Am I My Genes? Perceived Genetic Etiology, Intrapersonal Processes, and Health

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Abstract

With the increasing popularity and affordability of DNA sequencing through direct-to-consumer DNA sequencing services, it has become apparent that researchers need to understand how the results of sequencing one’s DNA affects consumers psychologically and behaviorally. In this paper, the authors discuss several intrapersonal processes that may impact how learning about our own genetic predispositions affects us. In particular, this paper sets out to identify the interplay between three relevant perspectives: genetic essentialist biases, perceived identity, and need for certainty. These interrelated perspectives and the empirical research that supports relevant underlying predictions provide a useful basis from which researchers can further identify testable hypotheses on these intrapersonal perceived genetics effects. Such research has potential far-reaching implications, not the least of which are in the health domain.

Since the Human Genome Project began in the 1990s, scientists and the public have been fascinated by the seemingly imminent capacity to identify the genes responsible for every trait, behavior, and illness. Advances in genomics have allowed some such predictions to be realized with the discoveries of genes largely responsible for specific illnesses such as Huntington disease (MacDonald et al., 1993) and Tay Sachs (O’Brien, Okada, Chen, & Fillerup, 1970) or involved in a variety of behaviors and conditions such as violence (Moffitt & Caspi, 2001), obesity (Boutin et al., 2003), or various forms of cancer (e.g., Fishel et al., 1993; Miki et al., 1994), to name a few. These findings have been part of the exciting genetic science that seems to hold much promise to experts and novices alike, garnering much scientific attention and centrally featured by media outlets. These developments also have paved the way for the emergence of concepts such as personalized medicine (Hamburg & Collins, 2010), whereby medical decisions are meant to be tailored to individuals’ genetic makeup.

These scientific advancements were also paralleled by increased affordability of personal genomics (i.e., individual’s DNA sequencing). This increased affordability had contributed to the emergence of commercial direct-to-consumer (DTC) gene sequencing service providers such as 23andMe, Navigenics, and deCODEme, allowing people to access more of their own genetic information with greater convenience. In fact, a recent study showed that 64\% of a large sample of participants were interested in using personal genetic tests (McGuire, Diaz, Wang, & Hilsenbeck, 2009). This interest may be especially robust in relation to genetic predispositions to developing certain medical conditions, with more than 80\% of respondents showing keen interest in learning about their own genetic susceptibility to different cancers, for example (Andrykowski, Munn, & Studts, 1996; Croyle & Lerman, 1993). These studies and others (e.g., Sanderson, Wardle, Jarvis, & Humphries, 2004) clearly demonstrate that the public has a strong interest in learning about their own genetic constitution.

Given the public’s interest in learning about their genes, it is important that researchers understand how gaining knowledge about our own genetic makeup may affect us; that is, what
are the psychological and behavioral outcomes of learning about our own genes, and what are some processes that might explain these outcomes? Some debate exists as to whether such knowledge will lead to positive or negative outcomes for consumers of personalized genomics. On the one hand, some argue that equipping the public with knowledge about their genes will allow them to make more informed decisions about their own medical treatments and more well-reasoned life choices, and they can engage in more preventive health behaviors as a way to decrease health risks (e.g., Bloss, Schork, & Topol, 2011; Wojcicki, 2013). Such claims find support in prospective surveys in which many patients believed that genetic testing would encourage them to be more motivated to adhere to taking medications (Grant et al., 2009). Furthermore, some researchers also argue that providing such knowledge to the public is empowering and do not believe that people will be overly anxious by overestimating their risks (Helgason & Stefánsson, 2010). In short, proponents of this perspective champion widespread disclosure of individuals’ genetic proclivities.

