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Essentially Biased: Why People Are Fatalistic About Genes

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Abstract

We propose that people are genetic essentialists—that is, they tend to think of genetic attributions as being immutable, of a specific etiology, natural, and dividing people into homogenous and discrete groups. Although there are rare conditions where genes operate in these kinds of deterministic ways, people overgeneralize from these to the far more common conditions where genes are not at all deterministic. These essentialist biases are associated with some harmful outcomes such as racism, sexism, pessimism in the face of illnesses, political polarization, and support for eugenics, while at the same time they are linked with increased tolerance and sympathy for gay rights, mental illness, and less severe judgments of responsibility for crime. We will also discuss how these essentialist biases connect with the burgeoning direct-to-consumer genomics

industry and various kinds of genetic engineering. Overall, these biases appear rather resistant to efforts to reduce them, although genetics literacy predicts weaker essentialist tendencies.

By all accounts, the genomics revolution has arrived. Since the first human genome was sequenced in 2003, the cost of sequencing genomes has fallen by more than a million fold, with the technical advancements of genotyping technologies far outpacing the rapid speed of improvements in computer transistors that was famously encapsulated in Moore's law (National Human Genome Research Institute, 2016). As a result of these dramatic technical innovations, the new industry of direct-to-consumer (DTC) genomics companies has recently emerged, allowing individuals to have their genomes sequenced at a relatively affordable price. Customers can be provided with information that purportedly speaks to their likely genetic ancestry, their risks for developing various fatal diseases, and their children's future career possibilities. Alongside this development, a vast scientific enterprise that links specific genetic variants to various human conditions continues to receive wide coverage in the popular media. How will people make sense of this brave new world of genomics? A growing field of psychological research has emerged to address this question.

It may seem strange to ask how people will understand a new scientific revolution. Won't they just come to learn this new information in the same way that they learn other information? That is, we might expect that people will respond more or less like scientists themselves do, whereby they slowly integrate their new insights with their existing ones, and ultimately developing a richer understanding that empowers them to more effectively interact with their world (cf. Kuhn, 1962/1996). On the other hand, much research has revealed that people do not process information about genetic attributions in a rational and even-handed way (for a review, see Dar-Nimrod & Heine, 2011; Heine, 2017). Rather, genetic causes are often understood fundamentally differently from other kinds of causes, and as we'll elaborate later, this has significant implications for the ways that people make sense of their worlds.

As a case in point, consider the following study (Dar-Nimrod, Cheung, Ruby, & Heine, 2014; Study 3): participants were randomly assigned to read one of three newspaper articles that discussed actual research related to obesity. Those who were assigned to a "Genetics" condition read about research describing how "obesity genes" relate to one's weight. Another group was

assigned to an "Experiences" condition and read about research describing how people's social networks relate to their weight. A third group was assigned to a Control condition where they read an unrelated article about corn production. Following a series of food-related assignments to mask the purpose of the study, participants took part in a food tasting task; they were given some cookies to sample and evaluate their flavors. The key-dependent variable was the amount of cookies that people ate. The results revealed that those in the "Genetics" condition consumed approximately one-third more cookies than those assigned to either the "Experiences" or the "Control" condition, which did not differ significantly from each other. The results of this study, together with those from several correlational studies in the same paper, suggest that people became more fatalistic about their weight when learning about genetic causes for obesity, but not when learning about experiential causes for obesity. This relative overweighing of genetic causes occurred even though both genes and experiences contribute to risk for obesity. Why might genetic causes be perceived differently than experiential ones?



1. PSYCHOLOGICAL ESSENTIALISM

We can gain insight into how people consider genetic causes when we consider the broader question of how people make sense of why things in general are the way they are. Aristotle provided an answer to this metaphysical question by proposing that all entities are as they are because of an underlying *essence* that they possess which makes them so (Moravcsik, 2001). Essences were described by Locke (1671/1959) as "the very being of anything, whereby it is what it is." They are imagined to be deep, internal forces that form the basis of identity of entities, and cause the entities to function as they do. For example, essences are what give rise to a cat's agility, aloofness, hunting prowess, soft furry coat, curiosity, and penchant for catnip—without such an essence, cats would be very different creatures. Of course, trying to specify what precisely is the underlying essence of a cat, and how this essence leads to all those cat-like characteristics, is metaphysically intractable. But the question embraced by psychologists is not whether there actually *are* essences that undergird the reality which we live in, but whether people *believe* that these kinds of essences indeed make things so (Medin & Ortony, 1989). This belief that essences give rise to entities is termed *psychological essentialism*.

Evidence for psychological essentialism is widespread. Indeed, there are few psychological phenomena which have been identified in a broader array of samples than essentialism: studies have found support for essentialism in samples of Chaldean and Hmong immigrants in Detroit (Henrich & Henrich, 2007), Mongolian herders (Gil-White, 2001), Vezo children in Madagascar (Astuti, 2001), Menominee in Wisconsin (Waxman, Medin, & Ross, 2007), rural Ukrainians (Kanovsky, 2007), children from poor neighborhoods in Brazil (Sousa, Atran, & Medin, 2002), and several dozens of studies involving children and adults from Western countries (see Gelman, 2003, for a review). However, despite this universality in prevalence, there is evidence that some populations are more committed to essentialist thinking than others (e.g., Giles, Legare, & Samson, 2008). For example, higher SES Americans hold more essentialist views than those of lower SES (Kraus, Piff, Mendoza-Denton, Rheinschmidt, & Kelter, 2012). Likewise, some psychological phenomena related to essentialism, such as a tendency to make dispositional attributions (Choi, Nisbett, & Norenzayan, 1999) and to have more entity theories of self (Heine et al., 2001), are more prevalent among Western populations than among East Asians. This suggests that psychological essentialism could qualify as a functional universal, serving a similar function everywhere, although the degree of commitment varies across populations (Norenzayan & Heine, 2005). Some have argued that it was adaptive for an omnivorous foraging species like humans to categorize the natural world around them into different species on the basis of imagined essences, to facilitate their success at foraging (e.g., Atran, 1998).

Research has identified a number of characteristics associated with psychological essentialism, and as we will explain later, these influence how people think about genetic causes. First, essences are perceived to be *immutable*. The properties that they carry are seen to persist and remain somewhat inviolate to one's experiences (for a review, see Gelman, 2003). They are perceived to lie deep within an individual, beyond the penetration of outside influences. For an example of this quality of essences, consider a study by Gil-White (2001) who interviewed Mongolian herdsman from two tribes: the Uryankhais, who are believed to be able to cast curses, and the Torguuds, who are not thought to have any such curse-casting abilities. Gil-White posed a question to the herders: if a boy born to Uryankhai parents was raised by Torguuds, would he be able to cast curses? The interviewees responded that the boy would indeed have such capability, given his Uryankhai birth parents, although he would not know it, given his Torguud upbringing. The

boy's Uryankhai essence is perceived to remain intact and is not compromised despite his exclusively Torguud experiences. This sense that one's upbringing does not impact one's underlying essence is not likely the result of education, as it has been documented with young children across a variety of contexts (Giles, 2003; Hirschfeld & Gelman, 1997). For example, by the age of five, children show evidence of a belief in the immutability of essences even in the face of large-scale transformations, such as a caterpillar developing into a butterfly (Rosengren, Gelman, Kalish, & McCormick, 1991). Entities may encounter a variety of experiences and transformations in their lives, but these are perceived to be inconsequential to the underlying essence (Gelman, 2004).

Second, people think of essences as being *deep down and internal*, beyond the reach of external influences. We do not actually see essences; they lie beneath the surface far beyond our visual field. Yet people believe that essences give rise to all that they see around them. Experiments where people are presented with natural kinds that have encountered some kinds of modifications to their surface reveal that people still believe that the underlying essence remains intact (e.g., Rips, 1989). By the age of three, children view transformations of an object's insides as impacting the object's identity more than transformations to the object's outsides, as the basis of identity lies with its deeply buried essence (Gelman & Welman, 1991). However, despite lying outside of our ability to see them, people believe that the essences are potentially accessible to experts (Gelman & Markman, 1987).

Third, people think of essences as underlying the *natural world*; they play a much weaker role for our understanding of artifacts. So while people may perceive essences to differentiate between gold and fool's gold, they are less likely to employ essences to define artifacts. The distinction between gold and fool's gold is believed to be due to an underlying essence that is supposedly identifiable by experts; in contrast, the distinction between a station wagon and a sports utility vehicle is understood more to be a matter of convention (see also Gil-White, 2001; Malt, 1989). This distinction in the role of underlying essences between natural kinds (i.e., categories that exist in the natural world) and artifacts is also evident among children. For example, Keil (1989) presented kindergarten students with a thought experiment where a raccoon had been altered to look like a skunk, yet the children insisted that it remained a raccoon under its skunk dressing. On the other hand, the same children were quite willing to accept that a coffeepot had been transformed into a birdfeeder. Essences are defining elements for making sense of the perceived natural world and are less consequential for understanding artifacts.

A fourth feature of essences is that they are perceived to *draw the boundaries of categories*. They represent what members of a category share in common, and what differentiate them from members of different categories (Medin & Ortony, 1989). This is especially so in the way people turn to essences to understand different species (Atran, 1998); for example, the basis of modern scientific taxonomy developed by Carl Linnaeus hinged on an assumption that different species derive from distinct essences (Ereshefsky, 2004). Likewise, this assumption also generalizes to people's perceptions of ethnic groups (Gil-White, 2001; Rothbart & Taylor, 1992) and serves as one key foundation of prejudice (Allport, 1954). Essences seem to carve nature at its joints and as a result, people feel they can make inductive inferences about what a newly encountered individual may or may not be able to do based on the imagined essence of its group (Gelman, 2004).

Finally, essences can be *transferred* from individual to individual while preserving their original identity. One way we can see this is that objects are perceived to be able to acquire the essences of their owners; a notion that is apparent from a young age (Gelman, Frazier, Noles, Manczak, & Stilwell, 2015). For example, Nemeroff and Rozin (1994) found that some American undergraduate students balked at the idea of wearing Hitler's sweater, regardless of how much it has been dry cleaned, for fear of coming in touch with his contaminating essence. In another example, approximately one-third of recipients of heart-transplants feel that they have acquired traits from their donors (Inspector, Kutz, & David, 2004). Likewise, prior to any education about heritability, young children understand that a child comes to acquire some aspects of essences from their biological parents even if they were adopted (Heyman & Gelman, 2000). Essences thus are understood to be able to move from object to object and from parent to offspring. In sum, people's intuitive understanding of essences comes wrapped up with some particular ideas about the ways that essences undergird the natural world.

1.1 Genetic Essentialism

Despite having rather specific ideas about what essences are like, people have a harder time forming concrete mental representations of essences. Hence, they turn to an *essence placeholder* that serves as a scaffolding that affords explanations for how any observed characteristics have come to be (Medin & Ortony, 1989). People have turned to various essence placeholders throughout history for making sense of their worlds, such as the four humors of Hippocrates that were assumed to be critical for understanding health and

personality, Chinese conceptions of chi, or the yogic belief in prana. But genes make a particularly apt placeholder for essences, and this leads people to imagine that genes share many of the features that they associate with essences (for more description, see Heine, 2017).

