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ORIGINAL RESEARCH

Causal Reasoning About Human Behavior Genetics: Synthesis and Future Directions

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Abstract
When explaining the causes of human behavior, genes are often given a special status. They are thought to relate to an intrinsic human 'essence', and essentialist biases have been shown to skew the way in which causation is assessed. Causal reasoning in general is subject to other pre-existing biases, including beliefs about normativity and morality. In this synthesis we show how factors which influence causal reasoning can be mapped to a framework of genetic essentialism, which reveals both the shared and unique factors underpinning biases in causal reasoning and genetic essentialism. This comparison identifies overlooked areas of research which could provide fruitful investigation, such as whether normative assessments of behaviors influence the way that genetic causes are ascribed or endorsed. We also outline the importance of distinguishing reasoning processes regarding genetic causal influences on one's self versus others, as different cognitive processes and biases are likely to be at play.

Keywords Causation · Genetic essentialism · Psychological essentialism · Bias · Normativity · Science communication

Introduction

Thanks to advances in technology and information sharing, we are living in an age where genetic information is increasingly available. This information is also becoming more personalized, as direct-to-consumer genetic tests become more affordable and a wider range of tests are offered. However, more information does not always mean that people are better informed.

Instead, evidence suggests that the plethora of public information about genetics has resulted in biased and erroneous thinking (e.g. Christensen et al. 2010; Dar-Nimrod and Heine 2011a). These biases and errors can have important effects. For instance, experimental work has demonstrated that the perceived genetic etiology of a trait can affect behaviors related to self-control (Dar-Nimrod et al. 2014a), as well as cognitive performance (Dar-Nimrod and Heine 2006). Well-being, psychological distress, and self-stigma are also influenced by whether or not identity-related traits are perceived as having genetic causes (Morandini et al. 2015). In some instances, the way that people understand the genetic causes of behavior may be more powerful behavioral drivers than any actual specific genes related to the trait (Dar-Nimrod et al. 2014a). Although most of the research to date has identified deleterious effects of genetic essentialist thinking, research that demonstrates positive outcomes, such as reduced animosity between groups in conflict (Kimel et al. 2016), has been recorded as well. In this review, we show that the way in which people reason about genetic causation is influenced by general factors related to causal cognition, cognitive biases related to an individual's normative and moral beliefs, and biases that particularly relate to people's conception of the gene.

Causal cognition, the process by which some causes are selected as more salient or important than others, is influenced by multiple factors, which we outline in detail in "How people understand causation". Some of these factors relate to properties of the causal relationship observed; for example, how specifically a cause relates to an effect. Others are founded in personal belief systems about morality and normativity. Experimental work has established

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that causal information influences people's moral judgements, such as how blame and responsibility are attributed (Fig. 1a). Conversely, normative and moral judgements, such as whether a cause is thought to be unusual, morally good or bad, or whether an effect has a positive or negative outcome, has been shown to influence the amount of causal power ascribed, or the degree to which causal information is accepted (Fig. 1b). This is of particular interest in human behavior genetics, as some behaviors are judged to be more morally acceptable than others.

Contemporary research has begun to focus on how perceptions of genetic causation influences moral and normative attitudes about traits and the individuals possessing them (Fig. 1c). For example, when mental illness is thought to be genetically caused, individuals manifesting the trait are believed to be more dangerous (Kvaale et al. 2013), attitudes towards those individuals are more negative (Read and Law 1999; Walker and Read 2002), and those afflicted perceive less control and experience greater depression relating to their illness (Birchwood et al. 1993). These attitudes are thought to stem from both general principles of causal cognition, as well as specific genetic essentialist biases, outlined in "How people understand genetic causation".

It is important to note that the moral and normative attitudes elicited may differ depending on whether one is assessing the trait in others versus oneself. This is an underexplored area of research, and in "Genetic causes and normativity" we outline some preliminary evidence for this phenomenon and suggest areas for future investigation. Another area of research ripe for exploration is how pre-existing normative and moral attitudes about traits and behaviors influence the way in which genetic causes are perceived (Fig. 1d). When an effect is perceived to be negative, the cause is imbued with greater potency (Knobe and Fraser 2008); that said, as genetic causes have been argued to illicit their own set of cognitive biases (Dar-Nimrod and Heine 2011a), it may be prudent to explore such potential effects for them specifically. For example, are people more likely to accept genetic explanations for traits and behaviors that are perceived to be morally negative or abnormal? (Fig. 1d). In "Genetic causes and normativity" we discuss some preliminary associative evidence for this, and suggest new ways that these factors could be investigated, and potentially intervened upon as an approach to combat biases in genetic causal reasoning.

How people understand causation

To understand how people think about genetic causes, one must first understand how people reason about causation. Very young children are apparently sensitive to cause and effect relationships (Gopnik et al. 2004), though philosophers continue to debate the precise nature of these relationships (Schaffer 2016). One reason for this continued debate is that for any given effect a near infinite number of causes can be identified. This problem of profligate causes (Menzies 2004), which leads to individuals selecting particular causes as more salient or important than others, is termed causal selection (Hesslow 1988), attribution (Kelley 1973), or causal privileging (Oyama 2000a, p. 31, 201).

