1-1-2008

Espousing Interactions and Fielding Reactions: Addressing Laypeople's Beliefs About Genetic Determinism

David S. Moore
Pitzer College

Running head: Interactions and Reactions

The content of this open-access pre-print article is the same as that contained in the published article with the following reference:

Moore, D. S. (2008). Espousing interactions and fielding reactions: Addressing laypeople's beliefs about genetic determinism. *Philosophical Psychology, 21,* 331 – 348.

ESPOUSING INTERACTIONS AND FIELDING REACTIONS: ADDRESSING LAYPEOPLE'S BELIEFS ABOUT GENETIC DETERMINISM

David S. Moore[1]

[1] David S. Moore is a Professor of Psychology at Pitzer College and Claremont Graduate University.
Correspondence to: David Moore, 1050 N. Mills Avenue, Claremont, CA 91711 USA. Email: dmoore@pitzer.edu

Abstract

Although biologists and philosophers of science generally agree that genes cannot determine the forms of biological and psychological traits, students, journalists, politicians, and other members of the general public nonetheless continue to embrace genetic determinism. This paper identifies some of the concerns typically raised by individuals when they first encounter the systems perspective that biologists and philosophers of science now favor over genetic determinism, and uses arguments informed by that perspective to address those concerns. No definitive statements can yet be made about why genetic determinism has proven so resilient in the face of empirical evidence pointing up its deficiencies, but conveying the essential interdependence of "nature" and "nurture" to the general public will likely require deployment of the arguments that systems theorists ordinarily use to reject genetic determinism. In addition, the elaboration of new metaphors that focus attention on the dynamic nature of trait construction will likely prove valuable, because re-conceptualizing notions like "genes" and "nature" will probably be one of the most effective ways to help students and the general public abandon the genetic determinism that biologists now recognize as indefensible.

Espousing interactions and fielding reactions:
Addressing laypeople's beliefs about genetic determinism

Genetic determinism, in all of its various forms, has proven remarkably resilient in the face of both theoretical and empirical work showing it to be a poor way of explaining how biological and psychological characteristics emerge during development. It appears that merely comprehending what genes actually do does not necessarily lead to a rejection of genetic determinism, because in spite of evidence to the contrary, even some biologists continue to write as if developmental processes can be genetically determined (e.g., Gehring, 1998). There are likely several factors that contribute to most people's strongly held convictions that genes can deterministically produce at least *some* of our characteristics; this paper will offer a non-comprehensive inventory of the concerns often raised by individuals when they first encounter the systems perspective championed by many philosophers of science (Griffiths & Gray, 2001; Moss, 2003; Robert, 2006; Stotz, 2006) and now ascendant in biology (Gilbert, 2001; Jablonka & Lamb, 2005; Lewontin, 2000; Nijhout, 2003).

Systems theorists advocate a probabilistic view of trait development that holds that all of our biological and psychological characteristics are co-constructed during development by genetic *and* environmental factors operating in collaboration with one another; this approach rejects the possibility that either type of factor is more agentive or provides developmental information that is more causally important than the other. In this paper, after identifying particular questions often asked by those encountering the systems perspective for the first time, arguments informed by the systems viewpoint will be deployed to address these concerns. Those psychologists, laypeople, and biologists who

embrace genetic determinism probably each have different reasons for doing so, but similar sorts of explanations are likely to be required when trying to disabuse these various populations of their deterministic ideas; nonetheless, the discussion below is intended to be particularly useful for psychologists.

The idea that genetic factors might be able to determine the form of biological and psychological traits has been with us since the beginning of modern theorizing about genes. Although Gregor Mendel did not use the word *genes* to name the "heritable factors" that he inferred must be responsible for observed variations in his experimental pea plants, the notion of a deterministic "germ plasm" had appeared in several late 19th century writings on biology—most notably in the work of August Weismann—and because of the close conceptual similarity between Mendel's "heritable factors" and Weismann's deterministic "germ plasm," it is little wonder that just a few decades later, Mendel's factors came to be thought of as deterministic "genes." T. H. Morgan's early 20th century discovery that genes are located on chromosomes eventually led to the development of the modern gene theory, which holds that genes are responsible for the development of inherited traits; this conclusion was based on the finding that the presence of particular genetic factors is highly correlated with the presence of particular traits. But even though such correlations do *not* support the contention that genes operate deterministically, modern gene theory nonetheless retained the genetic determinism that 19th century "germ plasm" theorists relied on to explain the intergenerational transmission of evolutionarily adaptive characteristics. This sort of conceptualization continued to inform theoretical biology well past the middle of the 20th century, as biologists embraced François Jacob and Jacques Monod's operon model of how genes regulate development (Keller, 2000).

Deterministic theories in psychology, likewise, have a long history. Psychology was informed by theories of biology at its inception (Sulloway, 1992), but the popularity of the belief that evolutionary mechanisms—operating proximally through deterministic genes—can produce behaviors grew significantly with the publication of the works of Konrad Lorenz (e.g., Lorenz, 1965) and E. O. Wilson (e.g., Wilson, 1978) shortly after the middle of the last century. As there were in biology (Driesch, 1908, cited in Gottlieb, 1992; Sturtevant, 1915, cited in Carlson, 1966), there were behavioral scientists who understood from very early on that genetic-deterministic theories would ultimately fail to explain the origins of our psychological traits (Kuo, 1976; Lehrman, 1953; Schneirla, 1966); nonetheless, psychological theories rooted in the deterministic ideas of 19th century biology continue to be fashionable in branches of contemporary psychology such as evolutionary psychology and quantitative behavior genetics. The recent popularity of nativism in theories of language acquisition (Chomsky, 1975; Pinker, 1994, 2002), social cognition (Baron-Cohen, 1995; Cosmides, 1989), cognitive development (Spelke & Kinzler, 2007), and other domains illustrates how widely accepted deterministic accounts of psychological phenomena remain.

