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Evolutionary biology looks at behavior genetics

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ABSTRACT

One leading edge of intellectual exploration that Tom Bouchard significantly contributed to lies at the interface of behavior genetics and evolutionary biology. Behavior geneticists have amply demonstrated that most important psychological individual differences owe part of their variance to genetic variants. An interesting issue from an evolutionary perspective concerns why meaningful genetic variation persists. Evolutionary biology offers a number of possible answers. I examine arguments and currently available data that speak to their application to variation in personality. Some likely answers (e.g., stabilizing selection is opposed by generation of new mutations) were conjectured by Bouchard and Loehlin (2001) in a classic review. Additional new possibilities (e.g., the importance of copy number variants) deserve close scrutiny.

1. Evolutionary biology looks at behavior genetics

It is my pleasure and honor to be part of an event honoring Tom Bouchard, his career, his contributions, the impact of those contributions. I recently discovered a remarkable fact. Bouchard's work—already extremely well-cited—was cited more times last year than ever, though this year may set a new high again. So the impact of his work not only continues; by this metric, its rate has yet to peak, six months from his official retirement. I'm sure that this achievement is not unheard of. But given a mean half-life of citations for scientific articles of a few years and typical decline in researchers' rates of productivity prior to retirement, I imagine it's far from typical. Bouchard's still-upward rate, I suspect, is testimony to the fact that much of his work has been at the leading edge of subsequent big knowledge expansions, ones that continue.

One such edge lies at the interface of evolutionary biology and behavior genetics. Naturally, everyone knows that Minnesota has a leading program in behavior genetics. Far fewer know that it also offered one of the first graduate seminars on evolutionary psychology in the country. Spring, 1994, I was on sabbatical at the University of Minnesota. I saw that Bouchard was teaching this course. I sat in. It was a wonderful intellectual exploration. Bouchard's interests in evolutionary biology at that time were broad but also targeted. In wide-ranging psychological domains—intellectual ability, personality, psychopathology, interests, values, and so on—one finds substantial genetic variation. Why? What's the evolutionary explanation? Bouchard recognized this as a deep, penetrating question, and one fundamental to putting behavior genetics in a broader theoretical context. As the final paragraph of his classic 1994 paper in *Science* (Bouchard, 1994) put it: "Unraveling the role human individual differences play in evolution is the next big hurdle, and its solution will turn the behavior genetics of human personality from a descriptive discipline to an explanatory one" (p. 1701).

2. What explains genetic variation in personality?

So do we now have an evolutionary biology of human behavior genetics? Only in outline form, though progress is being made. Bouchard and Loehlin (2001) offered that outline—in just a page or two, a general and very sensible framework within which to understand individual differences from an evolutionary perspective. A number of other important papers have followed (e.g., Buss, 2009; Keller & Miller, 2006; Nettle, 2006; Penke, Denissen, & Miller, 2007; see also Wilson (1994) and Buss and Greiling (1999)). I'll use Bouchard and Loehlin's paper as a touchstone to offer a few things about how evolutionary biology can inform the study of individual differences.

The core evolutionary question at its interface with behavior genetics is: What evolutionary processes explain genetic variation? As I hope I can illustrate, evolutionary biology can also generate useful thinking about proximate phenomena of core interest to psychologists and behavior geneticists: e.g., what genes underlie behavioral variation?; what physiological processes are involved?; what gene–environment interactions are involved?

2.1. What accounts for genetic variability in general?

Fisher's fundamental theorem states that the rate at which the mean fitness in a population increases due to selection at a given

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time is equal to the genetic variance in fitness. Because, in doing so, selection decreases genetic variance as well, the theorem implies that directional selection "uses up" or exhausts additive genetic variation under ideal conditions. On just about any phenotypic psychological trait imaginable, of course, genetic variation exists. Hence, the ideal conditions under which directional selection exhausts genetic variation do not exist. But in what ways? Broadly speaking, there are five possibilities (e.g., Crow, 1986; Roff, 2005).