Just as with essences, genes are perceived to be deep down and internal and thus share the same features as a nonmaterialistic placeholder, namely, not being constrained by any visible limitations in a manner that would bound future causal inferences (Medin & Ortony, 1989). Likewise, just as people believe that essences are potentially knowable to experts, people will often readily accept scientific claims of a novel genetic causal explanation for various phenomena (Dar-Nimrod & Godwin, 2016). Moreover, similar to how they perceive essences, people view genes as far-reaching causal factors—they offer reasons to explain a diverse array of phenomena and provide a succinct account for why people behave in the ways they do (Jayaratne et al., 2009). In addition, as with essences, genes are understood to be transferred from one generation to another (Gil-White, 2001). And, just as with essences, genes are perceived to be stable and unchanging throughout a person's lifetime, facilitating a sense of self-unity despite the overwhelming physical and psychological transformations that occur across their development (Chandler & Proulx, 2008). Genes are therefore remarkably well suited to serve as essence placeholders, given that people's understanding of genes aligns well with how people conceive of essences. Because of this overlap with people's essentialist intuitions, we submit that when most people are thinking about genes they are *not* really thinking about genes—they are thinking about metaphysical essences.

Conceiving of genes as essence placeholders suggests that people's understanding of genetics may be somewhat distorted. In line with this, a body of research documents an enduring, limited public understanding of basic genetic science (Condit, 2010; Henderson & Maguire, 2000). For example, a survey of American adults (Lanie et al., 2004) found that fewer than half could correctly answer the question, "Where in your body are your genes located?" (the correct answer is in your cells). Likewise, based on a US nationally representative survey of American adults, Christensen, Jayaratne, Roberts, Kardia, and Petty (2010) found that 76% incorrectly believed that "single genes directly control specific human behaviors." Moreover, with regard to evaluating US high school students' knowledge of genetics, the National Assessment of Education Progress (NAEP) revealed substantial deficiencies in genetic proficiency (O'Sullivan et al., 2003), most commonly observed in students' interpretation of genetic materials and

understanding of genetic diseases. The NAEP included eight questions probing students' understanding of genetics and genetic engineering (e.g., what is a gene? what is it made out of?). It also provided an article discussing the use of viruses in genetic engineering and asked the examinees to utilize their own knowledge along with the materials in the article, in answering relevant questions. Only 1% of 12th graders provided accurate responses that reflected appropriate integration of their knowledge and the genetic essays they read, demonstrating broad misconceptions among soon-to-be high school graduates. Even among arguably the most informed students, those who voluntarily submitted an essay to the American Society of Human Genetics' National DNA Day Essay Contest, more than half demonstrated a common misunderstanding of basic genetic concepts and essentialist biases (Shaw, Van Horne, Zhang, & Boughman, 2008).

These various misconceptions about genes may be facilitated by the pervasive representations of characteristics and conditions as genetically derived which appear across various socialization agents (families, media, the arts, or schools). Frequently, simple OGD (one gene, one disease; Conrad, 2002) accounts are offered in making sense of complex phenomena, facilitating the assumption that there is a single corresponding genetic cause underlying every human trait. Whether one reads a newspaper article entitled "Fat gene found by scientists" (Henderson, 2007), watches a Hollywood blockbuster in which genes are presented deterministically (e.g., *Gattaca*, *The Hulk*, *X-Men*), or is told that the "gene for alcoholism runs in my family," these commonplace exposures contribute to the implicit endorsement of genes as the essence of personhood. These simplified representations are straightforward, simple to digest, and are commonly tainted by an erroneous fatalistic flair (Conrad, 1999; Dar-Nimrod & Heine, 2011). A genetics curriculum focusing on Mendelian models that highlight deterministic inheritance only exacerbates such reductionist notions (Dar-Nimrod, 2012; Dougherty, 2009; Radick, 2016).

Regardless of how people come to acquire their understanding of genetics, it is common for them to perceive genes in a simplified and almost mystical, agentic fashion (e.g., Nelkin & Lindee, 1995; Sheldon, Pfeffer, Jayaratne, Feldbaum, & Petty, 2007), ignorant of the complex, interactive processes that invariably occur between genes and environmental factors (for thoughtful discussions, see Pinker, 2003; Ridley, 2003; Turkheimer, 2000). Despite harboring misconceptions about genes and their operations (e.g., Christensen et al., 2010; Lanie et al., 2004), people readily invoke genes to explicate a broad range of human afflictions, capabilities, and

behaviors (e.g., Condit, Parrott, Harris, Lynch, & Dubriwy, 2004; Gelman & Welman, 1991; Jayaratne et al., 2006; Sheldon et al., 2007). As people's understanding of genetics is extremely limited, it seems that genes are often invoked as the embodiment of a metaphysical essence rather than as components of a biological process for building proteins (Dar-Nimrod & Heine, 2011).

This is not to say that essentialist explanations are the sole kind of causal explanations that people consider (e.g., people readily accept that smoking is causally related to lung cancer), nor that all essentialist explanations rest on a foundation of genes (for other exemplars, see Gil-White, 2001; Haslam, 2011; Rangel & Keller, 2011). Rather, given the substantial conceptual overlap between people's lay understanding of both genes and natural kinds' metaphysical essences, genetic attributions often activate or strengthen certain essentialism-derived cognitive biases.

The Genetic Essentialism Framework (GEF; Dar-Nimrod & Heine, 2011) offers a theoretical foundation for describing cognitive processes set in motion once a person perceives genes to be a relevant causal factor. The GEF suggests that genetic attributions for various traits, conditions, or diseases activate four specific psychological processes termed genetic essentialist biases.

The first bias, *immutability/determinism*, specifies that thinking about genetic attributions lead people to view relevant outcomes as less changeable and more predetermined (see Gould & Heine, 2012). To the extent that a phenomenon is perceived to be immutable, it will be perceived to be beyond someone's control; and indeed, genetic attributions decrease perceptions of control over relevant outcomes (e.g., Parrott & Smith, 2014) and limit the perceived capability of other means, such as environmental manipulations or individuals' volition, to modify the outcome (e.g., Jayaratne et al., 2009). For example, research indicates that endorsement of genetic etiological explanations for disease is negatively associated with perception of control over a disease (e.g., Dar-Nimrod, Zuckerman, & Duberstein, 2013; Jayaratne, Giordimaina, & Gaviglio, 2012; Shiloh, Rashuk-Rosenthal, & Benyamini, 2002). Discounting one's personal control also leads to a reduction in perceived capability to execute a desirable behavior, which may lead to decreased domain-specific self-efficacy (Bandura, 1977). In line with that assertion, a study of inactive university students found that exercise-related self-efficacy is lower after exposure to a genetic attribution for inactivity compared with an experiential attribution (Beauchamp, Rhodes, Kreutzer, & Rupert, 2011).

Perception of immutability is not the only relevant cognition that affects perceived behavioral control when genetic etiology is implicated. The second genetic essentialist bias, the tendency to discount additional causal explanations once genetic attributions are made (termed *single* or *specific etiology*), also increases the likelihood that people will disregard alternative causal attributions for complex phenomenon (Dar-Nimrod & Heine, 2011). In accordance with this bias, identifying a particular gene can become conflated with a diagnosis of the related condition. Genes seem to be especially suited for narrowing the search for causes, because when genes are implicated as potential etiological explanations, they are viewed as more specific than experiential explanations to the outcome (Dar-Nimrod, Cheung, et al., 2014, Study 2). For example, someone who learns that she does not have “the gene for breast cancer,” might then view herself as not needing to engage in any future screening efforts, which would be a grossly incorrect conclusion from the genetic testing results. In line with this bias, research indicates that whereas environmental causal attributions are positively correlated with a sense of personal choice, greater endorsement of genetic attributions are negatively associated with both environmental attributions and a sense of choice (Jayaratne et al., 2009).

Whereas the first two genetic essentialist biases focus on individuals, the third one extends the attention to groups. The metaphysical essence is at its core a category-enabling construct; that is, it is the identifying facet that determines membership in a specific natural group (e.g., being a cat). It follows that essentialist thinking leads a person to focus on the central identifying features that are common to all the members, drawing attention away from ingroup differentiating features. A focus on the commonalities should lead to viewing individual members of a category as more homogeneous because they share the identifying features (e.g., “catness”); as such, essentialist thinking also brings to the fore the distinctiveness of members of a category from those who are not in it and do not share these defining features (e.g., a whale). Hence, genes, like essences, can be seen to carve nature at its joints. The same principle seems to operate with social categories, such as race or gender (Haslam, Rothschild, & Ernst, 2000; Rothbart & Taylor, 1992). And indeed, this third genetic essentialist bias, *homogeneity/discreteness*, asserts that genetic explanations for (social/natural) group differences increase the likelihood that each group will be viewed as homogeneous and more discrete from each other. Consistent with this bias, a large public opinion survey indicated that conservatives, who are more likely to see racial groups as homogenous and distinct from each other than liberals

(e.g., Gillborn, 1997), are also more likely to view racial differences as caused by genetic dissimilarities (Suhay & Jayaratne, 2012). Utilizing an experimental approach, Brescoll and LaFrance (2004) found that highlighting biological/genetic differences between men and women increased adoption of stereotypical descriptors.

The final genetic essentialist bias is *naturalness*, that is, genetic attributions increase the likelihood that a relevant outcome is perceived as a natural outcome. There is long-standing evidence in psychology that viewing an outcome as natural has an important evaluative component attached to it. Research on the naturalistic fallacy (e.g., Frankena, 1939; Moore, 1903; see also the related longevity bias: Eidelman & Crandall, 2014) has consistently demonstrated that elements which are perceived as natural are also perceived as inherently good and desirable. Whether it is the romantic notion behind Rousseau’s uncorrupted noble savage, the large “all natural ingredients” labels on goods at the supermarket, or political ideologies harnessing the term “natural order” to perpetuate their control, the evidence for such a tendency is all around us. By offering genes as causal explanation, the outcome is implicitly viewed to be natural and, by extension, appropriate. And indeed, men show increased moral acceptance of undesirable behaviors such as date rape when genes are even remotely implicated as opposed to societal forces (Dar-Nimrod, Heine, Cheung, & Schaller, 2011, Study 2). Furthermore, consistent with the naturalness bias, despite the consensual scientific position that genetically modified organisms (GMOs) are safe to eat, a majority of consumers wish to avoid them, often justifying their opposition by concluding that these products are “against nature” (Blancke, Van Breusegem, De Jaeger, Braeckman, & Van Montagu, 2015, p. 415). In that sense, genetic attributions may be used to increase acceptance of specific outcomes by appealing to our tendency to conflate what naturally “is” with what “ought to be,” although, as we shall see in later sections, attempts to strategically use this bias do not produce straightforward outcomes.

Recently, a new measure, the Genetic Essentialist Tendencies Scale (GETS; Dar-Nimrod, 2014), was constructed to assess these four biases. With six items addressing each of the biases, the 24-item measure targets the different facets of genetic essentialism (e.g., Immutability: “People with a genetic predisposition to a certain personality are destined to behave in a certain way”; Specific Etiology: “The environment does not affect the chances of getting cancer for someone with a genetic susceptibility to cancer”; Homogeneity: “People with a gene associated with risk taking are

probably quite similar”; Naturalness: “It is natural to behave aggressively if one has a genetic predisposition to aggression”). Data collected from community samples in several studies confirm the fit of a four factor underlying structure of the measure. They also uncover moderate to strong positive correlations ($0.30 < r < 0.60$) between the different biases and a factorial structure that supports the notion that genetic essentialism can be conceptualized as a unitary, second-order construct comprised from these separate biases (Dar-Nimrod et al., 2016). These studies revealed that the biases, as measured by the GETS, predicted varied outcomes such as fatalism, negative views of human nature (i.e., social cynicism), various forms of prejudice, health pessimism (both in general and when genes are implicated for a specific disease), and reduced intentions to engage in healthy behaviors, among others (Dar-Nimrod et al., 2016).

