Are pleiotropic mutations and Holocene selective sweeps the only evolutionary-genetic processes left for explaining heritable variation in human psychological traits?
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Published as:
Miller, G. F. (2010). Are polygenic mutations and Holocene selective sweeps the only evolutionary-genetic processes left for explaining heritable variation in human psychological traits? In D. M. Buss & P. H. Hawley (Ed.), The evolution of personality and individual differences, pp. 376-399. NY: Oxford U. Press.

We evolutionary psychologists pride ourselves on applying the latest evolutionary biology to illuminate human nature. Yet most of us have not kept up with the last decade's astounding progress in human evolutionary genetics. We're still focused on kin selection, reciprocity, sexual selection, and costly signaling as ways to explain the psychological adaptations that (supposedly) don't vary much across people. But when it comes to explaining individual differences, we have not yet discerned how 21st century evolutionary genetics clarifies heritable variation in cognitive abilities, personality traits, or psychopathologies. Those of us over age 35 especially need the humility to acknowledge to that genetics Ph.D. students typically know more than we do about the state of the art in multivariate behavioral-genetic modeling, how to run genome-wide association studies using DNA chips, or how to make inferences about ancestral selection pressures from molecular-genetic data.

I'm no exception. Until this sabbatical year when I started trying to catch up, I had no idea what I was missing. I assumed, like many evolutionary psychologists, that vague memories of out-dated undergraduate biology classes, plus some acquaintance with genetic correlations, life-history trade-offs, and frequency-dependent selection, would suffice to understand individual differences. Now I think we need to do better. We have been blind-sided by new genomic technologies, databases, and theories. These are only somewhat relevant to explaining universal psychological adaptations, but they are crucial to explaining heritable variation in psychological traits.

Here's a little test – a few basic questions that might appear on a typical graduate course exam in human evolutionary genetics (e.g. one based on the excellent textbook by Jobling, Hurles, & Tyler-Smith, 2004). Consider how many you can answer coherently.

1. Explain the evolutionary importance of the different types of mutations, including CpG transitions and transversions, indels, microsatellites, L1 and Alu retrotransposons, and segmental duplications
2. Explain the effects of gene conversion and genetic admixture on linkage disequilibrium
3. Explain how increased male reproductive variance affects the effective population sizes and genetic drift rates of X, Y, autosomal, and mitochondrial genes
4. Explain the five main measures of selective neutrality: the McDonald-Kreitman test, Tajima's D, the HKA test, ω, and H.
5. Explain how 'wombling' can help detect genetic boundaries in phylogeography

If you scored only 3 out of 5, that's 60%, a D-. Yet this is the sort of material that we evolutionary psychologists need to master – and to teach to our own students. We can't rely anymore on the view that heritable individual differences are just genetic noise arising as a side-effect from host-parasite coevolution (Tooby & Cosmides, 1990). This re-tooling will be tough,

but it's our job as the self-appointed disseminators of Darwinian theory in the behavioral sciences. If evolutionary psychologists don't make the connections between current evolutionary genetics and individual differences research, who will? So far, the other likely candidates – behavioral genetics, psychiatric genetics, and clinical neurogenetics – have not been filling the gap.

This is our disciplinary challenge, and once we face it, we immediately confront a daunting puzzle: most human psychological traits show far more heritable variation than would be expected if trait variation depended on just a few genes of major effect, and if evolution imposed stabilizing selection favoring a single optimal value of the trait (Carey, 2002; Pagel & Pomiankowski, 2007; Plomin, DeFries, McClearn, & McGuffin, 2008). This is true for all three main classes of psychological traits that are stable, heritable, widely predictive, and cross-culturally universal:

- personality traits such as the Big Five – openness to experience, conscientiousness, extraversion, agreeableness, and emotional stability (Bouchard & Loehlin, 2001; John, Robins, & Pervin, 2008; Matthews, Deary, & Whiteman, 2004; McCrae, Terracciano, et al. 2005; Miller, 2009) – and more specific traits such as sexual promiscuity (Gangestad & Simpson, 2000) and political engagement (Fowler & Schreiber, 2008);

- psychopathology traits, including the general dimensions of internalizing and externalizing (Krueger & Markon, 2006), and more specific dimensions such as the schizophrenia spectrum (Shaner, Miller, & Mintz, 2004; Sullivan, Kendler, & Neale, 2003), autism spectrum (Shaner, Miller, & Mintz, 2008; Veenstra-VanderWeele, Christian, & Cook, 2004), and psychopathy spectrum (Markon & Krueger, 2005; Moffitt, 2005);

- cognitive traits such as general intelligence (in the sense of the *g* factor arising from the all-positive correlations among mental abilities – Deary, Whalley, & Starr, 2008; Jensen, 1998), and its subordinate factors such as verbal ability, spatial ability, creativity (Kaufman, Kozbelt, Bromley, & Miller, 2007), social intelligence (Emery, Clayton, & Frith, 2008), emotional intelligence (Matthews, Zeidner, & Roberts, 2004), and mating intelligence (Geher & Miller, 2007).

