# Sociogenomic Personality Psychology

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**ABSTRACT** In this article, we address a number of issues surrounding biological models of personality traits. Most traditional and many contemporary biological models of personality traits assume that biological systems underlying personality traits are causal and immutable. In contrast, sociogenomic biology, which we introduce to readers in this article, directly contradicts the widely held assumption that something that is biological, heritable, or temperamental, is unchangeable. We provide examples of how seemingly unchanging biological systems, such as DNA, are both dependent on environments for elicitation and can be modified by environmental changes. Finally, we synthesize sociogenomic biology with personality psychology in a model of personality traits that integrates this more modern perspective on biology, physiology, and environment that we term sociogenomic personality psychology. We end the article with a discussion of the future directions of sociogenomic personality psychology.

With the decoding of the human genome and technological breakthroughs, such as Functional Magnetic Resonance Imaging (fMRI), personality psychology is poised to rediscover its biological roots. It is now codified as "law" that personality traits are heritable (Turkheimer, 2000). Theoretical models have mapped out the pathways between neurophysiological structures and temperament (Depue & Lenzenweger, 2001). Brain imaging studies are beginning to reveal the physiological correlates of traits, such as extraversion (Canli, 2004), agreeableness (Haas, Omura, Constable, & Canli, 2007), and

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neuroticism (Hariri et al., 2002). Molecular genetics studies have demonstrated the association between specific genetic markers and personality traits (Sen et al., 2004). The resurgence of research on the biological foundations of personality harkens back to the work of Eysenck (1967) and a time when personality traits—relatively enduring patterns of thoughts, feelings, and behaviors—were thought to be rooted in biology.

These advances raise a number of interesting issues, especially in light of the intervening decades in which the modal perspective on personality psychology was situationist and openly skeptical of the importance or existence of personality traits (Mischel, 1968). It appears that the situationist critique of personality traits has been put to rest. There are few disputes about the stability of personality over time, as this has been shown to exist even when using different judges of personality at different ages (Block & Block, 2006). The perspective on the cross-situational consistency of personality has changed, such that researchers have shown theoretically meaningful levels of correlations across disparate situations (Borkenau et al., 2004). Moreover, even the argument that personality traits are of little use in predicting important behaviors has been soundly refuted. Personality traits predict numerous, concrete, and important life outcomes across multiple domains (Caspi, Roberts, & Shiner, 2005; Ozer & Benet-Martinez, 2006). In fact, a recent review shows that personality traits predict mortality, divorce, and occupational attainment as well as, if not better than, socioeconomic status and intelligence (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007).

With advances demonstrating the power of personality and the pendulum swinging toward biological perspectives, it is time to reexamine underling theories of biological personality psychology. The current state of biological thinking in personality psychology is surprisingly consistent with Eysenck's perspectives espoused over 30 years ago. This is not to say that much work has not occurred in biological personality but rather that the overarching biological model underlying even modern biological investigations of personality share key features with Eysenck's original models. Alternatively, personality psychology has the opportunity to take advantage of the spectacular advances made in biology in the last few decades, which as we describe below, provide an alternative biological model for personality psychology to consider. In this article, we will review Eysenckian biological models of personality and how they still

inform biological thinking in personality psychology to this day and provide reasons for implementing an alternative perspective derived from biology called sociogenomics (Robinson, Grozinger, & Whitfield, 2005).

## Current Biological Models in Psychology

Eysenck's approach to the biological basis of personality still resonates in today's conceptualizations of biological personality psychology. This is exemplified in Figure 1, in which we have reproduced one of Eysenck's (1972) models depicting the interface between biology, personality, and society. In this model, Eysenck proposed that at the foundation of personality traits were physiological systems that are inherited and often associated with specific brain functions and structures. These physiological systems cause behaviors that then make up personality traits, which in turn cause social phenomena such as crime. We do not intend to discuss the intricacies of Eysenck's model or the validity of its structure. We invoke it here as a representative example of models that place the causal primacy of physiological systems over psychological systems.