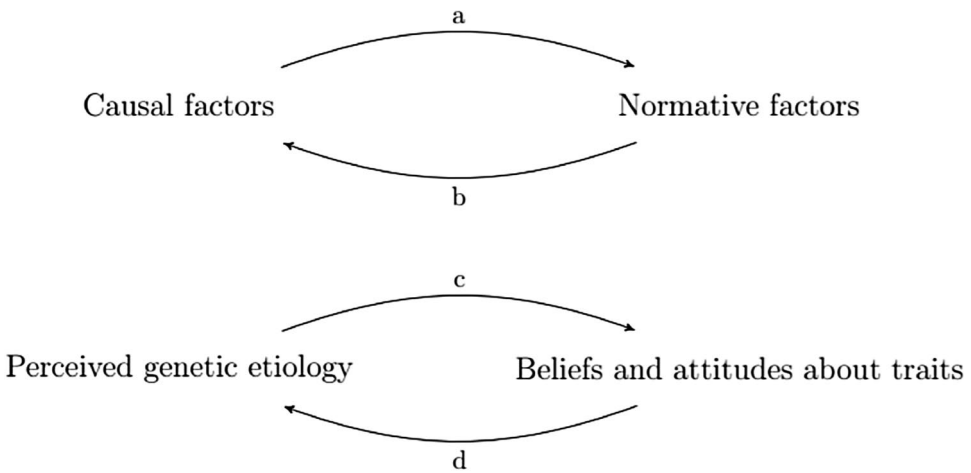

Fig. 1 The bi-directional relationship between perceived causes and normative ascriptions. (a) Represents how moral judgements are made based on causal data, a well-established precondition for ascriptions of blame and responsibility (for instance see Sloman et al. 2009). (b) denotes the effect of normative factors such as norm violation and moral valence on ascriptions of causation. (c) illustrates how perceptions of genetic etiology can influence attitudes and beliefs about traits, and bearers of traits, as has been demonstrated in empirical research. (d) represents are largely unexplored area of research: how pre-existing attitudes and beliefs about traits and trait-bearers might influence causal reasoning about genetics

The literature suggests that multiple characteristics are assessed when selecting or privileging causes. These characteristics fall into two broad categories: The 'person as a scientist' model of causal reasoning, whereby people reason about causation in a systematic way, using the causal information at hand (Kelley 1967); and the 'person as a moralist' model, where people reason about causation using pre-existing moral and normative judgements (Knobe 2010).

Person as a scientist

Specificity

An array of information is thought to be presented when an assessment of causation is made. The specificity of a causal relationship is thought to factor into an assessment of causation or causal strength. Causes that are specific to their effects, as opposed to factors that play a causal role in many different processes, appear to be more salient. For example, both the on/off switch and the position of the frequency dial are causes of the music received by a radio, but only the position of the dial is a specific cause of the music (Woodward 2010). This notion of 'causal specificity' has been explicated formally and informally in a number of different ways (Pocheville et al. 2017; Stotz 2006; Waters 2007; Weber 2006; Woodward 2010, 2011). The specificity of causal relationships is related to the perceived control that the cause exerts over the effect, a notion that has been linked to causal attribution and culpability (Alicke et al. 2011).

Stability and inevitability

Causal relationships which hold under a large range of contexts or background conditions are also thought to be more salient (Hitchcock and Woodward 2003; Mitchell 1997, 2000; Woodward 2006, 2010). In fact, the stability of a causal relationship across background conditions has been proposed as a quantitative criterion for scientific law-hood (Mitchell 1997, 2000). Stability captures how easily a causal relationship can be altered by other variables (Pocheville et al. 2017), and thus is likely to be related to the perceived inevitability (Lynch and Bourrat 2017) and ease of intervention (Woodward 2001) of causes. As a result, the stability of a causal relationship is related to the proximity of the cause and effect in both time and space. The more distal a cause and effect are from one another, the more causal links lie between them, and the greater the chance that the relationship could be disrupted by background perturbations (Woodward 2006).

Directness

Related to proximity, how direct or indirect a cause is to its effect seems to play a particular role in causal attribution in human behavior. Block and Dworkin (1976) emphasize the special causal role played by genes acting 'directly' on phenotypes, sustaining their effects within the boundary of the body, compared to genetic effects that influenced behavior through environmental intermediaries. Thus whether or not a cause is internal or external to an individual's physical body impacts on how it is assessed causally. Lynch (2017) shows how this distinction has made its way into the field of behavior genetics in the scientific discussion of gene-environment correlations. Both active and reactive gene-environment correlations have the same causal structure, yet are distinguished based on an internal/external divide. Further, Monterosso et al. (2005) found that physiological explanations of undesirable behaviors were exonerated more than experiential ones. A meta-analysis (Kvaale et al. 2013) indicated that biogenetic explanations for mental illness are associated with reduced blame related behaviors compared with other explanations.

Application in the sciences

Causal selection based on dimensions like specificity, stability, and proximity parallels the way in which causes are selected in the sciences. Generally, a drug treatment is picked out as a candidate cause of illness recovery if the relationship between the drug and recovery holds under a range of contexts, like demographic variation; specifically targets the illness it is treating; and it produces its effect through the body. It is thought that genetic causes are routinely privileged by scientists above other cellular causes of phenotypic development because their relationships to traits are perceived to be specific (Griffiths and Stotz 2013; Woodward 2010), stable and inevitable (Block and Dworkin 1976; Dar-Nimrod and Heine 2011a; Kaebnick 2006) and direct (Block 1995; Block and Dworkin 1976; Lynch 2017; Lynch and Bourrat 2017).