A central question for any Darwinian analysis of such a trait is: what evolutionary processes have maintained the trait's surprisingly high heritable variation? The simplest answers require an equilibrium assumption – that all of the alleles underlying the trait's current genetic variation have been at some sort of evolutionary equilibrium for at least the last several hundred generations (such that the trait's current heritability, genetic correlations, and other quantitative features perfectly reflect their pre-Neolithic values). Assuming equilibrium, then there are just three key possibilities: the trait's genetic variation is fitness-neutral, or adaptive, or maladaptive (Keller & Miller, 2006; Mitchell-Olds, Willis, & Goldstein, 2007; Penke, Denissen, & Miller, 2007). Each possible explanation is discussed in turn below; after that, we'll see what happens if we relax the equilibrium assumption.

Perfect neutrality: Implausible for psychological traits that predict anything interesting

The perfect neutrality model for any given trait posits that the trait's variation is exactly fitness-neutral. This means that trait has had *no* significant fitness consequences in *any* domain of life (survival, growth, mate attraction, fertility, parenting, or socializing) across recent generations. In principle, mutations are free to accumulate in fitness-neutral traits, potentially yielding the heritable variation that we see today. In practice, traits are only fitness-neutral if

they are subject to a selection coefficient smaller than $1/N_e$, where N_e is the effective population size that represents the effects of genetic drift (Jobling, Hurles, & Tyler-Smith, 2004). This N_e is estimated to be about 10,000 for ancestral hominids (Eyre-Walker & Keightley, 2007), so any trait that decreases reproductive success by even 0.01% (1/10,000) would not have been neutral. It would have been eliminated by selection.

Such a perfect degree of fitness-neutrality is implausible for all human traits that psychologists care about. This is because traits that don't predict behavioral outcomes in any domain of life are not considered to have any predictive validity, so don't attract any scientific attention. The traits that we want to understand – personality, psychopathology, and cognitive traits – are studied precisely because they do predict success, failure, or variation in some important life-domains (Buss & Greiling, 1999; Nettle, 2006), as shown by a formidable range of empirical research (Deary, Whalley, & Starr, 2008; Jensen, 1998; Krueger & Markon, 2006; Matthews, Deary, & Whiteman, 2004).

Balancing selection: Three empirical problems that arose in the last few years

The balancing selection model posits that the trait's variation is adaptive. According to this model, the trait had fitness consequences and was under selection, but the optimal trait value varied across space, time, ecology, population, age, sex, health, social status, mate value, and/or some other contextual variable. If each observed trait value had exactly equal average fitness payoffs under different circumstances, selection could have maintained a polymorphic mixture of alleles underlying trait variation (Gangestad & Yeo, 1997). Special cases of this phenomenon include frequency-dependent selection (different alleles are favored depending on their commonality versus rarity), host-parasite coevolution (different alleles help defend the organism against different fast-evolving parasites), sexually antagonistic co-evolution (different alleles are favored in males versus females), and speciation (different alleles are favored in different breeding populations, such that separate species form).

Balancing selection is an ideologically attractive way for liberal academics to explain individual differences: it suggests equal evolutionary adaptiveness across psychological variants, so it seems to validate the full range of human psycho-diversity. Inspired by evolutionary game theory models of alternative stable strategies (Vincent & Brown, 2005), evolutionary psychologists have often used balancing selection to explain heritable variation in human psychopathology (Mealey, 1995), human personality (Nettle, 2005), and animal personality (Nettle, 2006). In an earlier paper (Penke, Denissen, & Miller, 2007) my co-authors and I suggested that balancing selection may explain heritable variation in the Big Five personality traits. However, I'm not so confident any longer, since balancing selection has three key empirical problems that have only become apparent in the last couple of years.

Problem 1: The failure of genome-wide association studies (so far….)