1. *No selection:* This is unlikely: no meaningful selection on a trait whatsoever. It might apply to genes with no phenotypic expression, but the probability that it applies to any phenotypic trait—certainly any major behavioral trait—seems low (see Penke et al., 2007).
2. *Mutations:* Mutations may act as opposing evolutionary forces. On traits with directional effects on fitness, the effects of mutations are more likely to be dysfunctional than enhance reproductive success. At equilibrium, a point referred to as mutation-selection balance, the rate at which deleterious mutational effects enter the population equals the rate at which they are removed. The number of mutations affecting fitness varies across individuals. Hence, substantial genetic variation in fitness itself is probably maintained by mutation-selection balance (e.g., Burt, 1995; Houle, 1992).At equilibrium, many phenotypic traits are not directionally selected (i.e., linearly associated with fitness); rather, the extremes are selected against, and the middle selected for (i.e., the trait curvilinearly relates to fitness). This is referred to as stabilizing selection. For example, human height may be partly under stabilizing selection, with very short and very tall people being less fit than moderate-sized people (e.g., Nettle, 2002). Were there no mutations, selection would eventually eliminate variability around the intermediate optimum. But with mutation, at equilibrium the rate at which mutational effects on the trait are eliminated equals the rate at which they are introduced.
3. *Variable selection:* Selection itself may be variable, either spatially or temporally. If selection for particular genes is variable across space, gene flow at spatial boundaries (or across spatial gradients in selection) or occurring through migration results in mixing and, hence, variability. If selection temporally varies, selection may never drive variance to zero or positively select new beneficial mutations more frequently (but see Roff, 2005, for qualifications).
4. *Negative frequency-dependent selection:* This is a form of variable selection that exists only in specific situations. When it does, variants that are rare in the population tend to succeed, making multiple alleles persist stably. Some immune system genes, e.g., certain Class I and Class II major histocompatibility complex (MHC) genes, appear to work this way: All else equal, individuals may be better off when possessing a relatively rare form of immune defense, one that pathogens are not used to encountering. As a result of selection for alleles when they are rare (as well as heterozygote superiority [non-additive effects on fitness]), some MHC genes are high polymorphic and, hence, genetically variable. Social selection can also promote diversity if different personality "types" fit different social niches.
5. *Non-additivity:* Genetic variance that is non-additive, at least with respect to its effects on fitness, is not typically removed by selection. Most non-additive forms of variance do not generate parent-offspring correlation and, hence, selection on a parental generation does not change the distribution of non-additive effects on the offspring generation.

So what accounts for genetic variation in human personality? Bouchard and Loehlin (2001) concisely summed up their view: Stabilizing selection against extremes around optima, with some variation within extremes possibly maintained by variable selection (spatially variable, temporally variable, or frequency-dependent). They are likely right; after all, these constitute the main possibilities noted above. What current evidence speaks to each? And what is their relative importance?

2.2. Variable selection

The view that past variable selection explains genetic variation in personality has become a particularly popular one in recent years. Nettle (2006), for instance, discussed how each of the Big Five personality dimensions has perhaps been subject to variable selection (possibly augmented by frequency-dependent selection). He couched his arguments in terms of trade-offs. Anxious people, for instance, may do particularly well at some tasks, emotionally stable people well at others. For a particular environment, there may be an optimal trade-off between these task performances and, therefore, optimal level of anxiety-proneness. If environments varied ancestrally, either spatially or temporally, the optima favored may have varied along a continuum. As well, populations may support mixes of individuals specializing in performing particular tasks (see other examples in, e.g., Figueredo et al., 2005; McDonald, 1995; Mealey, 1995).

Some empirical studies are consistent with the idea that selection varies across spatially distributed socioecologies. Camperio Ciani, Capiluppi, Veronese, and Sartori (2007) found that mainland Italian and small island populations reliably differ in personality, consistent with local selection and adaptation. If different optima are favored in mainland and island populations and migration is non-negligible, within-population variation could be maintained. Worldwide, some reliable differences in Big Five traits across regions exists (e.g., East Asians tend to be less agreeable, conscientious, and open; Schmitt, Allik, McCrae, & Benet-Martínez, 2009).

Penke et al. (2007) argued that the genetic architecture of traits may contain signatures of "balancing selection" (largely, in their view, forms of variable selection—across time, space, allele frequencies): Such traits, they claimed, should tend to be affected by polymorphic genes with intermediate allele frequencies, each with moderate effects, and may possess substantial non-additive genetic variance. And, they suggested, personality traits tend to possess these genetic architectures.