1.2 Are These Biases Irrational?

We describe the aforementioned intuitions about genetic essentialism as *biases*. But what if instead these intuitions are a good-enough approximation of the ways that genes actually influence our phenotypes? On what grounds can we call them biases as opposed to accurate descriptions of the nature of genetic causes?

Indeed, it is not difficult to come up with examples of genetic causes which map nicely on to the intuitions that we described earlier. For example, going right back to the origins of genetics research, we can consider the observations of Gregor Mendel’s original studies with pea plants that were conducted in the gardens of St. Thomas’s Abbey in the present day Czech Republic. Mendel found seven characters of peas that were passed faithfully from parent to offspring, and ultimately provided the scientific world with the key notions of genetic segregation and dominance (Henig, 2000). For example, a pea that inherited two copies of a particular allele from its parents would produce a pea with yellow pods—100% of the time. The causal forces that produce a yellow pod do indeed appear to be immutable (there’s no indication that this causal series could be interrupted to produce a green pod instead); it has a specific etiology (the distinction between whether the pod is yellow or green is determined solely by the particular alleles that the plant inherits from its parents); all yellow-podded pea plants of this species are homogenous to the extent that they all share the same alleles and are discrete in that they are genetically different from their green-podded neighbors; finally, the yellowness of the pod would appear to be natural—emerging

as it does from its default state and is not the product of any kind of artificial engineering. So are not the intuitions identified in the GEF just another means for describing the way that genes produce traits in pea plants?

We agree that these intuitions are a good-enough description of the causal forces at work in Mendel’s pea plants. However, these pea plants are not at all representative of the ways that genes influence phenotypes more generally. Turkheimer (1998) highlights a continuum that lies between two broad classes of genotype–phenotype relations. On the one extreme of the continuum is what he calls *strong genetic explanation*. Strong genetic explanation means that a particular biological process has been identified and localized which can explain a large part of the phenotype under question. The link between the dominant allele and yellow pod color is a clear case of strong genetic explanation, as are the variations of the CFTR gene which have been found to deterministically cause cystic fibrosis (Pearson, 2009). For traits that can be characterized as having a strong genetic explanation, the aforementioned genetic essentialist intuitions are reasonably accurate descriptions.

However, this direct one-to-one relation between a particular genetic variant and a particular phenotypic character is not at all common. For example, the DTC genomics company, 23andMe, provides information on 60 human traits, ranging from eye color to likelihood of going bald. Only one of these traits—whether or not you have wet or dry earwax—is a Mendelian trait that emerges in this direct one-to-one way (Heine, 2017). Indeed, when students learn about genetics in high school, they may well encounter examples that are described as Mendelian characteristics, such as eye color, or whether or not you can roll your tongue into a tube. Neither of these are Mendelian traits, and the fact that high school curricula rely so much on these inaccurate examples reflects just how rare easily observed monogenic traits actually are.

Rather, human traits are more typically characterized by what Chabris, Lee, Cesarini, Benjamin, and Laibson (2015) refer to as “the fourth law of behavioral genetics,” which states that “a typical human behavioral trait is associated with very many genetic variants, each of which accounts for a very small percentage of the behavioral variability” (p. 305). For example, researchers have identified more than 100 common genetic variants that predict schizophrenia, which account for only a fraction of the genetic variability in the condition (Schizophrenic Working Group of the Psychiatric Genomics Consortium, 2014). Likewise, although height is highly heritable, researchers estimate that you would need to consider

approximately 294,000 common genetic variants to account for half of the variability in human height (Yang et al., 2010). Similarly, despite the relatively strong heritability of IQ, there are no single gene that predict IQ to any appreciable degree (the strongest single predictor is associated with about 0.3 IQ points; Rietveld et al., 2014); instead, approximately 500,000 variants are estimated to be implicated in predicting one half of IQ variability (Davies et al., 2011). These examples all represent what Turkheimer (1998) calls *weak genetic explanation*. In these cases the phenotype (e.g., whether or not one develops schizophrenia) is associated with a genotype (e.g., risk of schizophrenia is heritable), but not in a direct or deterministic way. Many genes interact, perhaps thousands of them, and each contributes to the likelihood of the phenotype. Phenotypes are influenced by genes only when the genes express proteins, and this expression is dependent on experiences in the environment. Moreover, this expression varies across the organism's developmental trajectory. Further complicating matters, various epigenetic markers can influence when and how often particular proteins are expressed (for a review, see Cole, 2009). Although genotypes correlate with phenotypes, in cases of weak genetic explanation the correlation remains largely unexplained, and the vast complexity of the interaction of the multiple forces at work may even be in principle unknowable, at least with the current limits of our technologies. Thus, explaining these complex phenomena by referring to the unspecified involvement of genes remains the wrong level of analysis to understand them (see Turkheimer, 1998, for discussion).

The error that people commonly make, and what leads us to call people's genetic essentialist intuitions "biases," is that people overgeneralize from the straightforward, easy to understand, yet rare examples of strong genetic explanation to explaining the vast majority of other phenotypes which emerge from weak genetic explanation. It is incorrect to assume that traits that are a product of weak genetic influence can be understood better by thinking of them as immutable, natural, of a specific etiology, and forming homogenous and discrete groups.

1.3 Genetic Essentialism Is Widespread and Distorts People's Understanding

Our central thesis is as follows: when people consider that genes are involved in a trait, they come to think differently about that trait. They come to

conceive of it as though it was caused by an essence, and they consider the trait in ways that are consistent with their essentialist biases. For example, consider how people's genetic essentialist biases can manifest in the way they think of learning styles. Many people endorse the idea of learning styles, or the belief that different people rely on distinct styles for most efficiently learning new material (e.g., visual learners vs auditory learners; Dunn, Griggs, Olson, Beasley, & Gorman, 1995; Joy & Kolb, 2009). An extension of this idea is the *meshing hypothesis*, which suggests that optimal learning occurs when an educator's teaching style matches with a student's learning style. Despite the lack of robust empirical evidence for such concepts (see Pashler, McDaniel, Rohrer, & Bjork, 2008), many people still endorse them, which can be predicted based on the extent to which people essentialize genes.

For example, American MTurk participants were asked to report the extent to which they thought learning styles were genetic vs environmental in origin, along with various perceptions of learning styles. The more the participants perceived learning styles to be genetically caused, the less control they expected one to have over what learning styles one has, the less malleable they thought learning styles were, and the more they thought that learning outcomes are best when learning styles and teaching styles match (i.e., endorsement of the meshing hypothesis). Crucially, mediation analyses revealed that both perceived control and perceived malleability of learning styles significantly mediated the relation between perceived etiology of learning styles and people's endorsement of the meshing hypothesis (Cheung, White, Sumitani, Truong, & Heine, 2016). Overall, this study provides a simple illustration of the role that genetic essentialist biases may play in our lives.

The notion that our essentialist biases influence our thinking when people encounter genetic explanations is not something that we should expect to be limited to a small set of rare or inconsequential traits, because evidence for heritability is extremely broad (e.g., Olson, Vernon, Harris, & Jang, 2001). Heritability refers to the proportion of a trait's variability within a specific sample that is due to genetics, and it is typically summarized by a heritability coefficient which varies from zero to one. Notably, heritability does not indicate the extent to which a trait is caused by genes in some direct way. Evidence for heritability (i.e., a heritability coefficient that is significantly greater than zero) has been demonstrated for such diverse traits and behaviors as whether one disapproves of student pranks (Martin et al., 1986), watches a

lot of television (Plomin, Corley, DeFries, & Fulker, 1990), donates blood (Pedersen et al., 2015), gets divorced (McGue & Lykken, 1992), or even gets mugged (Kendler & Karkowski-Shuman, 1997). In fact, evidence for heritability is so broad that Turkheimer (2000) proposed the “first law of behavioral genetics” which states that “all human behavioral traits are heritable” (p. 160). This law is meant to be the null hypothesis, rather than a fixed law, in the sense that heritability evidence is so extensive that we should assume human characteristics is heritable until proven otherwise. An example of an exception to this is that although one’s degree of religiosity is heritable (Bouchard, 2004), the particular religion that one belongs to (e.g., Buddhism, Islam, Catholicism) is not (D’Onofrio, Eaves, Murrelle, Maes, & Spilka, 1999). Of course, if almost everything is heritable, and to a fairly similar degree, then the utility of the very concept of heritability comes into question (Turkheimer, 2000). Nonetheless, given that so much of human behavior is heritable, and that—according to the fourth law of behavioral genetics—this heritability is usually the product of weak genetic influence (Chabris et al., 2015), our genetic essentialist biases can potentially distort how we understand much of the human condition.

Our essentialist biases are also at risk for interacting with a new kind of genetic information that is becoming more common. In recent years, dozens of DTC companies have emerged that provide risk information for more than 100 common diseases that are products of weak genetic explanation, such as most cancers, coronary heart disease, or Type II diabetes (Kaufman, Bollinger, Dvoskin, & Scott, 2012). The essentialist bias that people tend to understand genetic causes as emerging from a single etiology is particularly worrisome in the face of this DTC disease-risk information. After reviewing their genotyping results, a customer might conclude, for example, that they have “the Type II diabetes gene,” or “the Parkinson’s gene.” With a tendency to see genetic causes as ultimate causes, people may well find this kind of genetic information to be far more worrisome than the risk estimates actually communicate. Indeed, such faulty interpretations of genotyping results are not rare (e.g., Gordon et al., 2012; Wang, Gonzalez, & Merajver, 2004), and genetic counselors struggle to communicate genetic risk in a way that people can understand it (Austin, 2010; Evans, Blair, Greenhalgh, Hopwood, & Howell, 1994; Smerecnik, Mesters, Verweij, de Vries, & de Vries, 2009). An individual’s genotyping results may be viewed through the same set of essentialist biases that distorts the way people understand genetic causes more generally.



2. THE IMPACT OF GENETIC ATTRIBUTIONS ON PEOPLE’S PERCEPTIONS

The notion that people have genetic essentialist biases presupposes that genetic arguments have undue influence on people’s perceptions. As we discussed earlier, participants responded quite differently to an article arguing for a genetic cause of obesity compared with an article arguing for an experiential cause—it was only the genetic article that impacted people’s eating behavior relative to a control group. This biased reaction to genetic information is not limited to perceptions of obesity—as we’ll show, genetic attributions affect people’s perceptions across a broad array of different domains. Curiously, the domains which have shown biased responses to genetic information cut across topics that tend to be quite politically contentious. Frequently, essentialist thinking is bound up with intolerance, such as racism and sexism. Yet on other topics, essentialist thinking is seemingly paired with increased tolerance, as in the case of gay rights and criminal responsibility. In the following section, we’ll consider the evidence for how genetic attributions affect people’s perspectives on a variety of topics.

