Current Thinking About Nature and Nurture

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A devil, a born devil, on whose nature Nurture can never stick

William Shakespeare (1611 a.d., *The Tempest*)

1 Introduction

Curious people typically wonder at some point in their lives whether they might have been different if they had had different experiences while growing up. It is clear to all of us from casual observation that some of our characteristics are affected by our experiences; children growing up in Calais, France typically speak French, while children growing up just across the English Channel in Dover, England typically speak English, reflecting these children's exposure to French and English, respectively. In contrast, some of our characteristics are not obviously affected by our experiences at all; children often have facial features like their biological parents' facial features, regardless of whether or not they are adopted at birth. Likewise, some of our normal characteristics, such as five fingers on each hand, are present at birth, contributing to the impression that experiences play no role in the development of these traits. Such observations lead us to think that certain aspects of our behavioral characteristics, too—for example, a person's intelligence or personality—might not be affected by experience. But despite the intuitive appeal of such a perspective, empirical and theoretical investigations have now made it clear that this way of thinking misrepresents the development of both our biological and psychological traits (Bateson and Gluckman 2011; Blumberg 2005; Gottlieb 2007; Jablonka and Lamb 2005; Lewkowicz 2011; Lewontin 2000; Lickliter 2008;

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Meaney 2010; Moore 2008a; Noble 2006; Oyama 2000; Robert 2004). In fact, all of our characteristics are influenced by both biological and experiential factors.

The idea that some characteristics are caused by experiences whereas others are inborn has a long history, dating back at least to William Shakespeare's early seventeenth century work in the humanities and to Sir Francis Galton's late nineteenth century work in the sciences. As the first scientist to juxtapose the words Nature and Nurture (Plomin 1994), Galton defined Nurture as consisting of "every influence from without that affects [a person] after his birth… [including] food, clothing, education, or tradition […] all these and similar influences whether known or unknown" (Galton 1874, p. 12). In contrast, he used the word Nature to refer to the causes of traits that appear uninfluenced by experience. In large part because he was Charles Darwin's half cousin, Galton was interested in the transmission of characteristics across generations (Kevles 1995), and as one of the first individuals to investigate how experiences and heritages influence people's characteristics, the path he blazed strongly influenced modern conceptions. In particular, he believed that a sharp distinction between Nature and Nurture was justifiable (Gottlieb 1992). Galton's proposition that Nature and Nurture can be considered as dichotomous factors that contribute independently to our traits led directly to the modern characterization of Nature and Nurture as oppositional, as implied by the word 'versus' in the stock phrase *Nature versus Nurture.* Although Galton's conceptualization was ultimately unable to withstand close scrutiny, Nature and Nurture continue to be presented in some quarters as contrasting influences on development.

Galton's erroneous view has implications that go far beyond academic debates about biology. Having established the notion of "eugenics" based on his ideas about Nature and Nurture, Galton advocated policies wherein governments would "rank people by ability and authorize more children to the higher- than to the lower-ranking unions… [while the unworthy would] be comfortably segregated in monasteries and convents, where they would be unable to propagate their kind" (Kevles 1995, p. 4). The emergence of these kinds of ideas in the early twentieth century ultimately led to forced sterilizations in the United States and to genocide in Nazi Germany. As was appropriate, the rejection of eugenics after World War II did not entail the rejection of Galton's broader framework for the study of human characteristics; if Nature and Nurture really were oppositional factors influencing human development, people would simply have to come to terms with any implications of this reality, even if they found such implications politically distasteful. As it happens, scientists now know that Nature and Nurture collaborate to make us what we are (Moore 2002), but one of the lessons of the tragedies of the early twentieth century is this: our beliefs about these issues have important influences on our behaviors in both the public and private domains.

Molecular biology is a relatively arcane science, but to the extent that discoveries in this field bear on questions of Nature and Nurture, they are likely to have implications for our political and personal actions. For example, if the public generally believed that obesity can be avoided with a vegan diet, their reaction to skyrocketing rates of obesity would likely be different than if they believed some people have genes that cause them to gain weight over time no matter what they eat. Of course,

molecular biologists understand that individual genes never single-handedly cause characteristics like obesity—or any other phenotypes for that matter (Noble 2006; Stotz 2006)—but some molecular biologists sometimes speak and write in ways that can confuse readers about this point. And regardless, the public does not get most of their information about genes directly from molecular biologists. Instead, they often receive information like the account in an article on the *Newsroom* website of the University of California, Los Angeles (Wheeler 2010), which reported that geneticists have made:

> the startling discovery that nearly half of all people in the U.S. with European ancestry carry a variant of the fat mass and obesity associated (FTO) gene, which causes them to gain weight – from three to seven pounds, on average – but worse, puts them at risk for obesity… [and that the same gene] is also carried by roughly one-quarter of U.S. Hispanics, 15 percent of African Americans and 15 percent of Asian Americans.

Those uneducated in molecular biology could be forgiven for concluding—mistakenly!—that if a prestigious university like UCLA is reporting on the discovery of an "obesity gene" that *causes* weight gain and that is "carried by more than a third of the U.S. population," the obesity epidemic currently plaguing the U.S. need not be a reflection of the high-calorie diets and sedentary lifestyles typical of contemporary Americans. Such a conclusion could easily lead an obese person to attribute their condition to their genes and thereby rationalize continuing gluttony. Similar arguments could be made about people's beliefs in genes that determine IQ, which could lead to voting against the use of tax revenues for supporting public schools; why, some might argue, should we spend money on the education of children who might be "biologically" unable to learn?

Our beliefs about genetic and environmental contributions to people's characteristics influence what we do. For this reason, there is significant value in biology teachers being able to impart to their students an accurate understanding of how Nature and Nurture interact to produce our biological and psychological characteristics.