While some genetic variance in personality may well be due to variable selection, the strength of the current evidence may be questioned. First, personality variations across geographies and nations, even if reliable, are small. Differences between mainland and small island populations in Camperio Ciani et al.'s (2007) study account for, on average, <1% of the variance in Big Five traits. World regions explain, on average, ~3% (some due to sampling variability; Schmitt et al., 2009). Geographical variations are unlikely to account for much of the phenotypic variation in personality.

Second, major gene effects on personality are actually uncommon. A meta-analysis yielded no robust associations between 16 candidate genes and major personality dimensions once controls were instituted (Munafò et al., 2003). Later meta-analyses on two relatively promising candidates revealed little evidence of robust associations of 5-HTT (LPR genotype) with anxiety-related traits (Munafò et al., 2009) and evidence of, at best, weak associations between DRD4 (C-521T polymorphism) and novelty-seeking and impulsivity (accounting for, at best, 3% of the phenotypic variance on these traits; Munafò, Yalcin, Willis-Owen, & Flint, 2008).

Recent genome-wide scans offer no greater promises. Gillespie et al. (2008) detected no significant linkages with the four Eysenck dimensions (E, N, P, and L). And perhaps most impressively, Shifman et al. (2007) failed to find any loci accounting for >1% of the variance in neuroticism, despite having about 50% power to detect an effect of .5–1% of the variance. They concluded, "Since we failed

to find any loci accounting for more than 1% of the variance, the heritability of neuroticism probably arises from loci each explaining much less than 1%" (p. 302). This situation appears similar to that for height. Many robust associations have been found between specific markers and height, given that extremely large sample sizes are available (e.g., Perola et al., 2007), but the top 20 loci identified collectively account for less than 3% of the variance (Martin, 2009).

Third, polymorphic genes with meaningful effects on personality appear uncommon. Examples do exist; e.g., the 7-repeat allele of DRD4 associated with ADHD has a relative frequency of about 20% in European populations (e.g., Hattori et al., 2009). But are such polymorphisms common? It is perhaps hard to say; as noted above, studies have generally not successfully identified genes with noteworthy effects on personality. In a genome-wide search designed to assess whether there are regions in the human genome that are highly polymorphic, aside from MHC (whose polymorphism is described above) and ABO blood group regions, Bubb et al. (2006) found no more than expected under a neutral model—that is, no convincing evidence that balancing selection with deep historical roots has maintained high levels of polymorphism in genomic regions. This suggested that "long-term balancing selection may simply be rare in humans" (p. 2175). Obviously, polymorphisms at functional sites do exist. But most of these instances may be due to recent balancing selection, incomplete selective sweeps, or near-neutrality (see Hurst (2009) for a discussion of rare exceptions, e.g., other immune function genes).

Fourth, some personality traits do possess at least moderate levels of non-additive genetic variance (due to dominance effects or epistasis; e.g., Keller, Coventry, Heath, & Martin, 2005; Lake, Eaves, Maes, Heath, & Martin, 2000; Rhee & Waldman, 2002). But, as Keller (2007) argued, presence or absence of modest levels of non-additive variance are not a signature of any particular form of historical selection.

The hypotheses put forward by Nettle (2006), Penke et al. (2007), and others are important and worthy of future efforts to identify and test predictions that follow from them. I argue only that compelling evidence for them is lacking at this time.

2.3. Stabilizing selection and mutation-selection balance

Stabilizing selection, once again, exists when selection favors a trait value that is intermediate within the range represented within a population. Absent mutation, selection eventually drives out all additive genetic variance on such traits. If mutations that affect trait values arise, however, they generate variance on the trait, with values deviating from the optimum selected against. At equilibrium, selection removes mutational effects on the trait at the same rate at which they are introduced, and a fairly constant level of genetic variance on the trait persists in the population.

Perhaps many instances of stabilizing selection can be thought of in terms of trade-offs. As Nettle (2006) noted, anxiety-proneness may foster performance on some tasks, interfere with others. Within a particular environment, an optimum trade-off between task performances is favored. Variation in anxiety-proneness around that optimum, however, may be maintained by mutation-selection balance.