DNA is the root of physiological systems in Eysenck's model (Eysenck, 1997) and is often considered an immutable influence on phenotypes throughout the lifespan. Many theories assume that since genetic polymorphisms do not change, then the influence of DNA on behavior must not change. This would result in a constant genetic effect throughout the lifespan. This thinking is exemplified by

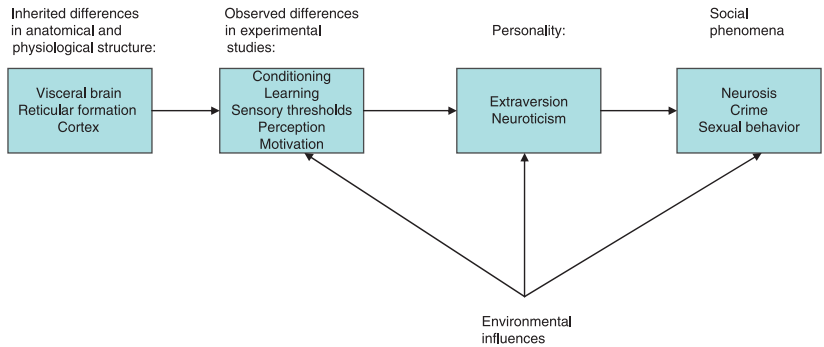


**Figure 1**
Eysneckian model of personality.

theories of personality that dichotomize personality into temperamental (biological) and characterological (learned) components. Temperament is seen by many as individual differences that reflect "expressions of neurobiological mechanisms that have a strong genetic basis" (De Fruyt et al., 2006, p. 539); see also Mervielde, De Clercq, De Fruyt, & Van Leeuwen, 2005). Personality traits have been defined similarly (e.g., McCrae et al., 2000), which would equate personality traits with constructs rooted in physiology and driven by genetic causes—a conceptualization quite similar to Eysenck's. Even those who distinguish between temperament and trait still argue that they are linked such that temperaments are the "biologically based" developmental precursor to adult personality traits and that the two sets of constructs are viewed as "biological" and linked through a common genetic basis (Deal, Halverson, Havill, & Martin, 2005). For example, "temperament has been equated to the hard ice ball around which the softer snow of personality accumulates developmentally" (p. 1267, Graziano, Jensen-Campbell, & Sullivan-Logan, 1998). These definitions of temperament and personality traits are quite similar to Eysenck's model in which the biological substrate to personality is unaffected by environmental influence and that which is biological is presumed to be causal and not caused.

We think it is important to examine more closely what researchers mean when they use the terms "biological," "genetic" and "highly heritable." First, it is nonsensical to describe something as "biological" because there is nothing psychological that is not biological. That is to say, if a person is thinking, feeling, or behaving, multiple biological systems have to be engaged. What we suspect people really mean when they refer to personality traits or temperaments as "biological," "genetic," or "highly heritable" is that there is some component to the trait or temperament that is unchanging. Moreover, the implicit assumption is that this unchanging influence is either DNA or some physiological structure that is largely the result of genetic polymorphisms. As the metaphor alluded to above—temperament is the ice ball—it does not change despite socialization. Or, to invoke a different metaphor, temperament is the biological set point at the core of any given personality trait. People can vary around this set point, but when left to their own resources they will naturally gravitate back toward their biologically driven modal tendency.

At first glance, the biological personality model that Eysenck espoused seems eminently reasonable. If personality traits and temperaments are heritable (Turkheimer, 2000), and there are known biological correlates of personality (Eysenck, 1967; Gray, 1970; Wacker, Chavanon, & Stemmler, 2006; Zuckerman, 1991), then it would not be a radical leap to assume that there is some unchanging biological system at the core of personality traits. However, the heritability of personality traits and the fact that personality traits have biological correlates does not mean that the environment does not play a role in determining personality traits and in shaping the biological systems assumed to underlie traits. As will be seen below, one of the key elements of sociogenomic biology is that it brings a new perspective to the relation between the biology and environment.

## Sociogenomic Biology

Sociogenomic biology is based on one theoretical assumption and two scientific discoveries that are by themselves innocuous but when combined provide a different perspective on the meaning of biology and potentially the meaning of biological personality psychology (Robinson et al., 2005). The assumption, derived from evolutionary theory and sociobiology (Wilson, 1975), is that all behavior is influenced by genes and forces of evolution. The latter is necessarily true for any heritable behavior that has some impact on survival or reproduction, even if it is small (Penke, Denissen, & Miller, 2007). Thus, sociogenomic biology focuses on the behavior of animals that live in groups in which members must cooperate and compete in order to survive and thrive. Clearly, humans fall into this category.