2 Cultural Lag

Among those who have considered the issue in great detail, thinking about Nature and Nurture has not changed significantly in the past few decades. Certainly by the turn of the millennium, it was already clear that construing Nature and Nurture as discretely different influences on development was an obsolete way of approaching questions about the origins of biological and psychological characteristics (Moore 2002). In fact, 10 years ago, the biologist Sir Patrick Bateson chose the title "The corpse of a wearisome debate," for his review of Steven Pinker's (2002) book *The blank slate: The modern denial of human nature*. From his review, it is clear that Bateson already believed in 2002 that books like Pinker's are not a valuable contribution to our understanding of "human nature." Nonetheless, as is evident from the recent publication (or re-issuing) of books such as *The mirage of a space between*

Nature and Nurture (Keller 2010), *The Nurture assumption: Why children turn out the way they do* (Harris 2009), or *Kids: How biology and culture shape the way we raise young children* (Small 2011), theorists continue to write about "the Nature versus Nurture debate" and publishers continue to believe there are people interested in reading about it. One sensible question we can ask is: why?

One reason this "debate" continues to generate interest is captured by the words "cultural lag," which Bateson (2002) used to refer to the fact that some people remain unaware of theoretical advances in a field long after the new way of thinking has become canonical in that field. Because of cultural lag in some quarters, reiteration of the essential interdependence of Nature and Nurture can still be merited, which is why a book like *The mirage of a space between Nature and Nurture* (Keller 2010) continues to be a valuable contribution to the literature on this topic. However, the recalcitrant persistence of Galton's outmoded perspective is not merely a function of passive cultural lag but rather is, in some cases, actively maintained. For example, in *The blank slate,* Pinker argued that "another book on nature and nurture" (Pinker 2002, p. vii) was warranted, *not* because of how important it is to debunk the simplistic Nature-versus-Nurture idea, but because of his perceived need to defend the idea that certain characteristics—for instance, intelligence (Herrnstein and Murray 1994) and rape (Thornhill and Palmer 2000)—are influenced by biology. In writing such a book, Pinker succumbed to the temptation to "pour scorn […] on those people suffering from cultural lag" (Bateson 2002, p. 2212), namely those people who continue to cling to the indefensible idea that some human characteristics are completely *un*influenced by biology. But in so doing, Pinker (perhaps inadvertently) perpetuated the beliefs that Nature and Nurture are separable and that they are independently measurable influences on our characteristics. Thus, although a nuanced understanding of how genetic and non-genetic factors *really* interact has obviated the Nature-Nurture debate, the debate lives on because some writers preserve it (whether they intend to or not). Books like *The blank slate* encourage a false understanding of the determination of our characteristics, by claiming that even if Nature and Nurture typically interact in complex ways, "in some cases, an extreme environmentalist explanation is correct … [whereas in] other cases […] an extreme hereditarian explanation is correct" (Pinker 2002, p. viii). In fact, neither of these extreme views is ever correct, and claims to contrary themselves reflect a form of cultural lag.

So, there are multiple forms of cultural lag, all of which need to be addressed by writers who can reiterate what has been accepted for decades in some corners of the biological and social/behavioral sciences (Beach 1955; Blumberg 2005; Gottlieb 1997; Johnston 1987; Lehrman 1953; Lewontin 1983). To those who would argue that Nature is more powerful than Nurture in determining our characteristics (i.e., cultural lag dating to Galton in the nineteenth century), the case must be made that Nature and Nurture are equally influential during development. To those who would argue that Nurture is more powerful than Nature (i.e., cultural lag dating to the 1950s, when behaviorists held sway in American psychology), the same case must be made. To those who would argue that Nature-Nurture interactionism "might turn out to be wrong" (Pinker 2002, p. viii)—a form of cultural lag dating only to the

early twenty-first century, but which is nonetheless significant—the case must be made that Nature and Nurture are now known to *always* interact during development. To those who would argue that it is a reasonable goal to attempt to measure *how much* Nature and Nurture each contribute to the development of particular characteristics (e.g., Plomin 1994), the case must be made that this question does not actually make sense once we acknowledge that Nature and Nurture are both *essential* to the development of those characteristics (a point considered in more detail in the next section). Once these various forms of cultural lag have been addressed, scientists can turn their attention to the truly consequential question of *how* Nature and Nurture interact in the production of particular characteristics. That is, rather than spending time answering nonsensical questions about how *much* Nature or Nurture influences the development of a characteristic, the question that should be driving our research programs and that should be situated at the center of our life sciences curricula is: *how is it* that genetic factors, proteins, cells, organs, organisms, populations of individuals, cultural factors, and other aspects of an organism's environment co-act to produce the organism's traits (i.e., phenotypes) in development?

3 Definitions and Conceptual Problems

Making the case that Nature and Nurture are both always essential—and therefore equally important—contributors to development requires clear definitions of these words. Early in the scientific consideration of Nature and Nurture, Galton adopted a decidedly vague definition of Nurture (cited previously), and considered everything else to be Nature. More than a century later, after biologists elaborated their understandings of molecular (i.e., genetic) contributions to inheritance, things became clearer; in the latest edition of their textbook *Behavioral Genetics*, Plomin et al. (2008) effectively defined Nurture as "environment" and Nature as "genetics" (p. 2). Because Galton was primarily concerned with the extent to which characteristics could be inherited and thereby run in biological families, it makes sense that his intellectual heirs—quantitative behavioral geneticists like Plomin and colleagues—would define Nature as "genetics;" after all, biologists for the past 100 years have generally believed that only DNA—the genetic material—is transmitted from generation to generation (Jablonka and Lamb 2005). Numerous theorists have recently argued that this belief reflects an unhelpfully narrow understanding of inheritance, and that a convincing case can be made that non-genetic factors can be inherited from our ancestors too, albeit via different mechanisms than those responsible for transmitting genetic factors (Carey 2011; Gottlieb 1992; Griffiths and Gray 1994; Harper 2005; Jablonka and Lamb 2005; Johnston 2010; Laland et al. 2001; Lickliter and Honeycutt 2010; Moore 2013; Uller, this volume). But regardless, if we accept the definition of Nature as "genetics" and Nurture as "environment," two problems with Galton's foundational conceptualization of the Nature/Nurture issue immediately become apparent.