In evolutionary theory, balancing selection can maintain only a small number of genes with moderate to strong effects, such that most of the genetic variation is concentrated on a small number of loci (Kopp & Hermisson, 2006). Functionally, we might expect that traits maintained by balancing selection should evolve to be controlled by one or a few major polymorphic loci (Penke, Denissen, & Miller, 2007), and these key loci should evolve to act as master developmental switches. For example, the sex ratio is maintained by balancing selection, so sexual differentiation in mammals evolved to be controlled by just one master gene, the SRY gene on the Y chromosome. Also, the biochemical variation underlying immune system defenses are under (frequency-dependent) balancing selection against fast-evolving pathogens, so these variants have evolved to be controlled by a localized cluster of about 140

'major histocompatibility locus' (MHC) genes on chromosome 6, spanning about 3.6 Mb between the flanking markers MOG and COL11A2 (Piertney & Olivier, 2006). Consistent with balancing selection, MHC diversity is higher in human populations exposed to higher pathogen loads (Prugnolle, Manica, Charpentier, Guegan, Guernier, & Balloux, 2005). In general, balancing selection creates an 'allelic spectrum' biased towards a few high-frequency alleles at each of one or a few major loci (Reich & Lander, 2001).

We might expect similar outcomes for any psychological traits under balanced selection: genes of major effect should be easy to find in linkage and association studies, especially in the genome-wide association studies (GWASs) that seemed so promising in the early 2000s (Stoughton, 2005). GWASs of complex human traits are becoming ever more successful in identifying a few loci per trait that might lead to useful biomedical investigations of diseases associated with that trait. However, GWASs of complex human traits have been very disappointing so far in the proportion of genetic variance that the identified loci explain – typically less than 2% (Maher, 2008; Weiss, 2008). The Affymetrix Genome-Wide Human SNP Array 6.0, a widely-used DNA chip for GWASs, can identify 1.8 million genetic markers for each individual's genotype, including about 900,000 single nucleotide polymorphisms (SNPs) and about 950,000 copy number variants (www.affymetrix.com). So far, despite intense GWAS efforts in the last four years, even the most enthusiastic reviews (e.g. Altshuler, Daly, & Lander, 2008) note that only about 150 out of these million-odd SNPs have shown any reliable associations with any human trait or disease. For example, a high-profile *Nature* paper with over 150 co-authors, which claimed to represent "a thorough validation of the GWA approach" and which has been cited more than 600 times in the 18 months since publication, actually found only 24 SNPs (out of 500,000 sampled) that showed any statistically significant associations with any of 7 major mental and physical diseases (Burton, Clayton, Cardon, Craddock, Deloukas, Duncanson, et al. 2007). The few replicated alleles that have been found in GWASs account for only a tiny percentage of trait variance, even when they are aggregated. This is true for the morphological trait of height (Visscher, Macgregor, Benyamin, Zhu, Gordon, Medland, et al, 2007), and for the psychological traits of intelligence (Butcher, Davis, Craig, & Plomin, 2008), and the Big Five personality traits (Gillespie, Zhu, Evans, Medland, Wright, & Martin, 2008; Terracciano, Sanna, Uda, Deiana, Usala, Busonero, et al., 2008; Wray, Middeldorp, Birley, Gordon, Sullivan, Visscher, et al. 2008). Such elusive alleles are not what we would expect from traits under balancing selection. More direct genetic methods have also found very few loci outside the MHC complex that seem to have been under balancing selection (Bubb, Bovee, Buckley, Haugen, Kibukawa, Paddock, et al. 2006; Hendrick, 2006).

The GWAS revolution is still very much underway, and some replicable genetic variants will be found sooner or later that, in aggregate, might explain 5 to 10% of the heritable variance in some psychological traits. Yet even the most ardent GWAS researchers recognize that there is a big problem of 'the missing heritability' (Maher, 2008): if most psychological traits are at least moderately heritable, why is it proving so hard to find the specific genes that account for their heritability?

Problem 2: Pervasive inter-correlations and fitness-related correlations

Traits maintained by balancing selection should not correlate very much with each other, if they were shaped by disparate selection pressures favoring different polymorphic strategies in distinct domains of survival and reproduction. For example, if extraversion variance reflects a balanced trade-off between sexual benefits and accident risks (as suggested by Nettle, 2005), but openness variance reflects a balanced trade-off between out-group social interaction benefits and out-group pathogen-infection dangers (as suggested by Schaller & Murray, 2008) and if those four factors (sexual benefits, accident risks, social benefits, pathogen dangers) did

not reliably co-vary under ancestral conditions, then extraversion should not be correlated with openness.