In spite of the ongoing popularity of these ideas in psychology, recent decades have seen the publication of overwhelming empirical support for the idea that genetic factors cannot deterministically cause either biological or psychological/behavioral characteristics (Stotz, 2006); this view has now become relatively mainstream among philosophers of biology. Contributing to the widespread acceptance of this idea have been data collected in several disparate fields, including developmental psychobiology, the neurosciences, embryology, and molecular biology. Neuroscientists, for example, have discovered that

brain structure is more dependent on experience than was previously thought (Cohen et al., 1997; Greenough, Black, & Wallace, 1987; Pantev et al., 1998). Molecular biologists have determined that a gene can be spliced in several alternative ways as a function of the (non-genetic) cellular *contexts* in which the product of that gene will be constructed (Smith, Patton, & Nadal-Ginard, 1989). Embryologists have found that basic information about which pole of a developing organism will develop into the head and which will develop into the tail can be extracted from chemical gradients present in the cytoplasm—not in the genes—of the developing zygote (Gilbert, 2006; Wolpert, 1992). And developmental psychobiologists have learned that experiences early in development can have epigenetic—and inheritable—effects wherein particular genes in a newborn animal can be turned "on" or "off" as a function of the behavior of the animal's mother (Harper, 2005; Meaney, 2007; Meaney & Szyf, 2005; Weaver et al., 2007). Consequently, it is extremely rare at this point to find a scientist willing to publicly endorse genetic determinism, because it is no longer tenable to argue that some genes can single-handedly cause the development of specific traits independently of the contexts in which development takes place.

Nevertheless, the now-discredited belief that genes can determine the form of some of our characteristics remains widely held by journalists, politicians, students, and the general public—and surprisingly, by many social scientists. This idea probably owes its tenacity to different things in different people. For some, its persistence probably reflects no more than straightforward ignorance of the significant facts. For others, genetic determinism might retain some currency because of the apparent elegance of its simplicity; the emerging understanding of how genes interact with non-genetic factors during development is extremely complex, and so perhaps less appealing to these individuals. Yet

other people might find it difficult to overcome a lesson learned at a young age, particularly when a deep appreciation of how traits really do develop is best obtained with exposure to empirical data from many diverse fields, including embryology, developmental psychobiology, the neurosciences, and developmental psychology (to name just a few). In the absence of relevant data, we cannot know how important each of these reasons is for different people, but anecdotally, it is clear that scientists trying to explain these ideas to their students often find them quite unwilling to believe that our biological and psychological characteristics *always* develop from the interactions of genetic and non-genetic factors.

To understand why genetic determinism is so tenacious, it will be helpful to consider some of the questions that naïve—but nonetheless resistant—audiences typically ask when first exposed to the essential facts of gene-environment interdependence. The following sections will each consider a question that such audiences commonly raise when encountering a systems viewpoint for the first time. While the questions themselves will serve to illuminate some of the major obstacles that hinder understanding of the fundamental interdependence of genetic and non-genetic factors during development, a presentation of the *arguments* that systems theorists ordinarily use to address these questions will aid those attempting to help students relinquish strongly held ideas about genetic determinism.

Several Questions Naive Audiences Typically Raise When Confronted With Gene-Environment Interactionism

If I look like my parents regardless of the environment in which I grew up, my appearance must have been determined by my genes, right?

One reason often given by those convinced that some traits must be genetically determined is the physical similarity of offspring to their parents. This objection to gene-environment interactionism typically involves references to the fact that children often resemble their biological parents even if they are raised by adoptive parents. As this argument is usually advanced, the physical characteristics of an adopted child who resembles her biological parents must be genetically determined, because the biological parents are thought to have contributed nothing to the child other than her genes. However, biologists now understand that parents provide several types of non-genetic "information" to their offspring prior to birth (and certainly prior to adoption), some of which play important roles in the development of the offspring's characteristics (Harper, 2005; Jablonka & Lamb, 2005). Examples of these types of "information" include the patterns-of-distribution of chemical factors in the ovum's cytoplasm, methylation patterns that affect the functioning of the DNA in the ovum and the sperm, and the specific chemical environment of the uterus (in which the characteristics of offspring first begin to develop), among others.

A common objection to this observation is that these types of "information" do not, in most cases, account for the physical *differences* that give individual offspring their unique appearances; normal development usually entails very little cross-individual variability in either zygotes' internal structures or in the characteristics of their prenatal environments. Some evidence suggests that differences in such factors can account for behavioral differences between individuals (Meaney & Szyf, 2005; Weaver et al., 2007), but these data, while exceedingly important, ought not distract from the following general point: developmental factors that never vary—for example, the presence of oxygen or

gravity in human developmental environments—while often ignored when people consider the origins of traits, are ignored in error.

For example, human developmental environments are always characterized by linguistic input that influences normal language development. The fact that such input is invariably present does not reduce the importance of this input in the development of linguistic competence. Of course, it remains the case that developmental factors that affect all individuals cannot account for the *differences* among those individuals; but just because a particular factor might not explain why you and I have different physical appearances does not mean that that factor did not contribute importantly to the development of your (or my) physical appearance.

The importance of this point is so often lost on those who have never before considered these issues that some additional comment on it is warranted. At times, the argument is made that if a particular causal factor is always constant in a particular context, it can be safely ignored when assigning causation; although the presence of oxygen in a building is a necessary background condition for the building to burn, it is far more common to assign causal power in such a scenario to the arsonist striking a match than to the presence of the oxygen in the vicinity of the flame. This is because the presence of oxygen is usually *assumed*, because it is so invariable. This line of argument is not unreasonable; perhaps when a researcher refers to "the gene for trait X," she means to say—and is typically understood to be saying—that the gene in question causes trait X *assuming a particular (normal) developmental environment*. But such a position effectively encourages us *not* to study the developmental mechanisms by which genes interact with other factors to produce the trait in question. And in so doing, we are more likely to miss

possible routes by which the development of the trait could be influenced, possibly with beneficial consequences.