First, there are a number of biological components that lie between genes and environments, and although these components occupy levels at which crucial phenotype-building interactions occur (Johnston and Edwards 2002), they are typically ignored in Galton-style behavioral genetics investigations. For those who have not studied biology, it can be easy to forget that genes can be considered to be physical structures with specific spatial locations and that they operate, therefore, within specific contexts (see Burian and Kampourakis, this volume). Genes can be thought of as analogous in some ways to the smallest elements in a set of nesting Russian *matryoshka* dolls; our genes constitute parts of our chromosomes, which are located within the nuclei of most of our cells, which constitute our organs, which are surrounded by hormones, fluids, and other organs, all of which are located within our bodies. Because genes and the environment outside of the body are both able to influence the states (or existence) of the various bodily components that lie *between* the genes and the environment (Gottlieb 1991a, 2007; Lickliter and Honeycutt 2010), it follows that an understanding of trait development that references only Nature and Nurture—and not these other in-between levels of biological systems—must be an incomplete understanding. In fact, a gene does what it does in part because of molecules present in its local environment (i.e., inside the nucleus of a cell). The simplistic idea that genes and environments are independent contributors to trait development fails to capture the complex reality that one gene's products can constitute the "environment" of another gene, and that environmental factors (e.g., a specific nutrient, a specific person, an altered light cycle, etc.) can have their effects on a trait by influencing biological factors that lie between genes and environments (e.g., hormones, epigenetic marks, neurons, etc.). When one considers the space between an animal's genes and its environment, it becomes rather more difficult to define Nature and Nurture in a way that clearly distinguishes between them (see Bateson and Gluckman 2011, for additional examples that strengthen this argument).

A second, related point arises when Nature is defined strictly as "genetics." Galton famously claimed that "when nature and nurture compete for supremacy on equal terms […] the former proves the stronger" (Galton 1874, p. 12), but this claim becomes utterly inconceivable when we define Nature as "genetics." Although modern behavioral geneticists, too, sometimes imply that genetic factors can be "stronger" than environmental factors in the development of some traits (e.g., see Deater-Deckard et al. 2006; Yamagata et al. 2006), the fact is that genetic factors, when isolated from their cellular and broader contexts, are inert (Noble 2006; Keller 2010); independently of other factors, genes *per se* have no "strength" at all. Instead, genetic and environmental factors *collaborate* to build traits (Moore 2002; Lewkowicz 2011), and when two or more factors are both *required* to produce an outcome, none of the factors can be more important—stronger—than any other. By analogy, consider the internal combustion engine under the hood of most automobiles. Such engines require fuel and an ignition spark to operate normally, and the absence of either of these components renders the engine non-functional. Just as it makes no sense to ask if the gasoline or the spark has the "stronger" effect on the functioning of the engine, it makes no sense to conceive of Nurture and genetics as

factors that "compete for supremacy" with one another (Moore 2011). Of course, different observers in different contexts might have reasons for choosing to focus on one factor over another, but it would be a mistake to believe that either factor ever actually has a *stronger* influence than the other on an outcome in a given situation. In their natural contexts, genes are essential contributors to processes that require essential non-genetic contributors as well.

4 Heritability and Its Weaknesses

Modern quantitative behavioral geneticists understand what Galton did not, namely that "the environment plays a crucial role at each step" (Plomin et al. 2008, p. 305) in the development of our psychological/behavioral characteristics. Nonetheless, a research method Galton pioneered to tease apart Nature and Nurture—studies of identical and fraternal twins—provided the data for 5,000 articles on behavioral genetics published between 2001 and 2006. Thus, even though modern behavioral geneticists understand that genetic and environmental factors always both play vital roles in trait development—which necessarily means that neither can ever be more important than the other—they continue to rely on a century-old technique that Galton devised specifically to "appraise [Nature's and Nurture's] relative importance" to the appearance of traits (Galton 1907, p. 131). Moreover, in their empirical research reports, modern behavioral geneticists write about statistical "heritability estimates," which are the primary product of twin studies, in ways that make it seem as if it is possible to measure the relative importance of Nature and Nurture. To give one of many possible recent examples, the authors of a twin study on impulsivity in adolescence concluded that their calculated heritability estimates were "consistent with estimates from […] past studies, suggesting that impulsivity is influenced around 40–45 % by genetic factors" (Niv et al. 2012). Such a claim would imply to many readers that an accurate measurement has been made of the relative strength-of-influence of genetic factors on impulsivity. But although numbers like these suggest that traits can be more influenced by genetic or by non-genetic factors, it is actually not possible to apportion causation of traits to such factors in this way.

A reasonable question to ask, then, is why our modern research literature is littered with what appear to be estimates of the relative importance of Nature and Nurture to trait development when the facts of molecular biology clearly indicate that both factors are always indispensible, and that therefore, it is never possible to evaluate which is the more important factor. The answer to this question likely has to do with the fact that the products of twin studies—heritability statistics—are notoriously misleading, in that they *appear* to reflect the relative importance of genetics in trait development even though they really do not (Block 1995; Keller 2010; Moore 2006, 2008a, 2013). Rather than revealing anything about the extent of genetic influence on trait development, these statistics (e.g., the 40–45 % reported by Niv et al. 2012) actually reflect the extent to which *variation in a trait* across a population can be "accounted for" by variation in genes across that population.

At first glance, a factor that accounts for the variation in a trait seems like it must be the cause of the trait, but in fact there are crucial differences between *causing* a trait and *accounting for variation* in that trait. Quantitative behavioral geneticists use an approach that can reveal *statistical* interactions that account for variation, but these kinds of interactions are very different from the "causal-mechanical" interactions (Griffiths and Tabery 2008, p. 341) known to characterize the developmental process itself. For this reason, even if a twin study of a characteristic reveals *no* statistical interaction between genetic and environmental factors, it is still the case that the development of the characteristic in individuals is *caused by mechanical interactions* between such factors (see Griffiths and Tabery 2008, for additional consideration of these two very different meanings of the word "interaction"). Because heritability statistics are about accounting for variation and not about causation, they do not actually reflect the strength of influence of genes on the development of a trait, even if it seems like they do. Moreover, it is not clear that there are interconnections between accounts of trait variation across a population and explanations of trait development in individuals (Moore 2008b), so the heritability estimates generated by twin studies do not even necessarily point the way toward genetic factors that might warrant further study (see Block 1995, for additional consideration of these issues).

