and maladaptive extremes of normal personality traits produced by rare genotypes and raise the question if these behavioural phenotypes have been adaptive in some environments or during some periods of human evolution. In other words, we cannot exclude that not only normal personality differences but also personality disorders are the product of a set of varying selection pressures favouring different phenotypes under different environmental conditions (Troisi, 2005).

Epidemiological data on personality disorders also suggest that gender and age configure different socio-environmental niches. The DSM-IV general criteria for a diagnosis of personality disorder require that the 'enduring pattern' (as defined in criteria A-C) be 'stable and of long duration...' and '...onset can be traced back at least to adolescence or early adulthood' (criterion D). Such a definition reflects the traditional view of personality disorders as persistent, enduring and stable patterns. However, available data suggest that some personality disorder diagnoses demonstrate only moderate stability and that they can show improvement over time. Cluster B personality

disorders (antisocial, borderline, narcissistic and histrionic personality disorders) tend to become less evident or to remit with age (van Alphen, Engelen, Kuin, & Derksen, 2006). In particular, the behaviour characteristics of antisocial personality disorder (ASPD) first appear during adolescence and often disappear during the 5th decade, and all large-scale epidemiologic surveys of ASPD confirm that at least 80% of those meeting criteria are men. If ASPD is viewed as a risk-taking behavioural strategy, its improvement with age and higher prevalence among males fits with the pattern one would predict from a life-history theory perspective.

Patterns in risk-taking are related to life-history variables, which include gender, age, marital and parental status, amount and predictability of resources and rates and sources of mortality. Among patients with ASPD, ages 15–29 are those of most severe manifestation of the disordered personality traits, including impulsivity, aggressiveness, irresponsibility and sensation seeking. Among males in the general population, these are the years of highest risk for motorcycle accidents and arrest for assault. From a life-history theory perspective, the common explanation for these clinical and socio-demographic findings lies in the role of risk-taking in reproductive competition, which is typically more intense for young men than for women or older men. During the teens and young adult years, competition for social and economic resources is acute, and one's fate in the mating market is being determined. For males at younger ages, the optimal strategy is to take risks to acquire resources for immediate use in reproductive effort, especially when environmental characteristics are uncertain and unpredictable (Hill & Chow, 2002).

In line with this argument, it is not surprising that personality disorders reflecting an internalising dimension (i.e. mood and anxiety), such as for example dependent personality disorder, tend to be more prevalent among women (Torgersen, Kringlen, & Cramer, 2001). In contexts where infant survival would usually depend on the mother's survival more than the father's, women are expected to have been selected for a greater tendency than men for self-preservation (Campbell, 1999).

Another crucial question addressed by Penke et al. is the validity of current measures for studying personality differences from the perspective of evolutionary genetics. The authors appropriately draw attention to the limits of self-report questionnaires, recommend changes based on the assessment of behavioural reactions to specific fitness-relevant situations and argue for a wider use of the endophenotype approach. However, they seem satisfied with the Five-Factor Model of personality and consider attachment styles as non-genetic personality traits. In effect, attachment research has generally presumed environmental mechanisms explaining individual differences in attachment security without, until recently, testing for possible genetic effects. However, in recent years, several behavioural genetic and molecular genetic studies have been conducted, and there is preliminary evidence for gene-by-environment interactions in the development of attachment styles. Recently, the first study combining molecular genetics with measurement of environmental influences (i.e. mothers' unresolved loss/trauma or frightening behaviour) on disorganised attachment has been conducted in children of 14–15 months of age (van IJzendoorn & Bakermans-Kranenburg, 2006). Results showed that the DRD4 polymorphism (short vs. long) and the −521 C/T promoter gene were not associated with disorganised attachment. However, a moderating role of the DRD4 gene was found: Maternal unresolved loss or trauma was associated with infant disorganisation, but only in the presence of the DRD4 7-repeat polymorphism. The increase in risk for disorganisation in children with the 7-repeat allele exposed to maternal unresolved loss/trauma compared to children without these combined risks was 18.8 fold. The T.7

haplotype showed a similar interaction effect: an elevated risk for infant disorganisation in the case of maternal unresolved loss (odds ratio 3.24).

If these preliminary data will be confirmed and expanded, attachment styles could be included among personality profiles amenable to an evolutionary genetic analysis. Such a possibility has been already suggested by Belsky (1999). In contrast with the traditional perspective of clinical psychology that views insecure attachment patterns as reflecting some kind of personality pathology, Belsky has advanced the hypothesis that, in the ancestral environment, all the patterns of attachment were equally adaptive in terms of promoting reproductive fitness in the ecological niches that gave rise to them. According to his hypothesis, the main evolutionary function of early social experience was to prepare children for the social and physical environments they were likely to inhabit during their lifetimes. Thus, attachment patterns could represent evolved psychological mechanisms that used the quality of parental care received during childhood as a cue for optimising adult reproductive strategies, as indicated by the strict association of each adult attachment style with different sexual and parental behaviours.

The Need for Inter-disciplinary Research in Personality Studies

KEES VAN OERS

Department of Animal Population Biology, Netherlands Institute of Ecology, The Netherlands
k.vanoers@nioo.knaw.nl

Abstract

The target paper demonstrates the value of evolutionary genetics for personality research. Apart from a summing-up of concepts, the authors validate their theory with evidence from studies on both human- and animal personality. In this commentary, I want to show the need for inter-disciplinary research to answer questions on personality in psychology and biology. Copyright © 2007 John Wiley & Sons, Ltd.

The target paper provides the reader with a very comprehensive review on how both traditional and modern evolutionary genetics may help us understand the maintenance of personality variation. The paper gives us elaborate explanations of evolutionary genetical processes in combination with clear predictions for personality. Moreover, apart from a sum-up of concepts, the authors critically evaluate the theories of others, and validate their own with evidence from a wide range of studies. Where the authors were not able to confirm their argument with data from human personality research, they easily shifted to work on non-human animals. This clearly shows the importance of studies across disciplines.

Although a recent discovery of animal personalities was suggested in the target paper, several animal psychologists had already started using methods from human personality

research in the 1960s. Studies were mainly on primate species (see Buirski, Plutchik, & Kellerman, 1978). Yet, in spite of the obviousness of personality differences within many animal species (Gosling & John, 1999; Wilson, Clark, Coleman, & Dearstyne, 1994), very little work was carried out in evolutionary research because of the fear of being accused of anthropomorphism. And although the use of animals for studying personality is still controversial (Gosling & Vazire, 2002), animal models have now proven to be a useful tool for studying the underlying physiological and genetical mechanisms of personality (e.g. Koolhaas, de Boer, Buwalda, van der Vegt, Carere, & Groothuis, 2001). These, mainly rodent studies, however were all on captive-bred populations and therefore give no insight into the evolutionary processes that shaped these traits (Merilä & Sheldon, 2001).