For example, although most members of the medical community consider phenylketonuria (PKU) to be a genetic disorder (Cole, Cole, & Lightfoot, 2004)—because those with PKU have genetic constitutions that differ slightly from the rest of us—the fact remains that the disorder can be effectively treated with an *environmental* manipulation. If newborns at risk for PKU are fed a diet free of phenylalanine (an amino acid normally present in all human diets) the symptoms of PKU can be virtually eliminated. In this case, an understanding of the developmental mechanics that produce the symptoms of PKU gave rise to a subsequently confirmed hypothesis about how to effectively treat the disorder by manipulating an environmental factor that is relatively constant in normal human developmental environments. Such are the benefits of remaining aware of the effects of such "invariable" developmental factors on the emergence of our characteristics. If the goals of the behavioral and social sciences are pragmatic to the slightest degree, the causal roles of invariable "background" factors in the development of our traits simply must not be ignored.

Of course, it should go without saying that an awareness of non-genetic contributions to development ought not blind us to the important roles *genetic* factors play in development; however, an explicit statement of this fact is necessary, because those offering explanations of development informed by systems theories are sometimes erroneously seen as denying the importance of genetic factors in development, simply because they insist on the importance of non-genetic factors as *contributors* to this process. If we want to know why two unrelated children raised by the same adoptive parents will

typically resemble their own biological parents, we need merely point out that genetic factors obviously *do* contribute in essential ways to the development of our characteristics. But developmental outcomes are not *pre-determined* by such contributions; non-genetic factors contribute to these outcomes in ways that are just as essential, even if it is difficult for us to see their effects when such factors are unvarying. Although the proverbial fish that has spent her entire life in the sea does not know that she lives in a watery environment (because she has never experienced anything else), it nonetheless remains the case that fish are as they are at least in part because of the peculiar qualities of water.

If my brother is outgoing and I'm shy even though our parents treated us the same way, our personalities must have been determined by our genes, right?

Genetic-deterministic explanations for our characteristics seem to be readily accepted in some cases because of erroneous folk-beliefs regarding the environmental factors that contribute to those characteristics. If a "folk theory" of personality holds that easily observable parental behaviors are the important non-genetic contributors to personality development, the subsequent discovery that there are no differences in such behaviors across parents might lead one to conclude that the important contributors to personality development are genetic. Of course, this is a fundamental error: in fact, *other* non-genetic contributors to personality development might be the important ones, and we cannot know in advance of developmental analysis if they are or are not. Time and again, developmental analyses have revealed that among the important influences on the development of particular characteristics are some that are, in the nomenclature of Gilbert Gottlieb (1991a), nonobvious; such contributing influences are factors that would not have been thought important prior to developmental analysis. The discovery that some of the

non-genetic contributors to development are nonobvious means that until we understand exactly what factors are involved in the development of a particular characteristic—and to date, there are virtually *no* psychological characteristics that develop in ways that are so well understood—we cannot effectively evaluate the extent to which two developmental environments are similar. Although it might *seem* as if the environment in which I developed is identical to the environment in which my brother developed, we cannot know if our environments were identical *with regards to the factors that really matter*, if we do not know in advance which factors actually are the ones that matter. The bottom line is that no two bodies can be raised in identical environments—even monozygotic twin fetuses in a single uterus are in slightly different positions relative to one another, and one of them will be born prior to the other—so the intuitions resulting from a casual observation that two individuals were reared in similar environments ought always be considered suspect.[2]

Haven't controlled studies of twins-reared-apart proven that genes can cause traits?

In the process of simplifying scientific reports so that they will be comprehensible to the general public, the popular press has contributed in significant ways to the widespread notion that genes can deterministically cause some of our traits. But the primary source of data that has contributed to this discredited notion has been the field of quantitative behavior genetics, which has drawn its most compelling conclusions from studies of fraternal and so-called "identical" twins who have been adopted into "different"

---

[2] Strong evidence that no two bodies ever develop identically—even if they are reared in identical environments—has been generated in studies of a relatively simple type of worm, the nematode *Caenorhabditis elegans*. Such studies have determined that genetically identical worms reared in identical environments can nonetheless develop different behavioral characteristics. As Gilbert & Jorgensen note, "organisms with the same inheritance…and the same environment…still [wind up with] behavioral differences as a result of chance events during development" (1998, p. 263). If differences like these can characterize such relatively simple animals reared under such controlled conditions, we ought to be very suspicious indeed of our intuitions that differences between human individuals reared in "the same environment" must have been caused by differences in their genes.

environments. These sorts of studies have been the subject of several extensive critiques (Joseph, 2001, 2006; Lewontin, Rose, & Kamin, 1984), many of which have been decidedly negative. These cogent critiques need not be recited here, given their accessibility elsewhere. Rather, for the purposes of the present paper, it is enough to note that the design of quantitative behavior genetics studies, by its very nature, is unable to demonstrate genetic determinism, even in those cases in which the general public is most convinced of its existence (e.g., in the case of eye color). This inability reflects the fact that such studies are always correlational, so the data they generate can at best reveal that variation in a particular factor (say, the presence or absence of a particular gene) is associated with variation in another factor (say, the presence or absence of a particular phenotypic characteristic). Of course, as all first year graduate students in the social sciences are aware, co-variation need never indicate that two factors are causally related; consequently, it is, frankly, far-fetched to think that these studies could even *lend support* to the hypothesis that particular genes determine the development of particular traits. In fact, quantitative behavior geneticists have conceded that their studies cannot be used to support claims of genetic determinism; as one leading evolutionary psychologist put it, no "sane biologist would ever dream of proposing" that human behavior is genetically determined (Pinker, 2002, p. 112). Nonetheless, when the results of studies of quantitative behavior genetics are passed on to journalists, textbook writers, students, and the general public, they are often misinterpreted in ways that perpetuate the idea that some of our characteristics can be genetically determined.[3]

