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

OPEN PEER COMMENTARY

Out of the Armchair

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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.

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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?

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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

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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

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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

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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.

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,

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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,

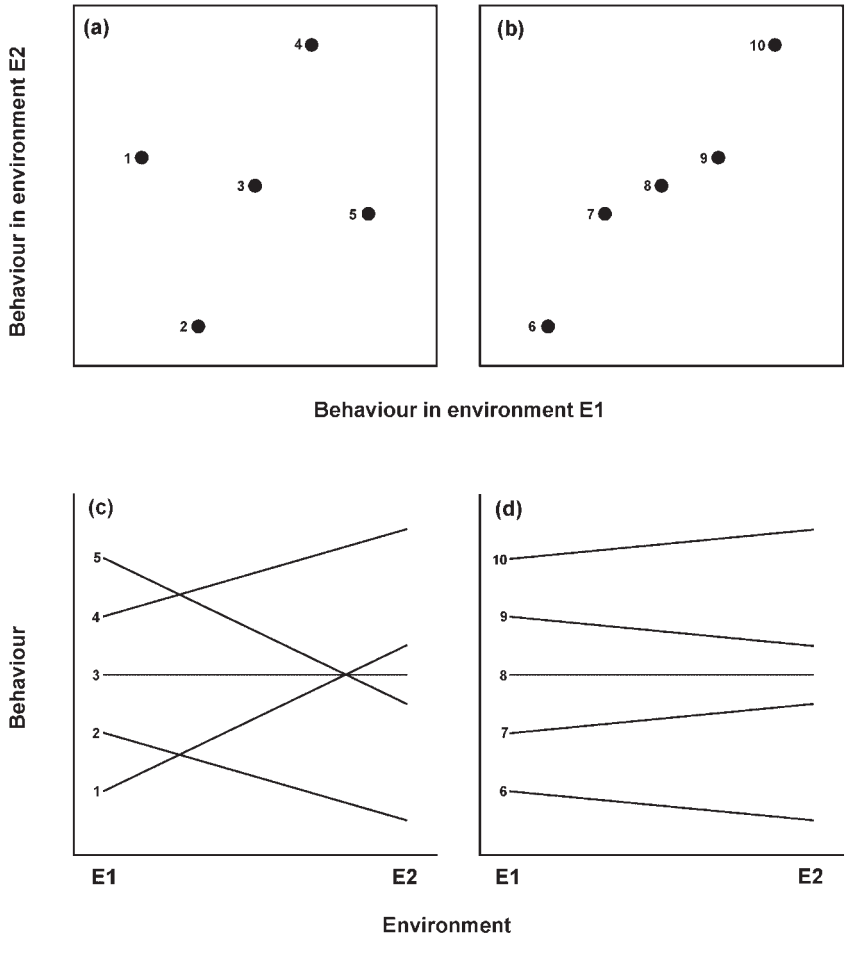

Figure 1. Graphical illustration of genetic variation in personality as viewed from (a–b) a character state approach (both panels plot the breeding values of two behaviours) or (c–d) a reaction norm approach (both panels give breeding values of the same behaviour expressed in two different environments). 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).

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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).

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Consilience is Needed, and Consilience Needs Bipartisan Expertise

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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

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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

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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

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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

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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

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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.

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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).

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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

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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.

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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

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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.

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Behaviour Genetics' Neglected Twin: Gene-Environment Correlation

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Abstract

The target paper posits that the driving force behind balancing selection is gene-environment interaction ($G \times E$) that describes environmental control of genes. It is argued that $G \times E$ 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 ($G \times E$) and recent empirical research that has shown to exert a major influence in personality and psychopathology. $G \times E$ 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 $G \times E$ 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

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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.

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Don't Count on Structural Pleiotropy

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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

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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.

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Standards of Evidence in the Nascent Field of Evolutionary Behavioural Genetics

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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.

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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?

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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?

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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.

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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

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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

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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

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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

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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

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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

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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.

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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?

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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

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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*,

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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?

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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.

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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

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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.

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Personality: Possible Effects of Inbreeding Depression on Sensation Seeking

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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

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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'. *$p < .05$; **$p < .01$.

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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

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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

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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

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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

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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?

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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 × 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

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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

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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

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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

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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 × E: the 'environment' of conflict with parents changes the 'genetic' effect on negative emotionality. However, the finding does not fit neatly into the G × 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

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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.

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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

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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.

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The Relevance of Personality Disorders for an Evolutionary Genetics of Personality

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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

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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

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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

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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

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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.

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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. Interdisciplinary 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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