

REPLY

Some Thoughts on Essence Placeholders, Interactionism, and Heritability: Reply to Haslam (2011) and Turkheimer (2011)

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In the target article (Dar-Nimrod & Heine, 2011), we provided a social–cognitive framework which identified genetic essentialist biases and their implications. In their commentaries, Haslam (2011) and Turkheimer (2011) indicated their general agreement with this framework but highlighted some important points for consideration. Haslam suggested that neuroessentialism is a comparable kind of essentialist bias and identified similarities with the genetic essentialism framework. In response, we acknowledge similarities but also identify qualitative and quantitative differences between genetic essentialism and other kinds of essentialist biases. Turkheimer challenged us to extend our discussion to address the question of how *should* people respond to genetic etiological information, critiqued the use of heritability coefficients, and identified a new construct ($1 - r_{MZ}$), which may be termed a *free-will coefficient*. In response, we emphasize the need to transform interactionist explanations from being empty platitudes to becoming the default conceptual framework; we wholeheartedly accept his critical view of heritability coefficient estimates (but acknowledge a more limited utility for them); and we are intrigued by his conceptual interest in identifying free-will coefficients yet warn against falling into pitfalls similar to those that were stumbled into in the past.

Keywords: genetic essentialism, gene by environment ($G \times E$) interaction, heritability coefficients, free will, genetic etiology

In our article (Dar-Nimrod & Heine, 2011), we argued that people’s conceptualization of genes increasingly has come to represent a placeholder for the psychological essence that has been described in the essentialism literature (e.g., Gelman, 2003; Medin & Ortony, 1989). We offered a social–cognitive framework to account for genetic essentialist biases and reviewed empirical research that demonstrates some implications of these biases across six different social categories. We wish to thank Haslam and Turkheimer for their largely positive responses and for pressing us to take the ideas underlying genetic essentialism in new directions. Because the issues they raised are nonoverlapping, we address their comments separately and sequentially.

Reply to Haslam (2011)

Haslam (2011) provided a compelling elaboration on the implications that genetic essentialist biases have for mental illnesses. This in-depth examination offered nuanced implications that were not emphasized in the target article. This elaboration indicates that even superficially positive effects of genetic essentialism (e.g.,

reduced blame for the mentally ill, reduced perceived responsibility for undesirable behaviors) may exacerbate negative effects (e.g., prognosis pessimism, increased desire for social distance). The emphasis on the connection between the seemingly positive effects and their negative correlates is of particular importance in this discussion as the geneticization of mental illness has been shown to have undesirable consequences not just among the lay public (e.g., Phelan, 2005) but also among the mentally ill (Rüsch, Todd, Bodenhausen, & Corrigan, 2010) and the professionals in charge of their care (Langer & Abelson, 1974).

More important, Haslam highlighted a number of similarities between genetic essentialism and neuroessentialism. The concept of neuroessentialism was not addressed in our target article, but we wholeheartedly agree with Haslam that it deserves careful consideration in future research. Genetic concepts are not the only placeholder that can prompt essentialist thinking (although we suggest they are an especially powerful placeholder); biochemical substances more generally (such as the nutrients and toxins that one consumes, bacteria and viruses, hormones), and any neurological mechanisms may serve as effective primes for essentialist biases as well. These all share the features of having a largely unobserved, underlying, nontrivial causal influence on people’s behaviors and outcomes. In the context of our focus on genetic essentialism, we discuss some conceptual and empirical similarities and differences between these biases.

Some findings seem to suggest that biologic attributions (such as hormonal imbalances or brain processes) activate essentialist cognitions in a manner similar to that of a perceived genetic

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etiology. For example, Monterosso, Royzman, and Schwartz (2005) found that undesirable behaviors that were attributed to either brain chemistry or genetic predispositions were judged to involve less volition and incur less blame compared with the same behaviors attributed to experiential elements. However, some indications suggest that a perceived genetic etiology may produce quantitatively more determinism compared with other biological processes. For example, Shiloh, Rashuk-Rosenthal, and Benyamini (2002) found that people judged illnesses as less controllable when the cause of the illnesses were specifically attributed to genes compared with other biological factors. It would be interesting and informative to compare the magnitude of essentialist biases between genetic concepts and other potential essence placeholders such as neurological mechanisms. Whether any essence placeholder prompts the same degree of essentialist thinking remains largely an open empirical question.