On the other hand, other scholars have expressed doubts over the claims that proponents of personalized genomics disclosure have made. Whereas proponents argue that people can engage in preventive measures to counteract their genetically predisposed health risks, opponents argue that the evidence for such behaviors as a general response to genetic testing is equivocal at best (e.g., Hunter, Khoury, & Drazen, 2008). Furthermore, researchers have argued that the general public is likely to understand genetic expression in overly simplistic terms (Nelkin & Lindee, 1995), introducing detrimental biases to the way they may interpret the results of their DNA sequencing (Dar-Nimrod, 2012; Dar-Nimrod & Lisandrelli, 2012; Lebowitz, Ahn, & Nolen-Hoeksema, 2013). Such biases are compounded by the public’s poor understanding of genetics. For example, Christensen, Jayaratne, Roberts, Kardia, and Petty (2010) found that fewer than half of their participants could identify the correct answer for six of eight basic questions on knowledge about genes (e.g., “Single genes directly control specific human behaviors” and “Our genes tell us which race we belong to”, neither of which are true). This lack of knowledge is magnified by the fact that many people who choose to get their DNA sequenced will not consult a physician in interpreting the results (Kaufman, Bollinger, Dvoskin, & Scott, 2012). Even when individuals consult their physician, there is concern about whether physicians can give accurate information given the paucity of replicable gene-association studies (for a review, see Charney & English, 2012). This problem is further exacerbated by the tendency for some researchers and the media to prematurely report on lab findings and oversell their impact and utility, and the commonplace replication failures of genetic findings, which make even published evidence inconclusive (Burke, Kuszler, Starks, Holland, & Press, 2008; Dar-Nimrod & Heine, 2011). These issues highlight some of the concerns over the potential for consumers of personalized genomics to misinterpret their personal genetic information.

To understand the potential effects of revealing personalized genomics to consumers, we will look at these effects through the lenses of intrapersonal processes. Our departure point for the assessment of such processes is rooted in psychological essentialism and more specifically, genetic essentialist biases. These biases capture cognitive fallacies resulting from learning about genetic explanations for various human behaviors, traits, and more germane to this paper, health conditions. We then extend the discussion about these biases to integrate what we consider the most central intrapersonal processes affected by the genetic essentialist biases. One such central process, perceived identity (how people view their individual and group identities), is intimately tied to personalized genetics as genes are often viewed as the seat of one’s essential characteristics (e.g., Dar-Nimrod & Heine, 2011; Nelkin & Lindee, 1995). Another central process, the need for certainty (a desire to feel certain about ourselves and our social environment), is intimately affected by personalized genetics as vast research indicate that people equate the presence of genes with certain outcomes (e.g., Dar-Nimrod, 2012; Jayaratne et al., 2006). In the rest of
the paper, we argue that these interrelated intrapersonal processes have important implications for how one might respond to personalized genomic information by drawing on various discrete research lines and identifying important future directions.

**Genetic Essentialist Biases**

Many scholars observed that people demonstrate skewed understanding of genes and the processes by which genotypes affect phenotypes (Charney, 2012; Lippman, 1992; Nelkin & Lindee, 1995). This phenomenon has been observed across various domains (e.g., Brescoll & LaFrance, 2004; Dar-Nimrod, Cheung, Ruby, & Heine, 2014; Keller, 2005) demonstrating its pervasiveness. More specifically, Dar-Nimrod and Heine (2011) elucidated four cognitive biases that people often engage in when they encounter genetic explanations. These biases were argued to increase people’s tendency to view outcomes associated with genetics (compared with non-genetic etiologies) as more: (i) immutable and determined (decreasing sense of self-efficacy and control); (ii) having a specific etiology (leading to a devaluation of alternative/contributing causes); (iii) establishing perceived homogenous and discrete categorizations (prompting stereotypical/prototypical evaluative processes); (iv) natural (increasing the likelihood of committing the naturalistic fallacy – the tendency to conflate “ought” and “is” for occurrences deemed natural). In short, people who show strong genetic essentialist biases endorse the perspective that genes create fundamental differentiations between people, are often the fundamental causes of traits and behaviors, and likely will lead to their associated outcomes – outcomes that may be seen also as being more morally acceptable because of the naturalistic fallacy.

At the broadest level, genetic essentialism provides an important framework to understand how learning about our genetic predispositions may directly affect our behavior, particularly with regard to the immutable/deterministic bias, which affects, among others, one’s sense of inevitability or certainty. Indeed, research indicates that the concept of genetics is implicitly associated with fatalism and inevitability (Gould & Heine, 2012). Such association was also demonstrated empirically to affect people’s attitudes – offering a genetic explanation for undesirable behaviors results in reductions of blame and culpability of the individuals who displayed such behavior more than if environmental or psychosocial explanations were provided (Dar-Nimrod, Heine, Cheung, & Schaller, 2011; Dar-Nimrod, Zuckerman, & Duberstein, forthcoming; Monterosso, Royzman, & Schwartz, 2005).