These are not novel points. For example, nearly 40 years ago, Lewontin (1974) pointed out that it is possible for variation in genetic factors to account for a high percentage—even 100 %—of the variation in a trait in a population, but that this does not mean genetic influences on that trait are any "stronger" than non-genetic influences. The development of a trait with a heritability of .80 (or even 1.0) can be influenced by environmental factors just as much as can the development of a trait with a heritability of .05 (Moore 2006, 2013). Of course, quantitative behavioral geneticists (e.g., Plomin 1990) understand this distinction between what heritability statistics can do (account for variability) and cannot do (explain the cause of a trait), but the distinction appears to be virtually impossible to maintain as they write about their findings. As a result, these researchers report their calculated heritability estimates, but then often misconstrue them as meaning something about the strength-of-influence of genetic factors—Nature—on trait development. In a masterful treatment of this problem, Keller (2010) has considered both the causes and consequences of this sort of conceptual "slippage" (p. 34), which, she argues, has arisen from the fact that the word "heritable" has come to have more than one meaning. Without reciting her arguments, it might be enough to note here that although it seems like the heritability estimates generated from twin studies should tell us something out how *inheritable* various traits are, they actually cannot.

Because heritability statistics have been the subject of unrelenting criticism from philosophers, biologists, and psychologists for nearly four decades, it is unnecessary to recount here why they are widely recognized as being unable to address the kinds of Nature vs. Nurture questions Galton and his followers in behavioral genetics hoped they would. In virtual unanimity, theorists have come to question the value of heritability statistics, particularly in studies of human beings. Heritability, which is almost always the metric referenced by those attempting to argue that Nature or Nurture are more important in the development of a given a trait, is a

statistic that is at worst meaningless and at best deceptive. Even leading behavioral geneticists now acknowledge that "heritability estimates are no longer important" (Johnson et al. 2009, p. 217).

A small army of scientists and philosophers of biology have identified a variety of misunderstandings that heritability statistics perpetuate. In an effort to protect unsuspecting readers from these common misinterpretations, I have pointed out in other publications (Moore 2002, 2006, 2008a, 2013) several things to keep in mind when one encounters these statistics. For instance:

- Heritability estimates tell us nothing about what causes an individual's traits (Johnson et al. 2009),
- Heritability estimates do not reflect the extent to which a trait is genetically determined and cannot be understood to reflect the *importance* of genes in the production of a person's traits,
- Heritability estimates are not measures of a trait's "openness" to environmental influence—they do not tell us how easily a trait can be affected by environmental factors (Lewontin 1974),
- Heritability estimates do not provide an accurate measurement of the likelihood that a trait will be "passed down" in a natural (i.e., not experimentally controlled) environment, so even 100 % heritable characteristics need not develop in the children of parents with that characteristic,
- Because some characteristics—for instance, the number of fingers present on normal human hands—are influenced by genetic factors that do *not* vary widely in human populations, these characteristics are not very heritable (Block 1995); no matter how counterintuitive it might seem, five fingers per human hand is not a heritable trait, given how behavioral geneticists define heritability,
- Heritability estimates reflect *environmental* variability, so the heritability of a trait in a population that develops in variable environments will be lower than the heritability of that same trait in a population that develops in less variable environments; thus, the heritability of a trait is not a characteristic of the trait at all, but is instead a characteristic of a studied population (Eisenberg 2004; Moore 2013).

As should be clear from this last point, heritability estimates cannot be generalized from the population that produced them to another population. Because this point has been misunderstood in the literature (Sesardic 2005), it warrants additional attention here. I have previously called attention to the fact that this caveat applies regardless of how similar two populations might appear; accordingly, I wrote "if alcoholism is [highly] heritable among Iowans, it need not be the case that it is [highly] heritable among Ohioans […] heritability estimates calculated for one population *do not apply* to another population" (Moore 2002, p. 47). Sesardic has argued that because I also believe genes and environments influence development symmetrically (i.e., they are always equally significant), it follows that "the non-generalizability of heritability implies the non-generalizability of environmental influences as well. Therefore, it would follow from Moore's pessimism about state-to-state inferences that if a new teaching strategy had good effects in schools in Ohio there would be no reason whatsoever to expect that the strategy would work in Iowa. This consequence is absurd…" (p. 80).

The absurdity here arises from Sesardic's misunderstanding of the central fact that *heritability estimates do not tell us anything about influences on trait development*; they tell us only how we can account for variation in a population. So, an environmental manipulation that *influences* the development of a scholastic competence in Ohio is likely also to influence the development of that scholastic competence in Iowa (just as a fictional genetic manipulation capable of influencing the development of a scholastic competence in Ohio is likely also to influence the development of that scholastic competence in Iowa). But because the *heritability* of a scholastic competence tells us nothing about what *influences the development* of that competence, it need not be the case that a study of the heritability of this competence in Ohioans would generate similar statistics as a study of the heritability of this competence in Iowans. If the factors that influence the development of a competence might not be equally variable for two different populations, the heritability of that competence in the two populations will differ, no matter how similar they (or their environments) might otherwise seem.

In spite of the fact that the heritability statistics generated by twin studies are unable to satisfactorily address questions about the relative importance of Nature and Nurture to the development of any of our traits, it remains the case that "twin studies […] provide the bulk of the evidence for the widespread influence of genetics in behavioral traits" (Plomin et al. 2008, p. 78). Of course, the fact that genes have important effects on behavior in general is now apparent; because behavior is a product of a brain, and because a brain is *built* using genes that contribute to the brain's structure, chemistry, and functioning, anyone thinking about the relationship between Nature and Nurture should understand that when it comes to behavior, genes are always influential. But this insight does not rely on twin study data; as Johnson et al. (2009) note, "Once we accept that basically everything—not only schizophrenia and intelligence, but also marital status and television watching—is heritable [READ: associated with genetic factors], it becomes clear that specific estimates of heritability are not very important" (p. 220). Twin studies confirm the importance of genetic influences on behavior, but the heritability statistics they generate mislead many readers by suggesting that some characteristics are *more* influenced by genes than by environmental factors, or that some characteristics are *more* influenced by genes than are other characteristics. But Nature and Nurture always play essential roles in the development of all of our traits, so neither of these suggestions is accurate. Given this insight, why is it that some of our traits (e.g., the languages we speak) are *obviously* influenced by environmental factors whereas others (e.g., the structures of our faces) are not?