3 To give just one of many possible examples, The New York Times reported in a 2005 article that "some researchers believe there is likely to be a genetic component of homosexuality because of its concordance among twins." Although studies of twins are not able to produce valid conclusions about the extent to which genetic factors contribute to the development of particular characteristics (Moore, 2001, 2006), such studies

Part of the trouble with the data collected by quantitative behavior geneticists is that they are typically used to compute heritability statistics, extraordinarily misunderstood statistics that, despite their misleading name, do not reflect how *inheritable* various characteristics are (Block, 1995; Moore, 2001). Heritability statistics can only account for *variation* in characteristics across populations; they cannot explain the development of characteristics in individuals, and they do not measure the likelihood that a parental characteristic will also be present (i.e., develop) in descendant generations. Under certain controlled circumstances (e.g., animal husbandry projects), heritability statistics have proven useful, in large part because when developmental environments are strictly controlled—as they are whenever heritability statistics have any practical value—variations in developmental outcomes can be accounted for only by those factors that remain free to vary (i.e., genetic factors alone).[4] However, heritability statistics have never been shown to have any predictive utility at all when applied to human beings and their offspring; this should not be surprising given what we now understand about how environmental factors influence developmental outcomes.

One reason heritability statistics computed for human populations have proven useless is that they cannot be appropriately generalized beyond the study population that generated the statistics in the first place. Generalizing the results of a heritability study

---

were presented to the public in this article in the context of a sentence that began "If sexual orientation has a genetic cause…," as if it might actually be possible for genetic factors to determine sexual orientation.

[4] Of course, the converse is true as well: when genetic variation in a population is virtually absent, variations in developmental outcomes can be accounted for only by environmental factors. This feature of heritability has given rise to some heritability estimates that strike most people as remarkably counterintuitive. For example, although no one would question the importance of the role that genetic factors play in the development of 5-fingered hands in human beings, the heritability of this characteristic is nonetheless extremely low (Block, 1995). This result reflects that fact that variations in finger numbers across human populations are better accounted for by variations in the frequencies of industrial accidents than by variations in the frequencies of particular genes (because there is very little variation across human populations in the frequencies of genes that contribute to the numbers of fingers on our hands).

from a population of research participants to a much broader population requires confidence that the variation in developmental environments experienced by the broader population is no different than the variation in developmental environments experienced by the research participants. But, because we typically do not know which factors play important roles in the development of a particular characteristic (as discussed above), it is not possible to know if the variation in those factors is the same across the broader population as it was in the original population of research participants; the variation in developmental environments experienced by the research participants could be greater than, equal to, or less than the variation experienced by the broader population (Moore, 2006). Consequently, heritability statistics do not provide us with a useful measure of the extent to which a characteristic is influenced by genetic factors independently of environmental factors, our intuitions about it notwithstanding.

I learned in school that eye color is genetically determined; how can that satisfying and predictively useful explanation be wrong?

One of the most widely disseminated insights in biology has been Gregor Mendel's interpretation of his pea-plant studies, brought to bear as an explanation of human eye color. Virtually any student with even a minimum of exposure to biology learned at some point in school that human beings have two alleles for eye color, the dominant brown allele typically being represented by a "big B" and the recessive blue allele typically being represented by a "little b;" this lesson has no doubt helped countless schoolchildren understand how a blue-eyed child might be born to two brown-eyed parents. This example has the obvious virtue of explaining people's readily observed experiential data (i.e., their anecdotes) in a way that feels intuitively reasonable to them.

Unfortunately, although the Mendelian explanation of the intergenerational transmission of eye color is readily comprehensible and appears to have predictive utility, it remains the case that it is a misleading simplification of the actual developmental processes that give rise to eye colors. As early as 1915, Sturtevant wrote:

> Although there is little that we can say as to the nature of Mendelian genes, we do know that they are not 'determinants'…The difference between normal red eyes and colorless (white) ones in Drosophila [fruit flies] is due to a difference in a single gene. Yet red is a very complex color, requiring the interaction of at least five (and probably of very many more) different genes for its production…we can then, in no sense identify a given gene with the red color of the eye, even though there is a single gene differentiating it from the colorless eye…all that we mean when we speak of a gene for pink eyes is, a gene which differentiates a pink eyed fly from a normal one—not a gene which produces pink eyes per se…" (Carlson, 1966, p. 69).

Thus, the explanation proffered for eye color in schools around the world has never been one that biologists have meant to be understood as genetic-deterministic. Rather, the Mendelian explanation continues to be taught in schools because it has predictive utility, owing to the fact that many of the genetic and non-genetic factors that contribute to eye color during development are relatively constant from individual to individual.[5] As noted

[5] It is worth noting here that it is not merely a happy coincidence that the non-genetic factors required for the development of some of our characteristics are so common in our developmental environments. Developmental environments are *typically* reproduced in each generation, ensuring that the presence of these non-genetic factors is relatively stable across generations. Griffiths and Gray (1994) have identified several types of "developmental resources" that are reliably reproduced in the developmental environments of successive generations, including (among others) parental, population-generated, and persistent resources. For example, the homes that parents build prior to the birth of their offspring impose important structure on the

above, a factor that does not vary during normal development might *appear* to make no contribution to development, simply because the factor does not contribute to observable *differences* in developmental outcomes. But just because a factor does not contribute to differences in an outcome does not mean that it does not contribute to the development of the outcome *per se*.

But don't some traits develop without any experiential input at all? And aren't some traits impervious to any such influences?