Person as a moralist

Morality

The 'person as a scientist' model is in opposition to the 'person as a moralist' model, under which moral factors influence causal selection and attribution (Fig. 1b; Alicke 1992; Alicke et al. 2011, 2015; Hitchcock and Knobe 2009; Knobe 2010; Kominsky et al. 2015). In empirical settings, morally negative actions such as stealing pens are attributed more causal power for a given effect (a lack of pens) than morally neutral or positive ones such

as taking pens with permission (Knobe and Fraser 2008). Further, these effects differ in correspondence with differences in prescriptive judgements of morality, such as on issues like abortion and euthanasia (Cushman et al. 2008). Additionally, the *motivation*s or *perceived culpability* of individuals make a difference to the causal attribution imparted upon their behavior. For example, an individual involved in a car accident after speeding home in order to hide a present for his parents was judged not only to be less morally responsible, but less causally responsible for the accident compared to one who was speeding home to hide a vial of cocaine (Alicke 2008; see also Samland and Waldmann 2016). The moral valence of the effect also mediates causal attribution: people are more inclined to attribute greater causality to morally negative outcomes (Alicke et al. 2011; Icard et al. 2017).

Normativity

An alternative interpretation of this phenomenon is that judgements of normality, rather than morality influence causal attribution (Driver 2008; Halpern and Hitchcock 2015; Hitchcock and Knobe 2009; Knobe and Fraser 2008; Philips et al. 2015). Morally negative actions, such as pen-stealing and cocaine hiding, are generally also more abnormal, and these authors suggest that people are more inclined to attribute causation on the basis of abnormality. This is the case for both statistical abnormalities (Hilton and Slugoski 1986), and events or actions which are viewed as prescriptively abnormal (Icard et al. 2017; Knobe and Fraser 2008; Solan and Darley 2001), with some indication that prescriptive norms have a greater influence on perceptions of causality (Roxborough and Cumby 2009; cf. Bear and Knobe 2017).

Causal cognition

Current research indicates that people operate both as scientists and as moralists when making causal assessments. Characteristics of causal relationships, such as the specificity, stability, inevitability, ease of intervention, and directness of cause to the effect, work in concert with pre-existing beliefs about the morality and normativity of causes, effects, and agents. This array of influences on causal reasoning are listed in Table 1. In the next section we review what is known about influences on genetic causal reasoning. The current model for understanding genetic causal reasoning is the Genetic Essentialist Framework (GEF) (Dar-Nimrod and Heine 2011a). We show in the next section how many elements of the GEF can be understood in terms of the general influences on causal thinking outlined in this section. By comparing this framework with what is known about causal cognition, we also illuminate some gaps in the literature where one framework may inform the other, indicating new areas of potential research.

How people understand genetic causation

Popular perceptions of genetics are dominated by the idea of 'genes for' specific behaviors. A nationally representative survey of adults in the United States found that 76% believe that "single genes directly control specific human behaviors" (Christensen et al. 2010, p. 470). Mass-surveys like this one consistently suggest low levels of public understanding (Durant et al. 1989) and the idea that the public simply fails to understand science has been termed the 'deficit model' (Ziman 1991, p. 101). The deficit model has been much-criticized and most science communication researchers reject it, arguing that the adequacy of public understanding of science cannot be reduced to the degree to which laypersons share

Table 1 A comparison of elements from the genetic essentialist framework and factors known to influence causal reasoning

Genetic essentialist elements (Dar-Nimrod and Heine 2011a)	Influences on causal reasoning
Immutable and determined	Stability (Mitchell 1997; 2000)
	Inevitability (Lynch and Bourrat 2017)
	Ease of intervention (Woodward 2001)
Specific etiology	Specificity (Stotz 2006; Weber 2006; Woodward 2010)
	Direct versus indirect (Lynch 2017; Lynch and Bourrat 2017)
	Internal versus external cause (Block and Dworkin 1976; Lynch 2017)
Naturalness	Abnormality of cause (Hitchcock and Knobe 2009; Knobe and Fraser 2008; Halpern and Hitchcock 2015)
	Moral valence of cause (Alicke 1992, 2000; Knobe and Fraser 2008)
Homogeneous and discrete	
	Moral valence of effect (Alicke et al. 2011; Icard et al. 2017)

Empty cells indicate a gap in the literature where one framework may inform the other

understanding with scientific experts (Turney 2003). These critics argue that "the public articulates complex understandings of genetic research" (Bates et al. 2005, p. 340), albeit not technically correct ones. However, it is not necessary to endorse the deficit model in order to be concerned about the public understanding of genetics, as one of us has argued at length elsewhere (Griffiths 2017). The cognitive biases we describe can affect both laypersons and scientific experts (Knobe and Samuels 2013). Our concern about the effects of these biases on public understanding is not that they lead the public to disagree with experts, but that they lead people, whether expert or public, to draw conclusions not warranted by the evidence presented to them.

Although specific mutations can wreak havoc on a host of physical and/or cognitive capabilities (e.g., as in the case of PKU), single genes do not control specific behaviors. Instead, genes influence behavior via a complex web of causal influences, as elaborated elsewhere (Dar-Nimrod and Heine 2011a; Heine 2017; Turkheimer 2000). Even among less complex organisms, it is hard to find examples of single genes controlling specific behaviors. The nematode worm *C. elegans* has been developed over many decades as a tractable model organism in which to elucidate the basic principles by which genes give rise to behavior. It has around 13,000 genes and 1000 cells of which 300 are neurons. Reviewing current knowledge about this important model organism, Schaffner (1998, 2016) points out that many genes are involved in the development of each neuron; that many neurons are involved in each behavior, and that these circuits frequently overlap; that is, any one gene is involved in the genesis of many neurons and can affect many behaviors, as can any one neuron. Furthermore, the worm's environment has a large influence on both the development of neural networks and the behavior produced by those networks, and the process by which genes (and environment) act to wire together the neurons is stochastic rather than deterministic. If there are very few 'genes for' specific behaviors even in the worm, it is unlikely that there will be 'genes for' complex human behaviors.