Gradually the view changed that measured individual differences are only characterised by an adaptive mean flanked by non-adaptive variation, into the idea that variation in itself can also be maintained by natural selection (Wilson, 1998). Moreover, behavioural ecologists who usually studied one trait at a time now realised that traits do not evolve independently, but from an evolutionary compromise to optimise fitness over a range of traits. Therefore, more and more biological studies now try to integrate personality into evolutionary biology (Sih et al., 2004; Réale et al., 2007). In contrast, psychologists are now trying to integrate evolutionary theory (e.g. Buss, 1991) and evolutionary genetics (presented in the target paper) into the present knowledge on human personality. Evolutionary biology thereby has a long standing tradition in interest in fitness consequences, mostly directly measured by the response to selection on life-history traits (Stearns, 1997).

Two different approaches for studying trait evolution can thereby be recognised, phenotypic and genetic (Lessells, 1999). In a phenotypic approach questions about the adaptive value of a trait are asked and the genetic approach considers the effect of selection, but mainly how selection will affect gene frequencies (see e.g. de Jong & van Noordwijk, 1992; Via & Lande, 1985) and the genetic structure of traits (see e.g. Roff, 1997). The authors show the value of the second approach for understanding evolutionary processes in humans and the similarity with animals. However, as the authors state: 'the central question for an evolutionary personality psychology is: how do psychological differences relate to fitness (the *f*-factor in Miller, 2000c)'. Although in humans, personality has been shown to influence the success of an individual, by affecting social relationships, school- and career success and health promotion and maintenance (e.g. Caspi, Roberts, & Shiner, 2005), the phenotypic approach has still been neglected. What is lacking, are studies that link variation in individual success due to phenotypic variation in personality with life-history characteristics; aiming to explain genetic changes over generations. Since the target paper shows that similar selection profiles are present for humans and non-human animals, similar approaches in measuring fitness should be feasible. Yet, only one study has looked at fitness aspects of human personality traits by comparing reproductive fitness among different groups (Eaves et al., 1990).

One example where direct measurements of selection pressures are needed is presented in the studies of Camperio Ciani et al. (2007). Italian coast-dwellers were compared to people living on three small islands off the coast of Italy. Personality differences were studied and population differences were ascribed to genetic differences due to dissimilar fitness payoffs. However, populations may differ from each other because of many reasons (Roff, 1997). It is therefore even more likely that the differences are not caused by genetic change, but are due to, e.g. differential dispersal patterns, founder effects and genetic drift.

I am conscious of the difficulties in measuring selection in a direct way in studies on human personalities, although I believe that it is mainly disbelief that prevents us doing it. Twin studies could be immensely valuable in this, but they have some methodological limitations, especially since natural experiments do not permit full experimental control. Also the alternative approach suggested in the target paper (the use of endophenotypes) may have a serious drawback: underlying mechanisms like hormonal mechanisms may on hand be used to assess personality differences, on the other hand they also present a context dependent expression of personality (see e.g. Carere & van Oers, 2004).

Animal studies could, however, be helpful in answering questions on selection pressures. They are able to measure the actual consequences of personality differences on life-history characters such as reproduction and survival by manipulating the social and/or non-social environment. Animal studies may thereby profit from the substantial knowledge on personality development and the molecular genetic background of human studies. We need, however, to evaluate current methods how personality is measured, validate similarities between humans and non-human animal personalities and compare relevant selection processes. Promising starting points are a common molecular genetic basis (Ebstein et al., 1996), underlying physiological mechanisms.

In conclusion, many proximate and ultimate factors underlying personality differences remain to be tested in both humans and non-human animals. The two distinct areas (biology and psychology) have built up their own specific knowledge, but the target paper shows that these findings can successfully be combined in building a shared theory. Inter-disciplinary work combining these efforts in cooperative projects would thereby enhance this process and will allow us to measure micro-evolutionary processes that play a role in shaping personality variation in humans and other animals.

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Authors' Response

Evolution, Genes, and Inter-disciplinary Personality Research

LARS PENKE[1,2]*, JAAP J. A. DENISSEN[3] and GEOFFREY F. MILLER[4]

[1]*Humboldt University, Berlin, Germany*
[2]*International Max Planck Research School LIFE, Berlin, Germany*
[3]*Utrecht University, Utrecht, The Netherlands*
[4]*University of New Mexico, Albuquerque, NM, USA*

Abstract

Most commentaries welcomed an evolutionary genetic approach to personality, but several raised concerns about our integrative model. In response, we clarify the scientific status of evolutionary genetic theory and explain the plausibility and value of our evolutionary genetic model of personality, despite some shortcomings with the currently available theories and data. We also have a closer look at mate choice for personality traits, point to promising ways to assess evolutionarily relevant environmental factors and defend higher-order personality domains and the g-factor as the best units for evolutionary genetic analyses. Finally, we discuss which extensions of and alternatives to our model appear most fruitful, and end with a call for more inter-disciplinary personality research grounded in evolutionary theory. Copyright © 2007 John Wiley & Sons, Ltd.

We were gratefully impressed to learn that our target paper received 22 commentaries, coming from disciplines as diverse as traditional personality psychology (**Funder**, **Matthews**, **McCrae**), molecular behaviour genetics (**Bates**, **Lee**, **Strobel**), quantitative behaviour genetics (**Jang**, **Johnson**, **Livesley**, **South and Krueger**, **Rebollo and Boomsma**), evolutionary behavioural ecology (**Dingemanse**, **Réale**, **Sih and Bell**, **van Oers**) and evolutionary psychology (**Campbell**, **Euler**, **Figueredo and Gladden**, **Gangestad**, **Keller**, **Saad**, **Troisi**). This shows the scientific community's high level of interest in understanding heritable personality differences within an evolutionary framework. The volume of commentary is also a testament to the inter-disciplinary challenge such an endeavour entails. We would like to thank all commentators for their thoughtful remarks and constructive criticism.

*Correspondence to: Lars Penke, Humboldt University and International Max Planck Research School LIFE, Berlin, Germany. E-mail: lars.penke@staff.hu-berlin.de

The overarching goal of our paper was to provide a theoretical introduction to evolutionary genetics for personality psychologists. Therefore, we found it especially pleasing that most commentators appeared open to an evolutionary genetic approach to personality, or even applauded it. We take this as an affirmation that our most central message—that personality psychology can benefit from an evolutionary approach grounded in evolutionary genetics—is already widely acceptable, if not fully accepted.

Our second major goal was to try to infer the mechanisms that maintain genetic variation in personality differences, given the predictions from different evolutionary genetic models, and the phenotypic and genetic evidence available from personality psychology. Most commentaries focused on specific assumptions, conclusions or details of our resulting evolutionary genetic model of personality. As **Keller** states, such healthy discussion is crucial in strengthening the relatively young scientific movement of evolutionary behavioural genetics. Of course, our evolutionary model of personality is only one possible reading of the current state of evolutionary genetic theory and the empirical research on human personality. It should be regarded as an initial working model that should be challenged, refined and extended.

