Available online at www.sciencedirect.com

ScienceDirect

Current Opinion in Psychology

# The evolutionary genetics of personality revisited

Lars Penke[1] and Markus Jokela[2]

Like all human individual differences, personality traits and intelligence are substantially heritable. From an evolutionary perspective, this poses the question what evolutionary forces maintain their genetic variation. Information about the genetic architecture and associations with evolutionary fitness permit inferences about these evolutionary forces. As our understanding of the genomics of personality and its associations with reproductive success have grown considerably in recent years, it is time to revisit this question. While mutations clearly affect the very low end of the intelligence continuum, individual differences in the normal intelligence range seem to be surprisingly robust against mutations, suggesting that they might have been canalized to withstand such perturbations. Most personality traits, by contrast, seem to be neither neutral to selection nor under consistent directional or stabilizing selection. Instead evidence is in line with balancing selection acting on personality traits, probably supported by human tendencies to seek out, construct and adapt to fitting environments.

**Addresses**
[1] Georg Elias Müller Institute of Psychology, Georg August University Göttingen, Gosslerstr. 14, 37073 Göttingen, Germany
[2] Institute of Behavioural Sciences, University of Helsinki, Siltavuorenpenger 1A, 00014 University of Helsinki, Finland

Corresponding author: Penke, Lars (lpenke@uni-goettingen.de)

**Current Opinion in Psychology** 2016, **7**:104–109

This review comes from a themed issue on **Evolutionary psychology**

Edited by **Steven W Gangestad** and **Joshua M Tybur**

For a complete overview see the Issue and the Editorial

Available online 3rd September 2015

http://dx.doi.org/10.1016/j.copsyc.2015.08.021



Personality traits are relatively stable dimensions of individual differences in cognition, affect and behavior. Human personality traits can be organized around five independent dimensions: extraversion, agreeableness, neuroticism, conscientiousness and openness to experience. These dimensions are of interest, as they are substantial predictors of important life outcomes, from educational and occupational attainment to lifetime reproductive success and longevity [1••,2,3]. The strongest single predictor of any personality trait is the standing of one's biological parents on the same trait: about 50% of the variation in broad personality traits is genetically heritable [4]. The finding is hardly surprising, as virtually all human traits ever studied with quantitative behavior genetic designs (e.g., twin and adoption studies) show substantial genetic components [5•] — a finding so robust that it has been enshrined as the first law of behavior genetics [6]. This raises the question of how genetic variation in personality traits has been maintained in populations over evolutionary time. In 2007, Penke, Denissen and Miller [7,8] proposed that the maintaining evolutionary forces can be inferred from the genetic architecture of traits as well as their associations with reproductive fitness. After summarizing the evidence available at that time, they concluded that personality traits are unlikely to be neutral to selection. For general intelligence, a balance between steadily occurring deleterious mutations and directional selection toward higher intelligence appeared to be the best explanation. Personality traits, by contrast, seemed to be under balancing selection, where selection pressures in different directions affected the same traits at different times or in different places, in a way that no genetic variant underlying personality traits is consistently favored over others.

## Recent evidence on the genetic architecture of intelligence

A lot of progress has been made since the publication of Penke and colleagues' article [7,8], particularly in the field of genomics. We now have an ever more elaborate toolkit to infer selective regimes from genetically informative data, and the necessary data is increasingly becoming available [9••]. For general intelligence, we know that several hundreds of rare mutations with large effect explain a substantial amount of the variation at the very low end of the distribution, that is, in cases of intellectual disability. However, other genetic variants in the same genetic regions that cause low intelligence when mutated do not contribute to individual differences in the normal range [10]. Neither do overall burdens of rare copy number variants (variation in how often a genetic region is repeated in the DNA), exomic mutations (those that can alter the amino acids produced by genes) or *de novo* mutations (that occurred from one generation to the next) explain any substantial amount of variance of intelligence in the general population, though one recent study suggested lower burden of exomic mutations in extremely intelligent individuals (IQ > 170) compared to those with average intelligence [11]. Thus, the genomic mutations studied so far have not been associated with intelligence in the normal range. Instead, genome-wide complex trait analyses (GCTA), which estimate the overall contribution of genetic variants with common frequencies in the