Philosophers and historians of genetics have hypothesized that unrealistic expectations about the role of genes arise from the view that genes exert causal influence in a special way—'the notion that some influences are more equal than others' (Oyama 1985, p. 31; see also Keller 2000; Moss 1992). One hypothesis is that only genes are regarded as *information* or *instructions*, whilst other factors are regarded as merely causes (Griffiths 2001, 2006; Griffiths and Gray 1994; Oyama 1985, 2000b). Griffiths (2002) offered another suggestion, stating that the 'privileging' of genetic causes over others might arise because genes are treated as the 'essence' of an organism in the manner described by Medin and Ortony (1989) (see also Dar-Nimrod and Heine 2011a; Nelkin and Lindee 1995).

Essentialism

The concept of a metaphysical essence that makes organisms what they are has traditionally been ascribed to Aristotle (e.g. Hull 1965), but has much older roots and is arguably a fundamental aspect of 'folkbiology' (Atran 1990). This essence is a category-enabling construct; that is, it is perceived to *produce* the characteristics shared by members of a natural category, such as an animal species, and is not the *outcome* of such membership. The human tendency to behave as if categories are based on defining and productive essences is termed *psychological essentialism*. Whilst essence as a metaphysical concept is intractable (Medin and Ortony 1989), evidence for the ubiquity of psychological essentialism as a psychological phenomenon is quite striking. It is clearly found in different cultures, both among children (e.g. Astuti 2001; Gelman 2003; Sousa et al. 2002) and among adults (e.g. Gelman 2003; Gil-White 2001; Haslam et al. 2000; Henrich and Henrich 2007; Waxman et al. 2007), although it seems like there are meaningful variations in essentialism-proneness among populations (e.g. Giles et al. 2008; Kraus et al. 2012).

The perceived essence of an organism, which is largely resistant to environmental and experiential alterations (for a review see Gelman 2003), has been argued to be incorporeal (Medin and Ortony 1989). Such an immaterial nature affords the capacity to make inferences, accurate or otherwise, relying on prototypical or stereotypical cognitions (Chao et al. 2013; Chao and Kung 2015; Plaks et al. 2004; Rothbart and Taylor 1992). Given such abstractness, the psychological essence can assume various forms to ease the discomfort of using what Medin and Ortony (1989, pp. 184–185) termed an "essence placeholder." Such a placeholder has been historically replaced with concrete (yet still abstractly defined) elements such as the Hippocratic humors in Ancient Greece, the energy force encompassed by the concept of Ch'i in China, or the Indian yogis' conceptualization of life force—Prana (Heine et al. 2017). Genes are material, rather than immaterial as Medin and Ortony's placeholder concept suggests, but lay people's perceptions of them suggest that they are not seen as such, instead they have been argued to represent a meme, or a cultural icon (Nelkin and Lindee 1995), that embody the same descriptors that underlie the metaphysical essence. They are viewed as natural, innate, causal, stable, and immutable; thus, Heine et al. (2017) argue that genes are particularly fit as an essence placeholder in contemporary society (see also Cheung et al. 2014; Dar-Nimrod and Heine 2011a; Heine 2017).

Despite harboring misconceptions about genes and their operations, people readily invoke genes to explicate a broad range of human afflictions, capabilities, and behaviors (e.g. Condit et al. 2004; Dar-Nimrod et al. 2014b; Gelman and Wellman 1991; Sheldon et al. 2007). For example,

explaining why a child who was born to unintelligent parents but was raised from day one by intelligent parents will have intellectual difficulties, a fifth grader announced that "It will have trouble. It's in its genes" (Heyman and Gelman 2000, p. 672). Representing characteristics and conditions (such as obesity, criminality, mental illnesses) as genetically derived, despite ample scientific evidence to the contrary, is pervasive in cultures exposed to accounts of scientific advances in genetics (Condit 1999; Nelkin and Lindee 1995). These simplified misrepresentations are palatable, simple to digest, and are commonly tainted by an erroneous fatalism (Alper and Beckwith 1993; Conrad 1999; Dar-Nimrod and Heine 2011a). Such misrepresentations are not created in a vacuum—much research has been directed towards understanding the formation of such misrepresentations with agents such as the media, the entertainment industry, and even scientists themselves (Conrad 1997, 1999, 2002; Dar-Nimrod and Heine 2011a; Nelkin and Lindee 1995). A genetic education curriculum focusing on Mendelian models that highlight deterministic inheritance only exacerbates the problem (Dar-Nimrod 2012; Donovan 2016; Dougherty 2009).

It has been argued that there is nothing distinctive about the failure of laypersons to understand genetic causation—human beings prefer simple, often mono-causal explanations in many other domains (Kitcher 2001). Hence the claim that genes are treated by laypersons as essence placeholders stands in need of empirical verification (see Dar-Nimrod and Heine 2011b; Haslam 2011; Turkheimer 2011, for a related discussion in the psychological literature). Following Griffiths' 2002 hypothesis, some 'experimental philosophy' (X-phi) research (Griffiths et al. 2009; Linquist et al. 2011) was conducted to confirm that labelling behavior 'innate' or 'in the DNA' expresses the idea that the behavior is a manifestation of an essence that characterizes a species (typicality), that it is hard to change (fixity), and is meant to be (teleology). In the meantime, the same hypothesis had been proposed in psychology, where it was elaborated in greater depth. The extensive empirical literature examining and developing the hypothesis is outlined in the next section.