In this response to commentaries, we will first reply to objections to the theoretical reasoning and use of empirical evidence in our target paper, and then discuss more general issues—the optimal levels at which we should study the evolutionary genetics of personality, how our model should be extended in the future and which alternatives could be explored further. Because so many researchers from diverse backgrounds made comments that were often quite specific, space limitations did not permit us to reply in detail to every point. However, we tried to address the key recurring themes in this rejoinder, and hope that such debate leads multidisciplinary research on the evolutionary genetics of personality to flourish in the future.

EVOLUTIONARY GENETICS AS A THEORETICAL FRAMEWORK FOR PERSONALITY PSYCHOLOGY

Funder and **McCrae** applauded our approach as being a healthy departure from the early days of 'evolutionary' accounts of personality that could not be refuted by empirical evidence. In contrast, **Bates** criticised our attempt as using 'armchair' evolutionary theorising instead of hard empirical 'field work' to settle the evolutionary history of traits. We think this 'data-first' bias, shared by **Bates** and many other personality researchers, is an understandable reaction to the peculiar history of personality psychology, but is now inhibiting progressive research. Before the trait approach integrated factor-analytic, cross-cultural and behaviour genetic studies of personality structure, personality psychology was a mess—a hodgepodge of Freud, Rogers, Maslow and other 'classic figures' who were long on theory and short on data. Frustration with this history (in which theory has more often retarded research than advanced it) has inoculated many personality psychologists against anything that sounds like theory. Here, we simply point out that evolutionary genetic theory has quite a different status than Maslow's hierarchy of needs, or any other traditional 'personality theory'. Evolutionary genetic theory is the dominant formal way that biologists use to think about the effects of selection, mutation, drift and migration on the genetic structures of traits and populations. It is the mathematical heart of biology, and is rooted in 140 years of progressive research. Well-established evolutionary genetic theories do not share the same limitations of traditional 'personality theories'. In any case,

we repeatedly descended from theory's armchair to compare evolutionary genetic predictions against the current state of empirical knowledge on human personality differences.

CAN WE ALREADY TELL SOMETHING USEFUL ABOUT THE EVOLUTIONARY GENETICS OF PERSONALITY?

To infer evolutionary histories and selective regimes from personality data is indeed a big step, dependent on the quality of both available data and theoretical models. **McCrae** asks if we really know enough to take this step, and **Keller** reminds us to be careful and critical before claiming firm conclusions. Evolutionary genetics, while well-established and intimately intertwined with quantitative genetics in evolutionary biology (**Gangestad**), is a rather new area for most psychologists and behaviour geneticists, who have only just begun to recognise its potential. We would hate to derail such a development through premature conclusions. It is also true that most theoretical models still provide at best ordinal predictions about trait characteristics for realistic evolutionary conditions (**Euler**), and that the relevant empirical data are still incomplete, though maybe not as indecisive as suggested by **Bates**, **McCrae** and **Keller** (a point to which we will return below). Therefore, the model we proposed is not the only possible one, and it should not be understood as conclusive. However, as the Table 1 in our target paper shows, even though the theoretical predictions for individual characteristics of traits shaped by certain evolutionary mechanisms are sometimes vague, the *pattern* of predictions that emerges across various characteristics clearly discriminates between them. Similarly, even though the quality of available empirical evidence for the individual characteristics varies widely, it was the overall pattern of data that struck us and led us to propose the model that general intelligence is under mutation-selection balance, whereas personality traits are under balancing selection.

Before we discuss how decisive the different predictions and lines of evidence really are, we would like to address the usefulness of an 'inconclusive' evolutionary genetic personality model. After all, **Keller** called for an exceptionally high standard of evidence at the current stage. We agree that it is likely too early to draw a *conclusive* model, but we see the merits of proposing a sufficiently *plausible* model to help generate new hypotheses, guide empirical research, and inform theories about personality in general (see **Funder** and **Matthews**). The important point is that a plausible model should be explicitly labelled as such and should not blind researchers to alternatives. Nor should it constrain empirical endeavours, which could lead to scientific myopia. Contrary to **Bates**' reading of our target paper, we did not call for a theory-driven moratorium on any particular kind of research, even including molecular genetic studies on the genetic bases of general intelligence (g). Instead, we explicitly stated that such studies should be done to test the predictions of our model, though they might benefit from being more theoretically informed.

CAN WE ALREADY MAKE INFERENCES FROM GENETIC ARCHITECTURES?

Keller questioned our use of genetic architecture information to infer mechanisms of genetic variance maintenance. This criticism has a theoretical and an empirical aspect that

are somewhat mixed up in his commentary. On theoretical grounds, we have to agree with **Keller** and also with **Figueredo and Gladden** that it is hard at the moment to discriminate between mutation-selection balance and balancing selection based on the relative contribution of non-additive genetic variance (V_{NA}) to the total genetic variance of a trait (i.e. the coefficient D_α). We acknowledged the inconclusiveness of the current literature, but were less explicit about it later on (especially in Table 1, where we simply stated the prediction we regard as most likely). As **Keller** rightly stated, the prediction that the proportion of V_{NA} will be medium for traits under mutation-selection balance and large for traits under balancing selection might be considered as the weakest in Table 1.

However, we do not follow **Keller**'s sudden dismissal of the prediction that V_{NA} will be higher in traits under selection (including mutation-selection balance and balancing selection) than in neutral traits. The argument here is that selection tends to deplete additive genetic variance (V_A), while V_{NA} is largely robust against selection. (On a side note, **Lee** is right that this is an extrapolation from Fisher's fundamental theorem, but a widespread one that is correct under many conditions, e.g. Roff, 1997.) Nor does **Keller** provide a theoretical counter-argument. Instead, he points to the rather independent issue of *empirical* difficulties with the establishment of V_{NA} estimates, a topic on which we totally agree. In humans, most inferences about genetic architectures come from twin studies (e.g. **Livesley**), where the traditional design confounds V_A and V_{NA}, and V_{NA} can only be estimated when shared environmental influences are neglected. In line with Keller and Coventry (2005), we think that studies with large extended twin-family designs would provide the best solution.

Furthermore, **Keller** noted that the unknown scale properties of most personality questionnaires and ability tests render rather uncertain even the existing estimates of genetic variance components from more powerful designs. As support, he cited a recent study by Lykken (2006) in which a scale transformation eliminated the V_{NA} component of skin conductance level, a psychophysiological measure of arousal. However, Lykken (2006) argues that this correction actually served as a statistical control for all kinds of confounding factors beyond arousal that influence skin conductance (e.g. individual differences in the density and reactivity of sweat glands). In such cases where the scale transformation decreases the complexity of the measured construct, a reduction in V_{NA} is what should be expected. This does not undermine the general validity of the untransformed score or the scale of the applied measure; it just shows that the untransformed score reflects a construct that is influenced by several interacting heritable components (Lykken, 2006). In our model, such a transformation would correspond to a statistical control of all but one of the interacting endophenotypic personality mechanisms. If a transformation like that becomes ever possible for personality traits, we would also predict a decline of V_{NA}. We agree with **Keller** (see also **Bates**) that the development of new personality measures with improved scale properties (esp. ratio scales) is highly desirable, but we put more trust in the V_{NA} estimates from extended twin-family designs than **Keller** does. While these results might not help us very much to discriminate between different forms of selection on personality differences (mutation-selection balance vs. balancing selection), they do suggest that selective neutrality of personality, as favoured by **Campbell**, is unlikely.