In some instances, trade-offs can be thought of in terms of energetic expenditure. Consider height. One might think that bigger would be better (e.g., in some physical contests, in some foraging tasks). But growing bigger costs energy, as does maintaining and repairing a bigger body. All else equal, a smaller individual has more energy to invest in other fitness-enhancing activities or capacities (e.g., a bigger brain, immune function, transfer of energy to offspring). In theory, for a given organism there is a "right size" in light of energetic trade-offs. Similarly, certain behavioral traits (e.g., dominance, surgency) may require substantial energetic investment to attain, and for a given organism there may be an optimal level of such traits in light of energetic trade-offs. (Because individuals in better condition may have a greater optimum of some traits than individuals in worse condition—e.g., be able to afford to grow bigger—some of these traits may partly be under directional selection too. Indeed, some evidence suggests that extremes of men's height are selected against, but a value greater than the mean is associated with greatest fitness; see Nettle, 2002. This may also be the case with some personality traits.)

So what evidence can distinguish mutation-selection balance from stabilizing selection? Recently, Jeff Simpson, Randy Thornhill, and I attempted to test one set of predictions derived from a form of stabilizing selection. Low-level mutations may have, as byproducts, effects on developmental instability—the imprecise expression of developmental design. Fluctuating asymmetry (or FA: absolute right-left asymmetry on bilateral traits that are symmetrical at the population level) is the most widely used measure of developmental instability (e.g., Møller & Swaddle, 1997). Consistent with the idea that mutations affect developmental instability, Carter, Weier, and Houle (2009) found that inbred lines of fruit flies had greater FA of wing dimensions than outbred lines. Inbreeding increases mutational effects in offspring by substantially increasing the number of loci that are homozygous for mutations. A "double-dose" of a mutation at a particular site (the mutation in a homozygous state) typically has deleterious effects on fitness much greater than double the deleterious effect of a "single dose" of the mutation (the mutation in a heterozygous state).

In work on humans, researchers typically measure FA in up to 10 of these traits—e.g., ears, elbows, wrists, finger lengths, ankles—and sum up the small asymmetries on all for each individual—to get a composite measure of FA. In a study of Minnesota Twins Reared Apart, h^2 of such a composite was found to be about .3 (Johnson, Gangestad, Segal, & Bouchard, 2008). The FA composite has a reliability much lower than the typical psychometric measure, however (almost certainly <.5); hence, the heritability of developmental instability underlying FA in this population may be much higher >.5.

We currently do not know what specific processes generate subtle asymmetrical growth and hence "developmental instability" tapped by FA. One possibility my colleagues and I have explored recently is oxidative stress. Cellular respiration produces as necessary byproducts reactive oxygen species (ROS) including free radicals, which can damage cellular membranes and DNA. Organisms produce antioxidant enzymes to neutralize them and render them harmless, and repair damage as well. But they do not do so perfectly. (Given typical diminishing returns to effort to maintain the soma, an organism that resisted and repaired the effects of ROS perfectly would almost certainly have higher fitness by reallocating some of that effort to another productive effort, such as reproduction; hence, organisms will not be selected to resist degradation of tissue perfectly; see Kirkwood (1977), on the "disposable soma" theory of senescence.) Hence, energy production necessarily entails a cost of oxidative stress—damage due to oxidants. Energy production can vary in how efficient or "clean" it is, however. Partly for this reason, some individuals experience higher levels of oxidative stress than others. Individual differences in oxidative stress may partly explain differential aging. Oxidative stress, for instance, appears to be implicated in development of Alzheimer's disease (Lin & Beal, 2006).

Biomarkers of oxidative stress can be measured in urine. In a recent study on college men, we sampled two, each on two different days—malondialdehyde, a marker of lipid peroxidation, and 8-OHdG, a marker of DNA damage. We summed up unit-weighted values to obtain a composite index of oxidative stress. One finding of interest is that this composite significantly correlated with

reports of familial Alzheimer's; some of the genetic variation in Alzheimer's disease may be due to genetic variation affecting oxidative stress.

More particular to our immediate interests, however, was the association between oxidative stress and fluctuating asymmetry. Higher levels of FA were associated with higher levels of oxidative stress (Merriman, Gangestad, Emery-Thompson, & Muller, unpublished data). At this time, we do not know whether oxidative stress is a cause or a consequence of developmental instability. An intriguing possibility is that differences in oxidative stress, as partly affected by mutations, generates at least some of the asymmetrical growth that constitutes FA.