Yet recent evidence suggests that all psychological traits show at least modest inter-correlations (ranging from less than r = .1 for single test items to r = .3 or so for higher-level aggregate scales). These correlations all seem to be positive if traits are measured on a worse-to-better scale of quality, whether indexed by social attractiveness, sexual attractiveness, social status, academic grades, economic success, or reproductive success (at least in natural fertility populations). For instance, the all-positive inter-correlations among cognitive abilities give rise to a general intelligence (*g*) factor (Jensen, 1998). Similar hierarchical factor models also seem necessary for both personality traits and psychopathology traits. If the Big Five personality traits are not forced into an orthogonal factor rotation, they show weak but generally positive correlations, and these can be best represented by two higher-order factors of Stability (spanning conscientiousness, agreeableness, and emotional stability) and Plasticity (spanning openness and extraversion) (DeYoung, 2006; Digman, 1997), which themselves are positively correlated, yielding a single General Factor of Personality (GFP) (Figueredo, Vasquez, Brumbach, & Schneider, 2007; Musek, 2007; Rushton, Bons, & Hur, 2008; Rushton & Irwing 2008, in press). However, this GFP is clearly not as strong as the g factor: it doesn't explain nearly as high a proportion of variation in the Big Five as *g* does for cognitive traits, at either the phenotypic or genetic levels (Yamagata, Suzuki, Ando, Ono, Kijima, Yoshimura et al., 2006). Also, debate continues about whether this GFP is an artifact of socially desirable responding or a genuine superordinate trait with predictive validity (McCrae, Yamagata, Jang, Riemann, Ando, Ono et al., 2008), and whether this GFP correlates with intelligence (Gladden, Figueredo, & Jacobs, 2008).

Similarly, the widespread comorbidities across categorical psychopathologies can best be represented by hierarchical dimensional models in which personality disorders reflect extremes of the Big Five personality traits (Markon, Krueger, & Watson, 2005; Widiger & Trull, 2007), and other disorders reflect high values on 'externalizing' and 'internalizing' dimensions, which are themselves positively correlated (Krueger & Markon, 2006).

Even across the domains of personality, psychopathology, and intelligence, phenotypic correlations are ubiquitous. For example, general intelligence correlates positively with openness to experience at both the phenotypic and genetic levels (Wainwright, Wright, Luciano, Geffen, & Martin, 2008); neuroticism (reverse-scaled emotional stability) correlates positively with the internalizing (or 'negative affectivity') dimension of psychopathology at both the phenotypic and genetic levels (Clark, 2005; Hettema, Neale, Myers, Prescott, & Kendler, 2006); and the externalizing dimension seems to reflect a combination of low intelligence, low conscientiousness, and low agreeableness (Lynam & Widiger, 2007; Saulsman & Page, 2004; Vitacco, Neumann, & Jackson, 2005).

So, some of the emerging evidence suggests that all major dimensions of human individual differences may fit into a unified hierarchical factor model, in which a general 'fitness factor' (representing general genetic quality) is superordinate to all three factors of *g*, the GFP, and mental health (Keller & Miller, 2006; Miller, 2007). This general fitness factor also seems to be superordinate to developmental stability as manifest in body symmetry (Prokosch, Yeo, & Miller, 2005) and physical attractiveness (Zebrowitz & Rhodes, 2004), and to general physical health as manifest in longevity (Deary et al. 2008) and fertility (Arden, Gottfredson, Miller, & Pierce, 2009). However, debate continues about relationships among *g*, the GFP, mental health, physical health, developmental stability, and sexual attractiveness. Nonetheless, without positing a hierarchical fitness model that represents a substantial portion of psychological and physical variance, it is very hard to explain apparent 'good genes' mate choice for mental traits among humans, since a single 'goodness' dimension implies a general fitness factor in psychometric analysis, and a general 'mate value' factor in subjective judgment (Miller & Todd, 1998; Neff & Pitcher, 2005).

Problem 3: Adaptive flexibility of behaviour is often better than hard-wired variation

For clever, big-brained primates like us, balancing selection seems especially weak at explaining heritable variation in psychological traits. Suppose there is some ecological or social domain that favors different strategies under different conditions. The alternative strategies could evolve as 'hardwired' genetic polymorphisms, or as 'softwired' developmental trajectories sensitive to early environmental cues during some sensitive period, or as flexible behavioural tactics that remain sensitive to current environmental cues throughout life (Figueredo, Vasquez, Brumbach, Schneider, Sefcek, Tal, et al., 2006). Hardwired polymorphisms make sense for alternative strategies that require a very early developmental commitment to growing a particular phenotype, such as a male or female body, but they make less sense for behavior. A key function of big, clever brains is to register a huge array of current environmental cues when deciding what to do (Tooby & Cosmides, 2005).