While it is hardly possible to distinguish between mutation-selection balance and balancing selection based on just V_{NA} estimates, data on inbreeding depression can be more decisive. This is because polygenetic traits under mutation-selection balance should always show inbreeding depression, while only traits under balancing selection through

overdominance will (**Keller**), and overdominance is actually rare and evolutionarily unstable. In this case, the problem is on the empirical side: Experimental inbreeding studies are only possible in non-human animals, and strong natural experiments (e.g. children from cousin marriages) are rare. Fortunately, this kind of inbreeding data exist for intelligence (supporting mutation-selection balance), but is lacking completely for personality traits. In a noteworthy first attempt to fill this gap, **Rebollo and Boomsma** reinterpreted Camperio Ciani et al.'s (2007) study, and also reported their own data, on personality differences between parents and their children who mated with a spouse from a geographically close or distant region. Both studies together suggest that those who mate within the same regions (which may reflect stronger inbreeding effects) have children who are lower on sensation seeking (esp. excitement seeking) and openness to experience, while results are unclear for extraversion and there was no effect for neuroticism, agreeableness, conscientiousness, anxiety or anger. The problem with these results is that they are very indirect and allow for various alternative interpretations. It is especially striking that effects were found exclusively for traits (i.e. sensation seeking and openness to experience) that can be directly associated with migration tendencies and active niche selection. This is most obvious in the worldwide distribution of DRD4 polymorphisms, which suggests that carriers of the allele that has been associated with high sensation seeking are more likely to migrate (Chen, Burton, Greenberger, & Dmitrieva, 1999). It is also striking that **Rebollo and Boomsma** found the sensation seeking difference already in the parent generation, even though we know nothing about the geographical mating habits of their parents. As these authors themselves state, migration might be a plausible alternative explanation for these particular results. What we need next are studies of inbreeding effects on personality traits with stronger designs (some suggestions are given by Mingroni, 2004).

Similar conclusions can be drawn for the other aspects of genetic architecture we discussed: while the theoretical models are specific enough to make predictions that distinguish at least one of the major evolutionary mechanisms for the maintenance of genetic variance from the other two, most empirical evidence on the number of genetic loci, the number of polymorphic loci and the average effect size of loci is still rather indirect. Again, the overall pattern of results allows us to evaluate which mechanism is the most plausible for a given trait, but better data is needed to substantiate these conclusions.

IS MATE CHOICE SIMILAR FOR INTELLIGENCE AND PERSONALITY TRAITS?

On the phenotypic level, evolutionary genetic theory suggests that traits under mutation-selection balance, but not traits under balancing selection or selectively neutral traits, should be sexually attractive in a general, species-typical way. The logic here is that choosing sexual partners based on reliable indicators of low mutation load will endow potential offspring with 'good genes'. In our target paper, we argued that studies on human mate choice support general mate preferences and assortative mating for intelligence, but not for personality traits. **McCrae** doubts this claim. He remarks that studies on self-reported mate preferences often find strong preferences for personal attributes such as 'honest', 'considerate' and 'affectionate', which can be ascribed to the

agreeableness domain. However, aside from the problem that self-reported preferences often do not reflect actual mate choices (Penke, Todd, Lenton & Fasolo, in press), it is important to distinguish between sexual attraction *per se* and pragmatic preferences for long-term mates. Long-term relationships are, ideally, cooperative relationships, so people prefer honest and trustworthy partners for long-term mating relationships, just as in other social relationships (Cottrell, Neuberg, & Li, 2007). The likely reason for this, however, is not sexual attraction *per se*, but the pragmatic avoidance of exploitation, distress, inconvenience and inefficient coordination (called 'relationship load' by Buss, 2006). This becomes obvious in studies where preferences are assessed across different mating contexts and relationship durations (e.g. Kenrick, Groth, Trost, & Sadalla, 1993). These studies show that the preference for agreeableness-related attributes vanishes when a mate is chosen for a sexual affair or a one-night-stand, where not much cooperation is necessary. Furthermore, it is unclear whether the long-term preference for warmth and trustworthiness really reflects the ideal of an agreeable mate personality (i.e. a trait of an individual), or the ideal of a secure attachment relationship (i.e. a dyadic trait) (Penke et al., in press). At least from an evolutionary theoretical perspective, people should seek a long-term partner who is faithful and supportive within the context of the relationship, but people should be less concerned their partner's behaviour towards, for example, alternative mates, rivals or out-group members. Exceptions might be traits like benevolence, generosity, heroic virtues and magnanimity. These agreeable characteristics seem to be sexually attractive in short-term and long-term mates, but apparently because they are reliable indicators of good condition and low mutation load (Griskevicius, Tybur, Sundie, Cialdini, Miller, & Kenrick, in press; Miller, 2007). However, the important point here is that high agreeableness *per se* is not sexually attractive, but some specific forms of agreeableness are generally attractive if they can only be displayed by individuals in good condition. Similarly, only people with high intelligence will be able to convert a high openness to experience into sexually attractive degrees of creativity (Haselton & Miller, 2006; Miller, 2000a). These personality traits are not always sexually attractive in themselves, but can be attractive under certain circumstances, when they advertise good condition and genetic fitness.

McCrae also noted that some degree of assortative mating has sometimes been shown for conscientiousness and openness to experience, but other studies (e.g. Watson, Klohnen, Casillas, Nus Simms, Haig, & Berry, 2004) failed to show assortative mating on these traits. We are not aware of a meta-analysis of the large human assortative mating literature, but the general picture is that assortative mating for intelligence is a well-established phenomenon, while findings are rather weak and inconsistent for individual personality traits.

Finally, **McCrae** mentions that assortative mating can result from social homogamy (i.e. choosing a mate from within one's self-selected social environment, such as college, job or neighbourhood), not just from direct assortment on perceived traits within competitive mating markets. More sophisticated research designs are able to disentangle these two alternatives, and they reveal that direct preferences exist independent of social homogamy, especially for intelligence (e.g. Reynolds, Baker, & Pedersen, 2000; Watson et al., 2004).

Overall, we think it is fair to say that intelligence is very often directly preferred in mate choice, while the evidence does not support such a general conclusion for personality traits.

IS PERSONALITY EVOLUTIONARILY RELEVANT AT ALL?