The idea that genes can deterministically cause some of our traits is attractive in part because we have all noticed that some of our characteristics appear to develop without any experiential input. We often consider characteristics to have been "nurtured" if they have been *learned*, that is, if there are obvious experiences that an individual is required to have had in order to develop the characteristic. In contrast, some of our characteristics—for example, our basic body plans (i.e., two arms and two legs, etc.) or our secondary sexual characteristics (e.g., my facial hair)—do not appear to require any particular sort of experience for their development. Likewise, some traits seem impervious to environmental influence because they develop in all normal members of a species, seemingly independently of the environments in which those individuals are reared. In both cases,

---

offspring's developmental environments; while enhancing an offspring's survival prospects, such parental resources also ensure that offspring are exposed to a limited portion of the stimulation the world has to offer, a portion that is, in the words of Lickliter and Berry (1990) "structured, organized, and specific to the organism." Likewise, cultural artifacts (e.g., spoken language in human beings) are present in the developmental environments of successive generations of organisms, and resources such as these, generated not by particular parents but by a *population* of ancestors, can structure the environment in ways that contribute importantly to the development of adaptive characteristics in individuals. Finally, environmental factors that are not actively produced by organisms at all but that are—by virtue of where a particular species reproduces—persistently present in those organisms' developmental environments can also provide successive generations with stable non-genetic information that contributes to the development of species-typical characteristics. For example, the National Academy of Sciences (1999) reports that "the larvae of the fly *Drosophila carcinophila* can develop only in specialized grooves beneath the flaps of the third pair of oral appendages of a land crab that is found only on certain Caribbean islands;" clearly, extremely specific developmental environments can, in effect, be passed from generation to generation in a way that ensures the stable transmission-across-generations of non-genetic developmental information.

however, what appears to be the case at first glance turns out not to be the case once we have studied the developmental mechanics that actually give rise to the traits in question.

To choose just two of many possible examples, Müller (2003) has written about the importance of experiencing particular mechanical forces during embryonic development in order to develop normal skeletal structures, and the work of Kaas (1991) and Merzenich (1998) with non-human primates and human beings has highlighted the important roles that normal sensory and motor experiences play in the development of the sensory and motor maps that characterize all normal human brains. In such cases, the experiences *required* for the development of these characteristics are nonobvious, perhaps because all normally developing individuals encounter the requisite experiential stimuli. If we define "experience" very narrowly as "learning," it is true that some of our characteristics do not require experience for their development. But such a definition is unnecessarily narrow, because there are many non-genetic factors that make essential contributions to development, and many of these would be considered "environmental" by anyone's definition of that word (e.g., nutrition). If we define "experience" more broadly as exposure to any non-genetic stimulus that can influence development, we can safely say that there are no traits that are impervious to experiential input.

As we have learned more about the biological mechanisms underlying the development of the structural and behavioral features of animals, it has become clear that just as genetic factors can only influence development by affecting cellular processes "above" the level of the genes, all experiences must have their effects by influencing events in the body's cells, whether those experiences entail learning or not. Experiential factors are now known to influence gene expression through several mechanisms, including (but

not limited to) those involving the actions of steroid hormones and those involving the actions of a class of genes known as immediate-early genes (IEGs). For example, testosterone levels change as a function of sexual experience, and hormones like testosterone are known to be able to diffuse across both cellular and nuclear membranes where—once they have been bound by specific receptors—they can bind with DNA to regulate gene expression (Yamamoto, 1985). In contrast, IEGs can be influenced even more directly by experience; IEGs located in the nuclei of neurons begin to be expressed only when specific experiences produce neural activity in those cells (Michel & Moore, 1995). As these examples make clear, experiences can influence development in many ways other than simply via "learning," as it has traditionally been understood.

Although for many of our characteristics, we have not yet discovered which experiential inputs influence their development, persistent investigators have repeatedly found that characteristics previously thought impervious to experiential input *are*, in fact, influenced by an organism's experiences during development. In a classic demonstration of this phenomenon, Gottlieb (1981) demonstrated that mallard ducklings' natural tendency to move toward the source of the mallard maternal call—a tendency that had previously been described both as innate, and uninfluenced by experience—can be affected by particular sensory experiences; in fact, Gottlieb (1991b) was able to get mallard ducklings to move toward the source of a chicken's call (a stimulus that normal mallard ducklings would not approach), simply by exposing them to particular auditory stimuli at the right time during embryonic development. Likewise, West and King have shown that a behavioral characteristic of male cowbirds previously thought to emerge from a species-specific innate template—namely the songs they sing during courtship rituals—is, in fact, importantly

affected by the males' experiences. Specifically, female cowbirds influence the structure of developing male cowbirds' songs by responding to segments of their immature, variable vocalizations with a very specific, non-vocal, social behavior that ultimately shapes the songs into the stereotyped, potent form that is typical of mature males (West & King, 1988; King, West, Goldstein, 2005). Given the number of characteristics that were once thought to develop in a way that is impervious to experiential input, but that have since been shown after extensive developmental analysis to be affected by such input, it is now the emerging consensus of developmental scientists that there are no traits whose development is impervious to experiential influence.

Doesn't the theory of evolution require adaptive characteristics to be transmitted across generations by the genes? Aren't species-typical, adaptive traits—those that develop in all normal members of a species—necessarily caused by genes alone?