Genetic essentialism

The seeming fit of the lay conceptual understanding of genes as a proxy for the metaphysical essence is termed genetic essentialism. Dar-Nimrod and Heine (2011a) offered a theory to delineate the cognitive processes set in motion once a person perceives a genetic etiology. Termed the GEF, the theory posits that genetic attributions for various traits, conditions, or diseases activate psychological biases (Fig. 1c), *Genetic Essentialist Biases*, leading to a surge in the likelihood that people view outcomes as: (1) *immutable* and *determined*, (2) having a *specific etiology* (which leads to a devaluation of other contributing causes), (3) resulting in different groups that are perceived as *homogenous* and *discrete*, and (4) *natural*.

A multitude of studies (e.g. Dar-Nimrod et al. 2013, 2014b; Lebowitz and Ahn 2014; Parrott and Smith 2014) now provides evidence supporting the GEF in a variety of areas ranging from individual characteristics to social groups and from explicit measures to implicit indications (for a recent comprehensive review see Heine et al. 2017). For example, Gould and Heine (2012) found an association between the concept of genetics and fatalism using implicit measures. Using explicit methods, No et al. (2008) demonstrated that people who more strongly endorse genetic-based racial theories see greater personality differences between racial groups. In a more expansive demonstration, Keller (2005) not only showed that stronger belief in the deterministic power of genes was associated with racial stereotyping, but also other indicators of prejudice, as well as entitativity and nationalism. In sum, the GEF suggests that when people label phenomena (e.g., traits, behaviors, diseases) as "genetic" these phenomena are, in turn, not only viewed as having a specific cause, but they are also viewed as more natural, discrete, and immutable, all of which could have profound consequences for future behaviors.

Genetic causal reasoning and the GEF

The four defining elements of genetic essentialism identified by Dar-Nimrod and Heine (2011a) can be compared to the factors listed in "How people understand causation" relating to perceived causal strength and causal attribution (Table 1). This comparison provides a novel synthesis, indicating how general influences on causal reasoning, which have been shown to work across contexts, also play a role in instances of genetic causation.

Immutable and determined

Implicit in the GEF are causal notions. Genes are not assessed in isolation, but in a causal relation to their phenotypic effects. For instance, that genes influence phenotypes in a way that is immutable and determined undergirds the type of causal relationship between genes and phenotypes. A gene determining its phenotype could mean that the phenotype occurs no matter the other factors in the causal background, that is, the causal relationship is *stable*, something which has been suggested as a general factor influencing causal attribution. Immutability also refers to the fact that the gene-phenotype relationship is inevitable, which as shown in "How people understand causation", is related to stability. In genetics, this inevitability is likely related to the fact that, at least until very recently, it has been difficult to intervene on genes or genotypes, and comparatively easy to intervene on environmental variables.

Specific etiology

The perceived specific etiology of genes refers to specificity of the causal relationship between genes and a trait. As described in "How people understand causation", specificity is a general characteristic that influences causal reasoning, and a range of specificity concepts are thought to impact how causes and causal relationships are assessed. This includes the notion that causes are particular to their effects, something exemplified in the 'gene for' discourse of genetic explanations. Specificity has been explicitly invoked as a reason for privileging genes over others causes (Waters 2007; Weber 2006). However, the specificity of genes to their effects has been disputed (Griffiths and Stotz 2013; Griffiths et al. 2015; Stotz 2006). In particular, models utilizing a formalization of the specificity concept demonstrated that the specificity of causal relationships between genes and their products compared to other causes of those products, such as splicing factors, depends on the details of the causal model used (Griffiths et al. 2015; see also Calcott 2017). This indicates the 'perceived specific etiology' element of the GEF is related to broader implicit notions about the causal relations of genes to their effects.

In the GEF, specific etiology is thought to lead to the devaluation of other causes, leading to the tendency to discount additional causal explanations once genetic contributions are made. For instance, an item on the Genetic Essentialist Tendencies Scale (GETS) (Dar-Nimrod et al. 2018) measuring specific etiology is: "The environment does not affect the chances of getting cancer for someone with a genetic susceptibility to cancer". This seems related to the general tendency to downplay environmental (external) causes when internal ones are present. Block and Dworkin (1976) have explicitly argued that scientists should treat genetic effects which exert their influence through internal mechanisms as different kinds of causes than others. That is, they claim that genetic effects that act 'directly' through internal biochemical pathways alone, as opposed to 'indirectly', where genetic influences extend into the environment beyond the body, are the only kind of effects that should be taken into account when estimating the heritability of a trait. This is despite the fact, as demonstrated by Lynch (2017; also see Lynch and Bourrat 2017), that both direct and indirect heritability cases are causally identical, except for the internal/external feature. This kind of causal reasoning also appears to occur amongst the broader public. Monterosso et al. (2005) showed that causal explanations of behavior stemming from internal biological factors such as a chemical imbalance in the brain are treated differently to those that are environmentally derived. The behavior is more likely to be considered automatic, and the individual involved less blame-worthy for their actions.

Naturalness

Dar-Nimrod and Heine (2011a) suggested that the perceived 'naturalness' of genes could prompt the naturalistic fallacy; whereby ethical properties are derived from the suggestion of natural ones. In particular, occurrences that stem from natural causes are judged as more morally acceptable than ones that stem from other causes. For example, homosexuality may be viewed more positively if perceived as a natural developmental outcome (Haslam and Levy 2006; Hegarty and Pratto 2001). If genes are judged with a positive moral valence because of preconceptions about naturalness, then it is likely that the way in which genes are causally regarded will also be influenced by these normative influences, as we have seen in "How people understand causation".