The first scientific discovery that guides a sociogenomic perspective is that the genome has been highly conserved across species. Therefore, understanding social behavior becomes a cross-species effort as the same genes are involved in analogous behaviors in distinctly different animals. This is consistent with the argument that personality psychologists can benefit by becoming intimately familiar with personality processes and structures of other species (Gosling, 2001, 2008; King, Weiss, & Farmer, 2005; Mehta & Gosling, 2006). Sociogenomics provides a profoundly compelling reason to do so—other animals most likely share molecular pathways and genetic

polymorphisms with humans that contribute to similar behavioral syndromes.

The second scientific finding that is most relevant to our argument that biological models of personality need to be updated is that genes are intrinsically dynamic in the way they transact with the environment, which contrasts starkly with the perspectives on temperament and personality traits provided above and directly contradicts an Eysenckian model of personality. One of the most significant recent findings in biology is that the genome, whether it is human or not, is intrinsically dependent on the environment for activation and maintenance. DNA consists of tens of thousands of genes that are responsible for coding different proteins, which are the building blocks of life (Penke et al., 2007). A gene, made of up a segment of DNA, does not directly produce behaviors, emotions, or thoughts. Moreover, genes do not continuously produce proteins. A common misconception is that DNA regulates when the production of protein occurs, since DNA is thought as the prime mover for all things biological. In fact, gene expression can be switched on and off and altered in response to both genetic and environmental factors. This is a result of both biology and the environment being able to influence the complex pathway that exists between DNA and protein production. In a flashback to introductory biology, the production of protein is the result of RNA, not DNA. DNA directs the synthesis of RNA, known as transcription. After transcription, RNA is translated into polypeptides, which ultimately form proteins. When, where, and how much of each protein is produced is contingent on the cellular environment. At each step in the process the cellular environment can be influenced by the environment outside an individual, ultimately affecting the production of proteins. Thus, the regulation of gene expression links the influence of DNA with the environment (Robinson, 2004).

Broadly speaking, differences in gene expression arise from at least two different processes, which may work in concert together. First, variation in gene activity may be inherited through variations in the DNA sequence (genetic polymorphisms). This is what commonly is thought of as "nature." Secondly, gene expression may be influenced through variation in environments. This is commonly considered to be "nurture." These processes work in tandem, resulting in not nature versus nurture, and not even nature and nurture, as commonly construed. Despite the common acknowledgment that

both nature and nurture are important, few conceptualize how the two work together. What is apparent from recent discoveries in biology is that the dichotomy between nature and nurture is not only false but a gross oversimplification (Balaban, 2006; Robinson, 2004). It turns out that both act on the same substrate, the genome. Both can influence gene expression and both impact the brain similarly, with the effect being that nature and nurture should not be thought of as two distinct processes but rather as two sides of the same coin. To demonstrate the transactional relation between environment and gene function, we will provide a few examples drawn from animal biology and behavior genetics studies of humans where both genes and the environment work together to influence behavior.

One of the most dramatic examples of how the environment affects biology comes from the life-course ecological analysis of the blue-headed wrasse (Stearns, 1992). The blue-headed wrasse is a reef fish found in the tropics. The males of the species are typically larger than the females and are, as the name implies, bright blue. Females in contrast are smaller and dull brown. Typically, males gather a harem of females that they guard and with whom they mate. Of course, being bright blue and large makes the males conspicuous. In the eternal reality of fish, this means the male is likely to be eaten by a larger fish. Interesting things happen after the male is consumed by a predator. The females in the harem do not initiate a search for a new male consort. Rather, literally overnight, one of the females transforms into a male. The effect is of course genetically mediated but entirely caused by the loss of the male or a change in the environment. That is to say, environmental change can change the sex of a fish.