Biologists' received view of evolution is known as the Modern Synthesis; this theory was produced by a collection of biologists writing in the first half of the 20th century, and was a remarkably successful fusion of Darwin's ideas about evolution and the theory of the gene that emerged following the scientific community's rediscovery in 1900 of Mendel's seminal work on heredity. In order to produce a comprehensive theory that actually worked, the architects of the Modern Synthesis felt compelled to exclude developmental processes from their theory, despite the fact that many of them—Theodosius Dobzhansky, Julian Huxley, and Sewall Wright, for example—recognized the central role that such processes rightly should play in a comprehensive theory of biology. One consequence of this decision was the subsequent definition of evolution in terms of changing gene frequencies across generations; the Modern Synthesis is a theory about

genes and their role in evolution (Jablonka & Lamb, 1995). However, as more recent data have discredited the notion that genetic factors can deterministically cause traits (adaptive or otherwise), the Modern Synthesis has become an increasingly unsatisfactory theory of biology.[6]

Although consensus has not yet been reached on how the Modern Synthesis should be altered, it is clear that any theory that *requires* characteristics to be transmitted across generations via exclusively genetic mechanisms will ultimately fail, because genetic mechanisms alone are not capable of doing the work that such theories require of them. Therefore, one of the several revisions to the Modern Synthesis that must be implemented if it is to remain a viable theory of evolution would replace the strictly genetic transmission of adaptive traits with transmission mechanisms that provide both the genetic *and* non-genetic resources that developing organisms require to build the traits that proved adaptive to their ancestors. Among the many theorists who have been trying to figure out how such mechanisms might operate are biologists (e.g., Jablonka & Lamb, 2005), philosophers of biology (e.g., Griffiths & Gray, 2001; Robert, 2006), and behavioral scientists (e.g., Lickliter & Berry, 1990; Lickliter & Honeycutt, 2003; West & King, 1987). These theorists acknowledge that nature has provided mechanisms by which organisms reliably develop adaptive characteristics during development; such species-typical, adaptive characteristics obviously play important roles in the evolutionary process. But it has become clear that such characteristics need not be genetically determined, in part because each species has

[6] Scientists dissatisfied with the Modern Synthesis have identified a variety of problems with the theory, only one of which is its underlying assumptions regarding the genetic determination of adaptive characteristics. Some of the most trenchant critiques of the Modern Synthesis have emerged from Evolutionary Developmental Biology, the branch of biology sometimes known as 'evo-devo'. For instance, evo-devo researchers have argued that the Modern Synthesis does a poor job of explaining how novel adaptive characteristics of animals emerge in the first place, a question that arguably should be at the center of any theory purporting to explain evolution. However, given the focus of this paper on genetic determinism, a broader critique of the Modern Synthesis that considers such concerns is beyond the scope of this work.

evolved in a particular context—a developmental niche—and these contexts can now be *expected* to be reproduced during the development of descendant generations (Greenough, Black, & Wallace, 1987). Thus, evolutionarily adaptive traits can be developmentally stable and universally acquired by members of a species, but still not be specified in genes that deterministically dictate phenotypic outcomes prior to the actual unfolding of development. In fact, it now appears that some genes *require* reliable developmental niches in order to be stably expressed across generations (K. Stotz, personal communication, January 28, 2008).

Why is it so hard to convey to students and the general public the full extent to which nature and nurture are fundamentally interdependent on one another?

Beyond the reasons alluded to above, there are likely additional reasons that students, journalists, and social scientists unfamiliar with the current facts of biology are skeptical when they first hear that genes cannot determine the forms of even relatively simple biological characteristics. But given the questions that usually arise when laypeople learn of the non-deterministic nature of genetic influence, it is clear that the major sources of this skepticism include 1) previous exposure to teachers who asserted that genes *can* determine some characteristics (however incorrect those assertions might have been), 2) exposure to mass media reports about studies (including twin and adoption studies) that, when simplified, seem to support claims of genetic determinism, and 3) personal observations of the development of characteristics that appear to be unaffected by experiences. In contrast, it seems unlikely that simple ignorance of the relevant facts of biology should be considered the primary source of skepticism in this case, because if it was, then one ought to be able to convince a skeptic that genes cannot independently

produce characteristics like hair color, alcoholism, height, or breast cancer by simply explaining what genetic factors *do* during development: that is, provide a portion of the information needed to sequence proteins. However, explaining that this is the one thing that genes actually can contribute to development generally does not lead immediately to acceptance of the idea that genes are unable to operate deterministically.

The resistance typically encountered by a scientist trying to explain how these processes work might be no different than the resistance physicists probably meet when they inform naïve listeners of the fundamental equivalence of matter and energy; as in that case, convincing listeners that genes do not operate deterministically would be difficult if they are ignorant of background information required to evaluate the relevant data. Similarly, although students can be taught about the limits to the conclusions one can appropriately draw when interpreting the correlational data generated in human twin studies, a deep appreciation of how little such studies can actually tell us about genetic contributions to our traits is more difficult to obtain; even professional social scientists occasionally jump to inappropriate causal conclusions on the basis of correlational data.

Two other features of this particular problem render it exceptionally difficult. First, even though biologists now *know* that genetic factors always interact with other factors to produce our characteristics, in most cases we are still poorly informed about the specific developmental mechanisms that produce those characteristics. Indeed, it is the very need to illuminate these developmental mechanisms that has motivated some of the more vocal critics of genetic determinism (Gottlieb, 1995; Lehrman, 1953; Lickliter & Honeycutt, 2003). Until such mechanisms have been elucidated, however, it is difficult to convince students to give up a sense of understanding (however ill-founded it might be) in favor of a

more honest sense of ignorance; generally, students are discomfited by the very large gaps that still characterize scientific knowledge in this domain. Second, just as Albert Einstein was troubled by quantum mechanics because of its probabilistic nature—he famously expressed this uneasiness in a 1926 letter to Max Born, writing "I…am convinced that He [God] does not throw dice"—it is not particularly surprising that students would prefer deterministic theories to probabilistic theories. The fact that the developmental processes that give rise to our characteristics are fundamentally probabilistic (Gottlieb, 1998) renders them harder to understand for students more familiar with simple cause-effect relationships.