The most important kind of evidence to distinguish between selective neutrality and any selection-based account for heritable personality differences (including mutation-selection balance and balancing selection) is the empirical link between personality and fitness. Only if personality differences have behavioural consequences that influence fitness, can we posit that some form of selection acts directly on personality. Fitness is ideally operationalised as the relative long-term (multi-generational) reproductive success of genotypes in populations, but phenotypic selection studies have established more practical operationalisations of fitness, such as measured reproductive success over a single lifespan or even shorter periods such as breeding seasons (**Réale**). Since the necessary data are not hard to gather for human personality, it is both surprising and unfortunate that human phenotypic selection studies are extremely rare. More of these studies are desperately needed to inform an evolutionary genetic approach to personality (**Dingemanse**, **Réale**, **van Oers**).

But does this mean that we have to fall back to the most 'parsimonious' baseline model of selective neutrality, as suggested by **Campbell**? We do not think so. As calculated by Keller and Miller (2006), the correlation between a truly neutral trait and fitness must not be greater than $\pm .0055$ (i.e. the square root of the maximal .003% fitness difference under which genetic drift is a more important factor than selection, given typical ancestral human population sizes). This effect size is greatly exceeded in the few studies that directly link personality differences to general reproductive fitness in humans (e.g. Eaves et al., 1990) and other animals (see Dingemanse & Réale, 2004), and in the much more numerous studies that link personality differences to specific components of human fitness (such as survival, social status, mating success and reproductive strategies; see our target paper for references). Even if it turned out that genetic drift had been somewhat stronger throughout our evolutionary history than assumed in Keller and Miller's calculation (**Réale**), which would allow somewhat greater effect sizes for neutral traits, and even though the effect sizes for single fitness components should be interpreted with caution because of evolutionary trade-offs (**Réale**), it seems highly unlikely that all of the well-documented behavioural consequences of personality differences are invisible to selection.

Finally, note that the selective non-neutrality of personality differences contradicts not only Tooby and Cosmides' (1990) neutrality account for the maintenance of genetic variance in personality, but also their pathogen-defence hypothesis (**Campbell**, **Livesley**). This hypothesis assumes that the *behavioural* consequences of personality differences are so invisible to selection that their genetic foundations can vary freely, such that the organism's proteome is more distinctive, unpredictable and harder for pathogens to exploit. Even if all personality-related polymorphisms (such as DRD4 or 5-HTTLPR) had pleiotropic effects at the level of organismic biochemistry that are relevant to anti-pathogen defence, any such anti-pathogen effects would need to be larger than the behavioural fitness payoffs of personality differences, in order for the pathogen-defence model to be applicable. The same logic must hold for any similar hypotheses that regard heritable personality differences as by-products of other adaptations (**Keller**). We would also like to add that, despite the ingenuity and prominence of the Tooby and Cosmides (1990) paper, we are not aware of studies that have directly tested the pathogen-defence model. So far, we regard our model as a more plausible alternative.

ENVIRONMENTAL CHALLENGES FOR AN EVOLUTIONARY
PERSONALITY PSYCHOLOGY

Our model suggests that future phenotypic selection studies should pay special attention to the way that human personality traits interact with specific environments. If spatio-temporal environmental heterogeneity is responsible for maintaining genetic variance in personality traits, then the correlation between a trait and fitness should reverse across some environments. Thus, certain environmental variables should act as statistical moderators of the relationships between personality traits and measures of survival, reproductive success and/or kin success. A methodological implication is that we need more precise, valid and evolutionarily informed ways of categorising and measuring the environmental factors that interact with personality traits to yield adaptive or maladaptive behaviour (**Funder, Matthews, Saad**). Characterising environmental structure at a useful level of description is a rather old problem that psychologists recognised long ago (see Meehl, 1978), but still struggle to solve (for a notable attempt see Holmes, 2002).

While we cannot offer a panacea, we suggest that an evolutionary framework for personality, richly informed by mid-level adaptationist theories (e.g. concerning kin selection, multi-level selection, reciprocity, sexually antagonistic coevolution, parent-offspring conflict and life-history theory) might help to isolate relevant environmental features. This is because environments can vary in many ways (**Johnson**), but not all of them are equally relevant for understanding the fitness payoffs of particular traits. A useful exploratory heuristic might be to consider 'Which variable environmental factors create different adaptive problems that are solved better or worse by individuals with certain personalities?' For example, big cities with high population densities and anonymous interactions might give Machiavellian cheaters more chances to exploit others than small villages would, in which reputations spread faster through gossip; thus, cities may offer higher fitness payoffs for disagreeable individuals than small villages do. Living in big cities might also imply frequent changes in people's social networks, which lead to persistent uncertainty about one's social status and mate value, and about the pool of available mates, friends and allies. Neurotic fears of social rejection might be as maladaptive in this context as an indiscriminate tendency to strive for the alpha rank all the time. Harsh and dangerous physical environments likely make social cooperation and mutual support necessary, as do intergroup conflicts over limited resources, so both may favour agreeableness and neuroticism. More generally, the differences in styles of social interaction that are at the core of many personality traits suggest that we should pay special attention to social-environmental factors that may mediate and modulate relationships between personality traits and fitness payoffs. Such a research program is already exemplified by work on sociosexuality as a personality trait with different mating payoffs in different environments (Gangestad & Simpson, 2000).

Saad emphasised the four different roles that environmental factors play in an evolutionary genetic approach to personality. So far, this section only discussed environmental niches, which provide selection pressures. Two of Saad's other environmental roles, the ontogenetic environment of personality development and the current real-time situational context of personality functioning, are combined in our reaction norm model as the 'environment' that interacts with the genotype to evoke a behavioural response. These two different functions of the environment as (1) the source of selection pressures and (2) one of the interacting factors in reaction norms (which correspond to the two 'Environment' boxes in Figure 3 of the target paper) appear to be

mixed up in one of **Funder**'s remarks: The ontogenetic and real-time environmental factors that evoke personality differences (an interaction effect) might or might not be the same across different environmental niches that select for or against these differences (a main effect). In future evolutionary genetic studies of personality, it should be helpful to distinguish more carefully between the environmental factors that shape a phenotypic personality trait and the environmental factors that make this trait have certain fitness cost and benefits.

The fourth role of the environment that **Saad** acknowledges is the ancestral EEA. Contrary to the commentaries by **Livesley** and **Bates**, the more evolutionarily remote and ancient forms of this environment play a negligible role within an evolutionary genetic perspective on current heritable variation in human personality. Understanding the remote Pleistocene EEA is very useful to explain non-heritable conditional strategies and universal sex differences (**Troisi**, **Saad**), as in mainstream adaptationistic evolutionary psychology. However, genetic variation in contemporary human populations depends on much more recent selection pressures over the last few hundred generations, within the Holocene. Thus, an evolutionary personality psychology may end up paying much more attention to the environment-specific payoffs for personality traits during recent (e.g. Neolithic) prehistory, and even within historically documented civilisations. For example, the divisions of labour and diverse social roles that emerge within complex hierarchical societies may have permitted a much wider range of personality traits to flourish than would have been possible under small-scale, egalitarian, hunter-gatherer conditions in the Pleistocene.

AT WHICH LEVEL SHOULD WE STUDY PERSONALITY TRAITS FROM AN EVOLUTIONARY PERSPECTIVE?