Recent methodological advances have allowed a genetic examination of instances, like the blue headed wrasse, where the environment and biology seemingly work together. What has been uncovered is that differences in environment can result in differences in gene expression. For example, when born, worker honeybees start out as caretakers of the hive but eventually transition from caretaker to food gatherer (Robinson, 2002). Genes guide this entire process and timing of behavioral maturation. The transition from caretaker to food gatherer is mediated entirely at the noncognitive, cellular level. The behavioral maturation is associated with changes in expression of at least 2,000 genes in the bee brain (Whitfield, Cziko, & Robinson, 2003). Interestingly, the expression of a specific

gene, the *for* gene, has been linked to rather subtle shifts in the environment. In one such shift, expression of the *for* gene is activated in some of the caretaker bees when there is a shortage of food gatherers in the hive, creating a cascade of biological changes that transition the caretaker bee into a food gatherer (Ben-Sharar, Robichon, Skolowiski, & Robinson, 2002). Here an environmental change triggers the expression of a gene, which results in higher mRNA levels in the brain that ultimately lead to increased protein products that cause changes in behavior. In other words, the specific gene is similar across all bees, but the influence of the gene is contingent on an environmental stimulus.

Changes in gene expression that do not depend on variations in the DNA sequence (genetic polymorphisms) are referred to as epigenetic effects (for reviews, see Feinberg, 2008; Jirtle & Skinner, 2007; Whitelaw & Whitelaw, 2006). Epigenetic effects can occur through multiple mechanisms, one of the best studied being methylation. Methylation represses transcription of the gene, stopping the production of specific proteins. In a fascinating line of studies, methylation clarified the mechanism behind the putatively inherited behavior of stress reaction in rats (Francis, Caldji, Champagne, Plotsky, & Meaney, 1999). Specifically, rats that handled stress better had been treated differently by their mothers when young. These rats had been licked more by their mothers, and variation in licking behavior was heritable. Therefore, the individual differences in response to stress could be due to genetic factors that were passed down from the mother, or it could be environmentally mediated through the licking behavior. Through a cross-fostering design, researchers found that the causal pathway was through licking behavior and its effect on gene expression (Weaver et al., 2004). Maternal licking influenced DNA methylation, which resulted in differences in expression of the glucocorticoid receptor gene. These differences in genetic expression resulted in different activity levels in glucocorticoid receptors. Rats with more active glucocorticoid receptors are better able to tolerate stress than rats with less active receptors. Thus, the difference in stress response was not due to variations in gene sequence but through an epigenetic modification of DNA expression through methylation (Weaver et al., 2004). Again, the effect of the gene was conditional upon the environment.

There is now provisional evidence for epigenetic effects on the human genome. Recently, the methylation patterns of Catechol-

O-Methyltransferase (COMT) gene of 12 monozygotic twins were examined (Mill et al., 2005). While the methylation patterns were highly correlated, they were not perfectly correlated, despite the fact that these were identical twins. This finding indicates that some event occurred during the life course of the twins that changed the way identical genes were expressed. Moreover, epigenetic effects were replicated in a second, larger study of twins that also showed that the level of discordance was correlated with age and amount of time the twins had spent together in their lives (Fraga et al., 2005). Specifically, older twins and twins who spent less time together showed greater evidence of epigenetic effects. This means that even within identical twins, some environmental or biological factors can differentially change the way in which identical alleles are expressed (Mill et al., 2005). This highlights the fact that gene expression in humans, like that of other animals, is not solely based on sequences of DNA. Rather, the environment can shape gene expression in humans as well.

Genetic expression has been difficult to examine in humans because current technologies do not permit a direct examination of expression levels in the brain (with the exception of post-mortem autopsies, e.g., Thalmeier et al., 2007). However, a number of naturalistic studies of heritability and gene-by-environment interactions suggest that the effects of DNA are contingent on the environment. As we noted above, one of the general laws of behavior genetics is that almost any psychological construct is heritable (Turkheimer, 2000), with most estimates of the heritability of personality traits hovering around 50% (Krueger & Johnson, in press). Some have argued that the 50% estimate is conservative and that the true heritability is not only higher but high enough to rule out environmental effects on personality traits (McCrae et al., 2000). Many of the initial estimates of heritability were based on a model that did not objectively include environmental effects and was therefore misspecified. In contrast, when measures of environmental influence are explicitly modeled, estimates of heritability are moderated by the environmental effects (Krueger, South, Johnson, & Iacono, in press). For example, the heritability of negative emotionality decreases and the effect of shared environment increases at high levels of parental conflict (Krueger et al., in press).