In the health domain, for example, people who were asked to imagine that they were genetically at risk of developing certain conditions felt that the manifestation of the condition will be less preventable than people who imagined that they were at risk for non-genetic reasons (Senior, Marteau, & Weinman, 2000). Such increased expectation for the development of a condition may hinder engagement in preventive health behaviors that some proponents of personalized genomics expect to occur as a result of increased personal relevancy (e.g., Collins, Green, Guttmacher, & Guyer, 2003; Guttmacher, McGuire, Ponder, & Stefánsson, 2010). And indeed, learning that one is at genetic risk for high cholesterol has not been found to affect adherence to risk-reducing behaviors such as dieting, exercising, and taking medication (Marteau et al., 2004). Similarly, Lerman et al. (1997) found that participants who were told that they were genetically at risk for developing lung cancer from smoking did not differ in smoking cessation compared with participants who were told that they were not genetically at risk. Interestingly, these findings emerged despite genetically at-risk participants in both studies reporting that they perceived themselves to be at greater risk of developing the respective conditions compared with participants who were not genetically at risk. These results may indicate that people believe that there is little that one can do to counteract the developmental trajectory of a genetically-associated outcome, perceiving this association to be immutable and
deterministic. These findings and others (e.g., Botkin et al., 2003; Lynch et al., 2006) give further credence to the Audrain et al. (1997) observation that learning about genetic susceptibility rarely results in lasting modifications of health behaviors, even when behavioral changes can decrease risk (but see Brewer, Weinstein, Cuite, & Herrington, 2004; Weinstein et al., 2007, for some indications of behavioral changes).

One disease in which behavioral modifications are somewhat more evident is cancer. The recent self-exposure of Angelina Jolie’s preventative mastectomy, which was widely disseminated, represents more than just a celebrity’s idiosyncratic behavior. Botkin et al. (2003) found that about half of the women who were genetically at risk for ovarian cancer had gotten an oophorectomy within 2 years of being genetically tested, compared with less than 5% of the non-genetically at-risk group. This does not negate the impact of genetic essentialist biases—rather, it suggests that one potential boundary condition under which such biases diminish inaction is when a clear treatment can overcome the perceived inevitability of genetic-associated diseases (e.g., environmental treatments of the genetically-determined phenylketonuria). Similarly, one would predict that once gene therapy fulfills its great promise as the next big treatment (e.g., Dar-Nimrod, 2007; Pearson, 2009), certain biases-based inaction will decrease.

These findings support the proposed mechanism in which the immutability/deterministic bias is translated to behavioral (in)action. Dar-Nimrod and Heine (2011) suggested that perceived personal control is reduced as a result of this cognitive bias, leading to inaction. Once the medical establishment offers a clear way to successfully negate the disease threat, perceived control is restored, and evidently, individuals may be more likely to modify their behavior. The difference in the uptake of preventative oophorectomies between high-risk individuals with or without an identified genetic susceptibility also highlights the deterministic bias—it seems that individuals with identified genetic susceptibility to cancer may perceive the development of cancer to be more deterministic and immutable and thus are more willing to take extreme, one-off, measures to reduce their disease risk. This pattern of behavior has also been found in relation to preferred treatment options and perceived prognoses regarding various psychopathologies (Deacon & Baird, 2009; Lam & Salkovskis, 2007). Similar findings emerged when people who were told that they had an allele associated with alcoholism believed that they had less personal control over their drinking behaviors but were also more likely to enroll in a “responsible drinking” workshop expecting, perhaps, personalized risk reduction strategies compared with individuals who were told they do not have such an allele (Dar-Nimrod, Zuckerman, & Duberstein, 2013). Even more striking is the fact that people are willing to forego having children if they perceive their psychopathologies to be due to genetic reasons (Meiser et al., 2007), showcasing the strength of the immutability/deterministic bias regarding a genetic cause. Overall, it may be that the degree to which people are affected by genetic essentialist biases predicts their subsequent adherence to preventive health behaviors as long as, perhaps, these behaviors are not continuous in nature (e.g., exercising over time) but rather necessitate a one-off drastic decision—a point that will require additional empirical support.