Alternatively, the perceived 'naturalness' of genes may lead people to believe that they are more normal causes of traits compared to other factors. Some have argued that instead of morality, it is the perceived abnormality of causes which influence the way in which people causally reason (Hitchcock and Knobe 2009). If naturalness is conceptually related to normality, and genetic causes are regarded as more (or less) normal causes of behavior, then it may be this feature that is responsible for the way in which genes are causally assessed.

Homogenous and discrete

Dar-Nimrod and Heine (2011a) argue that because psychological essence is a category-enabling feature, perceiving genes as the embodiment of essence increases the likelihood that the groups boundaries are perceived as less permeable (as the essence is stable and immutable). Along the same line, the shared essence of members of the same social group increases the likelihood that these individuals will be judged as more similar to each other, or homogenous. Curiously, the categorization of effects into continuous and discrete categories, shown to relate to the concept of genetic causation in the GEF, has not been explored for causal reasoning in other contexts (Table 1). This suggests that either this is a feature specific to or pronounced in the context of genetic causation compared to other contexts, or, that it is a general factor in causal cognition that has not yet been identified in other contexts. Further work is needed in the domain of causal reasoning to determine whether or not this feature can be detected more broadly.

Moral valence of the trait

Integrating the literature on causal reasoning and the GEF also illuminates another potential gap ripe for investigation. Empirical work on causal reasoning has shown whether or not an outcome is perceived as normatively good or bad

influences causal attributions. For instance, Alicke et al. (2011) used vignettes involving a doctor whose decisions to administer a drug either caused a good (patient recovery) or a bad (patient death) outcome. They found that when the doctor was following hospital protocol, they were attributed more causal responsibility for the positive outcome. However, if the doctor's decision was thought to violate hospital protocol, then causal attributions were stronger in the negative outcome condition. This illustrates the interactive effect of the normativity of the cause (violating protocol or not), and the moral judgement of the effect. Beliefs about genetic causation, which may themselves be related to ideas about naturalness and normality, have been shown to affect normative judgements about their phenotypic effects (as reviewed below). Although scholars have speculated that pre-existing normative beliefs about traits may influence how people reason about their etiology, to date no experimental research has examined this relationship. That is, at this point we do now know whether existing judgements about the morality or normality of traits (i.e., the effects) affect the degree to which people will come to accept or ascribe genetic causes.

Genetic causes and normativity

Given that the moral valence of effects influences how people causally reason (Fig. 1b), the way in which the perceived moral valence of traits influence perceptions of genetic causality is of interest for those involved in understanding genetic causal reasoning. There is some literature indicating a relationship between the moral valence of traits and genetic causality, yet the work is largely associative, meaning that perceptions of genetic etiology influencing beliefs about traits (Fig. 1c) often cannot be distinguished from beliefs about traits influencing perceived genetic etiology (Fig. 1d). Below we review the evidence for the relationship between normativity of a trait and attributions of genetic causation, with particular reference to the genetic basis of sexuality.

Causal evidence

Perceptions of others

Beliefs about genetic causation appear to influence how people perceive social groups, as well as how people conceive of their own identities. The former has been a focus of significant theoretical and empirical work. Starting with Allport (1979), social psychologists observed that the belief in group defining essences (often assumed to stem from "genes") appeared to be part of holding prejudiced beliefs. In the GEF, Dar-Nimrod and Heine (2011a) argue that when group characteristics are understood to stem from genetic causes, the likelihood of seeing social group memberships as *immutable* and *determined* increases as genes themselves are viewed in such a way. As such, relevant negative group characteristics are more likely to be viewed as being inherent and unalterable (as opposed to ones resulting from social or environmental forces). Such beliefs may be used to justify social inequalities between groups as natural and inevitable and to rationalize discriminatory attitudes and practices toward these groups (Jost et al. 2004). Genetic essentialist biases also lead us to view social group membership as being *discrete, informative* (i.e., groups are viewed as a rich source of inferences about the characteristics of individuals within the group), and group members as seen being *homogeneous* (Dar-Nimrod and Heine 2011a; Haslam et al. 2002). By sharpening group boundaries, increasing perceptions of difference between one's own and other groups, and promoting stereotypical thinking about outgroup members, this set of biases is thought to result in more negative attitudes to outgroups (Haslam et al. 2002; Heine et al. 2017). An example of empirical support for this argument can be found in Williams and Eberhardt (2008). This study showed that White American participants who were primed with an essay that described race as a biological construct showed more prejudiced attitudes towards a Black American protagonist compared with White Americans who were primed with an essay that described race as a social construct.

A small number of experimental studies have examined the causal relationship between genetic theories and attitudes toward homosexuality. These studies have demonstrated mixed results and mixed effectiveness. In one study learning about biological causes of homosexuality reduced anti-gay attitudes (Piskur and Degelman 1992), while in another it increased anti-gay attitudes (Oldham and Kasser 1999), and in a third had no effect (Pratarelli and Donaldson 1997). A further study Boysen and Vogel (2007), found that exposure to biological theories led to attitude polarization (i.e., strengthening of pre-existing attitudes). Thus, those already tolerant found biological evidence credible and became more sympathetic to LGB individuals, whilst those holding anti-gay attitudes tended to view these theories critically, and became less tolerant (Boysen and Vogel 2007).