On a conceptual level, one can find many similarities between the effects of perceived genetic etiologies and perceived biological or neurological etiologies. Attributions to various underlying biological factors (such as genes or the brain) seem to reduce the perceived control that people have over their behaviors and potentially generate affective reactions that are distinct from those with psychosocial and environmental attributions (e.g., Weiner, Perry, & Magnusson, 1985). The similarities between neuroessentialism and genetic essentialism may also be seen by contrasting the 19th century (pseudo)science of phrenology (e.g., Tomlinson, 2005), which focused on the anatomical structure of one's head, with the 19th century (pseudo)science of eugenics (e.g., Kevles, 1985), which focused on one's heredity. Both of these shared much intuitive public appeal, and both point to underlying and unchangeable aspects of people that purportedly shaped much of their behavior and potential. Academics also often amalgamate neurological and genetics research, both in terms of how the research is conducted and distributed (e.g., neuroscience journals tend to publish articles describing either neural or genomic processes) and how they are both targeted for critiques of reductionism (e.g., Peele, 1981). However, we believe it is useful to maintain a distinction between genetic essentialism and neurological (or other forms of) essentialism.

While the self is arguably perceived as coterminous with the brain (e.g., the argument "My brain made me do it" triggers a dismissal at the lowest level of Cartesian dualism), genes have been perceived as puppet masters (the famous image from the cover of the German edition of Dawkins, 1976). Although genes are necessarily internal to the self, they are not always perceived that way, and attributions to them share some features with external attributions, as they may mitigate perceptions of individual responsibility (Shostak, Freese, Link, & Phelan, 2009). Granting genes agentlike status can produce a fundamental change in the perception of the causation process. Whereas genetic attributions are likely to be perceived as the ultimate cause ("My genes made me do it" or, in its more common form, "I have the gene for it"), neurological attributions occupy an intermediate level of causation as they raise the question, "What caused the neurological process?" Hence, while we feel that neuroessentialism is a worthy direction for future research and that it shares much in common with genetic essentialism, the two are not interchangeable.

Reply to Turkheimer (2011)

Turkheimer (2011) does not challenge the premise of genetic essentialism. Instead he focuses on the important question regarding how *should* people respond to learning about genetic influences on behavior? As he noted, in our article, we made a clear case for why a genetic essentialist response may often be problematic. But then, what is the most appropriate response to evidence of genetic etiologies, be they genotype–phenotype correlations or large heritability coefficients? Should we conclude that genetic information is simply irrelevant to our judgments, should we adopt a naïve environmentalist account, or should we judge everything as an expression of free will?

What Is the Appropriate Response to Evidence of Genetic Etiology?

We call attention to an unacknowledged similarity between etiological behavioral explanations and psychological essences. As etiological behavioral explanations are similar to essences in that they are fundamental, unobservable, underlying, nontrivial, inferred, and not directly cognitively represented, they also tend to be represented by way of placeholders. The question of what is the causal force that underlies a behavior shares much in common with the question of what is the underlying essence that makes group members the way they are. A good placeholder is one that is simple to use and accessible, and different etiological behavioral explanations vary in their ease of use and accessibility. Hence, although virtually all serious thinkers about behavioral etiologies would conclude that the cause of any behavior or outcome must always be interactionist in some way, interactionist accounts do not serve well as placeholders. Take the case of the well-documented example of how a single polymorphism interacts with certain kinds of childhood experiences to reliably increase one's risk of engaging in future criminal behavior (e.g., Caspi et al., 2002). Even though this etiological account of criminality is relatively straightforward as only a single gene is implicated and a single set of environmental circumstances is identified, it remains challenging to explain, imagine, or communicate how genes and environments can interact in ways that affect behaviors. This is even more so in the case of interactionist accounts involving polygenic and less clearly specified environmental variables. It is perhaps because of their complexity and intangibility that thoughtful researchers who are persuaded by interactionist accounts still often find themselves instead feeling compelled to argue for one of the two poles of nature versus nurture (with many notable exceptions; e.g., Caspi et al., 2002; Guo, Tong, & Cai, 2008; Kim et al., 2010; Richerson, Boyd, & Henrich, 2010). So a counter to a naïve environmentalist position typically starts off with a nuanced and interactionist perspective highlighting how "biology matters too," but it seems to us that frequently this kind of argument starts to morph into something that sounds more like "biology is the only thing that matters" (e.g., Thornhill & Palmer, 2000). Likewise, counters to nativist accounts also commonly start off as interactionist but often transform into what sound more like exclusively environmentalist positions (e.g., Eagly & Wood, 1999). The extreme nature and nurture positions have a magnetic draw, we argue, because their simplicity and accessibility allows them to fill the etiological placeholder so well, rendering the nature versus

nurture debates a seemingly permanent fixture of the behavioral sciences.