Ultimately, genetic determinism likely owes much of its endurance to the fact that it is so simple. However, although many philosophers and scientists consider simpler theories to be preferable to more complex theories, all other things being equal (Sober, 1981), a truly complex reality requires a complex theory, even if such a theory might initially be less appealing to those exposed to it. Unfortunately, it is not yet clear how we might best attempt to convey the essential interdependence of nature and nurture to students and the general public.

That having been said, some of the new concepts and metaphors introduced recently by theorists concerned with these issues (Moss, 2003; Stotz, 2006; Griffiths & Stotz, 2006) might be useful tools in this effort. Metaphors can powerfully influence how we think about complex phenomena; for example, if one thinks of a genome as analogous to a recipe, one is more likely to focus on matters of process than if one thinks of the genome as analogous to a blueprint. Many systems theorists remain as dissatisfied with 'recipe' metaphors for the genome as they were with 'blueprint' metaphors for the genome, but it is

clear from this example how the use of different metaphors can influence our conceptualizations. In addition to those theorists who have suggested new metaphors for re-conceptualizing the genome, other theorists (e.g., Moore, 2001; Thelen & Smith, 1994) have proposed metaphors specifically designed to dispense with the outworn and obstructive dichotomies (e.g., nature vs. nurture, gene vs. environment, etc.) that we inherited from our intellectual ancestors at the end of the 19th century. In much the same way as a "recipe" metaphor for the genome draws attention to the *processes* driving development, these metaphors serve to focus attention on the fundamental interdependence of "nature" and "nurture," an interdependence that runs so deep that the very distinction between the two ideas is rendered suspect. Perhaps re-conceptualizing notions like "genes" and "nature" will finally enable students and the general public to abandon the genetic determinism that biologists now recognize as indefensible. But regardless, effective education about these matters will no doubt improve as we come to understand why belief in genetic determinism has persisted so long in the face of evidence indicating that it is not a constructive way to think about the emergence of biological and psychological characteristics in development.

References

Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind*. Cambridge, MA: MIT Press.

Block, N. (1995). How heritability misleads about race. *Cognition, 56,* 99-128.

Carlson, E. A. (1966). *The gene: A critical history.* Philadelphia, PA: W.B. Saunders Co.

Chomsky, N. (1975). *Reflections on language.* New York: Pantheon Books.

Cohen, L. G., Celnik, P., Pascual-Leone, A., Corwell, B., Faiz, L., Dambrosia, J., Honda, M., Sadato, N., Gerloff, C., Catala, M. D., & Hallett, M. (1997). Functional relevance of cross-modal plasticity in blind humans. *Nature, 389,* 180-183.

Cole, M., Cole, S. R., & Lightfoot, C. (2004). *The development of children* (5th ed.). New York: Worth.

Cosmides, L. (1989). The logic of social exchange: Has natural selection shaped how humans reason? Studies with the Wason selection task. *Cognition, 31,* 187 – 276.

Gehring, W. J. (1998). *Master control genes in development and evolution: The homeobox story.* New Haven: Yale University Press.

Gilbert, S. F. (2001). Ecological developmental biology: Developmental biology meets the real world. *Developmental Biology, 233,* 1-12.

Gilbert, S. F. (2006). *Developmental biology* (8th ed.). Sunderland, MA: Sinauer.

Gilbert, S. F., & Jorgensen, E. M. (1998). Wormwholes: A commentary on K. F. Schaffner's "Genes, Behavior, and Developmental Emergentism." *Philosophy of Science, 65,* 259-266.

Gottlieb, G. (1981). Roles of early experience in species-specific perceptual development. In R. N. Aslin, J. R. Alberts, & M. R. Petersen (Eds.), *Development of perception: Psychobiological*

perspectives. Vol. 1. Audition, somatic perception, and the chemical senses (pp. 5-44). New York: Academic Press.

Gottlieb, G. (1991a). Experiential canalization of behavioral development: Theory. *Developmental Psychology, 27,* 4-13.

Gottlieb, G. (1991b). Experiential canalization of behavioral development: Results. *Developmental Psychology, 27,* 35-39.

Gottlieb, G. (1992). *Individual development and evolution: The genesis of novel behavior.* New York: Oxford University Press.

Gottlieb, G. (1995). Some conceptual deficiencies in 'developmental' behavior genetics. *Human Development, 38*, 131-141.

Gottlieb, G. (1998). Normally occurring environmental and behavioral influences on gene activity: From central dogma to probabilistic epigenesis. *Psychological Review, 105,* 792 – 802.

Greenough, W. T., Black, J. E., & Wallace, C. S. (1987). Experience and brain development. *Child Development, 58,* 539-559.

Griffiths, P. E., & Gray, R. D. (1994). Developmental systems and evolutionary explanation. *The Journal of Philosophy, XCI,* 277-304.

Griffiths, P. E., & Gray, R. D. (2001). Darwinism and developmental systems. In S. Oyama, P. E. Griffiths, & R. D. Gray (Eds.), *Cycles of contingency: Developmental systems and evolution* (pp. 195 – 218). Cambridge, MA: MIT Press.

Griffiths, P. E., & Stotz, K. (2006). Genes in the Postgenomic era. *Theoretical Medicine and Bioethics, 27*(6), 499–521.

Harper, L. V. (2005). Epigenetic inheritance and the intergenerational transfer of experience. *Psychological Bulletin, 131,* 340 – 360.

Jablonka, E., & Lamb, M. J. (1995) *Epigenetic inheritance and evolution: The Lamarckian dimension.* Oxford: Oxford University Press.

Jablonka, E., & Lamb, M. J. (2005). *Evolution in four dimensions: Genetic, epigenetic, behavioral, and symbolic variation in the history of life.* Cambridge, MA: MIT.

Johannsen, W. (1911). The genotype conception of heredity. *American Naturalist, 45,* 129-159.

Joseph, J. (2001). Separated twins and the genetics of personality differences: A critique. *The American Journal of Psychology, 114,* 1 – 30.

