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Research report

Can merely learning about obesity genes affect eating behavior? ☆

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ABSTRACT

Public discourse on genetic predispositions for obesity has flourished in recent decades. In three studies, we investigated behaviorally-relevant correlates and consequences of a perceived genetic etiology for obesity. In Study 1, beliefs about etiological explanations for obesity were assessed. Stronger endorsement of genetic etiology was predictive of a belief that obese people have no control over their weight. In Study 2, beliefs about weight and its causes were assessed following a manipulation of the perceived underlying cause. Compared with a genetic attribution, a non-genetic physiological attribution led to increased perception of control over one's weight. In Study 3, participants read a fictional media report presenting either a genetic explanation, a psychosocial explanation, or no explanation (control) for obesity. Results indicated that participants who read the genetic explanation ate significantly more on a follow-up task. Taken together, these studies demonstrate potential effects of genetic attributions for obesity.

Introduction

"Battle your biology? Fat chance" lamented a headline in the *New York Post*, which provided a range of evidence indicating that people's genes largely determine their weight, implicitly and explicitly suggesting that the attempt to control one's weight is a futile endeavor (Cohen, 2000). In the science sections of respectable newspapers, one frequently finds such deterministic headlines followed by fatalistic portrayals of genetic involvement in obesity (e.g., Devlin, 2013; Kolata, 2007).

The attractiveness of such genetic explanations for obesity is rooted arguably in people's common perceptions that genes are the locus of the essence of individuals and groups (Dar-Nimrod & Heine, 2011), but it may also be facilitated by the growing body of relevant obesity research. In fact, among the obesity-related research projects funded by the National Institutes of Health, the percentage of abstracts containing the term "gene" steadily increased from 15% during the 1991–1993 period to 37% during the 2009–2011 period (I. Dar-Nimrod, unpublished data; available upon request). This increase in funding is reflected in a plethora of genetics-focused articles on obesity continuously published in premiere scientific journals (e.g., Frayling et al., 2007; Pearce et al., 2013). The increase in research and media attention to the genetic underpinnings for obesity appears to have an effect on laypeople; a comparison between two national polls conducted 20 years apart shows that whereas in 1979, 36% of the respondents perceived heredity to be more important than the environment in determining whether a person was overweight, in 1995, 63% of the respondents endorsed the belief that being substantially overweight is largely determined by genes (Singer, Corning, & Lamias, 1998). Furthermore, these etiological perceptions prove to be important to people – Segal, Polansky, and Sankar (2007) found that some parents are interested in learning about their children's genetic susceptibility to obesity even before birth, and believe that such information should be shared with children around the age of 10.

But how do people respond to genetic explanations for obesity? Past research has found that people sometimes respond to genetic explanations for various phenomena in seemingly irrational and counterproductive ways (for a review see Dar-Nimrod & Heine, 2011). Research on genetic etiological beliefs indicates that people frequently associate genetic predispositions with reduced behavioral control in ways that preclude environmental effects on behaviors (Dar-Nimrod, Heine, Cheung, & Schaller, 2011; Frosch, Mello, & Lerman, 2005; Monterosso, Royzman, & Schwartz, 2005; Phelan, 2005). In particular, discussions of the genetic etiology of complex behaviors are associated with more fatalistic cognitions and a decrease in people's perceived freedom of choice compared with discussions of alternative etiologies (Dar-Nimrod & Lisandrelli, 2012; Gould & Heine, 2012). These claims are supported by much

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empirical research (e.g., Beauchamp, Rhodes, Kreutzer, & Rupert, 2011; Brescoll & LaFrance, 2004; Dar-Nimrod, Zuckerman, & Duberstein, 2013; Sheldon, Pfeffer, Jayaratne, Feldbaum, & Petty, 2007). For example, women who learned of a genetic attribution for men's alleged superiority in math performed more poorly on a math test than women who learned of an experiential account for the same phenomenon (Dar-Nimrod & Heine, 2006). Applied to the topic of obesity, the effect of perceptions of genetic etiology on perceptions of immutability and control may also have undesirable direct and indirect behavioral consequences.

The Theory of Planned Behavior (Ajzen, 1991, 2002) contends that attitudes toward specific behaviors (e.g., overeating) affect intentions to exhibit such behaviors (e.g., to overeat). Empirical evidence indicates a strong relationship between attitudes toward an obesity-related behavior such as eating a low-fat diet and intention to follow such a diet (Armitage & Conner, 1999). Relevant to the current focus, exposure to genetic attributions for obesity-related behaviors seems to affect people's attitudes toward such behaviors. In one study, participants read a vignette depicting an overweight person who was described as an over-eater (Monterosso et al., 2005). Participants who learned that the person had a gene associated with obesity rated the eating behavior as less controllable and less blameworthy than did participants who learned of an environmental correlate for the overeating behavior. The determinism was even more evident in statements that participants made when they were probed to explain their rating of volition. For example, one participant stated "(w)ell they said it was genetically so it [would] you know, be something she had in her genes that *she can't control it, even though she wants to*" (p. 152, italics in original). Strikingly, participants reported that they would be more likely to overeat if they shared the relevant allele rather than the environmental correlate, suggesting a potentially maladaptive behavioral implication of perceived genetic etiology for obesity. Demonstrating a potential outcome of such perception, a recent survey of a representative (USA) national sample found that holding the belief that inheritance has "a lot" to do with obesity was associated with lower levels of physical activity and reduced consumption of fruits and vegetables (Wang & Coups, 2010). Other lay theories of obesity have also been linked to people's BMI (e.g., McFerran & Mukhopadhyay, 2013).