2.1 Sex and Gender

“It’s a girl!” These three words often signal a delightful end of the strenuous efforts to bring a new person into the world. The beaming, exhausted parents are now armed with an answer to the first question they are likely to encounter upon delivering the great news about the expanding family. Although the appropriate call should have been “it’s a female” denoting the sex of the newborn, given that sex, rather than gender, denotes the biological differences captured by the visible indicators and their underlying chromosomes, research may reveal that the reason for this ongoing error may be our tendency to see gender as the most essentialized social category (e.g., Haslam et al., 2000); that is, gender is viewed as denoting group differences that are the most natural, discrete, and immutable of all social categories (in contrast to categories such as race, ethnicity, sexual orientation, SES) and therefore may be easily swapped with the term sex.

However, gender, the term that captures normative perspectives of femininity and masculinity, is highly culture specific. The host of roles, responsibilities, and behavior that capture appropriate ways to be a man or woman substantially differs from one place to another. For example, whereas the majority of people in gender-egalitarian countries view career importance

and sexual freedom of men and women as equal, the majority of men and women in gender-stratified cultures do not (e.g., Williams & Best, 1990). Such malleability of gender perceptions has led some to completely reject biological explanations for gender differences beyond genitalia (e.g., Fausto-Sterling, 1985); however, a complete rejection of the role of nature in forming gender-typical preferences and behavior has proven to be inadequate when empirically applied to the real world. For example, instances of involuntary sex-reassignment procedures that have been conducted on infants in error (e.g., circumcision complications) have consistently demonstrated that while genitalia can be easily altered, gender development is most likely to follow natal sex rather than the reassigned one (e.g., Diamond & Sigmundson, 1997; Reiner & Gearhart, 2004).

Natal sex, however, is not a deterministic predictor of a person's gender either. Discarding both the chromosomal indicator and socialization pressures, transgender individuals, some not much older than toddlers, adopt alternative gender identities. The willingness of such individuals to incur substantial social costs (e.g., Norton & Herek, 2013; Stotzer, 2009) in the process indicates just how psychologically painful it can be to be assigned a gender identity at odds with how one identifies oneself. As Stotzer (2009) asserted, transgender people's very existence is repelled by "a society that is unforgiving of any system of gender that is not binary" (p. 170). Such a reaction is not surprising when one considers that their presence pulls the rug from underneath this most essentialized category.

Perceiving clear boundaries between men and women—boundaries that transgender people's existence violates—allows for clearer assignments of gender roles, a priori suppositions about skills and limitations, and other forms of stereotypical thinking. As such, the GEF predicts that the tendency to view ingroup members as more homogeneous, on the one hand, and increasingly distinct from outgroup members, on the other hand, should be exacerbated when genetic attributions are involved; thus, one would expect that endorsing genetic attributions for gender differences are associated with indicators of stereotyping and prejudice. And indeed, the more a person believes that genes determine a host of behavioral and psychological phenomena, the higher they score on an inventory of sexism (Keller, 2005). Moreover, increased endorsement of biological over socialization explanations for gender is associated with greater self-identification with stereotypically gendered traits (Coleman & Hong, 2008), in line with increased perceptions of the homogeneity of one's own group.

The correlational nature of the above research limits causal inferences, because such findings may be interpreted as indicative of dispositional elements that give rise to both preferences for genetic explanations and endorsement of stereotypes (e.g., conservatism). Thus, experimental designs are needed to explore whether exposure to genetic attributions affects gender-related attitudes and behaviors. Utilizing this kind of experimental approach, Dar-Nimrod and Heine (2006) examined the effects of offering genetic causal explanations for alleged sex differences in math aptitude on women's math performance. In two studies, women were provided with either genetic or experiential explanations for a purported superiority in math performance among men; other women were led to believe that there are no such gender differences. Results indicated that women who were exposed to a genetic explanation for the purported gender disparity performed worse compared to those in the experimental or no difference conditions. Women who were given an experiential explanation for the alleged sex difference, on the other hand, did not show diminished performance, performing on par with the women who learned that men and women do not differ in math performance (for similar findings on a different trait, see Moè & Pazzaglia, 2010).

2.2 Sexual Orientation

Gender may be the most essentialized social category, but it is far from being the only one. With a prominent (Western) societal shift from pathologizing and criminalizing same sex attraction toward its reception as an acceptable form of sexual desire, recent decades have seen monumental changes in judgment of nonheterosexual preferences and behavior. That said, painful reminders of rejection of nonheterosexual preferences are still around us. Whether such rejection comes in a form of ongoing criminalization of homosexual behavior as is the case in dozens of countries around the world, or through hateful rhetoric as in the case of the Westboro Baptist Church, or outright violence and carnage as in the murderous rampage at an Orlando gay nightclub in 2016, the prominence of sexual orientation as an essence-bearing marker remains. As part of this prominence, much of the discussion (and some suggest the increased acceptance of same sex attraction) revolves around the *perceived origin* of one's sexual orientation; whereas some endorse the idea that sexual orientation is the product of personal choice or upbringing, others favor genetic explanations (e.g., Jayaratne et al., 2006).

Passionate scientific and popular debates rage around the causal question of sexual orientation. Whereas sex is chromosomally indicated, research has not discovered any genes that predict sexual orientation to a meaningful degree. The closest scientists have come to discovering such evidence came about in the early 1990s. Variations in a specific genetic marker (encompassing multiple genes) on the subtelomeric region of the long arm of the sex chromosome, Xq28, were implicated in influencing at least one subtype of male homosexuality (Hamer, Hu, Magnuson, Hu, & Pattatucci, 1993); however, independent replication efforts have provided conflicting results (Ramagopalan, Dymont, Handunnetthi, Rice, & Ebers, 2010; Rice, Anderson, Risch, & Ebers, 1999; Sanders et al., 2014). Moreover, when it comes to identifying specific genetic underpinning for female homosexuality, there is, as of yet, no reputable relevant evidence. Despite the inability of molecular biology to identify a specific gene as a conclusive indicator of sexual orientation, the term “gay gene” has been featured prominently in media coverage, as indicated, for example, by the hundreds of thousands of search results following a simple Google search. Molecular biology aside, a key controversy underlies debates on the causal determinants of sexual orientation: is sexual attraction inborn or does it arise from socialization experiences and/or personal choice? Although a full account of the scientific research and discourse about this question is beyond the scope of this chapter, there is much evidence for a significant heritable component for sexual orientation (e.g., Bailey & Bell, 1993; Bailey, Dunne, & Martin, 2000).

Regardless of the evidence for the role of genes in sexual orientation, this chapter is concerned with people’s etiological perceptions and their outcomes. One desirable feature of believing that genes underlie sexual orientation is that this belief is associated with more accepting attitudes toward LGB individuals than is the belief that it is socially determined or chosen (e.g., Haslam & Levy, 2007; Jayaratne et al., 2006). This seems to suggest that adopting genetic explanations for sexual orientation decreases negative attitudes toward LGB individuals. Indeed, some experimental research also supports a causal interpretation of these correlations, indicating that manipulating perceived etiology by emphasizing genetic explanations results in increased support for LGB causes (e.g., Falomir Pichastor & Mugny, 2009, Study 5; Frias-Navarro, Monterde-i-Bort, Pascual-Soler, & Badenes-Ribera, 2015). However, some key moderators for the effect of genetic attributions on LGB support have also been identified, such as college major (Oldham & Kasser, 1999), premanipulation attitudes (Boysen &

Vogel, 2007), or the nature of one’s opposition to equal rights (Frias-Navarro et al., 2015), suggesting that manipulation of etiological attributions does not necessarily lead to increased tolerance. Taken together, growing data indicate that the association between positive attitudes toward LGB individuals/causes and endorsement of genetic attributions for sexual orientation may, at least for some, be an outcome of motivated cognition; that is, ideology may play a substantial role in explaining these data. This suggestion is supported by research which demonstrated that whereas ideological conservatives tend to endorse genetic rather than social explanations for racial and class-related differences (e.g., intelligence, drive, violence) more than liberals, the opposite is true for sexual orientation (Suhay & Jayaratne, 2012). Thus, people may also use etiological explanations for group differences strategically rather than blindly apply broad theories about the role of genes in explaining human behavior.

The somewhat mixed pattern of causal effects of genetic attributions on attitudes toward LGB individuals and/or causes may also arise from the tension between different genetic essentialist biases. On the one hand, in line with attribution theory (Weiner, Perry, & Magnusson, 1988) and empirical evidence (e.g., Cheung & Heine, 2015; Dar-Nimrod et al., 2011), the immutability bias reduces perception of culpability; thus among those who view homosexuality as negative, endorsing relevant genetic explanations should lead them to assign less blame and harbor less negative attitudes. Similarly, a natural bias can further lead people to assume that homosexuality is more natural if it has a genetic basis, which would lead to more tolerant attitudes. On the other hand, the discreteness bias increases perceptions of distinctness between a heterosexual individual and a homosexual outgroup member, which is likely to lead to more negative attitudes. Such opposing pulls from the different biases are supported by research that indicated that whereas perceptions of increased immutability were predictive of reduced antigay attitudes, perceptions of discreteness of male homosexuality were a positive predictor of antigay attitudes (Haslam & Levy, 2007).

Whereas much research was conducted on the relations between essentialist beliefs and attitudes toward LGB individuals among heterosexual individuals, only recently has research begun to examine the nature of such relationships among LGB individuals themselves. Some scholars (e.g., LeVay, 1996) predicted that LGB individuals’ endorsement of genetic explanations for homosexuality will result in positive consequences; however, empirical research delivers a more nuanced picture. Recent studies do suggest that essentialist beliefs about sexual orientation have implications among

LGB individuals as well. Just as heterosexual individuals show clashing effects among their essentialist biases and attitudes toward homosexuality, the different aspects of essentialism also conflict among LGB individuals (Morandini, Blaszczyński, Costa, & Dar-Nimrod, 2016; Morandini, Blaszczyński, Ross, Costa, & Dar-Nimrod, 2015). For example, those LGB individuals who view sexual orientation as biologically based or immutable tend to experience less sexual orientation uncertainty (which positively predicts well-being). On the other hand, gay men and bisexual women who perceive sexual orientation as existing in discrete typologies report more internalized antigay/bisexual attitudes (Morandini et al., 2016, 2015). Among gay men, this may be because discreteness beliefs sharpen the distinction between straight vs gay identified individuals, leading to increased self-stereotyping and increased feelings of marginalization or otherness. In contrast, bisexual women, who have flexible patterns of sexual attraction and therefore more amorphous sexual attraction boundaries, do not show this effect. There is also evidence that sexual orientation beliefs are connected to an individual's experience of sexual orientation. Lesbian women who report being exclusively same sex attracted are more likely to view sexual orientation as biologically determined, immutable, and discrete, than lesbian women who are nonexclusively attracted to women (Morandini et al., 2016). As empirical research on this topic is just emerging, it is still unclear whether sexual orientation beliefs are formed by reflecting on the nature of one's own sexuality or adopting perceived LGB community or broader societal zeitgeists. Regardless, these findings suggest that essentialist beliefs may be utilized to satisfy personalized epistemic needs, as suggested by Cheung, Dar-Nimrod, and Gonsalkorale (2014).