Perceived Carrier Status and Perceived Identity

Genetic essentialism may also have specific implications for one’s perception of their own and others’ identities, which leads people to act in accordance with that identity (Rise, Sheeran, & Hukkelberg, 2010). In particular, the aforementioned homogeneity/discreteness bias allows for the perceived possession of certain genes to serve as substrates for social identities (e.g., creating or strengthening social categorizations based on shared/believed genetic markers). For example, race is one of the most essentialized categories, such that each race is seen as being fundamentally different from all other races, and belonging to one race precludes belonging to other races (Prentice & Miller, 2007). Gender is likewise highly essentialized. In line with the
genetic essentialism framework, much research supports the notion that people perceive gender and race, and differences underlying these categories (i.e., discreteness), to be genetic in nature (Christensen et al., 2010; Dar-Nimrod & Heine, 2011; Plaks, Malahy, Sedlins, & Shoda, 2012).

The overlap between perceived genetic etiology and socially-constructed categories may indicate that genetic similarities contribute to the formation of group identities – a point that has also received empirical support (Tenenbaum & Davidman, 2007; Weber, Johnson, & Arceneaux, 2011; Zeiler, 2007). To date, little empirical research has been conducted on identity and changes in self-perception as an outcome of personalized genetic information of the kind that is readily available through medical testing and DTC genomic reports. The potential effects of deriving identities out of genetic predispositions are thus extrapolated from work on the labeling effect and interpolated from research on the phenomenology of people who learned that they are genetically at risk.

The labeling effect is a phenomenon whereby people who, upon discovering that they have a certain (pre-existing) condition, subsequently act in accordance with that label, despite not having acted in that manner previously (Macdonald, Sackett, Haynes, & Taylor, 1984). For example, people who were previously unaware that they were hypertensive learned that they fulfilled the requirements for hypertension. After this revelation, these individuals began to have workplace absences at similar rates as people who were previously aware that they were hypertensive and also experienced similarly high levels of psychological distress and low levels of psychological well-being. Prior to having been labeled as hypertensive, however, these participants acted like normotensives (Birkenhäuser, 2003; Macdonald et al., 1984). Several other studies using self-report measures of patients have also found that receiving a diagnosis of hypertension, rather than objective physiological symptoms, was predictive of subsequent ailments such as depression and headaches (see Pickering, 2007, for a review). These studies show the extent to which labels can lead one to act in congruence with that related identity, highlighting the psychological and behavioral impact of obtaining a label not dissimilar to the kind of labels inferred by perceived genetic susceptibility (McGuire & Burke, 2008). In fact, the original conceptualization of the labeling effect, and its subsequent elaborations, places significant emphasis on its impact on one’s perceived identity (Link, Cullen, Struening, Shrout, & Dohrenwend, 1989; Matza, 2010; Tannenbaum, 1938). Furthermore, the assimilation of information into the core aspects of one’s identity may be particularly likely when the information deals with chronic health conditions (e.g., Jacoby, Snape, & Baker, 2005), which personalized genomic information is set to provide. More importantly, such an assimilation process could also extend the extent to which one experiences stigma associated with these chronic conditions (Field, 1976).

An alternative explanation for the labeling effect affecting one’s perceived identity is that it instead operates through self-fulfilling prophecies. Whereas the data to rule this possibility out are not available at this point, such mechanism does not preclude perceived identity from playing a role. Research revealed other psychological phenomena in which identification is an important precursor for self-fulfilling prophecies to manifest, such as stereotype threat and boost (e.g., Shih, Ambady, Richeson, Fujita, & Gray, 2002).

A more direct process by which learning about genetic predispositions impacts one’s identity-formation is the degree to which one engages with or are cognitively and emotionally involved with those new identities. Perhaps the most relevant research for this perspective is McAllister’s (2003) phenomenological study on people’s perceptions of their increased risk of developing hereditary non-polyposis colon cancer (HNPCC) based on family history. HNPCC is a dominantly inherited disorder, such that only one genetic mutation needs to be passed down from the parent for the child to be affected. Participants appeared to be psychologically affected differently based on McAllister’s (2003) categorization of engagement. There were people who focused on their similarities with family members who had previously developed cancer,
thereby expressing intense fear and anxiety over their elevated cancer risk—embodiment the homogeneity/discreteness bias. Compared with these participants, “partially engaged” individuals acknowledged the probabilistic nature of the genetic inheritance process. However, none of them indicated they believed they carry the relevant mutation, instead showing reduced emotional investment in their at-risk identity. Interestingly, unlike those who were intensely engaged, those who were partially engaged also tended to focus on how they were dissimilar to family members who had developed cancer, actively distancing themselves from that afflicted identity by avoiding the homogeneity trap.