Recent research has also shown that specific genetic polymorphisms interact with life experiences to predict such complex

psychological phenomena as depression (Caspi et al., 2003; Jacobs et al., 2006; Kendler, Kuhn, Vittum, Prescott, & Riley, 2005), delinquency (Caspi et al., 2002; Foley et al., 2004; Kim-Cohen et al., 2006) and aggression (Verona, Joiner, Johnson, & Bender, 2006). These gene-by-environment interactions (G $\times$ E) reflect that certain forms of a gene are more susceptible to the environment than others. For example, in the Caspi et al. (2003) study, a functional polymorphism in the promoter region of the serotonin transporter gene moderated the influence of stressful life events on depression. In each of these cases there was no genetic main effect; the association only came about when taking the environment into consideration. These studies illustrate that the effect of genes may often not be direct. Genetic polymorphisms do not determine behavior; rather, different alleles modulate responses to the environment.

These findings from both animal biology and human psychology provide clear evidence that DNA sequences are not the simple, unchanging causal mechanisms depicted in typical biological personality models. Moreover, consistent with a sociogenomic perspective, the co-action between environment and allelic variation is necessary for natural selection to occur (Ridley, 2003). As we have seen above, alleles do not directly code for behavior; rather, alleles affect responses to the environment. The DNA sequences that get passed on have evolved to work with specific environments. Thus, survival of an allele in biological evolution depends on the interplay between itself and the environment. Furthermore, since personality development is an environmentally dependent process, genes are involved in this process the entire time development occurs, which many have argued is lifelong (Gottlieb, 2003; Johnston & Edwards, 2002; Roberts & Woods, 2006). The critical point to take from these examples is that genetic effects, once thought to be a constant influence, are often dependent on environments to be triggered. Interestingly these environmental contingencies do not solely occur in childhood and young adulthood when the brain is still developing. The interplay between the environment and one's genome is lifelong. In terms of Eysenck's model, it is clear that environments can and do affect physiological systems, even one as basic as DNA.

If environments are capable of shaping genetic expression, and triggering dramatic physiological changes, then it would seem reasonable to expect that functional neuroanatomical structures should also change in response to environmental input. Consistent with this

perspective, there is ample evidence that human hormone levels, neurotransmitter functioning, and brain structures change in response to environmental intervention. For example, men who get married or engage in a committed relationship have a significant drop in testosterone (Mazur & Michalek, 1998; McIntyre et al., 2006). Recent research shows that cognitive behavioral therapy changes blood flow and functioning in the brain (Felmingham et al., 2007). Finally, long-term studies of the experience of stress has shown that structural changes in the brain result from the accumulation of what is called allostatic load, which is where the usually adaptive stress response functions improperly and works against the body (McEwen, Liston, & Morrison, 2006). Specifically, the prefrontal cortex is remodeled under long-term stress, with atrophy in the medial prefrontal cortex and the hippocampus and expansion in the orbitofrontal cortex and basolateral amygdala. Thus, just as is the case for nonhuman animals, human physiology can be affected quite significantly by environmental interventions.

Given the evidence that environments influence genetic expression across species and that both personality and physiology in humans change through environmental influence, it is impossible to endorse or support the original Eysenckian model or similar modern variants of deterministic biological personality models (e.g., McCrae & Costa, 1999). As a point of reference, we provide a new model that is associated with a sociogenomic personality position that folds in environment, biology, traits, and states into one comprehensive model (see Figure 2). In contrast to Eysenck's original model, traits are latent constructs, as is the environment. We have left biological effects as manifest variables because it is quite possible that some aspects of biological effects, such as neuoranatomical structure, may not fit a latent conceptualization. This model was created so as to account for the multiple pathways through which environmental effects could shape genetic expression, physiology, and personality traits.