Humans are clearly flexible enough to do so with regard to most classic dimensions of psychological variation across individuals. When cognitive challenges increase, we can think longer, harder, and more sombrely, increasing our effective intelligence (Andrews, Aggen, Miller, Radi, Dencoff, & Neale, 2007); when over-learned habits will suffice, we can afford to act automatically and unconsciously. Likewise, when a new situation calls for a different personality, we enter altered states of consciousness called emotions that instantiate the required temperament: angry (disagreeable) if fighting, friendly (extraverted) if socializing, free-spirited (open and impulsive) if courting. If we already have emotions that are adaptively flexible and environmentally contingent; why do we need personality traits, which can be seen as genetic biases in the relative frequency of experiencing different emotions and having different act propensities (Buss & Craik, 1983; Fleeson, 2001)? The flexibility of emotional states argues against the adaptive utility of heritable personality traits. More extreme situations can even evoke temporary states that resemble psychopathologies: short-term depression after failures, short-term psychosis during trances, or short-term obsessiveness during hunting, gathering, or grocery-shopping.

In some sense, the highest-fitness humans may not have stable cognitive, personality, or psychopathology traits – only an exquisitely adaptive flexibility in matching one's current response mode to one's current environmental challenges. The question then becomes: what evolutionary benefit could arise from hardwiring such behavioural flexibility at the level of a genetic polymorphism? Any situation that would impose balancing selection on the behavioural strategies of a small-brained insect should, among big-brained primates, simply favour the evolution of new psychological adaptations – new ways of being sensitive to environmental variables. Obviously, short-term adaptive flexibility cannot explain heritable variation in personality traits, but it casts doubt on facile arguments about balancing selection for balancing traits.

Pleiotropic mutations: The last equilibrium model left standing?

The pleiotropic mutation model posits that the trait's variation is maladaptive. The trait has fitness consequences and selection favors a certain optimal value or range on the trait, but the trait is so polygenic (depends on so many alleles at so many genetic loci) that harmful mutations are constantly eroding genetic quality, and creating deviations from the optimal trait value (Zhang & Hill, 2005). The few mutations with strongly harmful effects and high penetrance (e.g. dominance) – that is, major Mendelian disorders – are quickly eliminated by selection. However, the many mutations with very slightly harmful effects and weaker penetrance (e.g. that are partially or fully recessive) are under much weaker purifying selection, so persist much longer in the population (Eyre-Walker & Keightley, 2007). The result is that

every human carries at least several hundred old, mildly harmful mutations inherited from previous generations, plus at least a few new mutations, mostly due to spermatogenesis errors in the father (Ellegren, 2007). These mutations are surprisingly common in humans, and range in size from single nucleotide polymorphisms (SNPs) such as insertions, deletions, and changes in single DNA base-pairs (Boyko, Williamson, Indap, Degenhardt, Hernandez, Lohmueller, et al. 2008; Gorlov, Gorlova, Sunyaev, Spitz, & Amos, 2008), through tandem repeats, segmental duplications, and copy number variants for longer stretches of DNA, and larger inversions and translocations of chromosomal segments, up to whole-chromosome aneuploidies (e.g. trisomy-21) and uniparental disomies (UPDs) (Feuk, Carson, & Scherer, 2006). Traits may be affected by mutations not only in classic protein-coding regions of the genome ('exons'), but also in regulatory and promoter regions before and after genes (Keightley, Lercher, & Eyre-Walker, 2005; Wray 2007), and in regions that code for various non-coding RNAs that coordinate gene regulation (Amarall, Dinger, Mercer, & Mattick, 2008; Bartel, 2004). Traits subject to pleiotropic mutation should be influenced by rare, evolutionarily transient variants across a very large number of loci, which should produce mostly additive genetic effects in aggregate (Hill, Goddard, & Visscher, 2008), and which should prove very difficult to find in GWAS studies (Gorlov et al., 2008; Keller & Miller, 2006; Weiss, 2008). Thus, the pleiotropic mutation model can more easily explain the hierarchical structure of fitness and fitness components (Keller & Miller, 2006; Miller, 2007).