As Turkheimer correctly inferred, we think the most appropriate response to genetic etiological accounts (or to environmentalist etiological accounts) is an interactionist one: genes are relevant to all complex human behaviors, yet they do not determine any such behaviors. We imagine that few would disagree with this statement, but the challenge lies in making this statement anything beyond an empty platitude. We offered some relevant suggestions in our target article regarding how researchers might best frame their work to render it less essentializing but note that this remains a difficult challenge.

It should go without saying (but we will say it anyways) that etiological accounts should not determine moral evaluations of complex human behaviors. For example, our evaluation of the ethical standing of binge drinking should not be affected by a consideration of the genetic underpinnings for alcohol dependence anymore than by the proposition that St. Patrick's Day was extremely cold this year. Such a focus, as Pinker (2002, p. 179) put it, "is a confusion of explanation with exculpation." The mea culpa "My genes made me do it" is not an accurate account of the role that genes have in associated behaviors. As there are no known complex human behaviors in which genetics render the actor unable to resist performing a behavior, we contend that genetic etiological accounts should not serve as the basis for moral evaluations. Genes provide one source of influence (depending upon how those genes are expressed in interaction with other genes and experiences and following a developmental trajectory), but there are many other sources of influence at play, making the role of genes in producing any complex behaviors far from deterministic. Furthermore, the amount of influence that genes have on behaviors is considerably smaller than one might expect. For example, meta-analyses reveal that the β_3 -adrenergic receptor gene (ADRB3), the most studied polymorphism linked with obesity (which is commonly labeled "the obesity gene"), contributes on average less than 2 lbs. to the average person's weight and considerably less than this among Europeans (Kurokawa, Nakai, Kameo, Liu, & Satoh, 2001; Kurokawa et al., 2008). Likewise, a meta-analysis of DRD4, the so-called "novelty-seeking gene," has shown that the average association between the relevant polymorphism and novelty-seeking has a nonsignificant effect size ($d = 0.06$; Kluger, Siegfried, & Ebstein, 2002), which by the standards of the behavioral sciences, is a trivial effect (Cohen, 1988). Other etiological accounts such as naïve environmentalist ones should not affect moral judgments either, as they too provide only one small source of influence on behavior. However, the data we reviewed did not show support for such an environmental essentialist bias; for example, in a number of different paradigms, evidence of environmental influence was largely ignored by participants (e.g., Dar-Nimrod & Heine, 2006; Dar-Nimrod, Heine, Cheung, & Schaller, in press; Monterosso et al., 2005; No et al., 2008; Williams & Eberhardt, 2008). We suggest that genetic essentialist biases are more potent than environmental essentialist biases because genes make for a better essence placeholder than the environment in that they are perceived as offering an underlying, materialistic, immutable, and fundamental cause of an individual's nature (but see Rangel & Keller, in press). Likewise, interactionist accounts make for a poor placeholder, and we know of no evidence for an interactionist essentialist bias (e.g., Walker & Read, 2002).

Interpreting Heritability

The most direct source of evidence regarding the role of genes on behavior comes from gene-association studies; however, the more readily available and widely discussed sources of evidence derive from heritability estimates. Here, we agree with Turkheimer's criticism of the ways that heritability has frequently been used and interpreted. We also wish to call attention to other ways that heritability estimates have been misinterpreted.

First, we note that heritability coefficient estimates typically hover around .50, which suggests that even when they are interpreted in the erroneous ways that Turkheimer critiques, they should be viewed as far from deterministic. The magnitude of these values however, may be sufficient to lead to *causal discounting*, as people who are presented with a strong causal attribution tend to undervalue alternative causes (Kelly, 1972). Portraying genes as a strong explanation for many phenomena using such coefficients hinders adequate evaluation of other explanations (e.g., environment, choice).

Second, we would like to highlight one way that heritability estimates are grossly overestimated that is rarely acknowledged. One large component of environmental influence is never taken into account in these estimates: people's cultural background. Heritability estimates are most commonly derived from studies of adopted/biological siblings or of twins who typically share the same family and culture. Even in the more rare but powerful "twins reared apart" paradigm, there are no instances of studies of twins or siblings who were reared in different cultural environments. Because these experimental designs are not capable of accounting for the role of different cultural environments, this results in a substantial restriction of range problem for estimating the amount of variance accounted for by the environment. How large of an influence might culture have on these estimates? Take the case of self-esteem. Behavioral genetics studies have estimated the magnitude of the heritability of self-esteem to be around .50, with shared environmental effects accounting for a negligible amount of the variance (e.g., Roy, Neale, & Kendler, 1995). However, a meta-analysis of the magnitude of the effect size of cultural differences in self-esteem between Westerners and East Asians is a substantial $d = 0.91$ (Heine & Hamamura, 2007). This large cultural component of the variance in self-esteem is completely absent in the calculations of the estimates of environmental variance, meaning that the role of the environment in self-esteem is grossly underestimated while, correspondingly, the estimates for heritability are overestimated. Similar problems exist for calculating heritability estimates in other domains as well, yet rarely is this inherent confound of cultural bias in the design of behavioral genetics studies acknowledged. Heritability estimates are inflated for any phenomenon for which cultural experiences affect its outcome. Unfortunately for our ability to accurately estimate heritability, large effects of cultural experiences are the norm rather than the exception in many domains of psychological research (e.g., Henrich, Heine, & Norenzayan, 2010).