If a trait partly reflects the effects of low-frequency mutations, one might expect it to covary with FA. This line of reasoning led researchers to examine associations between FA (measured as a composite of multiple traits' unsigned asymmetries) and psychometric intelligence, *g*. Several studies have found that FA negatively predicts *g* in Western samples (Bates, 2007; Furlow, Armijo-Pruett, Gangestad, & Thornhill, 1997; Luxen & Buunk, 2006; Prokosch, Yeo, & Miller, 2005), though others report mixed (Rahman, Wilson, & Abrahams, 2004) or no (Johnson, Segal, & Bouchard, 2007) evidence for an association. More studies are needed. If the association does exist, even weakly, it may reveal that *g* is partly a function of rare mutations, and that genetic variance in *g* is due to mutation-selection balance of variants affecting a fitness trait. A number of proximate processes are consistent with this scenario: *g* may be directly diminished by instability of brain development (a DI-mediator model), developmental instability may be a byproduct of rare mutations that directly affect gene expression in the brain and compromise its performance (a direct causal model), or energetic investment in brain growth and maintenance underlying *g* may be contingent on condition, as broadly affected by mutations (a condition-dependent model).

If personality variations similarly represent (ancestral) fitness traits on which variation is maintained by mutation-selection balance, personality traits too might be associated with FA. But what if a personality trait has been under stabilizing, not directional, selection? No linear association can be expected, but a related approach can be applied. FA may relate to the *quadratic* component of trait variance, with trait extremes being associated with high FA. Simpson, Thornhill, and I examined these associations using measures of Big Five traits in three samples of men, with a total *N* over 250 (Gangestad, Simpson, & Thornhill, unpublished data). This study has not yet undergone critical peer review. I present our results as merely illustrative of findings that, if robust, are consistent with mutational variation subject to stabilizing selection.

Within each sample, we calculated partial correlations between each trait and the quadratic component of the trait (trait values squared), with linear effects of trait values controlled. For each trait, we estimated the mean effect across samples. Averaged across all traits, the partial correlations were positive: Individuals at the extremes of Big Five measures possessed greater FA than did individuals close to mean values. But effect sizes varied widely across traits: highly robust for agreeableness ($r \approx .3$), significant for conscientiousness and openness (with effects roughly half that for agreeableness), weakly and non-significantly positive for neuroticism, and near-zero for extraversion.

These findings are consistent with at least some personality variations having been subject to stabilizing selection, maintained by mutation-selection balance. One possibility is that developmental instability itself results in variable personality outcomes, sometimes leading to extreme levels. Consistent with this idea, Rose, Reed, and Bogle (1987) and Bogle, Reed, and Rose (1994) found that identical twins with greater developmental stability are less similar in personality than those with lower developmental stability. Alternatively, rare mutations both affect personality outcomes and have, as byproducts, effects on developmental instability. These processes are not mutually exclusive.

2.4. Summary

What evolutionary processes account for genetic variation in personality? Probably multiple ones. Authors such as Penke et al. (2007) and Nettle (2006) have bet on forms of variable selection being primary causes, and they may well be right. But at this point, I agree with Bouchard and Loehlin's emphasis on stabilizing selection. Future progress requires the development of new ways to test the possibilities.

3. What mutations affect personality?

As I suggested at the outset, evolutionary biology may generate useful thinking about proximate phenomena of core interest to psychologists and behavior geneticists, in addition to the big question of what evolutionary processes maintain variation. One such question may be what mutated genes underlie behavioral variation?

As illustration, I briefly discuss recent work on copy number variants or CNVs. It was once thought that the "normal" human genome could be defined by a shared reference genomic structure, one specifying all single nucleotide sites. In an ideal, extreme form of this view, all genetic variation between any two individuals would consist merely of the aggregate of base differences at all 3 billion or so single nucleotide sites (e.g., A vs. T, C vs. G). And mutations would overwhelmingly consist of single substitutions at a particular nucleotide site. Geneticists have long recognized the existence of exceptions—insertions, deletions, (variable repeat sequences?) or inversions of long chromosomal segments in individual genomes. Recently, however, they found that the purported "exceptions" are anything but unusual (e.g., Iafrate et al., 2004; Sebat et al., 2004). A substantial portion of the genome is subject to "copy number variation"—differences across individuals in number of copies of a chromosomal segment >1000 bases long (in rare cases >1 million bases long). Some individuals might have two copies (one inherited from each parent), whereas others have one (with no copy—a "deletion"—inherited from one parent), and yet others >2 copies (multiple copies inherited from at least one parent). Thus far, several thousand such sequences have been found in the human genome, comprising at least 12% of it (e.g., Kidd et al., 2008; Redon et al., 2006; Wong et al., 2007). Inter-individual variation, then, consists not only of differences at single nucleotide sites. Individuals differ in number of copies of particular DNA strands they possess. Because CNV strands consist of many bases, this "structural variation" may account for more total inter-individual genetic variation than all variation at single nucleotide sites combined (Beckmann, Estavill, & Antonarakis, 2007).