Several aspects of the new model are unique. Unlike Eysenck's original model, the proposed model involves reciprocity between many of the connected variables. For example, the environment can affect biological factors directly. Accidents can impart serious biological insults that affect personality, such as in the case of Phineas Gage, who after having a tamping rod removed from his brain became more aggressive and hostile than before his injury. Environments can also affect biological systems indirectly through

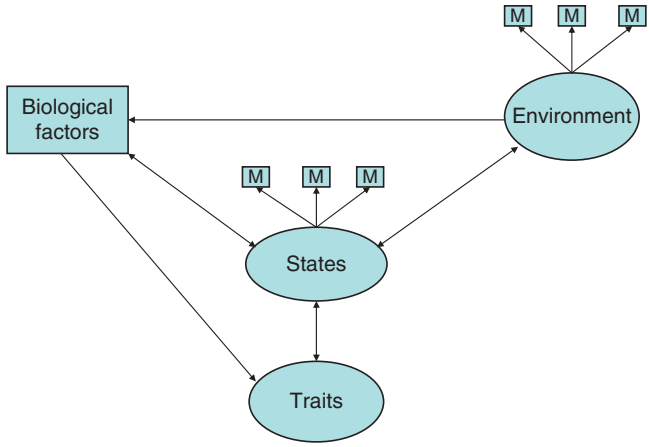


**Figure 2**
Sociogenomic model of personality.

the way environments make people think, feel, or behave in any given situation (e.g., states). For example, it is clear that environmental factors can affect state levels of thoughts, feelings, and behaviors, and in turn, how someone thinks, feels, and behaves can affect their biology. On the other hand, some pathways are direct and unidirectional. Biological factors will not affect the environment directly but indirectly through the personality trait or state. Viewed this way, personality serves as the conduit between biology and significant life outcomes. Furthermore, the trait can be directly affected by biological functions—children are born with a wide variety of temperamental starting values that are then shaped by environmental experiences. In turn, traits will not affect biological structures directly but indirectly through the effect of continuous state effects. For example, as noted in the McEwen et al. (2006) research, continuous states of anxiety and stress can lead to neuronatomical changes in brain structures. Stressful states likely interact with genes responsive to stress, which, in turn, will affect the neuroanatomy that shapes the habitual ability of the person to respond to future environmental insults—thus a trait-like phenomenon.

We also incorporate the fact that environments interact with genes to affect states and traits. For example, individuals with two copies of the short form of the serotonin transporter gene (5-HTT) are prone to higher levels of aggression when provoked in an experimental setting (Verona et al., 2006). We believe that similar

processes played out over long periods of time can account for the gene-by-environment effects on delinquency and depression that occur decades apart (Caspi et al., 2002, 2003). That is, the stable depression that emerges from the interaction between significant life events in adolescence and the 5-HT transporter gene occurs because of the continuous state experiences (e.g., rumination) that result in longstanding changes in neurotransmitter levels or neuroanatomical structures that then cause consistent, global depression.

One of the keys to the sociogenomic-inspired personality model that differs not only from Eysenck but from many other personality models is the placement and prominence of the role of states. The necessity of states being the prime mediator in the model became apparent when we attempted to answer the question of how environments literally get under the skin. As already noted, environments can cause change through gross physiological insults. On the other hand, environmental effects typically act on momentary thoughts, feelings, and behaviors—that is, environments cause changes in states that then affect changes in traits in a bottom-up fashion (Roberts, 2006). This bottom-up nature of the socialization effect of environmental experiences is an important distinction. Environments will not affect personality traits directly unless they involve some dramatic change in physiology. On the other hand, the slow incremental effects of environmental experience, which appears to be the most common pattern of development (Roberts, Wood, & Caspi, in press) can be explained by the mediation of states. Only through prolonged effects on momentary thoughts, feelings, and behaviors will neuroanatomical structures or gene expression be changed enough to change the model level of the system and thus change the personality trait (e.g., McEwen et al., 2006).

The structure of this new model is highly relevant to the presumption that if a personality trait is "biological," "heritable," or "genetic," it is immutable and therefore causal. As we saw above, many psychologists assume, explicitly or implicitly, that if something is biologically based, it is causal because it cannot be changed. The assumption that biology reflects the ultimate level of causality is built on the assumption that biological systems are distal causal factors that are not affected by any other link, as in Eysenck's original personality model. The evidence from animal biology, behavior genetics, and personality development research directly contradicts this notion. Just because something is rooted in DNA does not mean that

it is impervious to environmental influences, even after the DNA sequence is set. Thinking of personality psychology through this sociogenomic lens leads to a number of questions and insights that we turn to now.