Perceptions of self

Although considerable work has examined how lay theories of genetic causation influence our perception of others, less has examined how they may color perceptions of self. Roth and Lyon (forthcoming) report that the majority of individuals who learned of their racial/ethnic ancestry from genetic reports describe an impact of this information on their lives from changes in their identity, through support of sport teams in international competitions, to joining native tribes or learning a new language. One recent line of work suggests that genetic essentialism can influence self-stigma among

minority individuals. For instance, experimental work by Coleman and Hong (2008) has found that women who were exposed to biological explanations of gender differences were more likely to engage in self-stereotyping, reporting greater endorsement of "negative" feminine traits, than when exposed to accounts which emphasized social and environmental causes. Likewise, Dar-Nimrod and Heine (2006) found that when women were presented with claims that men are genetically superior at mathematics, they demonstrated poorer math performance than when exposed to non-genetic (experiential) causes of these gender differences.

Perceived etiology

For research involving causal manipulations, the focus so far has been to investigate how beliefs about genetic causation influence the way in which people form attitudes about phenotypes, and the individuals who manifest those phenotypes (self and other) (Fig. 1c). What is yet to be explored is whether pre-existing normative beliefs about phenotypes (i.e., whether a phenotype is perceived as a deviation from the norm or not) and the moral valence of the phenotype (i.e., whether the phenotype is deemed desirable or undesirable) influence the way in which people causally reason about genetics. That is, do beliefs about phenotypes influence the way in which genetic causal information is assessed, accepted, or selected (Fig. 1d)? To this point, no published studies have sought to manipulate the desirability of a phenotype to examine how it influences genetic attributions of that phenotype.

Associative evidence

Perceptions of others

Correlational studies have found that those who endorse genetic essentialist beliefs about certain social groups (including women and racial minorities), are more likely to endorse stereotypes and hold more prejudicial attitudes about these groups (Bastian and Haslam 2006; Jayaratne et al. 2006; Keller 2005; Martin and Parker 1995). For some other social groups, such as gay/lesbian individuals, genetic essentialist beliefs look to be a double-edged sword. On the one hand, perceiving homosexuality as biologically determined, immutable, and fixed early in life has been found to be associated with greater tolerance toward gay/lesbian individuals (Boza 2016; Haslam and Levy 2006; Haslam et al. 2000, 2002; Hegarty and Pratto 2001). This is commonly explained by citing causal perceptions influencing normative ones (Fig. 1c), via attribution theory in the context of stigma (Weiner et al. 1988) (i.e., when stigmatized characteristics are perceived as uncontrollable [as in having a "gay gene"], individuals are judged less harshly for behaving in line with the underlying genes). An alternative view, forwarded by Hegarty (2002), is that rather than genetic attributions of homosexuality causing more positive attitudes toward gay men and lesbians, they have become symbolic markers of pro-LGBT politics due to opponents of gay rights traditionally framing homosexuality as a lifestyle that is "chosen". However, it is also possible that these results are explained by normative perceptions influencing causal ones, such as if those which have pre-existing positive or tolerant attitudes about homosexuality are more likely to accept or believe in genetic explanations for the trait (Fig. 1d).

Perception of self

On the other hand, there is evidence that when socially undesirable traits are self-relevant, individuals will often emphasize genetic causes over environmental ones. For instance, evidence suggests that sexual minorities endorse biological explanations of their homosexuality (Morandini et al. 2015, 2017), and there is evidence that those with eating disorders will often attribute their difficulties to biological factors outside of their control (Easter 2012). In both cases, causal reasoning appears driven, at least partly, by attempts to alleviate self-responsibility for the undesirable trait or to strategically counter blame and stigmatization (from others) for possessing the undesirable trait.

Interestingly, self-serving biases dictate that explanations for one's own successes are attributed to internal, dispositional factors, whereas failures are attributed to external factors outside of one's control (Mezulis et al. 2004). This pattern is reversed for explanations of others' behavior, particularly when others are perceived to be outgroup members. In these cases, negative outcomes are judged to be caused by internal factors, and positive ones to situational factors (Pettigrew 1979). This suggests that despite the physical internality of genes, people may consider them as external to the self, and thus related to self-identity in a unique and interesting way. The idea that what is authentic for an individual is dictated by their genetic endowment is popular in the broader culture. In fact, various identity movements, including contemporary gay and transgender rights movements, often argue that their fight for legal and political recognition is necessary so that they can express their inborn phenotypes in an authentic manner.

However, viewing one's minority status as anchored in one's genetics has also been found to be associated with positive self-perception. For instance, many LGB individuals embrace genetic theories of their sexual orientation. Indeed, following Hamer et al. (1993)'s discovery of the Xq28 genetic marker of male homosexuality, a gay-pride T-Shirt became popular among gay men in the Bay Area of San Francisco which read "Thanks Mom for Xq28" (Hamer 2011). Relatedly the slogan "born this way" has become the

catch cry of contemporary LGB activists in recent struggles over gay rights (Jang and Lee 2014). Of course whether genetic theories of homosexuality actually changed gay men's attitudes toward their own sexuality is speculative—it is also possible that LGB individuals simply made use of scientific research to further their struggle for gay rights.

Recent correlational studies undertaken in our lab have found that gay men who perceive their sexuality as being genetically based and immutable report less uncertainty about their sexual identity, and in turn less self-stigma and greater psychological well-being (Morandini et al. 2015). Similar findings have been observed among lesbian and bisexual women, whereby biological determinist accounts of sexual orientation are associated with less self-stigma (Morandini et al. 2017). Morandini et al. (2015) speculated that much in the same way that attributing homosexuality to uncontrollable factors (such as "genes") may reduce heterosexual's prejudice toward sexual minorities, so too may attributing one's same-sex desires to genes relieve self-blame. Furthermore, it is possible that perceiving sexual orientation as inborn and immutable may reduce attempts to change one's sexual orientation (Tozer and Hayes 2004). However, it is possible to give a different causal account of these associations, whereby existing psychological well-being or lower self-stigma facilitate a greater belief in the genetic etiology of identifying traits, including perceived immutability (Fig. 1d).