Evolution out of equilibrium

A big problem with all three equilibrium models – perfect neutrality, balancing selection, and pleiotropic mutation – is that human traits are unlikely to have been at evolutionary equilibrium for the past several hundred generations. New genetic evidence shows that human evolution did not stop in the Pleistocene, but has accelerated throughout the Upper Paleolithic (50k to 10k years ago) and the Holocene (the last 10k years) (Hawks, Wang, Cochran, Harpending, & Moyzis, 2007; Kelley & Swanson, 2008; Meisenberg, 2008; Neilsen, Hellmann, Hubisz, Bustamante, & Clark, 2007; Nettle & Pollet, 2008). Selection pressures cannot have remained the same after the human dispersals out of Africa and the Upper Paleolithic revolution, especially since the rise of agriculture, domestication, money, and institutionalized monogamy. These changes may not have had enough time to produce complex, new, cross-culturally universal psychological adaptations (Andrews, Gangestad, & Matthews, 2002; Tooby & Cosmides, 2005), but they could have had dramatic effects on the patterns of genetic variance underlying human personality, psychopathology, and cognitive traits.

Cryptic genetic variation uncovered by modern environmental complexity?

The emergence of cities, complex cultures, stratified societies, and divisions of labor probably created many new social and sexual niches in which new and more diverse psychological traits could thrive. The proliferation of these new niches may have uncovered huge amounts of cryptic genetic variation (Gibson & Dworkin 2004) in psychological traits – variation that was not manifest phenotypically under ancestral conditions, but that is manifest under more diverse modern conditions. For example, polymorphic alleles that now create heritable variation in openness to experience may have been hidden away in the Pleistocene, when there simply weren't many new ideas, values, or experiences towards which one could be open or closed. The degree of openness manifest in prehistory may have been tightly constrained by the relative simplicity, conservatism, and insularity of small-scale hunter-gatherer societies (Marlowe, 2005). In effect, openness could have been developmentally canalized under ancestral conditions (Flatt, 2005). With no variation manifest in actual openness, any genetic loci that influenced potential openness in prehistory may have been invisible to

selection, and polymorphisms could have accumulated at those loci (Schlichting, 2008). The result can be described with a 'capacitor model' (Le Rouzic & Carlborg, 2008; Moczek, 2007): the latent trait of openness becomes 'genetically charged' with heritable variation under Pleistocene conditions, and this variation can become manifest and 'discharged' in novel environments, as when human cultures became more complex, dynamic, and permeable in the Holocene.

However, there are two serious problems with this cryptic variation model for explaining major human psychological traits. First, it is really a special case of the perfect neutrality argument, which doesn't work for traits that we care about. Cryptic variation could accumulate in prehistory only if it had *no* fitness consequences in *any* domain of life. For example, prehistoric propensities towards higher openness would not have been fitness-neutral if they had *any* effect on the likelihoods of migrating to new habitats, mating with out-group members, learning new survival or courtship skills, or trying new foods or drugs (cf. McCrae, 1996). If heritable variation in the trait influenced development, survival, mating, or parenting in any way, it could not have accumulated as truly cryptic variation. Second, even if cryptic variation became apparent only since the rise of Holocene civilizations a few thousand years ago, there would have been plenty of time for selection since then to shape current patterns of variation. Assuming an average 25-year generation time, five thousand years is about 200 generations. Previously cryptic alleles that turn out to impose even a 1% decrement in lifetime reproductive success in the Holocene will be reduced from 50% prevalence to 5% prevalence in 100 generations, and will virtually vanish within 200 generations (see Keller & Miller, 2006, Figure 2). Overall, cryptic genetic variation in important psychological traits is unlikely to have accumulated in a fitness-neutral way in prehistory, and even if it did, it is unlikely to have persisted under historical selection pressures.

Cryptic variation may have played one important role in recent human evolution – it could, in principle, have provided a vast reserve of potentially adaptive variants that could prove their fitness under evolutionarily novel conditions (Barrett & Schluter, 2008). When our ancestors faced the new challenges of Holocene living, they did not have to wait around for new mutations to accumulate. Cryptic variation, decanalized by new environmental conditions, could have offered a great head-start for genetic adaptation to civilized life. It just would have been subject to intense new natural and sexual selection pressures from that point onwards.

Ongoing selective sweeps since the Pleistocene

The last 50,000 years of human evolution are likely to have been driven by massive changes in selection pressures (Meisenberg, 2008), increased genetic admixture across populations (Klimentidis, Miller, & Shriver, in press), and an increased number of potentially beneficial mutations per generation due to dramatically increased population sizes (Hawks, Wang, Cochran, Harpending, & Moyzis, 2007). These changes would have affected not only the human populations that launched the Upper Paleolithic revolution and the rise of civilizations, but all populations that experienced any gene flow from those populations – including contemporary hunter-gatherers.