## Unfinished Business in the Move Toward a Sociogenomic Personality Psychology

If current trends are any indication, personality researchers will explore the biological foundations of personality in the coming years. Our hope is that personality psychologists not only move in the direction of biology but actually move all the way over to become more like some of our colleagues in biological sciences. It is clear from our limited forays into animal biology that many biologists do not suffer from naïve assumptions of nature versus nurture (Balaban, 2006; Robinson, 2004). Though many people endorse the perspective that it should be nature *and* nurture, few if any psychologists can articulate what that means (cf. Johnson, 2007). In contrast, sociogenomic biologists have provided personality psychologists keen insights through their work detailing how evolution, genes, physiological systems, and the environment work in conjunction with one another. We believe that a biological model of personality psychology that adopts a similar approach will avoid meaningless arguments over nature and nurture. It is not only both but most interestingly both.

What would a sociogenomic personality psychology look like? First, a sociogenomic personality psychology would be comparative from the start. Possibly one of the most important foundations of sociogenomic biology is the fact that the genome is conserved across species (Robinson et al., 2005). This means that many of the molecular pathways and genetic polymorphisms associated with human personality exist in other species and are often involved in analogous behavioral syndromes (e.g., Winstanley, Dalley, Theobald, & Robbins, 2003). This fact is even more significant given the fact that many animals exhibit similar personality traits as humans (Gosling, 2001; Gosling & Graybeal, 2007; Sih, Bell, Johnson, & Ziemba, 2004). It also has the potential to skewer "homocentric" perspectives on personality that arise from solely focusing on humans. For example, it is often assumed that "characterological" traits in humans, such as self-control, are primarily the result of socialization and act

as a control mechanism over more biologically driven emotional temperaments that are presumed to be in place from birth, such as neuroticism (Bell, Backstrom, Huntingford, Pottinger, & Winberg, 2007; Carver & Miller, 2006). On the other hand, individual differences in control mechanisms are identifiable in species that predate human evolution, such as stickleback fish, and these control systems are dependent on the environment for their development (Bell et al., 2007). Moreover, the same physiological system that involves the serotonin neurotransmitter appears to be involved in control of fish and human behavior (Carver & Miller, 2006). Therefore, it is a distinct possibility that humans have a control system that is analogous to control systems in many other species with whom we share similar genes that help to structure these systems. Integrating findings across species, such as these, may provide keen insights into human personality and human personality development (Gosling, 2001). In this case, it may dispel the myth that character traits are particular to human development and solely dependent on the environment.

Our point in invoking the animal examples is to illustrate the point made by sociogenomic biology that key insights into social behavior can be gleaned from an examination of multiple species (see also Mehta & Gosling, 2006). That is to say that humanity, per se, should not be the sole focus of personality psychology. Rather, the genome is the target we should be training our attention on. Since the genome has proven to be a remarkably conserved across species, when looked upon with a sober eye, it has to be given significant credit for both the existence of human psychological functioning and of the behaviors and actions of all of the successful life forms on the planet. Although it is important to know how the genome affects humanity, it is also important to keep in mind the immense capacities of the genome as exemplified in other species. Based on this, studying other species can shed light onto how the human genome operates. For example, technological limitations prohibit direct examination of gene expression in the human brain. However, brain expression can be examined in mice, which share similar genes and neural systems. By focusing on the genome across species we will come to a better understanding of the way in which genes interact with environments to cause personality and in turn shape the development of humans and other animals across their life course (Howell et al., 2007).

Of course, a sociogenomic personality psychology would embrace the current explosion of research on the genetic architecture of personality. Nonetheless, it would also argue for a different approach than the robotic pursuit of genetic associations with self-reports, which have resulted in single genes being associated with a multitude of diverse phenotypes but with few, if any, strong and replicable associations (Caspi & Moffitt, 2006; Ebstein, 2006). Rather than a direct genotype to phenotype account of functioning, a sociogenomic approach would integrate the environment into personality genetics and embed research questions in evolutionarily relevant contexts.