These kinds of deterministic responses would seem to be irrational given the relatively weak empirical link between specific genes and body weight in our current environment. For example, analyses of Body Mass Index (BMI) changes show that in the last 50 years, the proportion of overweight people in the USA has doubled and the proportion of obese people nearly tripled (Flegal, Caroll, Ogden, & Curtin, 2010). Such an increase cannot be explained by genetic changes, underscoring the substantial role that the environment has on people's weight. Furthermore, looking at the association of specific genes with obesity, meta-analyses of genetic association studies on obesity (see Speliotes et al., 2010) reveal a "modest" effect of the combined risk of all 32 identified variants associated with obesity (p. 939), with the strongest single common genetic predictor, the FTO gene, accounting for approximately an increased Body Mass Index (BMI) of 0.39 kg/m^2 – a difference of around 1 kg for an adult between the height of 160 and 180 cm, although the precise amount may well vary across individuals because of potential interactions with environmental factors. Various other genes have been identified with somewhat weaker links to obesity (Fujisawa, Ikegami, Kawaguchi, & Ogihara, 1998; Young et al., 2007). Hence, the degree to which these so-called "obesity genes" affect people's body weight is considerably smaller than people's deterministic responses would suggest (e.g., Monterosso et al., 2005; Singer et al., 1998; Wang & Coups, 2010).

The deterministic perceptions of genes discussed thus far potentially engender both positive and negative attitudinal and behavioral outcomes. On the one hand, the findings by Monterosso et al. (2005) indicate that a perceived genetic etiology for obesity may lead to a reduction in prejudice, which is a positive societal outcome. On the other hand, they also indicate that a perceived genetic etiology may serve as the basis for legitimizing such self-harming behaviors as over-eating, engaging in low levels of physical activity, and reduced consumption of fruits and vegetables, corresponding with real world associations between these beliefs and behaviors (Wang & Coups, 2010). To assess the potential behavioral implications of a perceived genetic etiology for obesity, the present studies: 1) evaluate associations between a direct antecedent of behavior (perceived behavioral control; Ajzen, 1991, 2002) and obesity-related etiological beliefs (Study 1); 2) experimentally assess the effects of different etiological explanations for metabolic rates on the strength of the cause–outcome associations (Study 2); and 3) evaluate actual eating behavior following exposure to different etiological accounts of obesity (Study 3). An institutional ethics committee approved all studies. Participants in all studies indicated their informed consent prior to taking part in the study and were thoroughly debriefed immediately after. Sample sizes were determined based on conceptually similar past studies on genetic essentialism (e.g., Dar-Nimrod et al., 2011; Monterosso et al., 2005).

Study 1

Method

The topic of interest for this study was part of a much larger study, which contained general questions about perceptions of genes (in various areas such as sexual identity, sexual orientation, and health) as well as the relationships between etiology, penetrance, and immutability in the health realm using vignettes which discussed fictitious diseases. Specifically, 131 undergraduate students (83 women, 43 men, five unreported) from a large Canadian university, ages 17–57 (M_{age} = 21.5, SD = 4.75) indicated whether they believed that obese people can control their weight with a categorical "yes" or "no" response. Later, they used a 6-point scale in response to the question "Do you believe that obesity originates from a genetic disposition or environmental causes (e.g., love of food, upbringing, no exercise, etc.)?" (1 – It's all due to genetics, 6 – It's all due to the environment).

Results and discussion

Seven individuals failed to complete at least one of the variables leading to a final sample of 124. A logistic regression analysis was conducted predicting a person's belief that obese people can control their weight from their etiological beliefs. As expected, an increase in endorsement of genetic explanations over environmental explanations for obesity significantly predicted a decrease in likelihood that one believes obese people can control their weight (B[SE] = –.60[.22], Wald = 7.33, p = .007, OR = 1.82). The same pattern was found after controlling for age and gender as well (B[SE] = –.65[.23], Wald = 7.87, p = .005, OR = 1.92).

This study suggests that a belief in genetic etiology for obesity is associated with a belief that obese people cannot control their weight. However, this was a correlational design, which limits causal inferences. To further explore such associations experimentally, in Study 2 we manipulated perceived etiological explanations for an obesity-related phenomenon (metabolic rate) and evaluated these explanations' effects on people's weight-related beliefs as well as their perceptions of different facets of the etiological explanations.