The notion of engagement could be useful in explaining the equivocal results found so far regarding people’s varied responses to genetic test results. It may be that people who become “intensely engaged” after receiving a positive genetic test result may respond by increasing engagement in preventive behaviors as long as group identification is associated with such behavioral intentions (Pierro, Mannetti, & Livi, 2003). Other people may become “partially engaged” after receiving a positive genetic test result and will not respond in line with such perceived afflicted-group-related behaviors. Moreover, combining engagement with the labeling effect may reveal that people who act in accordance with their label/identity are also likely to evince intense engagement when the identity seems to encompass behavioral elements.

Interestingly, McAllister (2003) also identified a group of individuals, the “disengaged”, whose engagement was so dominant that it had become too overwhelming to deal with. Ironically, this led to disengagement from the at-risk status and subsequent denial of this identity. If this is the case, then people who are disengaged may respond negatively by decreasing their engagement with preventive behaviors they perceive as indicators of the group identity in what can be viewed as an ostrich phenomenon—the overwhelming experience of threat leading the individual to ignore it completely. This is akin to research showing that images that induce high levels of fear may actually be counter-productive when trying to induce positive changes in health behaviors (Witte & Allen, 2000).

The above hypotheses all represent promising and important avenues for future research. However, the interplay between one’s perception of their own genetic proclivities and identity is not the only intrapersonal element of note to consider with the advance of the availability of genetic information. Motivational elements and epistemic needs may also play a notable role.

Perceived Carrier Status and the Need for Certainty

Finding out information about one’s own personal genetic susceptibility can provide a fertile opportunity to satisfy epistemic needs. The most relevant epistemic need in this context revolves around one’s desire for certainty. The interplay between one’s perceptions of their genetic susceptibilities (and their meaning) and one’s desire for certainty can manifest through several means: certainty through need for cognitive closure, certainty in perceived personal and group identity, and certainty through differences in neuroticism.

The need for cognitive closure (NfCC) construct (Kruglanski & Webster, 1996) has been identified as an important motive that affects information processing. When NfCC is high, people tend to show a preference for information that can be processed quickly and easily (Kruglanski & Webster, 1996). Thus, NfCC may serve to affect behaviors designed to attain personally-relevant genetic information as well as enhance genetic essentialist biases once such information is attained.

And indeed, previous research has revealed that the need for certainty attenuates information seeking related to undertaking a predictive genetic test for cancer. A study (Croyle, Dutson, Tran, & Sun, 1994) found that adding a description that emphasized the existing risk for people who do not carry the susceptibility mutation resulted in less interest in the test among women who were high on NfCC’s measures. The opposite pattern was observed among women with low scores.
As high NiCC individual prioritize easily-processed information, essentialist beliefs may be particularly strong because such beliefs are characterized by clarity and lack of ambiguity. Indeed, Roets and Van Hiel (2011) found that experimentally induced high NiCC led participants to endorse the beliefs that all members of a social outgroup were alike and that strong inferences about a person could be made on the basis of group membership. This suggests that adhering to essentialist beliefs may satisfy the need to avoid ambiguity (which is a hallmark of NiCC), enhancing the coherence and predictability of one’s social world. Thus, when considered within the context of personal NiCC, personalized genetic information may activate or exacerbate immutability/deterministic and homogeneity/discreteness biases, leading to psychological and behavioral responses in line with these epistemic preferences (i.e., increased fatalism among high NiCC).

The desire for certainty may take on even greater significance when applied to the self as is the case when one receives from personalized DTC genomic tests, not the least because the degree to which a person is certain about the self is fundamental to how one thinks, feels, and behaves. Along this line, we argue that receiving information about one’s genetics may alleviate certainty concerns and affect one’s behaviors (see Dar-Nimrod & Heine, 2011; Dar-Nimrod & Lisandrelli, 2012, for similar arguments). This assertion is supported by empirical findings; for example, Dar-Nimrod and Heine (2006) found that women showed stereotype-threat-related underperformance on a math exam when they were exposed to genetic etiological explanations for the stereotypical gender difference in math, but not when they were exposed to an experiential one. Similarly, Brescoll and LaFrance (2004) found an increase in self-stereotyping after highlighting a genetic explanation for gender differences in a fictitious trait but not after highlighting a psychosocial explanation.