We have apparently reached one of those intriguing points in the history of science when there is a mutual recognition between two fields that they have been working on the same problems in slightly different but complementary ways. In this case, the two fields are evolutionary behavioural ecology (the study of variation in animal behaviour) and personality psychology (the study of variation in human behaviour) (**van Oers**). Such times of mutual recognition are always accompanied by initial confusions over terminologies, assumptions, methods and objectives, before the two fields can take full advantage of each other's insights and findings. Evolutionary ecologists, who usually study animals that cannot report their thoughts or feelings, naturally must focus on observed behaviours, and their correlations, contingencies and fitness consequences across environments. Since personality psychologists have restricted their studies to a very talkative mammal, they usually prefer to ask their subjects to verbally report their thoughts and feelings, and to look for latent personality constructs that can explain patterns across these self-reports (**Réale**). In terms of the watershed model, evolutionary ecologists usually start their analyses more 'downstream' than personality psychologists (**Euler**)— by observing emitted strategic behaviour rather than by recording verbal responses about intended or remembered behaviour.

Evolutionary ecologists usually have a solid training in evolutionary genetics, and they know that selection does not operate on a single trait at a time, but affects all traits that are genetically intercorrelated at once. That is why one objective of animal personality studies is to find behavioural tendencies that are genetically correlated (the 'character state perspective', **Dingemanse**, **Sih and Bell**), to understand how patterns of genetic variance

and covariance in behavioural propensities fit into the genetic variance–covariance matrix (the 'G matrix') that describes all phenotypic traits, whether morphological, physiological or behavioural. The higher goal is to identify fairly independent dimensions in the G matrix, since these dimensions could also evolve fairly independent of each other. Consequently, these dimensions would constitute the most suitable units of analysis for evolutionary genetic studies (Mezey & Houle, 2003).

Personality psychologists are very familiar with looking for independent dimensions in variance–covariance matrixes, using methods such as factor analysis. However, they started doing so many decades before evolutionary ecologists did, and tended to use phenotypic correlations among cognitive tests, or among self- or peer-ratings on personality-descriptive adjectives or questionnaire items, rather than among field observations of actual behaviour. This search culminated in the discovery of independent, latent phenotypic dimensions in humans, of which the g-factor and the FFM of Personality reached the highest consensus. Most interestingly, these dimensions replicate fairly well on the genetic level (e.g. Plomin & Spinath, 2004; Yamagata et al., 2006), suggesting that research on human personality has already come close to characterising the genetically correlated dimensions that evolutionary ecologists are still seeking in other species. What is now called the 'character state perspective' in evolutionary ecology is so fundamental to personality psychology that we simply took it for granted in our target paper. Resolving such terminological and methodological confusions might be the most important first step for inter-disciplinary personality research.

When **Livesley** and **McCrae** suggested instead that lower-order, interdependent personality facets may be the best level of analysis for an evolutionary genetics of personality, they may have confused the heritable individual differences relevant to personality research with the species-typical, domain-specific adaptations studied by adaptationistic mainstream evolutionary psychology. A hallmark of adaptations is their complex functional design, which would break down when too much genetic variation is introduced. As a consequence, most heritable individual differences cannot be adaptations (Tooby & Cosmides, 1990) and they cannot be analysed using traditional standards of adaptationism. Rather, they are dimensions of genetic variation that are tolerated within systems of interacting adaptations. For example, humans are likely endowed with adaptations to regulate attachment relationships (**Troisi**), to discover signs of social rejection (Leary & Baumeister, 2000) and to monitor environmental dangers (Nettle, 2006). All these systems are under strong stabilising selection to function effectively (which maintains their complex adaptive design), but they are still all influenced by individual differences along a heritable dimension called neuroticism. This dimension of personality variation is not at the same level of description as the adaptations themselves, and is maintained by different selective forces—according to our model, by balancing selection given environmental heterogeneity—rather than stabilising selection for raw functional efficiency.

The lower-level facets of broad personality dimensions show substantial genetic intercorrelations (Yamagata et al., 2006) and will thus show correlated responses to selection. This makes them unlikely to be the most useful units of analysis in studying the evolutionary genetics of personality traits. That being said, we are open to ongoing debate concerning which and how many personality factors best represent independent dimensions of variation in the behavioural aspects of the human G matrix. We concentrated on the dimensions of the FFM, mainly because of their clarity and familiarity, and the rich literature on them. **South and Krueger** as well as **Figueredo and Gladden** suggested that there may be even higher levels of abstraction than the FFM, as

suggested by the evidence of modest phenotypic (Markon et al., 2005) and genetic intercorrelations between the FFM domains (**Johnson**). One problem with such jumping to a higher level of abstraction is that some genetic correlations may be different from zero at a statistical level of significance, but not at an evolutionary level of significance (cp. **Jang**): these genetic correlations may be caused by environmental factors through gene-environment interactions (GEIs), making them environment- and population-dependent. In the target paper, we adopted van Oers et al.'s (2005) argument that genetic correlations due to structural pleiotropy (i.e. shared mechanisms on the endophenotypic level) should not change signs across environments, whereas those due to GEIs should change signs across environments. **Johnson** noted that this criterion might fail to distinguish between types of genetic correlations because people select, create and evoke their own environments, leading to gene-environment correlations (r_{GE}s). The effects of GEIs and r_{GE}s can easily be confused in empirical results and are difficult to separate (but see Johnson, 2007). **Johnson** argues that r_{GE}s are problematic because they could lead to a homogenisation of the populations in certain environments with regard to the traits under study (if its result is that every niche harbours only individuals with exactly those personality trait levels that fit best to the niche's demands). In this case, the genetic correlations could indeed be attenuated by reduced trait variance—possibly down to zero, given perfect r_{GE}s. However, we do not see how the variance reduction within environments that could be caused by r_{GE}s can lead to artificial *sign changes* in genetic correlations across environments. But even if the discriminatory power of the criterion offered by van Oers et al. (2005) is limited in certain cases, we do not follow **Johnson**'s conclusion that this (possible) methodological issue with the detection of structural pleiotropies implies that they are rare in nature.

More critical is **Dingemanse**'s remark that the **G** matrix is not static and might differ between environments and populations as a result of local selection pressures. Genetic correlations that freely evolve between populations are likely not constrained by structural pleiotropy, but may be the result of selection for limited plasticity. While this does not make them less interesting from an evolutionary genetic perspective, some of our arguments would indeed be invalidated (see **Dingemanse**). We think that the key data to distinguish between structural pleiotropy and selected limits on plasticity would come from cross-cultural studies. If the factorial structure of the behavioural aspects of the **G** matrix replicate across populations around the world, it is unlikely to reflect recent, local selection pressures. Initial data suggest that the FFM shows good replication of genetic factorial structure across three populations from three continents (Yamagata et al., 2006). This and other studies also suggest that the structure of the behavioural aspects of the **G** matrix reflects fairly accurately the phenotypic structure of the FFM personality dimensions, which allows us, according to the protocol suggested by Roff (1997, p. 100), to use phenotypic structures as a surrogate for genotypic structures. Phenotypic data is available for a larger sample of cultures, and again they suggest that the FFM structure replicates rather well across populations (McCrae & Allik, 2002). While more cross-cultural (and within-culture cross-environmental) comparisons of **G** matrices would be desirable (preferably with designs that are able to differentiate between additive and non-additive genetic variance), these results suggest that the structure of the FFM is caused by structural pleiotropy across behavioural propensities within each of its main dimensions. It remains to be seen, however, if other genetic factor solutions replicate better across cultures, or if the FFM dimensions (and the *g*-factor, for which a similar logic holds) already are the best level to study the evolutionary genetics of personality.