Joseph, J. (2006). *The missing gene: Psychiatry, heredity, and the fruitless search for genes.* New York, NY: Algora Pub.

Kaas, J. H. (1991). Plasticity of sensory and motor maps in adult mammals. *Annual Review of Neuroscience, 14,* 137 – 167.

Keller, E. F. (2000). *The century of the gene*. Cambridge, MA: Harvard University Press.

King, A. P, West, M. J., & Goldstein, M. H. (2005). Non-vocal shaping of avian song development: Parallels to human speech development. *Ethology, 111,* 101 – 117.

Kuo, Z.-Y. (1976). *The dynamics of behavior development* (enlarged ed.). New York, NY: Plenum Press.

Lehrman, D. S. (1953). A critique of Konrad Lorenz's theory of instinctive behavior. *The Quarterly Review of Biology, 28,* 337-363.

Lewontin, R. C. (2000). *The triple helix: Gene, organism, and environment*. Cambridge, MA: Harvard University Press.

Lewontin, R. C., Rose, S., & Kamin, L. J. (1984). *Not in our genes.* New York: Pantheon.

Lickliter, R., & Berry, T. D. (1990). The phylogeny fallacy: Developmental psychology's misapplication of evolutionary theory. *Developmental Review, 10,* 348 – 364.

Lickliter, R., & Honeycutt, H. (2003). Developmental dynamics: Toward a biologically plausible evolutionary psychology. *Psychological Bulletin, 129,* 819 – 835.

Lorenz, K. (1965). *Evolution and modification of behavior*. Chicago, IL: University of Chicago Press.

Meaney, M. J. (2007, March). Adaptive phenotypic plasticity in rats: Maternal effects on programming of behavioral and endocrine responses to stress. In B. J. Ellis (Chair), *The stress response systems: Integrated evolutionary-developmental perspectives.* Symposium conducted at the meeting of the Society for Research in Child Development, Boston, MA.

Meaney, M. J., & Szyf, M. (2005). Maternal care as a model for experience-dependent chromatin plasticity? *Trends in Neurosciences, 28,* 456 – 463.

Merzenich, M. (1998). Long-term change of mind. *Science, 282,* 1062 – 1063.

Michel, G. F., & Moore, C. L. (1995). *Developmental psychobiology: An interdisciplinary science.* Cambridge, MA: MIT.

Moore, D. S. (2001). *The dependent gene: The fallacy of nature vs. nurture*. New York: W.H. Freeman.

Moore, D. S. (2006). A very little bit of knowledge: Re-evaluating the meaning of the heritability of IQ. *Human Development, 49,* 347 – 353.

Moss, L. (2003). *What genes can't do.* Cambridge, MA: MIT.

Müller, G. B. (2003). Embryonic motility: Environmental influences and evolutionary innovation. *Evolution & Development, 5,* 56 – 60.

National Academy of Sciences (1999). *Science and Creationism: A View from the National Academy of Sciences*. Washington, DC: National Academy Press.

Nijhout, H. F. (2003). The importance of context in genetics. *American Scientist, 91,* 416-423.

Pantev, C., Oostenveld, R., Engelien, A., Ross, B., Roberts, L. E., & Hoke, M. (1998). Increased auditory cortical representation in musicians. *Nature, 392,* 811- 813.

Pinker, S. (2002). *The blank slate: The modern denial of human nature.* New York: Viking.

Pinker, S. (1994). *The language instinct: How the mind creates language.* New York: W. Morrow.

Robert, J. S. (2006). *Embryology, epigenesis and evolution: Taking development seriously.* Cambridge: Cambridge University Press.

Schneirla, T. C. (1966). Behavioral development and comparative psychology. *The Quarterly Review of Biology, 41,* 283-302.

Smith, C. W. J., Patton, J. G., & Nadal-Ginard, B. (1989). Alternative splicing in the control of gene expression. *Annual Review of Genetics, 23*, 527-577.

Sober, E. (1981). The principle of parsimony. *The British Journal for the Philosophy of Science, 32,* 145 – 156.

Spelke, E. S., & Kinzler, K. D. (2007). Core knowledge. *Developmental Science, 10,* 89 – 96.

Stotz, K. (2006). With 'genes' like that, who needs an environment? Postgenomics's argument for the 'ontogeny of information'. *Philosophy of Science, 73*, 905 – 917.

Sturtevant, A. H. (1915). The behavior of the chromosomes as studied through linkage. *Zeitschrift für Induktive Abstammungs und Verersbungslehre, 13,* 234 – 287.

Sulloway, F. J. (1992). *Freud, biologist of the mind*. Cambridge, MA: Harvard University Press.

Thelen, E., & Smith, L. B. (1994). *A dynamic systems approach to the development of cognition and action.* Cambridge, MA: MIT Press.

Wade, N. (2005, May 10). For gay men, an attraction to a different kind of scent. *The New York Times*, pp. A1.

Weaver, I. C. G., D'Alessio, A. C., Brown, S. E., Hellstrom, I. C., Dymov, S., Sharma, S., et al. (2007). The transcription factor nerve growth factor-inducible protein A mediates epigenetic programming: Altering epigenetic marks by immediate-early genes. *The Journal of Neuroscience, 27,* 1756 – 1768.

West, M. J., & King, A. P. (1987). Settling nature and nurture into an ontogenetic niche. *Developmental Psychobiology, 20,* 549 – 562.

West, M. J. & King, A. P. (1988). Female visual displays affect the development of male song in the cowbird. *Nature, 334,* 224 – 246.

Wilson, E. O. (1978). *On human nature.* Cambridge, MA: Harvard University Press.

Wolpert, L. (1992). *The triumph of the embryo.* New York, NY: Oxford University Press.

Yamamoto, K. R. (1985). Steroid receptor regulated transcription of specific genes and gene networks. *Annual Review of Genetics, 19,* 209 – 252.