2.3 Health

A key impetus underlying the scientific quest to identify genes has been to improve people's health. An example of this quest can be seen in the sequence of three DNA bases, cytosine–adenine–guanine (CAG), which gets translated into the amino acid glutamine. Located along the short arm of chromosome 4, the wild-type (normal) form of the HTT gene contains between 6 and 35 such CAG repeats. However, among about 50–100 individuals of every million people, the number of these CAG repeats is greater than 40. For those individuals, the protein that the HTT gene produces leads to devastating outcomes; unless these individuals die at an early age, they will develop a ruinous disease, Huntington's disease, that will

ravage their cognitive capabilities, personality, and physicality, ultimately leading to their untimely death (e.g., Andrew et al., 1993). The number of CAG repeats does not only foretell whether one will or will not develop Huntington's, it also predicts, with remarkable precision, the age of onset of visible symptoms and, by extension, longevity (Walker, 2007).

The deterministic nature of the effects of the HTT gene shares many features with lay conceptualizations of destiny, but these kinds of fully penetrant monogenic disease-risk variants represent a small minority of humanity's disease burden. That said, many of the more common causes of death such as heart disease, diabetes, or various forms of cancer are directly influenced by genetics and/or are indirectly affected by genetic influences on relevant health behaviors (such as smoking or caloric intake). The influence of one's genes on health has been widely acknowledged for decades (e.g., Herzlich, 1973), and evidence of such common attributions also emerges in response to questions about specific conditions/illnesses such as alcoholism (e.g., Keller, 2005), obesity (e.g., Dar-Nimrod, Cheung, et al., 2014), cancer (e.g., de Vries, Mesters, Van de Steeg, & Honing, 2005), or mental illness (e.g., Schomerus et al., 2012), to name a few. Although such genetic associations for common diseases do indeed exist, they can best be understood as exemplars of weak genetic explanation, in that they are the product of many, many genes interacting with each other and with numerous environmental and psychosocial influences. Notwithstanding such complex networks of interacting etiologies, our essentialist biases make us prone to understanding disease risk in more deterministic ways. This raises the question of how people understand health outcomes if they are made aware of relevant genetic underpinnings. Specifically, how do genetic attributions affect the ways that people: (a) form attitudes toward those afflicted with particular conditions; (b) assess risk and personal control; and (c) ultimately behave?

Consider the role of genetic attributions in how people make sense of mental illnesses and those afflicted by them. Much evidence supports a substantial heritable component for all common psychopathologies, but actual determinist links between specific genetic variants and mental health conditions are limited to rare syndromes (WHO, 2001). Despite the general lack of substantial genetic risk predictors for mental health outcomes, much evidence suggests that people are affected by considering the role of genes in mental illness. One source of evidence for the ways that people understand genetic causes of mental illnesses can be seen in efforts by various advocacy groups to reduce mental health stigma. Such advocacy groups often

emphasize genetic and biological causes of common psychopathologies to reinforce their agenda, hoping that offering biological explanations will reduce stigma (e.g., Phelan, Cruz-Rojas, & Reiff, 2002). And indeed, research shows that people are less likely to blame individuals for their unwarranted symptomatic behaviors in the face of biological/genetic explanations (e.g., Phelan, 2005; Phelan et al., 2002). For schizophrenia, the most studied illness in this context, a metaanalysis indicates that biological/genetic explanations reliably predict less stigma and blame (Kvaale, Gottdiener, & Haslam, 2013). However, a closer look at the metaanalysis on the relations between these constructs suggests evidence for the double-edged sword of genetic essentialism. That is, although biological/genetic explanations may reduce blame, they are also associated with a modest increase in the perceived dangerousness of patients and a desire for greater social distance from the sufferers (Kvaale et al., 2013). On the one hand, the immutability bias implies reduced control by the actor and thus reduced blame, and the natural bias suggests that the illness is more acceptable and less stigmatizing. On the other hand, the discreteness bias highlights how the afflicted individuals are fundamentally different from others, making them a target for discrimination. Moreover, the sense of reduced control that comes with the immutability bias suggests that the individual may be unable to prevent themselves from engaging in behaviors that are dangerous to others. In addition, the specific etiology bias also raises the specter that the individual will never be free of their condition, thus leading to more pessimistic prognoses (Kvaale et al., 2013; Lebowitz, Ahn, & Nolen-Hoeksema, 2013). Thus, endorsing genetic explanations for mental illnesses yield decidedly mixed effects for how people view those with psychopathologies (Kvaale et al., 2013; Lebowitz & Ahn, 2014; Phelan et al., 2002). Although psychopathology is the most prominent topic in which perceptions of undesirable health-related symptoms as an outcome of biological/genetic explanations have been studied, similar essentialist tensions have been found in research on prenatal genetic testing (e.g., Blumberg, 1994; Kelly, 2009), smoking (e.g., Dar-Nimrod, Zuckerman, & Duberstein, 2014; Tercyak, Peshkin, Wine, & Walker, 2006), alcoholism (Dar-Nimrod et al., 2013), memory loss (Lineweaver, Bondi, Galasko, & Salmon, 2014), and overeating (Monterosso, Royzman, & Schwartz, 2005).

Genetic essentialist biases are implicated in a wide array of health behaviors, but they are not limited to questions of *whether* a relevant behavior occurs (e.g., abstaining from unhealthy eating, performing well on a memory test); they may also affect *which* of several competing behavioral options

is chosen as a goal. If an unhealthy behavior is seen as “genetic,” it is often presumed that biomedical interventions targeting genes would be appropriate to modify the behavior, while lifestyle interventions would not, in line with the specific etiology essentialist bias. Empirical evidence demonstrates that exposure to genetic attributions indeed increases preferences for physiological interventions to mitigate unhealthy behaviors or improve disease outcomes (e.g., Lebowitz & Ahn, 2014; Phelan, Yang, & Cruz-Rojas, 2006). For example, Wright, Weinman, and Marteau (2003) asked smokers to imagine being tested for genetic susceptibility to nicotine dependence. Half of the participants were told to imagine receiving a positive indication for genetic susceptibility and half were told to imagine receiving a negative indication. Respondents in the positive group were more likely to endorse medication as an effective means of smoking cessation than those in the negative group. In addition, smokers in the positive group were more likely to say they believed willpower to be less effective as a method of smoking cessation, demonstrating a potentially damaging effect of the specific etiology bias.

2.4 Race and Ancestry

Research on population genetics has provided evidence for the intersection of two rather obvious and widely shared intuitions: (1) people inherit traits (and genes) from their biological parents and (2) people’s mating preferences are not determined randomly. Although there are several factors that guide the way that people choose their mates, one particularly powerful factor rarely gets discussed, perhaps because it seems to go without saying. The *propinquity effect* refers to the fact that people are far more likely to form relationships with people who are geographically near than with those who are geographically distant (Festinger, Schachter, & Back, 1950), and this also holds true for mating. A key genetic consequence of this effect is that genetic variants tend to cluster geographically. This clustering occurs for two reasons. First, selection pressures can leave adaptive alleles more common in certain regions, such as how an allele of gene SLC24A5, which is associated with lighter skin color, became more common in areas of higher latitude, as it was adaptive for people in those regions to absorb more ultraviolet radiation to catalyze the production of vitamin D (Lamason et al., 2005). Second, random genetic drift will lead some neutral alleles to proliferate in certain areas because those who had more offspring in a particular region will have their own genetic variants become relatively more common in

subsequent generations. For example, the population of Norfolk Island has a number of genetic risk factors for cardiovascular disease that are more common than average because they descend from a small group of individuals who must have carried the same mutations (Bellis et al., 2005). Natural selection and genetic drift result in the frequencies of different alleles varying significantly around the world.

The uneven distribution of alleles across the globe makes it possible to identify the geographic origins of one's ancestors to a fair degree of precision by examining their genomes. For example, one study investigated a few thousand Europeans and was able to predict the location, within 310 km of precision, of the birthplace of approximately 50% of the participants by only examining the single-nucleotide polymorphisms of their autosomal DNA (Novembre et al., 2008). More pertinent to the theme of this chapter is the question of whether learning about the geographic distribution of genes affects the way that people understand themselves and others. There are numerous anecdotal accounts of the ways that people have experienced changes in their identity as a result of encountering unexpected genomic ancestry information, which has now been provided to over 3 million people through DTC genomics companies (Petroni, 2015). As an example of the effects of genes on identity, consider the case of Csanád Szegedi; he was elected to the European Parliament as a leading member of the anti-Semitic Jobbik party in 2009. However, upon unexpectedly learning that his maternal grandmother was Jewish, he became an Orthodox Jew in 2013 (Applebaum, 2013).

Although the kind of abrupt transformation exhibited by Szegedi is certainly unusual, many people do seem to experience a change in identity upon receiving unexpected information from their genomes. Roth and Lyon (in press) contacted more than 600 individuals who received genomic ancestry information from DTC companies. Among those who responded, only 26.1% reported that the test results had no impact on how they identify with their race or ethnicity, their identity more generally, or their activities or friendships. The vast majority claimed that the tests had an impact on some aspect of their lives. For example, upon receiving their genetic feedback some people began learning an ancestral language, chose new ethnic categories on the census, cheered for new teams in the World Cup, made new groups of friends, joined native tribes, and began to think of their identities differently. It should be noted that the ancestry information provided by DTC companies suffers from overpromising, is often full of errors, and is

currently not subject to any oversight or regulation (Bolnick et al., 2007; Heine, 2017; Royal et al., 2010).

Setting aside the question of how learning about the geographic ancestry of one's *own* genes affects an individual, one may also examine how learning about the geographic distribution of genes in general affects people. Reflecting on how genetic variants are distributed unequally across the globe would seem to resonate with the essentialist bias of conceiving of essences as carving nature at its joints. It highlights that humanity does not share a uniform genome, but that people from different regions of the globe have some identifiable genetic differences. Considering the genetic boundaries that partition the world may therefore lead people to conclude that people of different ethnicities have distinctive essences. And because a key component of ethnic prejudice is that it's founded on the sense that outgroups are of a different essence than ingroups (Allport, 1954; Yzerbyt, Judd, & Corneille, 2004), this raises the possibility that reflecting on the geographic distribution of genes may exacerbate feelings of prejudice.

There is considerable evidence for this effect. In a study by Keller (2005), German students were asked to read an essay that described how geographic ancestry can be revealed by one's genome or a control essay on an unrelated topic. Later, participants were asked questions about expanding the European Union and were asked to indicate their feelings toward people from various Western European countries and Eastern European countries. The results indicated that those who read about the geographic distribution of genes showed a larger ingroup bias in preferring Western Europeans over Eastern Europeans compared with those who read the control essay. Likewise, people who were exposed to arguments that the human population's genome varied significantly evaluated ingroup and outgroup faces in a more dichotomous way compared with those who read that human genetic variation is minimal (Plaks, Malahy, Sedlins, & Shoda, 2012; see also Kang, Plaks, & Remedios, 2015). Moreover, Kimel, Huesmann, Kunst, and Halperin (2016) compared how American Jewish participants responded to an essay outlining how Jews and Arabs were highly genetically similar in contrast to those who read either an essay arguing that Jews and Arabs were genetically distinct or a control essay. Those who read about the genetic similarities between Jews and Arabs were more in support of peace-making efforts in the Middle East than those who read either of the other two essays. Given that the human genome is remarkably homogenous in contrast to many other species (Templeton, 2013)—for example, whereas

genetic variation associated with the continental races accounts for only about 4.3% of human genetic variability (Rosenberg et al., 2002), the different “races” of chimpanzees account for 30.1% of chimpanzee genetic variability (Gonder et al., 2011)—perhaps getting this message out will lead to less ethnic prejudice more generally.