Group identification is another route by which the need for certainty may affect how one responds to information about personalized genetics. As suggested previously, the homogeneity/discreteness bias increases individual perceived similarity to people who may share the same genetic proclivity. We contend that such perceived similarity may produce group identification, which serves to promote self-certainty. Along similar lines, Hogg (2000, 2007, 2009) argued that self-certainty is a motivational mechanism underlying social identification. Consistent with this theory, when people are uncertain about the self, they are more likely to identify with groups, even if the quality of these groups is questionable (see Hogg, Siegel, & Hohman, 2011). These findings suggest that identifying with genetically similar others may be particularly effective in promoting self-certainty. Although empirical research on this assertion is scarce, it may prove to have translational implications in behavioral modifications. As identification with a group often provides people with norms for how to think, feel, and behave (e.g., Tajfel & Turner, 1979, 1986; Turner, Hogg, Oakes, Reicher, & Wetherell, 1987), interventionists can harness genetic essentialist biases in an attempt to increase adherence to preventative health behaviors by first shaping and then stressing the norms of such newly conceived genetics-centered groups.

Related to negative and aversive responses to uncertainty (Berenbaum, Bredemeier, & Thompson, 2008; Hirsh & Inzlicht, 2008), neuroticism, the tendency towards greater emotionality, fear, anger, and general psychological distress (Costa & McCrae, 1980), may also play an important role in the intrapersonal effects of personal genetics revelations. While researchers over the years have raised the potential relationship between this construct and the effect of receiving results from genetic testing (e.g., Van Oostrom et al., 2007), few studies have directly examined this link.

Several studies have demonstrated that one of the strongest predictors of psychological distress after receiving positive genetic tests is one’s emotional state prior to testing (Broadstock, Michie, & Marteau, 2000; Van Oostrom et al., 2003). Given that this may reflect a general tendency to experience psychological distress, particularly under conditions of uncertainty, researchers hypothesized that neuroticism may be a key construct underlying this important consequence of receiving genetic testing results (Ho, Ho, Bonanno, Chu, & Chan, 2010; Van Oostrom
et al., 2007). In accordance with this expectation, Reichelt, Møller, Heimdal, and Dahl (2008) found that participants’ neuroticism was predictive of general psychological distress before being tested, and both psychological distress and cancer-specific distress 18 months after receiving their test results. Unfortunately, the authors did not examine the interaction between neuroticism and cancer status to determine whether those who received positive genetic tests and who were high on neuroticism experienced greater distress than those who also received positive genetic tests but who were low on neuroticism. Despite that, it is apparent that neuroticism may be an important factor that moderates how learning about one’s genetic predispositions may impact someone psychologically. Taken together, the dearth of studies examining the role of neuroticism should not undermine the recognition of its importance in explaining how different people may have different reactions to learning about their genetic predispositions.

Conclusion

There is little empirical research on intrapersonal processes that follow people’s interpretations of their personal genetic information, although there are clear indications that these interpretations affect them psychologically and behaviorally (Dar-Nimrod & Heine, 2011; Dar-Nimrod et al., 2013; Kaufman et al., 2012). This paper highlighted three particular perspectives – genetic essentialist biases, self-identity, and the desire for certainty – all of which generate clear directions for future research.

Given the popularity in personal genetic testing, as is evidenced by much empirical research (e.g., Hall et al., 2009; McGuire et al., 2009), scholars have begun focusing on understanding how the results of these tests may impact people psychologically and behaviorally. Researchers must keep pace with the great advancements in human and particular behavioral genetics and step up efforts to understand the psychological effects and underlying mechanisms involved in responses to the revelation of such genetic-associated tendencies. If a goal of personalized genomics is to empower people to engage in more preventive or primary care behaviors (Helgason & Stefánsson, 2010; Wojcicki, 2013), then it is evident that we must understand the relevant interpersonal and intrapersonal processes that hinder people’s adherence to such beneficial behaviors.

Acknowledgement

Funding from the Australian Research Council (DP140104527) is acknowledged with gratitude.

Short Biographies

Benjamin Y. Cheung is a PhD Candidate at the University of British Columbia, where he also completed his MA in social psychology, and BA in psychology and in Asian language and culture with a specialization in Korean. His research broadly examines questions regarding how people interpret genetic information, particularly its implications for moral reasoning, prejudice, stigma, and perceived personal responsibility.

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Notes
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