An obvious example concerns the possibility of genetic admixture between expanding human populations and indigenous archaic hominid populations such as Asian Homo erectus (Cox, Mendez, Karafet, Pilkington, Kingan, Destro-Bisol, et al. 2008), European Neanderthals (Weaver & Roseman, 2008), or indigenous African 'Rhode-sioids' (Brauer, 2008). Genetic models suggest that our human ancestors could easily have 'poached' any useful genes that had independently evolved among other hominid lineages, and any such genes that gave even a small selective advantage could have swept through not only local human populations, but all human populations, assuming moderate rates of migration and gene flow (Garrigan & Kingdan, 2007; Hawks, Cochran, Harpending, & Lahn, 2008). The genetic and fossil evidence

concerning Neanderthal admixture is mixed (pro: Hawks et al., 2008; Trinkaus, 2007; con: Hodgson & Disotell, 2008), but completion of the Neanderthal Genome Project should clarify the issue.

More recent selective sweeps probably accompanied both the Upper Paleolithic revolution (c. 30,000 years ago in Europe) and the Holocene emergence of complex civilizations in the last 10,000 years. These cultural revolutions produced rapid expansions of human population, spatial range, ecological diversity, and social complexity, which created thousands of new ecological, economic, social, and sexual niches. These in turn must have imposed strong, dynamic, and local selection pressures that affected human gene frequencies in different populations.

For example, with the invention of money as a form of 'liquid fitness' came the possibility of sexual selection for wealth – and for any psychological traits that facilitated its acquisition, investment, arbitrage, and defense (Clark, 2007; Ellis, 2001; Nettle & Pollet, 2008). Ruling-class males in some populations attained unprecedented reproductive success through bride-capture, harems, slaves, prostitutes, and mistresses, perhaps driving rapid selective sweeps for certain traits associated with leadership, belligerence, oppressiveness, rapaciousness, and risk-taking (Earle, 2002; Summers, 2005). Very recent increases in the anonymity and mobility of some societies may have created more reproductively rewarding niches for psychopaths (Mealey, 1995; Moffitt, 2005). Conversely, religiously imposed monogamy in some populations may have increased selection for fidelity, conscientiousness, agreeableness, and parental investment (Miller, 2007), and may have greatly increased the strength of assortative mating for personality and cognitive traits, thereby amplifying genetic variance in those traits (Hooper & Miller, 2008).

With each Holocene selective sweep in favor of some new higher-fitness alleles, other phenotypic side-effects and linked loci would be carried along to higher population frequencies, which may explain genetic variation in apparently unrelated traits.

New mutations that happen to have beneficial effects on one trait are likely to have pleiotropic effects on other traits that reduce their net fitness benefits by a factor of two, on average (Otto, 2004). For example, if selection in favor of general intelligence suddenly became more intense in some population, this could have favored the spread of new IQ-boosting alleles even if those alleles have a range of harmful side-effects on physical or mental health that would have been counter-selected under Pleistocene conditions (Cochran, Hardy, & Harpending, 2006). Hypothetically, the same alleles that increase intelligence through modified sphingolipid metabolism and lipid storage in the brain could also disrupt a variety of bodily organs and tissues, perhaps creating higher risks of Tay-Sachs, Canavan disease, Niemann-Pick disease, torsion dystonia, and Mucolipidosis type IV. The result would be increased heritable variation in these side-effect traits, while the selective sweep is underway. Medical geneticists focused on the neurological disorder of torsion dystonia (painful muscle contractions resulting in uncontrollable distortions of posture and limb position) might then wonder how the genetic risk factors for this disease could have persisted ancestrally, and might speculate about hidden adaptive benefits to weird postures – without realizing that the disease is an evolutionarily transient side-effect of strong recent selection for intelligence.

Also, given one new mutation favored by selection, any nearby loci on the same chromosome would be carried along to higher frequencies due to linkage disequilibrium. This genetic hitch-hiking would spread not just a single allele, but a whole haplotype (set of alleles in linkage disequilibrium, which is broken down only slowly by genetic recombination), and the linked genes could increase the observed variance in physical or psychological traits that are functionally unrelated to the favored trait. This linked-loci effect is absolutely fundamental to the entire HapMap project, which traces the structure of haplotypes across human populations (International HapMap Consortium, 2007). For example, an ongoing selective sweep in favor of an openness allele could, in principle, increase the frequency of a schizotypy allele that is a few

million base pairs away on the same chromosome. The result would be a phenotypic and genetic correlation between openness and schizotypy (e.g. Miller & Tal, 2007), which could easily lead the unwary to posit a balancing selection model in which the fitness benefits of openness balance out the fitness costs of schizophrenia. Indeed, any such haplotypes during a selective sweep would tend to show a complex mix of fitness costs and benefits that could easily be mistaken for the outcome of balancing selection.