The genetic essentialist bias of seeing populations with different genes as being more discrete from each other can also interact with our other essentialist bias of seeing genetic causes as being of a specific etiology. That is, upon seeing phenotypic variation between human populations, people may conclude, erroneously, that genotypic differences between the populations must account for this. For example, this was a key argument of the controversial book, *The Bell Curve*, in noting that because African-Americans and European-Americans performed differently on some kinds of intellectual tasks, the difference in performance could be explained by imagined genes that distinguished these two populations (Herrnstein & Murray, 1994). Given how this notion resonates so well with our essentialist biases, it’s worth noting that just because genes may account for individual variability in a trait, this says nothing about whether genes underlie between group variability in the same trait (for thoughtful discussion of this issue, see Nisbett, 2009).

The degree to which people see population differences, such as those described in *The Bell Curve*, as having a genetic basis is something that we recently investigated (Schmalor, Cheung, & Heine, 2016). American MTurk participants were randomly assigned to read an essay either about a geographic distribution of genes, a description of human genetic homogeneity, or a control topic. They were presented with a list of ethnic stereotypes that covered a wide range of desirability (e.g., French have a more sophisticated palate; Africans have a better sense of rhythm; Asians are worse drivers). Participants were asked how accurate and how offensive they found the stereotypes, and then they were asked to indicate what percent of the population variability could be attributed to genes or experiences. On average, people viewed approximately 35% of the variability underlying these stereotypes as genetic. However, those who read about the geographic distribution of genes estimated that genes accounted for approximately 5% more of the population variability in comparison with the other two groups. That is, when people considered the argument that people vary in their genes around the world, they viewed genes to be a more significant component underlying ethnic stereotypes compared with those who did not encounter this argument.

This tendency toward seeing population differences on complex traits as an outcome of underlying genetic variability speaks to the controversy over what entails race. Is race something that is largely socially constructed, or is it grounded in biology? Although it is tempting to see race as biologically grounded, given the visible differences across the continental races, there is a strong consensus among both biologists and social scientists that race is something that is socially constructed rather than biologically based (Bliss, 2012; Boas, 1940; Cavalli-Sforza & Feldman, 2003; Hunley, Healy, & Long, 2009; Lewontin, 1972; Yudell, Roberts, DeSalle, & Tishkoff, 2016). Biologists maintain a set of criteria for identifying whether a population can be considered a subspecies (the nonhuman equivalent of race), and human races do not come close to meeting any of these criteria (Templeton, 2013). Moreover, we can see evidence of the social basis of race when we consider the various ways that race is defined. For example, people of disadvantaged races are often subject to the notion of hypodescent, where those of the lower-ranked ethnic groups are seen to have a contaminating influence. An extreme example of this was the “one drop rule,” which made the case that people would be deemed to be Black if they had any African blood. Evidence for hypodescent can still be found today; if one morphs photos of White and Black faces, people tend to identify the faces as Black even if the percentage of the contribution from the morphed Black photo is considerably less than 50% (Ho, Sidanius, Levin, & Banaji, 2011). Hypodescent reflects that our judgments as to what counts as being a member of the Black race is not proportionately based on genes. Regardless of whether human races really are socially constructed, however, more relevant to this chapter is the question of how people respond when they encounter an argument that race is grounded in one’s biology vs social conventions.

Williams and Eberhardt (2008) investigated this question by examining how White American participants would respond to a video of a Black target who was discussing how he had been laid off from his work. Prior to seeing the video, some participants had read an essay highlighting how race was a biological construct, whereas others read that race is a social construct. Those who read about the biological argument for race had more prejudiced attitudes toward a Black target than those who had read the social constructionist argument. Likewise, in another investigation, Asian-Americans who read an essay arguing for a biological account of race were found to identify less with American culture than those who read an essay arguing for a social basis of race (No et al., 2008; also see Chao, Chen, Roisman, & Hong, 2007).

A perceived biological basis of race makes one's ethnic identity appear fixed and responsible for characteristics of one's ethnicity.

2.5 Criminality

Academic inquiries into the biological bases of criminal behavior have long existed, most notably involving Lombroso's (1876/2007) ideas about the physiognomy of criminality and his thesis of the "born criminal." Although most of Lombroso's ideas have not survived to the present, the underlying motivation of seeking a biological explanation for criminality persists. This is apparent in the appeal of the XYY chromosome defense that appeared in the 1960s, whereby people believed that having an extra Y-chromosome predisposed men to have lower intelligence and greater proneness toward violence, despite a striking lack of evidence (Slabbert, 2006). More recently, and more germane to the central theme of this chapter, results from the Human Genome Project and advances in behavioral genetics have tantalized the public and researchers with the possibility that criminal behavior may be distilled down to one's genetic makeup (Friedland, 1998). Whereas criminality is most certainly a product of a complex interplay of both genetic and nongenetic factors (Alper, 1998), the scientific community has made great strides toward identifying various genetic markers that correlate with criminal or aggressive behavior (for a summary, see Beaver, Schwartz, & Gajos, 2015).

The fact that researchers are discovering genetic components of criminal behavior is helpful for our understanding of human behavior and for the accumulation of scientific knowledge. However, an important issue that these findings pose for the legal community is how members of the criminal justice system, both laypeople and experts, interpret and make use of such findings. One important tenet of the criminal justice system is that it assumes that people act out of free will, and that criminal deeds are the product of one's willful intent to act in contravention to the law. This is encapsulated in the core concept of *mens rea*, or "guilty mind," referring to one's mental state and volitional control during the commission of the crime (Aharoni, Funk, Sinnott-Armstrong, & Gazzaniga, 2008). The immutability bias, however, undermines this inference, which may engender the perception that "criminal genes" inevitably lead to criminal behavior, ultimately prompting mitigated judicial outcomes. The recognition of such biases has led to an ongoing debate among legal scholars regarding the place that genetic evidence has within the courtroom, particularly given that individual genes can

generally only account for a very small proportion of the variability for a given phenotype (Baum, 2013; Berryessa & Cho, 2013; Chabris et al., 2015).

Emerging evidence over the last decade has shown that genetic explanations of criminality lead members of the criminal justice system to think in ways consistent with genetic essentialist biases, in contrast to nongenetic, particularly environmental, explanations of criminality. In terms of impacting perceptions of a criminal, genetic explanations lead people to attribute less behavioral control to the perpetrator and less willful intention in terms of committing various crimes, relative to environmental explanations, reflecting the genetic essentialist bias of immutability (Cheung & Heine, 2015; Dar-Nimrod et al., 2011). Furthermore, people feel that such explanations of criminality are more persuasive and acceptable as excuses in criminal cases, compared to more experiential explanations such as exposure to violence or parental abuse (Heath, Stone, Darley, & Grannemann, 2003), leading them to perceive lower levels of overall criminal culpability (see also Monterosso et al., 2005). As a result, genetic explanations of criminal behavior lead people to be more accepting of insanity and diminished capacity defenses and to prescribe shorter prison sentences (Cheung & Heine, 2015), in accordance with the fact that such explanations appear to mitigate many concepts that are relevant for *mens rea*, including perceived control and intention (Dar-Nimrod et al., 2011). This finding generalizes even to state trial judges and Superior Court Judges (Aspinwall, Brown, & Tabery, 2012; Berryessa, 2016).

As noted in previous sections, the same genetic essentialist biases that underlie the mitigating perceptions resulting from genetic explanations for criminal behavior may also lead to aggravating perceptions. For instance, expecting a deterministic relation between genetic causes and criminal behavior leads people to engage in more stable causal attributions and expect a greater likelihood of recidivism (Cheung & Heine, 2015). This creates an enhanced perception that the criminal is dangerous, engendering greater levels of fear toward the criminal (Appelbaum & Scurich, 2014). Importantly, both the mitigating and aggravating perceptions of the criminal factor into people's prescribed prison sentences—in opposite directions. In other words, mitigating perceptions (e.g., less ascriptions of behavioral control) predict shorter prison sentences, whereas aggravating perceptions (e.g., greater expectations of recidivism) predict lengthier prison sentences (Cheung & Heine, 2015). These responses suggest that people's objectives for punishment may be influenced by the kinds of explanations that they

encounter. Genetic explanations trigger people's concerns about incapacitation and protection of the public as an objective for punishment, rather than deterrence, rehabilitation, or just deserts (Carlsmith, Darley, & Robinson, 2002). Overall, the extant data suggest that people have a double-edged perception of genetic causes for criminality, similar to their effects on evaluations of individuals with a psychopathology, leading to both mitigating and aggravating perceptions.

2.6 Political Orientation

From its inception, political psychology has focused on individual differences associated with variations in political affiliation. In the wake of WWII, these efforts took on greater urgency as psychologists turned to the emerging field of personality research to determine what, if any, core psychological mechanisms underlie adherence to fascist ideology, and extreme right-wing beliefs more generally. The first of these "political personality" constructs was outlined in *The Authoritarian Personality* (Adorno, Frenkel-Brunswick, Levinson, & Nevitt Sanford, 1950), which featured proposed dimensions of right-wing ideology that would be reified over the course of the century: conventionalism and antiintellectualism, which represent *resistance to social change*, and submission to authority, with a corresponding *preference for social hierarchy* (also see *Right-Wing Authoritarianism*, Altemeyer, 1981; and Social Dominance Orientation, Pratto, Sidanius, Stallworth, & Malle, 1994).

Many psychologists have intuited that fundamental biological differences undergird these personality constructs, which in turn are influenced by genetic factors (e.g., "Politics might not be in our souls, but it probably is in our DNA"; Hibbing, Smith, & Alford, 2014, p. 298). Initial evidence for this genetic influence came from twin studies suggesting a moderate degree of heritability (Martin et al., 1986; Olson et al., 2001). Using more powerful statistical techniques, subsequent research has supported the contention that social value adherence is partly heritable, along with the degree to which these values are maintained, even if the same cannot be said for specific political party affiliation (Alford, Funk, & Hibbing, 2005; Hatemi, Alford, Hibbing, Martin, & Eaves, 2008).

In recent years, these apparent links have spurred efforts to specify shared genetic markers underlying a possible conservative phenotype (Deppe, Stoltenberg, Smith, & Hibbing, 2013; Fowler & Dawes, 2008). In particular, these efforts have focused on the potential genetic underpinnings of a purported "negativity bias" demonstrated by ideological conservatives

(e.g., Hibbing et al., 2014, p. 22; but note a critical review of this argument, Charney & English, 2012). From this general perspective, political conservatism is itself seen as a genetically evolved response to threatening or uncertainty arousing stimuli (e.g., Jost, Glaser, Kruglanski, & Sulloway, 2003), which plays a role in both dimensions of conservatism as it is classically defined. First, a *resistance to social change*—and change, more generally—has been claimed to be the product of a relatively diminished neural response to error feedback that would motivate behavioral change (Jost & Amodio, 2012) caused by relatively decreased brain mass in areas associated with cognitive conflict detection (e.g., anterior cingulate cortex, Kanai, Feilden, Firth, & Rees, 2011). Second, a *preference for social hierarchy* has been argued to follow from the relative proclivity of conservatives to attend more (e.g., Dodd et al., 2012) and respond more strongly (Oxley et al., 2008) to aversive stimuli, and this bias has been implicated in the heightened derogation of perceived social outgroups (e.g., heightened disgust and attitudes toward gay men; Inbar, Pizarro, & Bloom, 2009).