EXTENSIONS OF OUR EVOLUTIONARY GENETIC MODEL
OF PERSONALITY

Evolutionary genetics is a rich and complex field, and offers much more to personality psychology than we could cover in our target paper. Since evolutionary genetics is novel ground to most personality psychologists, we chose to focus rather simply on the major evolutionary mechanisms that can maintain genetic variation in traits. Also, we tried to rely on theoretical arguments and models that are already well-established and relatively uncontroversial in evolutionary genetics. So, for example, we did not discuss the new but sketchy literature on the maintenance of genetic variance in reaction norms (**Sih and Bell**), where the current conclusions depend on the specific assumptions of complex models and are sometimes contradictory (see de Jong & Gavrilets, 2000 vs. Zhang, 2006). Also, we could only make parenthetical references to some other topics, such as niche picking (a form of active r_{GE}). In the future, our model should be extended by including, among others, a more explicit account of r_{GE}s (**Jang, Johnson, Sih and Bell**), reactive heritability beyond condition-dependency (**Gangestad**), indirect selection in social groups (**Sih and Bell**) and models of genetic variance maintenance in reaction norms (**Sih and Bell**). It should also be contextualised within the broader frameworks of evolutionary game theory (**Sih and Bell**) and life-history theory (**Gangestad**). We regard these extensions as generally compatible with our model, but more theoretical and empirical work is needed to see how exactly they would affect our conclusions about the origins and nature of genetic variation in personality. **Dingemanse** reminded us that our model and any future extensions should ideally be tested in formal mathematical models, not just as verbal descriptions. Furthermore, statistical models are needed that allow us to test these relationships against empirical data. First steps in this direction have already been made (**South and Krueger**, **Johnson**; Wolf et al., 1998), but there clearly is plenty of work that still needs to be done.

In itself, an evolutionary genetic model of personality cannot offer a complete theory of personality. It can provide an ultimate perspective on why heritable personality differences exist, how they change over evolutionary time and environments and which fitness effects they may have. This makes it an important building block of any comprehensive personality theory. In the end, however, any evolutionary genetic model of personality should be complemented by more proximate theories (such as **Matthew**'s) concerning the phenotypic structure, underlying mechanisms and lifespan development of personality traits. However, as **Funder** correctly noted, our evolutionary genetic model of personality is more compatible with some proximate personality theories than with others, and those theories that contradict it will have to provide alternative accounts for the existence of genetic variance in personality. In the following, we will compare our model to some alternatives suggested in the commentaries.

ALTERNATIVES TO OUR EVOLUTIONARY GENETIC MODEL
OF PERSONALITY

Recent selective sweeps

Mutation-selection balance models assume that within any given population, for any given trait, there is an abstract, idealised, mutation-free genotype that would show optimal

adaptation to the population's environmental demands and selection pressures. Applied to the case of human intelligence, mutation-selection balance models suggest that the highest possible *g* level can be attained only when all genes that influence cognitive functioning are free of harmful mutations. **Lee** called this a 'Platonic ideal'. Both **Bates** and **Lee** pointed to studies suggesting that human general intelligence has been subject to recent selective sweeps and in the midst of a genetic transition (Evans, Vallender, & Lahn, 2006; Wang et al., 2006; see also Williamson et al., 2007). We agree that the hypothetical optimal genotype for optimal intelligence is an oversimplification, and might be better conceptualised as a 'moving target'. Most mutations in protein-coding and regulatory regions of the genome are harmful, but beneficial mutations are more likely to occur when environments change. Given all the changes that have been occurring in the human ecology during the last 20 000 years, (including larger social groups and mating markets, novel habitats, agriculture and literacy), it is very likely that some *g*-related mutations have become beneficial and are currently on their way to fixation. These newly favoured polymorphisms might exist at any current prevalence level, and might have large phenotypic effects, so molecular genetic studies might be better able to identify them.

Beneficial mutations that are on the rise probably contribute to the genetic variance of *g*, but so does a load of many, rare, small-effect harmful mutations. This is not only a widespread empirical conclusion (Plomin, Kennedy, & Craig, 2006), but also a necessary implication if *g* has a large mutational target size. (**Bates** notes that several thousand rare polymorphisms with strong effects on general intelligence have been identified, but these evolutionary transient, harmful mutations usually cause severe mental retardations, not individual differences in the normal range; see Plomin & Spinath, 2004.) In our view, only a conceptualisation of *g* as a downstream trait that represents the functional integrity of large parts of the brain and the genome can explain why there are positive-manifold genetic correlations between different cognitive abilities, why *g* is linked to general phenotypic condition and why *g* is sexually attractive. It can also explain why trauma often reduces, but never raises, *g* (**Keller**; Keller & Miller, 2006). We do not see how these findings can be reconciled with recent selective sweeps as the only explanation for the heritability of *g*. In an effort to refute our mutation-selection balance account, Bates referred to unpublished evidence of a zero genetic correlation between *g* and fluctuating asymmetry. While we cannot evaluate this study, such a result would challenge only one possible mediator between mutation load and cognitive ability (the construct of 'developmental stability'), not the general claim that *g* is under mutation-selection balance. Contrary to **Bates** and **Lee**, we doubt that recent selective sweeps alone can explain most of the genetic variance in *g*, but we believe that such sweeps, in conjunction with mutation-selection balance, may be important, with their relative contributions to be determined by future empirical research.

Cognitive reaction norms

While the *g*-factor of intelligence seems to have a direct link to many components of fitness, **Strobel** noted that individual differences in certain lower-order cognitive processes show phenotypic and genetic relations to personality traits—which, in our balancing-selection model, should have net fitness neutrality when averaged across all relevant environments. He suggests that such lower-level traits that combine cognitive and personality characteristics may constitute a third category of traits to consider in extending our model. We do not think that such a fundamental modification is necessary. Our two

trait categories are basically defined by the selective mechanisms that maintain their genetic variance, not by their apparent psychological nature (i.e. 'cold' and cognitive vs. 'hot' and temperamental). If the lower-order cognitive processes discussed by **Strobel** are indeed under balancing selection and structurally linked to personality traits, they clearly fall in the 'reaction norm' category of our model and are likely best conceptualised as facets of certain personality traits. However, since they are usually assessed by cognitive tests that load on the g-factor, it might be advisable to control for g (which we suggest captures mutation load variance) when their genetic underpinnings and their associations with personality traits are studied.