Both of these effects – linked loci that create haplotype divergence across and within populations, and harmful pleiotropy that reduces fitness even in traits that are genetically correlated with favored traits – can produce substantial maladaptive genetic variance. The stronger selection has been in recent human evolution, the stronger these maladaptive side-effects are likely to have been, potentially explaining a lot of imperfection in human bodies and minds.

Genetic variance created by recent selective sweeps would, like that created by pleiotropic mutations, show an elusive molecular-genetic basis. Any alleles with very strong positive fitness effects would sweep to fixation rapidly in any given population, so would not leave behind much genetic variance. The alleles that do still explain genetic variance because they are still undergoing a sweep are likely to have milder fitness effects, so will show weaker – or perhaps undetectable – effect sizes in GWAS studies. Moreover, the specific alleles favored in Holocene sweeps may differ across populations, so any GWAS associations that do emerge may not replicate across populations. Finally, any haplotypes undergoing a sweep may contain thousands of genetic variants that might be detected in GWASs, but most of those will have no functional relationship to the trait being favored; they will simply be in linkage disequilibrium with the functionally relevant alleles. Here again, without a good understanding of the evolutionary genetics, GWAS studies of human intelligence, personality, and psychopathology may continue to yield results that are rare, weak, and unreplicable, and that explain only a small percentage of genetic variance.

Conclusion

If we take seriously the notion that human evolution has continued and accelerated in the Holocene, then we have to rethink two foundational assumptions of traditional evolutionary psychology: (1) the Pleistocene 'environment of evolutionary adaptedness' (EEA) as the most recent relevant evolutionary environment for understanding individual differences, and (2) evolutionary equilibrium models (perfect neutrality, balanced selection, and pleiotropic mutation) as the most relevant theories for explaining heritable variation in human traits. Our most important psychological traits are likely to have been subject to strong selective sweeps (rapid increases in the frequency of new fitness-increasing alleles) due to increased population density (Hawks, Wang, Cochran, Harpending, & Moyzis, 2007), more intense social and sexual competition (Summers, 2005), more virulent pathogens (Schaller & Murray, 2008), more technology-associated hazards (Gottfredson, 2007), and more selective and assortative mating (Miller, 2000). Evolutionary theorists don't yet know how to combine the genetic models that assume evolutionary equilibrium, and the genetic models relevant to human populations that have been evolving faster than ever in the last few hundred generations. In the next few years, the main challenge in evolutionary personality psychology will be to develop the relevant evolutionary genetic models that accept the importance of ongoing post-Pleistocene human evolution, and to understand and test their distinctive empirical predictions.

If pleiotropic mutations and Holocene selective sweeps do turn out to explain most individual differences in psychological traits, this would have implications for future research at several levels. At the molecular-genetic level, we might expect that most variation in intelligence, personality, and mental health traits will arise from differences in overall mutation

load (Keller & Miller, 2006), rather than from allelic variants at just a few loci (as expected from balancing selection models, and as assumed by most current gene-hunting methods, including GWAS searches for SNPs and copy number variants). At the neurogenetic level, we might expect overall mutation load to influence general neurodevelopmental stability (Prokosch, Yeo, & Miller, 2005), as assessed by overall brain size, anatomical typicality, neurochemical typicality, precision of neuronal growth patterns, and/or neural conduction velocities or signal-to-noise ratios, rather than expecting particular alleles to have clear effects on specific cortical areas, neurotransmitter systems, or fiber tracts. At the psychometric level, we might expect all reliably measurable psychological traits to have some position within a hierarchical structure that spans intelligence, personality, and psychopathology, and that encompasses aspects of psychological functioning that were previously pigeon-holed as 'cognition', 'emotion', 'motivation', and 'consciousness'. Finally, at the sociological level, a renewed appreciation that mutation load and recent selection are important in explaining human psychodiversity might lead researchers to reconsider a range of issues in moral philosophy, socio-political ideology, and bioethics. We may need to find radical new ways to reconcile the empirical facts about human diversity with our classical liberal values of equality, meritocracy, democracy, and multiculturalism. In short, evolutionary genetics is undermining the old anthropological assumption about 'the psychic unity of mankind', and we better learn the scientific and moral sophistication to deal constructively with the consequences.

Acknowledgements
For helpful feedback on drafts of this chapter, thanks to: Rosalind Arden, David Buss, Greg Cochran, A. J. Figueredo, Henry Harpening, Nick Martin, Dan Nettle, Lars Penke, Phil Rushton, Andrew Shaner, and Peter Visscher.

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