CNVs originate through mutational events, but not, of course, mutations defined as single nucleotide substitutions. One important cause is non-allelic homologous recombination (NAHR; see Bailey & Eichler, 2006; Kim et al., 2008). Through recombination, segments of homologous chromosomes inherited from the two parents are spliced and recombined to create new homologous chromosomes. NAHR occurs when a segment of DNA incorrectly matches up with a segment on the homologous chromosome during recombination, resulting in creation of a new chromosome that contains a deletion, duplication, or inversion. Chromosomal regions containing segmental duplications (SDs) are prone to NAHR. SDs are effectively copy number variations (specifically, duplications) that have gone to fixation; most everyone has the duplication. Specific regions of the human genome are very rich in SDs. NAHR is particularly likely to occur in the presence of SDs because

a long segment of DNA can readily "mismatch" with a similar DNA sequence (its duplication) on a different allele during the crossover process involved in recombination. In short, duplications beget more duplications as well as deletions; duplications yield genomic instability. CNVs, then, are 4–12 times more likely to appear in regions of the genome rich in SDs (e.g., Iafrate et al., 2004; Sebat et al., 2004; Wong et al., 2007; see also Bailey and Eichler (2006)).

The rate at which mutations creating CNVs occurs is several orders of magnitude greater (perhaps 1 in 10,000; e.g., Sebat et al., 2004) than the rate at which single nucleotide mutations occurs. Atypical CNVs (perhaps deletions more so than duplications) tend to be deleterious (Locke et al., 2006).

In the words of one pair of authors, psychiatry was recently hit by a "copy number variant tsumani" (Joober & Boksa, 2009). Whereas psychiatric geneticists have had difficulty identifying single nucleotide variations that robustly associate with major disorders such as schizophrenia, autism, or bipolar disorder (but see Crespi (2008), for a review of some such variations), research in just the last several years has convincingly linked these disorders to CNVs (particularly ones rare and newly arisen in the affected individual; for recent reviews, see Cook & Scherer, 2008; St. Clair, 2009; Zhang et al. 2009). Of course, single nucleotide variations may also predispose these disorders, but largely as a function of rare mutations at sites distributed throughout the entire genome (Keller & Miller, 2006).

Why do CNVs appear to be so important to an understanding of psychiatric disorders? One possibility is that CNVs have substantial effects because they reflect "big" mutations (variations at many nucleotide sites). But it is worth considering another possibility: CNVs are especially important to psychiatric disorders because they are important to uniquely or recently evolved human traits more generally.

3.1. Segmental duplications, CNVs, and human evolution

Segmental duplications, once again, are effectively CNVs that have gone to fixation because selection favored duplication. A duplication of a DNA sequence may bolster level of expression of a gene (or genes) contained within the segment, which selection may favor. Additionally, once a segment of DNA has been duplicated, selection may favor changes in that segment, such that it can serve functions partly distinct from the original copy (while the functions served by the original are preserved). Selection that copies-pastes-modifies is a common route to adaptation (e.g., Taylor & Raes, 2004).

And indeed, SDs appear to have been particularly important in the evolution of the great apes and humans. A rapidly advancing science of comparative genomics finds that there was a burst of increased SDs in the common ancestor of great apes, and SD content has continued to expand particularly in chimpanzee and human genomes (Marquez-Bonet et al., 2009). Though SDs account for only about 5% of the human genome, they account for more divergent evolution between chimpanzees and humans than all single base-pair changes combined. Segmental duplications near the centromeres (especially some chromosomes, e.g., 1, 9, 16) appear to be particularly core to the lineage-specific expansions of the human genome. Consistent with these duplications being adaptive, SD regions are richly inhabited by signatures of positive selection for substitutions within them (i.e., adaptive modification subsequent to duplication; Bailey & Eichler, 2006).