The reporting of this research has been widespread, with a particular emphasis on public dissemination of findings that suggest a genetically determined biological origin to political affiliation (e.g., Hibbing, Smith, & Alford, 2013). As we have discussed, attributing genetic origins to criminality (e.g., Cheung & Heine, 2015) and sexual orientation (e.g., Haider-Markel & Joslyn, 2008) may indeed increase tolerance for behaviors that are otherwise stigmatized, as these behaviors are seen as immutable and natural. Similarly, Hibbing et al. (2014) emphasize that the genetic basis of political differences should lead to increased understanding and acceptance. However, recall the tension we have identified in previous sections between the immutability and naturalness biases on the one hand, and the discreteness bias on the other hand. This latter bias suggests that the manner in which genetic attributions are portrayed in the "political genetics" literature could facilitate intolerance of political outgroups by highlighting how those with conflicting political views are ultimately made up of different underlying essences.

Political affiliations are often portrayed in a manner that emphasizes and exaggerates the extent to which they are bounded, immutable, genetically determined categories. "Conservatives" and "liberals" are described with discrete nouns, and as manifestations of homogenous natural kinds that ultimately originate "in our DNA" (Hibbing et al., 2014, p. 298). In reality, however, ideological commitments vary continuously across whatever dimensions they happen to be operationalized by, and are, at

best, marginally related to contemporary measures of genetic influence (Charney & English, 2012; Hibbing et al., 2013). Regardless, the commonly misapplied genetic attributions may motivate individuals to perceive those who predominantly identify with an opposing ideology in a manner that would discourage efforts to seek common ground, as they are understood to possess a fundamentally divergent essence. Similarly, efforts toward meaningful engagement with perceived political outgroups may seem pointless to the extent that ideologies are understood as immutable natural categories that resist social influence as they spring from a specific etiology.

Relative to liberals, genetic attributions may especially decrease tolerance toward conservatives, as the natural origin of conservatism is sometimes depicted as an atavistic response to disgust and fear (e.g., Jost et al., 2003), shaped by diminished brain structures associated with higher cognitive function, and exaggerated brain structures associated with threat detection (e.g., Kanai et al., 2011). In this depiction, the genetic pathways underlying conservatism were selected for during the Pleistocene epoch (Pinker, 2012), when primitive man faced constant mortal threat, in contrast to the present day. As such, conservative genetic markers may be regarded as an evolutionary dead end, insofar as “strong negativity biases were once selected for but now are not” (Hibbing et al., 2014, p. 32)—a depiction that resonates with eugenic beliefs, as we discuss in the next section.

Despite the prevalence of this depiction in popular discourse (e.g., Mooney, 2012), there is relatively little research that assesses the extent to which genetic attributions for political affiliations decrease ideological tolerance or increase political discourse (Hibbing et al., 2014). In one recent study (Suhay, Brandt, & Proulx, in press), measures of genetic attributions for ideology (e.g., “A person’s political beliefs are determined by their genetics”) were inversely correlated to items that assessed tolerance of those with divergent political identities (e.g., “I often spend time with people who have political beliefs different from my own”) among both liberals and conservatives. Moreover, this heightened ideological discrimination was especially prevalent among political liberals, which may be due to the explicitly negative portrayal of conservative genetics. These perceptions may play a role in the generally negative (Duarte et al., 2015) and discriminatory (Inbar & Lammers, 2012) attitudes toward conservatives among ideologically liberal social scientists.

2.7 Essences and Eugenics

Considering a genetic foundation for human traits does not just affect how we think about those specific traits. It is also implicated in broader efforts to change those traits. Here we consider what is arguably the most pronounced cost of our genetic essentialist leanings—it can be associated with support for eugenic social policies.

While eugenics may be associated primarily with the horrors of Nazi Germany and the holocaust, this social philosophy had much broader support prior to WWII. Indeed, discussions in favor of eugenics were commonplace throughout the industrialized world in the early 20th century, as the logic that governed the breeding choices made by livestock breeders began to be applied to humans (for a review, see Kevles, 1995; Paul, 1995). Some efforts were termed positive eugenics, in the sense that those who were seen as possessing good genes were encouraged to spread their bounty to the next generation. For example, “Fitter family” contests were held in state fairs in the United States, where medals were given to those designated as “Grade A Individuals,” alongside the other prize-winning livestock (Kevles, 1992). But these efforts were soon overtaken by much wider scale programs of negative eugenics, where the goal was to prevent the unfit from breeding. In the United States, the popular descriptor for the unfit was “feeble-minded,” a catch-all term that included any kind of perceived defect in intelligence or moral character. Negative eugenics was championed by a variety of progressive organizations, such as the Sierra Club and Planned Parenthood, as both sought the goal of reducing the world’s population by preventing births among the unfit (Paul, 1995; Stern, 2005). The eugenics movement cut across the political spectrum and was championed both by those on the right, who sought to increase the relative proportion of their own kind, and by the left, who viewed it as a necessary pillar for the establishment of a social welfare state (Spektorowski & Ireni-Saban, 2013). Mandatory sterilization was legalized in 1927 in the United States, which led to the forceful sterilization of more than 60,000 Americans, disproportionately minorities and women (Stern, 2005). Similar mandatory sterilization programs emerged across the industrialized world, in such diverse places as Canada, Sweden, Japan, and much of Latin America (Broberg & Roll-Hansen, 1996; Robertson, 2001; Stepan, 1991). These efforts rendered the decision of who would have children to rest ultimately with the state.

Support for eugenics was embraced by leading Western politicians (e.g., Winston Churchill, Tommy Douglas, Teddy Roosevelt) and public

intellectuals at the time (e.g., Alexander Graham Bell, W. E. Dubois, George Bernard Shaw, H. G. Wells; Kevles, 1995; Paul, 1995). But two disciplines stand out in their support for eugenics: first, psychology played an outsized role in the movement (which has received relatively little attention within the field), as the metric of genetic quality that was most widely targeted was IQ, and many psychologists were active in promoting eugenic policies to improve national IQ, including Carl Brigham, James McKeen Cattell, Robert Fisher, G. Stanley Hall, Karl Pearson, Charles Spearman, Lewis Terman, Edward Thorndike, and Robert Yerkes (Heine, 2017). Second, and most pertinent to the logic of genetic essentialism, was that eugenic support was widespread at the time among geneticists. Prior to the war, there was scant light that separated the fledgling field of human genetics from eugenics—indeed, the latter was often thought of as applied genetics (Paul, 1995). Evidence for this link can be seen from a variety of sources. For example, the founder of behavioral genetics, Francis Galton, was also the father of modern eugenics (Galton, 1875, 1883). Likewise, in 1916, every member of the founding editorial board of the journal, *Genetics*, endorsed the eugenics movement (Ludmerer, 1972). The link between the two fields is also evident in that half of academic biologists in Germany joined the Nazi party prior to the war, which was the largest representation of any professional group (Paul, 1995).

One reason that there was such a link between the study of genetics and eugenics in the early 20th century was that many early geneticists favored simple Mendelian accounts of human traits. For example, Charles B. Davenport, the leading American eugenicist at this time, maintained that many human traits, included feeble mindedness, a love for the sea, nomadism, shiftlessness, and innate eroticism were the product of single genes (Comfort, 2012; Kevles, 1995). To the extent that human traits could be viewed as simple and direct consequences of single genes, it is far more straightforward to imagine efforts to change the frequency of desired traits through controlled breeding.

We have investigated whether similarly deterministic perceptions of genetic causes predict support for eugenic ideas today. We created a scale to measure support for eugenic policies (Heine, Cheung, & Ream, 2015). It includes items such as “There should be laws discouraging people with low intelligence from having biological children” and “Anyone convicted of a violent crime should be sterilized as part of their punishment.” We correlated this scale with the Belief in Genetic Determinism Scale (Keller, 2005) and the GETS (Dar-Nimrod et al., 2016). Support for

eugenic policies correlated moderately positively with these measures of genetic determinism/essentialism (Heine et al., 2015). Moreover, the link between eugenics and simple accounts of genetic understanding was further demonstrated in that we found negative correlations between eugenics support and knowledge of genetics (operationalized either in terms of the number of genetics classes taken, or in terms of performance on a genetics knowledge test; Ream, Cheung, & Heine, 2016). These findings reveal that those who are more likely to understand the irreducibly complex ways that genotypes get translated into phenotypes are less in support of efforts by the state to improve the collective genome through strategic breeding.

2.8 Genetic Engineering

Studying the predictors of support for eugenics may seem like an outdated question, akin, perhaps, to investigating present day support for slavery. But with the rapid advent of several new genetic technologies, the idea of improving the genes of current and future generations has reemerged as a topic of public discourse (e.g., Hudson & Scott, 2002; Winkelman, Missmer, Myers, & Ginsburg, 2015). However, in contrast to the early 20th century when the state was petitioned to lead efforts to enhance a nation’s collective genome, this time eugenics is emerging through the backdoor (Duster, 2003). There are presently a variety of genetic technologies that are available for parents to make their own reproductive decisions which can potentially shape posterity (see Heine, 2017, for a review). Some of these technologies allow for the genes of fetuses to be genotyped (e.g., amniocentesis, chorionic callus sampling, cell-free fetal DNA screening), and thereby providing the parents with information about potential congenital disorders which may lead them to decide to terminate a pregnancy. Another alternative is to genotype a series of fertilized embryos using pre-implantation genetic diagnosis, and then select to implant those in the womb that are not carriers of particular alleles associated with genetic diseases, or even to select the sex of one’s baby. Parents can also select from sperm and egg donors, while perusing remarkably detailed catalogs describing the donors’ phenotypes. If this was done on a large scale, it could shape the collective genome because people seek different traits when selecting gamete donors than they do when selecting romantic partners—when people select donors, they place relatively greater weight on their health, attractiveness, height, and various abilities, whereas when choosing romantic partners, people place relatively greater weight on character (Scheib, 1994;

Scheib, Kristiansen, & Wara, 1997). Moreover, there is much fear over novel technologies that have not yet been used to create a human, such as cloning, or creating designer babies using genome editing by way of CRISPR/Cas9. In contrast to the negative relation between genetic knowledge and support for eugenic policies, there is a weak positive relation between genetic knowledge and support for new reproductive technologies to enhance the genomes of future children (Ream et al., 2016). But overall, these technologies strike many as deeply problematic, with a commonly expressed concern that these entail people playing God (Calnan, Montaner, & Horne, 2005; Condit, 2010; Winkelman et al., 2015). Moreover, aside from screening fetuses for congenital disorders, the other reproductive technologies are not yet commonly practiced.

Another aspect of genetic engineering is far more common in our lives. GMOs now play a substantial role in our diets. Approximately 80% of American processed food contains at least some GMOs (Lemaux, 2008). Yet, despite the pervasiveness of this technology, it remains bothersome to many: approximately three quarters of Americans are concerned with having GMOs in the food supply (Harmon, 2014), and approximately 70% of GMO opponents view GMOs as absolute moral violations (Scott, Inbar, & Rozin, 2016)—that is, they are opposed to them regardless of any documented benefits or harms that they might entail. GMOs seem bothersome because they violate the essentialist bias of genes as natural. A frequent criticism is that GMOs are abominations, and that they represent people playing God (Condit, 2010). Likewise, GMOs run afoul of our essentialist biases as genes carving nature at its joints. For example, people are more bothered by GMOs that involve transgenic modifications (i.e., introducing genes from unrelated species, such as a tomato receiving a gene from a fish) than they are of those involving cisgenic modifications (i.e., introducing genes from a related species, such as an orange receiving a gene from a lemon; Gaskell et al., 2010).