5 Overlooking Nurture's Effects

There are several reasons traits might appear to be unable to be influenced by environmental factors even when they can be. First, some of the factors that influence characteristics are present in *prenatal* environments, so we have little opportunity to directly witness their effects, which can be significant nonetheless. For example,

there is evidence that a mother's diet can influence her infant's preferences for particular flavors (Mennella et al. 2001) or can influence the likelihood of her adult offspring being obese (Davenport and Cabrero 2009) or schizophrenic (Hoek et al. 1998). Likewise, the sounds that fetuses hear *in utero* can influence their behavioral characteristics once they are born (DeCasper and Spence 1986). Morphological characteristics that develop prenatally—a category that includes things like the bones in the face—also emerge as a result of interactions between genetic and non-genetic factors that occur *in utero* (e.g., see Hall 1988).

Second, some of the factors that might influence our characteristics are constant across human developmental environments, making it difficult to observe their influences. Because every human being grows up in an environment containing, for instance, oxygen and gravity, and *almost* every human being grows up in an environment containing, for instance, certain nutrients and communicative adults, it is impossible to casually observe the effects of such environmental factors. Nonetheless, such factors are likely to have important effects on the development of our traits, even if they cannot be invoked to explain *differences* among individuals. For example, although specific nutrients are known to influence human hair color (McKenzie et al. 2007), the effects of these environmental factors are not readily apparent to us because in many parts of the world the relevant nutrients are so plentiful that no one is malnourished in the specific ways that would reveal dietary influences on hair color. Likewise, the important role of gravity in the development of normal mammalian motor systems was undetectable until it was possible to study the effects on rats of developing as neonates in the microgravity environment present in the space shuttle's low-earth orbit (Walton et al. 2005; for further discussion of the importance of factors that *could* account for differences between individuals but that ordinarily do not because they ordinarily do not vary across individuals' developmental environments, see Griffiths and Tabery 2008).

Third, some of the factors that influence our characteristics are extremely subtle and might simply have escaped our notice. Studies of diverse species have now revealed a variety of effects of environmental stimuli on trait development, effects that bear a decidedly non-obvious relationship (Gottlieb 1991a) to the stimuli that produce them. For example, exposing chicks to their own toes influences their subsequent consumption of mealworms (Wallman 1979), exposing squirrel monkeys to either grasshoppers or crickets in their food influences their subsequent fear of snakes (Masataka 1993), and exposing mallard ducklings to their own embryonic vocalizations influences their subsequent preference for their mothers' assembly calls, even though the mothers' calls sound nothing at all like the embryos' vocalizations (Gottlieb 1991b). Considering how difficult it is to discover associations like these that seem entirely unpredictable, it is likely that non-obvious environmental contributors to development will ultimately be found to be a category that includes a large number of influential environmental factors that have yet to be recognized (for another good example, see King et al. 2005).

Finally, some of the factors that influence the development of our traits are not genes, but are nonetheless biological; steroid hormones are a good example. Biological chemicals like these do not fit behavioral geneticists' definition of

"Nature" (because they are not genes), but because they are produced within our bodies, they do not fit our intuitions about what should count as Nurture, either. Consider testosterone, a steroid hormone known to influence psychological characteristics as diverse as aggression and spatial cognition (see Archer 2006, or Mehta and Beer 2010, for references to the literature establishing the link between testosterone and aggression, and see Aleman et al. 2004, for evidence that experimentally administered testosterone affects visuospatial ability). Testosterone's effects on these characteristics means that *any* experience an individual has that influences testosterone levels could potentially influence their behavior. Importantly, this would be true regardless of whether or not the experience is one we would ordinarily associate with Nurture. So for example, when salivary testosterone levels are influenced by the experience of athletic competition (Edwards et al. 2006), we recognize this as an effect of Nurture (because some children experience more athletic competition than others). In contrast, when testosterone levels increase at the onset of puberty, similar effects on behavior can be expected even though experiencing the onset of puberty would ordinarily *not* be associated with Nurture. (It is for this reason that Gottlieb (1991a) suggested a broad and relational definition of experience that includes experiences other than those involving obvious learning). Should testosterone be considered an aspect of Nature or Nurture? The question makes little sense in light of what scientists now understand about how the molecules in our bodies are affected both by our genes and our experiences.

In summary, some of the environmental factors that influence development operate *in utero*, some are invariably present in human developmental environments, some do their work in extremely subtle ways, and we simply fail to recognize others as environmental factors at all (because even though they are *not* genetic and *can* be influenced by the external environment, they are located within a person's body). In each case, the influences of these factors are not easy to detect. As a result, casual observation sometimes suggests that we have some characteristics that are completely *un*influenced by Nurture. However, because genes only express their products in *contexts* and because their contexts influence what they do, the genome must be thought of as being reactive (Gilbert 2003), and non-genetic factors must be understood to always play a role in the development of our characteristics.

6 Genes in Contexts

It is in the discovery that genes do different things in different contexts that we can see most clearly how dichotomous thinking about Nature and Nurture must be erroneous. If a genome is associated with a characteristic in context A and that same genome is associated with a different characteristic in context B, it is clear that it makes no sense to think about either of the characteristics as being caused more by Nature than by Nurture or vice versa; the particular characteristic that develops depends critically on both the genes in question (Nature) and on the context in which those genes are being expressed (Nurture).

A good example of this type of environment-dependent phenotypic plasticity (West-Eberhard 2003) can be found in the development of the honeybee. Large numbers of honeybee larvae in a single colony can be genetically identical to one another, but a small number of these clones will develop into queens while the rest will become workers. Remarkably, workers are often half the size of queens, and unlike queens, they have sting barbs, short lifespans, and a behavioral repertoire required for food collection, among other major behavioral and morphological characteristics that distinguish them from queens (Carey 2011). The factor responsible for these differences is one even Galton recognized as Nurture: diet. While the larvae that become queens are maintained on a diet of royal jelly, their identical twin sisters that become workers are switched to a different "worker diet" after they turn 3 days old (Shuel and Dixon 1960). Therefore, what the genomes of these clones *do* depends on their nutritional context. But can we think of royal jelly as the factor that contains all of the information required for the construction of, for instance, mature ovaries, which are present only in queens? Of course not; critical information for the construction of ovaries is contained in the bees' genomes as well. The normal growth of ovaries in queens requires particular DNA *and* a particular developmental context, and this kind of collaborative construction of phenotypes during development is the rule among mammals as well.