What are the implications for understanding human individual differences? If much of human evolution occurred through changes in SD-rich regions, it stands to reason that these regions play critical roles in the development and expression of many traits derived in the human lineage, phenotypically distinguishing us from close relatives. Some of these unique features are ones that rapidly evolve within coevolutionary systems (e.g., immune function, olfaction, reproduction). More interesting from an evolutionary psychological perspective, SDs also appear to contain genes involved in neuronal development or expressed in neural tissues, perhaps central to human-specific cognitive features (e.g., Dumas et al., 2007; Popesco et al., 2006; Sikela, 2006).

Though substantial levels of SD have likely been selected in the recent human lineage, SDs once again carry a special cost: They predispose genomic instability and hence deleterious CNVs. And what functions are these CNVs likely to disrupt? Naturally, ones facilitated by DNA in SDs, including psychological ones. From an emerging understanding of the evolution of the human genome, then, it is perhaps no coincidence that CNVs have been found robustly important to an understanding of major psychiatric disorders such as schizophrenia and autism.

Do CNVs affect "normal" variations in personality? No study has yet closely examined these associations. One tantalizing finding of a genome-wide scan for genetic markers associated with neuroticism, however, is that genetic markers in CNV-rich regions of the genome were significantly overrepresented in the set of markers with greatest associations (Shifman et al., 2007); variations within these regions may be particularly important to neuroticism. Might the same be true of many other personality traits? Future research will tell.

4. Processes mediating links between genes and phenotypes

Bouchard and Loehlin (2001) emphasized that physiological and psychosocial processes mediate associations between genes and phenotypes, and that we should aim to understand these processes. Oxidative stress is one possible process I have discussed.

More generative from an evolutionary perspective, however, is Bouchard and Loehlin's specific suggestion that psychological development may be adaptively contingent on various conditions an individual finds him or her self in—that what works depends partly on what internal and external resources one can draw upon and that evolution has accordingly favored contingent developmental "tactics" sensitive to possession of those resources.

The broad theoretical evolutionary framework pertinent here is life history theory. It views organisms as entities that harvest energy from the environment and allocate it to fitness-enhancing activities. But what activities? How big do I grow? As a plant, do I expend energy to overwinter or instead die and use that energy to produce seeds that can survive the winter? As a female bird, how many eggs should I produce per clutch? As a male bird, do I help feed offspring or look for more mates? Do I store energy as fat, use it to build muscle, or allocate it to my brain? How much should I invest in immunity against pathogens, how much to repairing my cells? These are all decisions about how to allocate resources. Life history theory offers sophisticated conceptual models that allow us to understand the selection pressures that would favor one decision or another in a particular species filling a particular niche within evolutionary economic frameworks, where fitness is the ultimate currency.

What's optimal at one time of life or given particular circumstances will not necessarily be optimal in other circumstances. Optimal decisions for the young and the healthy, for instance, differ from those optimal for the old and the sick.

Naturally, though I write as though these decisions can be made deliberately, that is not how natural selection has shaped us to make them. Selection has tuned physiological systems that render allocation decisions. For instance, endocrine systems modulate allocation of effort in coordinated ways (Finch & Rose, 1995). Testosterone specifically has been conceptualized by life history theorists as a hormone that modulates allocation of male effort into

traits that foster intrasexual competition—ultimately, a form of competition for mates—and other adaptive features such as immune systems, somatic repair, and perhaps good brains that pay off in survival or, given humans' traditional, ancestral reliance on difficult to extract, high density food packages, production capacities (see, e.g., Bribiescas, 2001; Ellison, 2003).

One major way that evolutionary biology can contribute to an understanding of personality variation is through application of life history theory. Which men, for instance, particularly invest in testosterone-faciliated traits? Why? What circumstances foster allocation of effort into them, and can these effects be understood in terms of adaptive contingent allocation? What are the consequences of these allocations for development of other traits, such as intelligence? This is but one example. I suspect that, in the future, life history theory will have major impacts on our understanding of psychological individual differences.

5. Summary

Fifteen years ago, Bouchard and other behavior geneticists had firmly established that virtually every behavioral trait imaginable was moderately heritable. Tom Bouchard was one of the few in this field who saw that these findings raised a highly important theoretical question: Why are behavioral traits heritable? Evolutionary biology can provide answers. Bouchard and Loehlin (2001) looked to evolutionary biology and sketched out some answers—but perhaps even more importantly, questions that demand answers and programs of research to provide answers. Answers are now coming to light.

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