We agree with Turkheimer that heritability coefficients are a product of the samples and environments from which they are drawn. Although this is how heritability is explained in leading behavioral genetics textbooks (e.g., Plomin, DeFries, McClearn, & McGuffin, 2008), in practice it is frequently grossly misinterpreted, even by behavioral geneticists. For example, it is not

uncommon to see articles in which the heritability coefficients are described as indicating the ceiling of the portion of the variance that is explained by genes. So, for example, if a given phenomenon has a heritability coefficient of .50 and researchers have identified gene-associations that account for 12.5% of the variance, then they sometimes misinterpret that their targeted genes as having identified one quarter (i.e., 12.5/50) of the genetic variance of that phenomenon (e.g., Derringer et al., 2010; Schunkert et al., 2011). Such misinterpretations of heritability by behavioral geneticists demonstrate that training in genetics is not always sufficient to protect one from essentializing genetic influences.

Turkheimer described why he believes that heritability is a “fool’s errand” (p. 826). We acknowledge here that probably no one has contributed more to an understanding about what heritability means than Turkheimer himself, and his writings have greatly shaped the ways that the two of us think about it. However, we find ourselves in the position (an unusual one for us) to be debating with someone with even more critical views on heritability than our own. We agree with Turkheimer that the absolute value of heritability coefficients is largely meaningless; however, we feel that the relative value of these coefficients are meaningful, provided that they are collected within the same contexts and samples, just as the relative magnitude of F values (but not the absolute magnitude) provides meaningful information when collected within the same contexts and samples. For example, that behavioral genetics studies find larger heritabilities for height than they do for divorce, personality, and anxiety disorders in the same populations and contexts does tell us that genetic influences play a larger role in influencing height in these contexts than it does for divorce, personality, or anxiety disorders. One nice empirical demonstration of the utility of relative differences in heritability coefficients comes from research investigating the heritability of attitudes. This research finds that some attitudes (e.g., whether one supports the death penalty or likes jazz music) have larger heritability estimates than other attitudes (e.g., whether one supports the use of straitjackets or is in favor of coeducation; Martin et al., 1986). Moreover, research comparing the characteristics of attitudes shows that those attitudes with the larger heritability estimates are more accessible, strongly held, and resistant to social influence than those attitudes with smaller heritability estimates (Tesser, 1993). Heritability coefficients are meaningful when their relative size is contrasted between characteristics assessed in the same samples and contexts. The problem with using heritability estimates occurs when the coefficients are reified by generalizing them to other samples and contexts.

Quantifying Free Will

Lastly, Turkheimer took on the ambitious task of trying to replace the conceptually faulty heritability coefficients with what perhaps could be termed *free-will coefficients* (i.e., $1 - r_{MZ}$). We think his efforts here are intriguing, although we will withhold our judgment until we can see these ideas developed more and see how they stand up to more rigorous empirical tests. Such an important and enormous endeavor deserves more space than a commentary, and we urge Turkheimer to develop this idea further, so it can be better evaluated. One potential problem with this endeavor may be its reliance on the summation of the estimates of the coefficients for the shared environment and heritability (i.e., r_{MZ}), which, as

Turkheimer argued, are problematic because they are constructed on a faulty premise. While the aggregation of shared environment and heritability answers some of the identified problems, it leaves other critiques unaddressed.

In sum, there are multiple genetic methods by which we can understand the complexity of genotype–phenotype relations. Heritability is one method, and it has many, often unrecognized limitations. We contend that one reason that heritability is so often misunderstood, in the ways articulated by Turkheimer, is that people’s genetic essentialist biases lead them to view heritability in an overly deterministic fashion. Identifying ways in which heritability could be communicated without activating essentialist responses is a desirable direction for future research.

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Received May 27, 2011

Revision received June 8, 2011

Accepted June 9, 2011 ■