Although theorists have thought of genes as providing information for trait construction at least since Francis Crick (1970) elucidated the "central dogma of molecular biology" in 1958, it is now clear that environments, too, provide information for trait construction (Lickliter and Berry 1990; Lickliter 2000). Thus, although the central dogma is still featured prominently in biology textbooks, its implication that DNA can be construed as single-handedly determining phenotypes is clearly wrong (Moore 2002). To the extent that textbooks represent genes as providing all of the information required for trait construction, they are masking what biologists currently understand about phenotypic development.

There are at least three different ways in which genes can be influenced by their contexts. First, genes can effectively be "turned on," "turned off," or rendered more or less active by chemical compounds that are normally involved in gene regulation. Because these compounds literally lie on top of genes, they are referred to as "epigenetic," and they have recently been the focus of an enormous amount of scientific attention (Bateson and Gluckman 2011; Carey 2011; Moore 2013; Uller, this volume). Although epigenetic phenomena have been observed since the early 1960s (e.g., Beutler et al. 1962), researchers have recently begun focusing on behavioral epigenetic phenomena, wherein specific *experiences* alter the activity of specific genes, thereby influencing subsequent behaviors. Among the most compelling findings in this domain have been those reported by Meaney (2010; Weaver et al. 2004). In this work, newborn rodents exposed to particular kinds of mothering grow up to be adults with particular ways of reacting to stressful situations. Meaney's lab has demonstrated that the parenting has its long-term effects by altering genetic activity in the offspring—not by changing the offspring's genes *per se*, but by epigenetically changing what those genes are *doing*. Although research on behavioral epigenetics in human populations is only now getting underway, several studies have already

reported effects in people that are consistent with those observed in rodents (Beach et al. 2010; Borghol et al. 2012; McGowan et al. 2009; Oberlander et al. 2008), so there is good reason to believe that the experiences we have as we develop have significant effects on the activity of our genes. The implications of these findings for discussions about Nature and Nurture are so profound that one epigenetics researcher (Weaver 2007) subtitled his article on the epigenetic "programming" of offspring by their mothers' behaviors "Nature versus Nurture: Let's call the whole thing off."

Second, it has become clear that there is a particular class of genes that begin to function in neurons when they are activated by specific kinds of environmental stimulation. These genes are known as "immediate early genes," and they have been found to be able to respond to changes in light cycles in hamsters (Rusak et al. 1990) and in cats (Rosen et al. 1992), and to species-specific birdsongs in zebra finches and canaries (Mello et al. 1992). Primates like human beings have immediate early genes as well, and at least one of them has been found to be associated with various forms of learning (Okuno and Miyashita 1996) and memory (Davis et al. 2003). Again, the discovery of genes that are responsive to environmental stimulation reinforces the fact that it is an error to imagine that our bodies and environments are not in constant communication as they collaborate in the construction of our phenotypes.

Third, molecular biologists (e.g., Pan et al. 2008; Wang et al. 2008) now estimate that as many as 95 % of our genes undergo a process known as "alternative splicing," which enables a given gene to perform different functions in different contexts. For example, Amara et al. (1982) discovered that the gene that contributes to the production of the hormone calcitonin in the thyroid gland also contributes to the production of an entirely different product—a neuropeptide—when it is "alternatively spliced" in a different context (the hypothalamus). The fact that the same exact gene is capable of doing two entirely different things in different cellular contexts controverts the idea that genes operate independently of their environments. But if genes are *typically* capable of doing *many* different things as a function of how they are influenced by different contexts, the belief that characteristics can be determined exclusively—or even primarily—by genes would become increasingly untenable.

As it happens, alternative splicing does appear to work like this, rendering dubious the textbook notion that particular stretches of DNA are best thought of as "coding for" very specific products or as "controlling" very specific processes. For the purpose of illustration, imagine that a particular segment of genetic material contains information in the order ABCD. Given what molecular biologists now understand about alternative splicing, this segment of DNA could be spliced to yield a variety of different products, including products associated with other orders, such as ACD, BCD, AD, AC, DCBA, BDCA, DA, etc. (Noble 2006). It is as if a sentence that reads "Madison drove Terry to see the dog" could, in different contexts, mean "Terry drove Madison to see the dog," "Madison drove the dog to see Terry," or even "The dog drove Terry mad." It is not yet known for certain if this extreme flexibility characterizes most genes, but molecular biologists acknowledge that alternative splicing is "a universal feature of human genes" (Trafton 2008, p. 6, quoting Burge),

so this kind of flexibility is certainly a possibility. Regardless, it has become clear that the idea that our genetic material contains a code that is capable of specifying particular predetermined phenotypic outcomes is false. In fact, genes typically behave as they do at least in part because of how they are effectively instructed to behave by the contexts in which they are operating. Simplistic notions of Nature and Nurture have no explanatory value in a system as complex as this one.

Given how extremely common alternative splicing is, it ought not be treated as a "special case" in biology curricula. Rather, by introducing students to multiple examples wherein different gene products—and consequently different processes and outcomes—are generated in different developmental contexts, such curricula could effectively emphasize that phenotype development is fundamentally a process involving the *co-action* of genetic and non-genetic factors. Such an approach would be an improvement over the still-popular approach that dogmatically emphasizes the one-way flow of information from DNA to phenotypes.

7 Rupturing Reaction Ranges

Because genetic activity is influenced by environmental factors, genes cannot determine the final forms of any of our characteristics independently of the contexts in which development is occurring. In the face of this conclusion, a common fallback position holds that genes can specify a *range* of possible phenotypes, and that the particular environment to which one is exposed dictates which phenotype within the range is the one that develops. In 1963, Gottesman put it this way: "A genotype determines an indefinite but circumscribed assortment of phenotypes, each of which corresponds to one […] possible environment" (p. 254). Thus, this position effectively holds that what we inherit from our parents is a particular "potential" that may or may not be realized, depending on the experiences we have as we develop. But as intuitively appealing as this so-called "reaction range" idea is, the observed facts of development suggest that thinking about things in this way is not helpful and can actually be quite misleading.

As Platt and Sanislow (1988) explain, "empirical support for the reaction-range concept is questionable" (p. 254); instead, there appear to be no knowable limitations that constrain any particular genotype. This sounds like a radical claim, because it seems obvious that human beings cannot develop from an elephant genome, no matter what sort of environment we allow it to develop in! And in fact, genetic factors do constrain developmental outcomes. But because it is impossible for us to know the limits of any individual's potential, the mere existence of such unknowable constraints cannot have any practical implications for us.