Areas for future research

As shown in "How people understand causation", normative beliefs about outcomes, such as the perceived moral valence of an effect influences the way in which people causally reason, and interacts with other features of causal reasoning, such as the perceived normality of the cause (Fig. 1b; Alicke et al. 2011; Cushman 2008). This phenomenon is yet to be demonstrated regarding the causal relationship of gene to phenotype.

At present, there remain a range of unanswered questions about the manner in which moral and normative beliefs about phenotypes influence how people reason about or endorse genetic causation. To our knowledge no published work has explicitly examined whether manipulating either of these factors influences perceptions of phenotypic etiology. To address this gap, one may want to assess differences in endorsement of genetic explanation for specific behaviors after manipulation of perceived normative beliefs (by offering for example, bogus information about normative behavior in a specific culture, including the endorsement of such norms in the population in question), or by manipulations of moral standing (by comparing, for example, evaluations of protagonists who exhibit praiseworthy versus condemned behaviors).

Conclusion

People do not always reason about the world rationally. Experts and laypeople are subject to biases in reasoning about cause and effect relationships, and so it should come as no surprise that these biases rear their heads in the understanding of genetic causation. Foremost among these is motivated cognition, whereby people's ontological beliefs about the word are influenced by their personal and group interests. Genes appear to be held in a particular regard when it comes to the causes of human traits and behaviors. It is thought that this is because they fulfil the psychological role of an essence placeholder (Griffiths 2002), and as such, are ascribed particular properties which relate to their causal influence on traits (Dar Nimrod and Heine 2011a).

The ascription of these properties to genes result in specific biases conceptualized by the GEF. This paper illustrates how the GEF biases can be understood within a broader context of factors which influence causal reasoning across settings. Our synthesis shows how multiple elements that are known to influence causal reasoning, such as stability, inevitability, specificity, moral valence, and normality, map to the four defined elements of the GEF (Table 1). Interestingly, one feature of the GEF—that genetic attributions for social groups' differences increases the likelihood that the individuals in the group are viewed as more homogeneous and discrete from members of other groups, shares no parallel in the broader literature on causal reasoning. This suggests the need for investigation as to whether this factor influences causal attributions across a range of other contexts.

That causal reasoning is influenced by moral and normative factors is of particular interest for understanding the way in which genes are perceived. There are two ways that morality has been demonstrated to influence causal reasoning. First, the perceived moral valence of a cause, whether it be an action, or the motivations of an actor, have been shown to make a difference to the degree of causation ascribed to that actor or agent (Alicke 1992, 2000; Knobe and Fraser 2008; Samland and Waldmann 2016). Second, the perceived moral valence of an effect has been shown to influence the way in which causes are ascribed (Alicke et al. 2011; Icard et al. 2017). In the context of genetic causation, moral attitudes about phenotypes are associated with judgements of genetic causation. This could be explained in two ways. The moral valence of a cause can be understood in terms of the GEF, where the naturalistic fallacy is employed, and genes are judged as having a positive moral valence. Thus the moral valence of the cause could influence causal ascriptions. However, the second relationship, where the moral valence of a phenotype influences causal judgements, is yet to be explicitly investigated in the domain of genetic causation.

There is also population-level variation on how some behaviors are morally judged, for example, with phenotypes like sexual orientation. An experimental investigation into whether the perceived moral valence or perceived abnormality of these traits influences the way in which genetic explanations are received or accepted will provide stronger evidence that it is associations with abnormality and morality, and not some confounding factor, which relate to judgements of genetic causation.

Also to note for future research is the potential for differences in assessment for one's own versus others' behaviors, and for ascriptions concerning others' from a different social group, as these factors have been shown to influence ascriptions of causation and blame across other contexts (Mezulis et al. 2004; Pettigrew 1979). Preliminary correlational evidence suggests that individuals will emphasize genetic causes over environmental ones for their own socially undesirable traits (Morandini et al. 2015), whereas they will emphasize environmental causes for undesirable traits in others (Haslam and Levy 2006). This is an apparent reversal of self-serving biases (Mezulis et al. 2004), and to some extent, the fundamental attribution error (Pettigrew 1979), whereby one's own negative behaviors are attributed to external causes, and others' negative behaviors are attributed to internal or dispositional ones. This suggests that how genetic causes relate to moral and normative beliefs could interact in interesting way, yet to be revealed.

Correlational literature supports the notion that people's reasoning about the causes of phenotypes is often linked to their moral or social evaluations of these phenotypes (e.g. Suhay and Jayaratne 2012). In relation to others, when phenotypes are perceived as particularly undesirable, individuals tend to downplay genetic causation, and instead foreground the role of choice and environmental factors. This may be in order to hold individuals morally accountable for undesirable or deviant behaviors (associated with these phenotypes) and/or to frame the phenotype as "unnatural". For example, those who hold anti-gay attitudes appear more likely to conceive of homosexuality as a deviant lifestyle choice, which is mutable and culturally specific, than as a stable disposition with a genetic etiology (Haslam and Levy 2006).

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Compliance with Ethical Standards

Conflict of interest Kate E. Lynch, James S. Morandini, Ilan Dar-Nimrod and Paul E. Griffiths declare that they have no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

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