population to traits based on around a million genetic markers across the genome, suggest that 28–51% of the variance in intelligence is due to the cumulative effect of thousands of common genetic variants, each with miniscule effect size. For a brief review, see [12]. Common genetic variants are unlikely to be deleterious mutations [7]. The GCTA estimates still leave a substantial proportion of heritable variance in intelligence unaccounted for, and it still remains possible that a burden of very specific rare mutations that are older and intronic (non-coding for amino acids) contribute to the unexplained fraction of the heritability [13]. Alternative explanations for the unaccounted heritability in GCTA include interactions between variants at different genetic loci (epistasis), or between genes and environments. The overall picture suggests that only the low and perhaps the very high end of the intelligence spectrum can be directly explained by mutation–selection balance. The rest of the spectrum might be effectively more neutral to selection than generally appreciated, perhaps due to trade-offs between benefits of higher intelligence and energetic costs of developing and maintaining a highly intelligent brain. Alternatively, normal-range intelligence could have been actively selected for robustness against mutational insults, which can result in both highly redundant (and thus compensatory) common genetic variation and widespread epistasis [11,12,14].

## Recent evidence on the genetic architecture of personality

Compared to intelligence, much less genomic data is available on personality traits. What can be said with high certainty is that none of the candidate genes for personality (including DRD4, 5HTT-LPR and COMT) have held up in meta-analyses. If these genes are associated with personality at all, their individual effects are tiny [15,16]. The lack of individual genetic variants with large effects is in line with genome-wide association studies (GWAS), which scan the genome for individual genetic variants linked to traits. So far GWAS of personality have not found a single replicable hit [17•,18,19]. These results suggest that a large number of genetic variants with individually tiny effects explain a substantial part of the heritability of personality, which is similar to what has been found for intelligence and indeed any human behavioral, clinical, and physical traits. As this seems to be a general pattern, it has recently been proposed as the fourth law of behavioral genetics ([20]; the first was discussed above, the second and third being that environmental influences do not contribute much to the similarity of family members, but substantially to their dissimilarity [6]).

A puzzling finding that diverges from the patterns generally found for other human traits is that GCTA estimates of the overall contribution of common genetic variants to personality traits are low: zero to 21% variance explained (highest for openness to experience and neuroticism, zero for agreeableness and conscientiousness), with confidence intervals often touching or including zero [17•,18,19,21•]. These estimates are markedly lower than the heritabilities for personality traits found in quantitative genetic studies [4]. Explanations for the surprisingly high proportion of 'missing heritability' in personality traits remain unclear. Some of it might be explained by rare mutations, which are not captured by GCTA estimates and have hardly been studied directly for personality, or by widespread epistasis and gene–environment interactions. Notably, while most quantitative genetic designs are not well suited to isolate epistasis, those few studies that have used appropriate designs have consistently identified substantial non-additive genetic components (including epistasis) for most, if not all, broad personality traits [4].

## Personality and reproductive success

Evidence from a dozen studies suggests that personality is related to the most direct indicator of reproductive success: how many children people have. Many associations, however, have not been consistent across studies (Table 1), and the effect sizes tend to be small. In the largest study to date from the contemporary United States [22], higher offspring number was associated with higher extraversion (+0.12 more offspring per one standard deviation change in the personality trait), lower neuroticism (−0.05), higher agreeableness (+0.07), lower conscientiousness (−0.06), and lower openness to experience (−0.19). Some of the associations between personality and offspring number may have emerged only recently, and thereby represent evolutionarily novel selection pressures. For example, in the United States higher openness to experience and women's higher conscientiousness were not associated with fertility among individuals born in the 1920s, but the associations strengthened in more recent birth cohorts [23]. These time-varying associations probably reflect societal changes, such as women's broader participation in the workforce and the adoption of less traditional lifestyles where people prefer to have smaller families. Other associations may represent more universal effects of personality on mating success and family formation. For example, extraversion and neuroticism have been associated with offspring number in various samples, extraversion particularly in men and most consistently in traditional small-scale societies (Table 1).

Except for one early study [24], no studies have found evidence for curvilinear associations where the highest number of children would be observed for intermediate levels of parent's personality trait. The lack of curvilinear associations speaks against stabilizing selection. Furthermore, there appears to be no intergenerational trade-offs between the numbers of offspring and grandoffspring that would negate the associations across more than one