In general, people have a rather poor understanding of what GMOs entail; only 57% of Americans and 36% of Europeans correctly recognized that non-GMO food products also contain genes (Hallman, Hebden, Aquino, Cuite, & Lang, 2003), and only 42% of Americans were aware that the addition of a fish gene would not necessarily make a tomato taste fishy (Hallman, Hebden, Cuite, Aquino, & Lang, 2004). Opposition to GMOs is associated with a lack of genetic literacy; there is a weak positive correlation between performance on a test of genetic knowledge and support for GMOs (Ream et al., 2016). Such an association seems to be telling when one

considers that, in stark contrast to the only 36% of Americans who support GMOs, 88% of American scientists are in favor of the technology (Pew Research Center, 2015). It appears that much of the opposition toward GMOs is not based on a weighing of costs and benefits, but is ultimately grounded in people being disturbed about the essences of their food being tampered with (see also Scott et al., 2016).



3. PERNICIOUSNESS OF GENETIC ESSENTIALISM

The previous sections of this chapter have shown that genetic essentialist biases are based on an overly simplistic understanding of genetic information, combined with a human tendency to imagine underlying essences, leading people to arrive at unwarranted conclusions, and to ascribe undue inferential power to these genes. Given that many consequences of our genetic essentialist biases are decidedly negative, it is important that efforts go into reducing these biases. These efforts may be short-term or long-term, with most attempts falling into the latter category. As this section will demonstrate, existing attempts generally try to address people's overly simplistic understanding of genes, yet these largely reveal the imperviousness of these biases.

3.1 Short-Term Efforts to Reduce Genetic Essentialism

Immediate efforts to eliminate genetic essentialist biases generally involve trying to get people to understand the complex relation between genes and their associated outcomes, with varying levels of success. Two ways of accomplishing this are to emphasize the epistatic and polygenic nature of most genetic causes, or to manipulate people's perception of the strength of a genetic effect. For example, Cheung (2016) sought to contrast how people would respond in their punitive judgments of someone convicted of murder depending on how the genetic risk information was presented. One study contrasted a monogenic cause vs a polygenic cause underlying violence. Another study varied the magnitude of the predictive strength of the putative violence-causing gene. Neither of these efforts significantly affected people's judgments about the applicability of a defense of diminished responsibility. In all of these cases, people in the genetic risk conditions assigned less responsibility to the accused compared with those who instead read of an experiential cause of his crime.

Another method of eliminating people's specific etiological bias is to highlight the role of external and nongenetic factors in an effort to

complicate the genetic causal story. The simplest method of accomplishing this is to discuss genetic causes in the context of interactions with the environment, downplaying the solitary role of genes. Current evidence is mixed on the effectiveness of this method, made more complicated by the fact that very little work has examined the impact of such causal interactions on people's perceptions. For instance, one study found that those who read an account that schizophrenia was the product of both biology and environment had significantly reduced perceptions of danger compared with those who had learned only of a genetic account of schizophrenia (Walker & Read, 2002). Extending this research to address mental health professionals, Lebowitz and Ahn (2014) exposed clinicians to nuanced etiological accounts for depression (i.e., accounts containing both biogenic and psychosocial explanations) with varied emphases, yielding a condition in which the clinicians read a predominantly biological/genetic account and a condition in which they read a predominantly psychosocial account. They found that compared with clinicians in the predominantly psychosocial condition, clinicians in the predominantly biological/genetic condition assessed medication as more effective and psychotherapy as less effective.

However, other research has found that people's stigma toward mental illness did not differ significantly between thinking about mental illness as being due to biological causes vs a gene-by-environment interaction (Boysen & Gabreski, 2012; also see Deacon & Baird, 2009). Similarly, a study on perceptions of violence found that a genetic account of violence elicited an equivalent response from participants as a gene-by-environment account of violence; both conditions resulted in people judging a perpetrator to be less responsible for his crimes, compared with those who learned of an environmental account of violence (Cheung, 2016; also see Lippa & Sanderson, 2012 for similar findings). In sum, causal accounts that point to gene-by-environment interactions are not necessarily perceived much differently than purely genetic accounts. Although it remains possible that there may be ways of making the environmental component of the gene-by-environment interactions more salient to participants (as did Lebowitz & Ahn, 2014), and that this might then weaken people's essentialist biases, the evidence in support of that approach is limited at this time.

Optimistically, there is some evidence that certain interventions can encourage behaviors that are reflective of weaker genetic essentialist biases. One such relatively successful attempt strongly emphasizes the important role that external procedures (e.g., applying sunscreen) can play in dampening or controlling the likelihood that underlying genetic risks will be

expressed (e.g., genes for melanoma), which can empower people to engage in more preventative measures in reducing their risks of developing melanoma (Taber & Aspinwall, 2015). This success, though, still capitalizes on one's genetic essentialist biases rather than eliminating those biases, because the external procedures mechanically prevent the genes from expressing themselves while likely leaving much of the underlying biases intact. Another effective method has utilized weight gain prevention intervention programs that focus solely on obesogenic behaviors rather than addressing the potential ways that genetic causes underlie obesity (McVay, Steinberg, Askew, Kaphingst, & Bennett, 2015). One particularly impressive part of the success of this intervention is in its ability to *decrease* the extent to which people make genetic attributions for weight loss. Despite these successes, a perusal of the nature of these programs underscores the difficult prospects of eliminating genetic essentialist biases: both programs rely on a disproportionately strong emphasis on the importance of nongenetic factors in order to overcome people's genetic essentialist biases, speaking to the strength and pervasiveness of these biases.

3.2 Long-Term Efforts

There are no experimental data to speak to the efficacy of long-term efforts on reducing genetic essentialist biases. The most promising evidence comes from work demonstrating that higher educational levels predict less prejudice, which is associated with having a less essentialized perception of racial categories (Jayaratne et al., 2006). These results are echoed by the finding that people who have taken more genetic courses (or who perform better on a test of genetic knowledge) tend to have weaker genetic essentialist biases (Dar-Nimrod & Godwin, 2016; Ream et al., 2016). Indeed, a large-scale international comparison of primary and secondary school teachers found that greater levels of biological training are associated with a weaker tendency to appeal to genetic essentialism and innatism in understanding group differences (Castéra & Clément, 2014). In particular, genetics training that emphasizes the interactive role of genes and experiences is associated with a less deterministic understanding of genetics, compared with a standard Mendelian curriculum (Radick, 2016). Collectively, these results suggest that an effective long-term strategy for stemming genetic essentialist biases is to improve and increase the public's education in terms of factual genetic information, which many researchers have pushed for (e.g., Burley & Harris, 1999; Castéra & Clément, 2014; Marks, 2009).

In sum, the general pattern of results from existing data suggests that although there might be short-term avenues for reducing genetic essentialist biases, they generally require that genetic causes be trivialized as much as possible. The mere inclusion of genetic causes, even as only a part of a more sophisticated set of mechanisms that lead to a certain outcome, is often sufficient to trigger people's genetic essentialist biases. One direction that is worth exploring is the more long-term option of ensuring that people are better educated on genetics so they have a more accurate understanding of the relation between genes and associated outcomes.

4. CONCLUSION

The genomic revolution has arrived, and people are encountering information about their own genomes and about scientific research on genetics more than ever before. Our review suggests that people's robust tendency for psychological essentialism makes them prone to conceive of imagined essences underlying the natural world. And because popular conceptions of genes make them such an effective placeholder for essences (Medin & Ortony, 1989), we propose that when people encounter these genetic attributions, they tend to understand them in ways similar to how they understand essences; that is, genetic causes appear immutable, of a specific etiology, and natural, and groups that possess them appear more homogeneous and discrete. These genetic essentialist biases are irrational responses for understanding complex human traits, and they have some potent costs, as they are associated with increased racism, sexism, fears about people with mental illnesses, deterministic and pessimistic thoughts about disease prognoses, fears of recidivism among criminals, unwarranted worries about GMO food products, and sympathy for eugenics, among others. On the other hand, these same biases are associated with more tolerance and sympathy for gay rights, people with mental illnesses, criminals, and the potential for international peace. Regardless of the valence of the outcomes, these biases represent incorrect ways of understanding how genes underlie complex human traits.

There are several key questions about genetic essentialism that remain largely unexplored. First, the majority of the findings that we discuss come from WEIRD (Western, educated, industrialized, rich, and democratic) samples; do these effects generalize across different contexts? On the one hand, evidence for psychological essentialism has been found in every culture in which it has been explored thus far in published studies (see Gelman, 2003;

Henrich, Heine, & Norenzayan, 2010), and this leads us to expect that the effects obtained here would be broadly found across other contexts. On the other hand, some constructs that would seem to relate to essentialism are more pronounced in some cultures than in others. For example, Westerners are more likely to make dispositional attributions over situational attributions (e.g., Choi et al., 1999) and are more likely to embrace entity theories of abilities (e.g., Heine et al., 2001), in comparison with East Asians. These differences would suggest that the kinds of effects described in this chapter would be weaker in East Asian societies. For example, one study found that Chinese were less likely than Canadians to incorporate biological information about a target in their behavioral predictions for that target (Lee, 2009). Another possibility is that more collectivistic cultures might be more prone to view essences underlying collectives (e.g., a Japanese genome), as opposed to essences underlying individual differences. Clearly, much research is in order to address the cultural boundaries of the effects reported here.

A second key question that emerges is how broad and enduring are the essentialist responses that we have documented? In terms of the breadth of these responses, do people only essentialize specific genetic attributions, such as the notion about genes underlying criminality, or does encountering the general concept of genes make people essentialize all possible domains (e.g., personality, disease, intelligence)? Likewise, if people learn that genes underlie a specific domain, such as criminality, are they more likely to see genes underlying other specific unrelated domains, such as obesity? These questions have not yet been addressed in the published literature. Furthermore, it remains unclear how long people will show essentialist responses to a genetic prime. All of the experimental studies that we reviewed documented effects that were measured in a time span of a few minutes. Could encountering genetic arguments have more enduring consequences?

Third, is it possible to reduce the magnitude of these kinds of genetic essentialist effects? Although our initial efforts to reduce these effects have not been especially encouraging (see Cheung, 2016), it seems reasonable to expect that there are likely ways of presenting genetic information such that it provokes a less essentialist response. For example, the evidence that genetics education is associated with weaker essentialist responses suggests that a richer understanding of how genes actually operate leads to less of a tendency to equate genes with essences. There may be other ways to reduce essentialist biases. For example, perhaps becoming aware that one possesses essentialist biases might itself reduce the impact of these effects.

If recent history can be a guide for how the next few decades will unfold, it suggests that people will be increasingly encountering genetic attributions. Given that people's essentialist biases seem to provide people with a fundamental misunderstanding of genetic causes, these encounters may well lead people to draw a variety of mistaken conclusions. In the face of this flow of new genetic discoveries and personalized genetic information, it is important to help people come to understand genetic causes better, so they can make more informed decisions about their own lives.

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