Addressing this issue empirically, Lewontin (2000) discussed studies in which populations of genetically identical plants (*Achillea millefolium*) were allowed to develop in three different environments, namely at either 30, 1,400, or 3,050 m above sea level. Similar studies of *Drosophila melanogaster* examined how animals that had had large portions of their genomes cloned would respond when allowed to

develop in a variety of different environments, namely at either 4, 21, or 26 °C. What is clear from all of these studies is that a single genotype, placed in a variety of different environments, can contribute to the development of a variety of different phenotypes. This finding on its own should not surprise anyone who has read this far into this chapter, but the *implication* of this finding is the surprising conclusion that genes cannot circumscribe phenotypes in any knowable way, rendering the range-of-reaction concept valueless. When faced with conclusive data in the mid-1950s that demonstrated that identical genomes react differently to different environments, Theodosius Dobzhansky—one of the key contributors to biology's modern synthesis of Darwinian evolution and Mendelian genetics—concluded that knowing what a particular genotype might be capable of would require empirically testing its development in *all* possible environments. Short of doing this impossibly comprehensive experiment, he wrote, "we can never be sure that any of these traits have reached the maximal development possible with a given genotype" (Dobzhansky 1955, p. 77). Thus, although the range of possible phenotypes associated with a genotype might be discoverable *in theory*, the fact remains that we can never know how a genotype might respond in some not-yet-tested environment; the limits of a genotype's reaction range cannot be known. And in case it was not obvious to readers why the range of all possible environments is infinite (and therefore untestable), Dobzhansky noted that "new environments are constantly produced. Invention of a new drug, a new diet, a new type of housing, a new educational system, a new political regime introduces new environments" (p. 75). As a result of this state of affairs, we can never confidently assert anything about genetic limits on an individual's developmental potential.

What is also clear from the kinds of studies presented by Lewontin (2000) is that different genotypes do not respond to different environments in similar ways. That is, it need not be the case that a genotype associated with the 'best' (or worst) phenotype in one environment is the same genotype associated with the 'best' (or worst) phenotype in a different environment. Instead, different genotypes have different environments that are optimal for them. Writing 16 years earlier, Lewontin addressed this issue directly using cloned corn plants as an example:

> … one genotype may grow better than a second at a low temperature, but more poorly at a high temperature […] modern corn hybrids are superior to those of fifty years ago when tested at high planting densities in somewhat poorer environments, while the older hybrids are superior at low planting densities and in enriched conditions. Plant breeding has then not selected for 'better' hybrids […] Thus genotype and environment interact in a way that makes the organism unpredictable from a knowledge of some average of effects of genotype or environment taken separately (Lewontin et al. 1984, pp. 268–269).

Because genotypes interact with their environments like this, we can never know *prior to performing the manipulation* how changing a person's environment might affect their development; manipulations that might have a desirable effect on one child cannot be guaranteed to have a desirable effect on a different child (or vice versa). Because a genotype associated with a "good" phenotype in one context could be associated with a "bad" phenotype in a different context, it is not possible to identify a particular genotype as generally "superior" or "inferior" to any other

genotype. Given this reality, saying anything absolutely true about the "Nature" of anyone's genes is, for all intents and purposes, impossible. What a gene does depends on the environment in which it is operating. As West-Eberhard (2003) summed up the last several decades of thinking in this domain, "evolving organisms are universally responsive to the environment as well as to genes" (p. 3), so the discovery of this kind of developmental plasticity—wherein organisms develop in different ways in different contexts—should not surprise any of us, and educators should begin trying to stress for their students that genes are merely non-deterministic *contributors* to people's physical and psychological characteristics.

8 Influencing Traits

At the end of her 2010 book on Nature and Nurture, Evelyn Fox Keller argued that what "people want to know about" when they ask Nature/Nurture questions is really whether or not a given characteristic can be influenced by the circumstances in which a person develops. Although the answer to this question is now understood to be "yes" in all cases, this is not the final word on the issue. Many people assume that some traits can be *more* influenced by Nurture than can other traits, and further, that some traits can be more *easily* influenced by Nurture than can other traits. These claims seem intuitively reasonable given our experiences with living things, but they are not strictly true.

In many cases when it seems like we cannot influence the development of a trait (or cannot influence its development very much, or very easily), it is only because we do not understand *how* the trait develops. Because we understand that infants growing up around French-speaking adults will learn to speak French, we can manipulate the language a child learns by moving to France. In contrast, in the 1950s, before scientists understood the nature of the metabolic disorder called phenylketonuria (PKU), it appeared as if the development of the mental retardation typical of untreated children with PKU could not be similarly manipulated. These days, it is common to hear the claim that "a single gene is necessary and sufficient to cause [PKU]" (Plomin et al. 2008, p. 32), but although PKU can be understood in this way, our understanding of what this gene *does* permitted the discovery of a dietary manipulation that allows treated individuals to experience normal mental development even if they have the genetic abnormality associated with PKU. Prior to the implementation of this Nurture-based manipulation, the heritability of PKU was high—because human diets are virtually invariable in the extent to which they contain the amino acid associated with PKU, so the presence of PKU was associated with genetic variation only—but now that researchers understand something about the Nature-Nurture interactions that give rise to mental retardation in these cases, influencing outcomes for PKU patients is not particularly difficult. The same will be true of other conditions as we learn more about their development. Traits are likely to appear unchangeable when we do not yet understand how to change them.

It is no accident that the tools of quantitative behavioral geneticists (e.g., twin studies, adoption studies, heritability estimates) have left us with a confused understanding of this fact. In a textbook intended to be the definitive introduction to behavioral genetics, Plomin et al. (2008) note that "quantitative genetics, such as twin and adoption studies, depends on Mendel's laws of heredity but does not require knowledge of the biological basis of heredity" (p. 40). However, it is precisely an understanding of how genes mechanistically do what they do—in interaction with their contexts—that is required to comprehend how it is that highly heritable traits can nonetheless be easily and profoundly influenced by environmental factors.