This poses interesting questions. What are the evolutionarily relevant contexts for personality trait development? Moreover, what are the effects of these environments on genetic expression and neurophysiology? If environments influence certain epigenetic mechanisms, then is it possible to reverse genetic effects or gene-by-environment interaction effects? What are the important times in development that certain environments are most potent? At this stage in our understanding of personality development, we know almost nothing about the relevant environmental stimuli responsible for the development of specific personality traits. This is at once appalling and invigorating. It seems an egregious oversight in our understanding of human nature that we cannot articulate the most relevant and important environments for personality development and their timing across the life course. At the same time, this is an opportunity, both theoretically and empirically, for a new generation of personality researchers to tackle a key research need.

A sociogenomic perspective would additionally lay out the complex pathway from genes to behavior. What are the genes that are expressed when someone goes through a traumatic incident, and how do allelic differences result in interindividual differences in responses to such an incident? Do these immediately expressed genes trigger other genes to be expressed causing a biological cocktail of factors that influence multiple brain areas? What if someone intervenes and helps to change that person's reaction to the traumatic event? Do the changed cognitions and emotions that result from the environmental intervention engage other molecular pathways to counteract these biological processes and possibly change associated traits? What physiological pathways are influenced by these thoughts and feelings? These are just some of the questions that must be laid

out in understanding how biological systems relate to manifest behavior and personality.

As the lines between biology and psychology blur, it is necessary to put these complex biological pathways into perspective. As neuroscience continues to unravel the complexities of our brains, there still exists the need to concentrate on the phenotypic structure of personality. A sociogenomic personality psychology would embrace a detailed understanding and differentiation of the constructs we measure. On this front, personality psychology has excelled. No other field cares more about nor obsesses more over measurement issues than personality psychology. Nonetheless, confronting biological systems may necessitate additional work on the measurement and assessment of personality. Take an example from recent research on the links between serotonin functioning and both impulse control and depression (Carver & Miller, 2006). Carver and colleagues argue that to understand the role of serotonin, one must differentiate between two modes of self-regulation. Specifically, they propose that, in the first mode, people have a quick, associative, emotionally based system organized around approach and avoidance emotions. The second mode is represented by an effortful control system that is used either to engage in voluntary behavior—working out to improve fitness—or inhibit inappropriate behavior—not hitting the person who makes you angry. Presumably, this effortful control system is synonymous with conscientiousness.

Using this system, one can gain a keen understanding of specific psychological syndromes and constructs. Depression reflects a relatively inactive approach system and inactive effortful control system—neither the negative emotions are controlled or the behaviors and thoughts necessary to shake the negative spirals symptomatic of depression. Analogously, impulse control conflates high approach and low effortful control. According to Carver, Johnson, and Joorman (2007), the serotonin system facilitates greater effortful control and thus has pervasive effects on seemingly unrelated psychological constructs, such as depression, angry hostility, and impulsivity. From a measurement perspective, we must ask ourselves whether our current crop of personality measures capture the systems like those proposed by Carver and others. It is likely that we will make further strides in personality science if we hone our measures to better capture physiological systems currently being identified.

Fully implementing a sociogenomic personality psychology will require training of new methods, the learning of new terminology, and the consilience of vastly different disciplines (Wilson, 1999). This prospect is both exciting and overwhelming. The field of personality psychology will undoubtedly become more cross-disciplinary in the future to aid in these advances. Of course, this does not mean that all personality psychologists must capitulate and get advanced training in biology. There are many questions that do not necessitate bridging levels of analysis. For example, investigating the predictive validity of personality traits, personality development, or the mechanisms that explain the effect personality has on behavior remain significant and important scientific endeavors despite not being intrinsically connected to biological systems.

In sum, we have discussed issues relevant to the reinvigoration of biological studies in personality psychology and in doing so have borrowed and elaborated on a theoretical model from biology. In our move toward a sociogenomic personality psychology, we see that DNA is not always at the helm of the causal ship. Environments interact with genes to produce the biology behind behavior. Given these findings, we believe that an integration of psychological and biological concepts that move beyond traditional biological models like those found in Eysenck's work will be instrumental to the future viability of a biological personality psychology.

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