Gene-environment correlations

It is hardly debatable that humans have been perfecting ways to modify their own environments for thousands of years. Due to technical and cultural innovations, modern humans seldom face unmodified natural ecologies; rather we confront complex built environments and social institutions that have been shaped as our 'extended phenotypes'. Consequently, r_{GE}s might be more important for humans than for any other species. **Jang** and **Johnson** argued that modern humans are so adept at creating, selecting and evoking their own ideal environmental niches that almost no genetic variance in personality is lost to selection now. We agree that modern selection-minimising environments might be one reason why 'maladaptive' genetic variants, like those leading to mental illnesses, are sometimes preserved in the population (**Jang**). However, we doubt that r_{GE}s can fully explain genetic variance in the normal range of personality. The reason is that mere survival is not the only adaptive problem—fitness also depends on success in social competition for resources, status and mates. In modern societies, few will die because they are ill or incompetent, but many will fail to maximise the quantity and quality of their sexual partners and offspring (e.g. Keller & Miller, 2006). As we argued in our target paper, it is likely that personality differences have their strongest effect on fitness in the social domain (see also **Matthews**). As long as diverging interests exist in social groups, no single individual will have full control over his or her social environment (**Sih and Bell**; Penke et al., in press). Some will do better than others, partly due to luck, but primarily due to individual differences in general fitness and variation in the fit between people's personalities and their (socio-)environmental niches. Thus, r_{GE}s may alter or attenuate the selection pressures on personality differences, but they are unlikely to eliminate them. Note also that if r_{GE}s indeed neutralised all selection pressures, personality differences would be under neutral selection, which is, as we argue in our target paper and above, inconsistent with empirical evidence. Accordingly, r_{GE}s alone cannot maintain genetic variance.

Antagonistic pleiotropy

Sih and Bell remark that antagonistic pleiotropy is still discussed as a viable mechanism for maintaining genetic variance, for example by Roff (2002). While it is true that the final word has not been said about this mechanism (especially when trade-offs between more than two traits are involved), even Roff, in a recent review (Roff & Fairbairn, 2007), regards antagonistic pleiotropy alone as very unlikely to explain persistent genetic variance. However, even if some genetic variance in some personality traits is maintained by antagonistic pleiotropy, it would not alter our model dramatically. All it would imply is that environmental heterogeneity is not necessary in these particular cases.

Continua of evolutionary stable strategies

Keller lists MacDonald's (1995) hypothesis of weak stabilising selection on personality traits (which allows for continua of evolutionary stable strategies) as a viable explanation for genetic variation in personality. However, stabilising selection, even if weak, can only erode, but never maintain, genetic variation (Roff, 2002). The same is true for the related mechanism of correlational selection (**Sih and Bell**; Roff & Fairbairn, 2007). In both cases, either personality traits must be selectively neutral, or the mutational target size of personality traits must be sufficiently large that enough mutational variance is reintroduced (see **Gangestad**), or some form of balancing selection must occur. This brings us back to the three main mechanisms we discussed.

The *K*-factor

The *r-K* continuum describes differences in life-history strategies between species. Each species has evolved a complex functional design that allows for its specific strategy of growth, mating and parenting. For example, many finely coordinated adaptations in a rat's phenotype interact to let it mature fast, reproduce early and often, develop a small brain, refrain from extensive parental investment, die early, etc., and these systems of adaptations are different from those in an elephant or human. Such an *r-K* continuum might apply not just to explain between-species differences in life-history adaptations, but to explain within-species differences in behavioural strategies and personality differences. **Figueredo and Gladden** suggested that the human **G** matrix might be characterised by just one principal dimension—the '*K*-factor'—corresponding to individual differences in life-history strategies and their associated personality traits. Our concerns with this suggestion are mostly theoretical.

We do not see how such an all-encompassing genetic dimension can be maintained by frequency-dependent selection or any other form of balancing selection. Selection cannot change the whole adaptive design of a species back and forth at the level of all genetic loci that influence life-history traits, since this would inevitably break up the complex functional coordination of the life-history strategy (Tooby & Cosmides, 1990). Instead, balancing selection can only maintain a small set of polymorphisms that act as 'switches' between different life-history or behavioural strategies (Kopp & Hermisson, 2006; Turelli & Barton, 2004). These polymorphisms must, through cascading effects in genomic regulatory systems, affect all adaptations involved in the strategy. One—and possibly the only—example for such a potent genetic switch in humans is the SRY gene that guides the sexual differentiation of males and females (Tooby & Cosmides, 1990). In the case of the *K*-factor, a similar master genomic regulatory switch would have to be identified (and we suspect it already would have been discovered if it existed, given the intensity of gene-hunting for loci with major behavioural effects). Such a master regulatory switch might, for example, affect a range of behavioural traits by regulating testosterone levels and receptor sensitivities during brain development and functioning, since testosterone affects a wide range of sexual, competitive, aggressive and parental behaviours (Ellison, 2001). However, testosterone-related polymorphisms alone cannot explain all the other traits subsumed in the *K*-factor, including general intelligence and the dimensions of the FFM. As long as there is no evidence for more potent genetic switches that affect all these traits, we regard *K*-factor theory as slightly over-ambitious in trying to explain human individual differences. Alternatively, **Gangestad** offers some more detailed considerations on the

evolutionary genetics of life-history strategies, including reactive strategy adjustment to one's own mutation load (i.e. condition-dependency). We encourage future studies to proceed in the directions he suggests.

CONCLUSION

Our target article introduces a way to study personality from an evolutionary perspective, based on evolutionary genetics. Thereby, it supplements adaptationistic evolutionary psychology with a toolbox for the study of individual differences, and it supplements behaviour genetics and personality psychology with a theoretical framework to understand heritable personality differences. We reviewed three theoretical models for the maintenance of genetic variance in heritable traits, and assessed the available empirical evidence to draw conclusions about the plausibility of each model as it might apply to human personality. While some aspects of the evidence remain weak, the overall pattern of results suggests that balancing selection is more plausible than its alternatives as an explanation for most heritable personality traits, as is mutation-selection balance for general intelligence. It remains to be seen whether our model can integrate future theoretical innovations and empirical findings. We are open to alternatives, extensions, modifications and most importantly empirical studies with more refined methods that test the predictions of our model.

Clearly, the development of a comprehensive evolutionary personality psychology is a big challenge that is still to be met. Many of the challenges and opportunities in this endeavour lie in its inter-disciplinary nature: neither psychologists nor biologists will be able to solve this problem on their own (**van Oers**). The commentaries are encouraging because they suggest that both sides are willing to learn from each other. If basic communicative issues (terminologies, etc.) can be resolved, we see many opportunities for fruitful inter-disciplinary cooperation, and maybe we can even come a little bit closer to the utopian ideal of consilience (**Euler**).

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