Of course, just because all characteristics can theoretically be influenced by the contexts in which development occurs does not mean that a knowledgeable scientist could completely control the development of someone's phenotype, because some environmental manipulations are practically impossible to implement. If Keller is right that people who ask Nature-Nurture questions really want to know how easily a characteristic can be influenced by an environmental manipulation, it will not matter to them that the correct answer is "very easily, if you know how the characteristic develops"; such a person really wants to know how easy it might be to implement the manipulation. And because implementation is not always equally easy, all characteristics are not *in practice* equally easy to influence; after all, changing someone's diet, for instance, is currently easier than changing the gravitational field in which they develop!

Similarly, although it is true that all characteristics develop from gene-environment interactions, it matters very much *when* in development various things happen. So even if scientists were able to discover a hypothetical environmental manipulation that, when implemented in infancy, increases the IQ scores that treated babies achieve once they are adults, it could still be the case that after a certain point in development, that manipulation might have no effect on IQ at all. That is, just because it is true that Nurture has a role to play in the development of all of our characteristics does not mean that anything is possible at any given moment. To use another hypothetical example, even if psychologists fully understood the developmental origins of violent behavior, an adolescent's violent behavior could be intractable either because it is too late in her development to significantly influence those behaviors or because the environmental manipulation required to alter her behaviors is technically difficult to implement. As Keller put it, "perhaps we should rephrase the nature-nurture question, and ask, instead, how malleable is a given trait, at a specified developmental age?" (2010, p. 75).

To the extent that what matters to us are these kinds of questions, there is plenty of research still to be done, because scientists currently understand very little about how malleable particular traits are (although this is changing, as suggested by the publication of Bateson and Gluckman's 2011 book on developmental plasticity and robustness). But note that this understanding of Nature-Nurture interactions changes our focus from questions about whether or not particular traits are "innate"—or about how powerfully genetic versus environmental factors influence those traits—to questions about *how* and *when* traits develop. Such a change of focus is bound to

help us as we grapple with individuals' and society's problems; in contrast to the correlational approach long used by quantitative behavioral geneticists, a developmental perspective encourages experimentation, and as such, it has the potential to reveal interventions that can actually be used in productive ways to influence developmental outcomes.

9 Conclusion

In the nineteenth and twentieth centuries, some scientists' ideas about Nature and Nurture were used to argue that certain people were inherently inferior to others; the belief that certain characteristics are determined by biology alone led in Germany to the systematic extermination of millions of people (Proctor 1988) and in the United States to large scale programs to sterilize individuals deemed socially "undesirable" (Kevles 1995). Ironically, the more we have learned about genetics in the past 50 years, the more we have come to understand that our characteristics are jointly determined by biological and environmental factors, that is, that all of our characteristics result from a unitary developmental process that relies on both "Nature" and "Nurture" for its functioning. Indeed, Bateson's characterization of the Nature-versus-Nurture debate as a "corpse" is appropriate, because it is clear now that Nature and Nurture are not oppositional influences on development; instead, they work collaboratively.

Although many theorists who read the academic literatures relevant to Nature and Nurture have understood for years that genes interact with their contexts to produce phenotypes, many high school students maintain misunderstandings about genes, for instance that genes operate deterministically (Shaw et al. 2008). It is likely that these misconceptions result from, or are perpetuated by, the content presented in introductory and advanced high school biology textbooks (Castéra et al. 2008; dos Santos et al. 2012; Gericke and Hagberg 2010). The idea that genes operate deterministically seems to be deeply ingrained in us, perhaps because Weismann proclaimed that "the germ-substance" (1894, p. 20) operates deterministically even before the world knew of Gregor Mendel, and 15 years before Johannsen (1911) had even coined the word "gene;" given this long history, it might not be surprising that some educators continue to teach genetics using Punnett squares and other tools that can be mistaken to support genetic determinism (see Jamieson and Radick, this volume). But because our conceptions about genes have such important consequences for all of us, it is important to find ways to teach genetics that convey how genes and environments operate collaboratively in the construction of phenotypes. An excellent way to ensure that this message is passed on to students would be to adopt a pedagogical approach that encourages study of the *emergence* of phenotypes in developmental time. To the extent that textbook writers and educators adopt such a developmental perspective, subsequent generations of students are likely to graduate from school understanding that DNA is merely one factor that contributes to the characteristics we observe in the living things around us.

As I was writing this chapter, the *New York Times* published an Opinion piece entitled "Sorry strivers: Talent matters" (Hambrick and Meinz 2011b), implying that people have some preordained level of competence—talent—that constrains what they can expect to achieve, whether in the arts, sciences, business, or sports, for example. As I indicated previously, it is certainly possible that some of us are in a developmental moment in which practice or striving might not have much influence on what we can achieve. In addition, there can be no doubt that scientists' understanding of how to improve people's performances in many domains is limited, so even if there are ways to improve people's skills, we might still be ignorant of those ways. But regardless of what is or is not possible for a given person to achieve from this moment forward, the idea that we are conceived with some quantity of competence that is predetermined by "Nature" is certainly false. "Talent," like all of our other characteristics, develops; it is not present in a fertilized egg any more than completely formed teeth are present in that same zygote. Thus, it is of as little value to talk about the extent to which "talent" contributes to a competence as it is to talk about the extent to which "Nature" contributes to a competence; what matters is how the competence *develops*. And it is only by studying the development of biological traits, psychological traits, and abilities—think eye color, IQ, or eye-hand coordination—that we can learn how to influence their emergence in individuals (in theory, either through genetic or environmental manipulations).

Hambrick and Meinz conclude their essay by noting pessimistically that "it would be nice if intellectual ability […] were important for success only up to a point […] But wishing doesn't make it so […] Sometimes the story that science tells us isn't the story we want to hear." Intellectual ability *is* important, of course, but we ought not make the mistake of earlier generations and conclude that this ability is somehow unaffected by the experiences we have as we develop. Rather than studying the extent to which competence is influenced by factors we cannot yet control—for example, "working memory capacity" (Hambrick and Meinz 2011a)—we would be much better served by studying the *development* of such factors, so that we can learn how to helpfully influence their emergence. A focus on developmental processes—how they normally work and how we can influence them—rather than on questions about Nature and Nurture, will yield such insights in the future. In this case, the story science tells us is one we very well might want to hear.

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