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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.
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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term "gene" steadily increased from 15% during the 1991–1993 period to 37% during the 2009–2011 period (I. Dar-Nimrod, unpublished data; available upon request).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.

Study 2

Method

One hundred and forty-three undergraduate students (36 men, 106 women, and one undeclared) from a large Canadian university, ages 17–45 (M_{age} = 20.5, SD = 3.88), participated in a study about attributions of positive outcomes for psychology course credit. Participants read a vignette portraying a protagonist, Jeremy, as a chef-in-training who learns that he has high metabolism in the course of his culinary education (see Appendix 1). Participants were randomly assigned to three experimental conditions offering divergent explanations for the protagonist's metabolic rate: control (n = 48); genetic (n = 53); and experiential (n = 42). The control condition attributed such differences to individual variation. Based on published research, the genetic (Haldar et al., 2012) and experiential (Armstrong & Reilly, 2002) conditions attributed such differences to the KLF15 gene, and whether one was breastfed as a child or not, respectively.

Following the vignette, participants completed a questionnaire assessing their beliefs about Jeremy's weight. Using 5-point Likert-type scales participants indicated perceptions of control ("How much control do you think Jeremy has over how much he weighs?"); validity ("How likely do you think Jeremy has higher metabolism than the average person?"); and immediate generalization ("Compared to the average person in the country, how easily do you think Jeremy's body can burn calories from fats?"). In addition, projected behavioral stability was measured using an open-ended question ("How much change in weight, in pounds, do you think Jeremy will have over the next 5 years?").

Attributional style questionnaire (ASQ)

We measured participants' causal attributions in relation to the etiological explanations using the ASQ (Peterson et al., 1982), adapted for this specific scenario. The questionnaire assessed causal locus (higher values denote greater internal attribution), causal stability, causal control, and causal generalization (i.e., how specific is the cause to the phenomenon). In a similar manner we assessed causal malleability ("Is the effect of the cause of Jeremy's higher metabolism something that can be changed or corrected?"). Participants used 7-point Likert-type scales to provide their ratings, with higher scores reflecting more of the specific element in question.

Results and discussion

We analyzed the results using multiple regression analyses. Our manipulations were dummy-coded into two variables, with the genetic condition being the comparison group. The first regression coefficient reported for each dependent variable does not include covariates. The second one includes age, gender (0 = Male, 1 = Female), and self-reported BMI as covariates. These covariates and all criterion variables have been standardized prior to analyses.

Beliefs about weight

As detailed in Table 1, compared to participants in the genetic condition, participants in the experiential condition felt that Jeremy had marginally/significantly more control over his weight [β = 0.35, p = .09; β = 0.42, p = .05], and viewed Jeremy's higher metabolism as nominally/significantly less valid [β = –0.32, p = .12; β = –0.43, p = .03]. Participants in the experiential condition also demonstrated significantly weaker tendencies to make proximal generalizations by indicating their belief that Jeremy can less easily burn calories [β = –0.53, p = .01; β = –0.64, p = .002], and they (only) nominally endorsed lower temporal stability estimates, indicating greater weight gain over the next five years [β = 0.31, p = .13; β = 0.34, p = .11]. Compared with the participants in the genetic condition, participants in the control condition were not significantly different in their evaluations of control, validity, or stability. They only differed in their proximal generalizations, indicating that Jeremy burns calories less ably than in the genetic condition [β = –0.53, p = .008; β = –0.60, p = .002].

Attributional style

Participants in the experiential condition perceived the cause of Jeremy's higher metabolism as being more external [β = –0.71, p < .001; β = –0.85, p < .001], and more malleable [β = 0.57, p = .005; β = 0.65, p = .003] compared with participants in the genetic condition. They also viewed the cause as significantly/marginally less specific to metabolism [β = 0.42, p = .04; β = 0.39, p = .06] and viewed Jeremy's control over the metabolic effect of the cause as significantly/marginally more substantial [β = 0.41, p = .05; β = 0.42, p = .06] compared with the participants in the genetic condition. There were no significant differences between these groups on causal stability evaluations. Compared with the participants in the genetic condition, participants in the control condition viewed the cause as marginally/significantly more malleable [β = 0.33, p = .09; β = 0.42, p = .04] and nominally/marginally less specific to metabolism [β = 0.32, p = .11; β = 0.36, p = .07]. No other significant differences emerged (see Table 1).

Taken together, the results of the study indicate that a genetic attribution for high metabolic rate is interpreted as more valid, consequential, and potent compared with an experiential attribution. The genetic cause is also evaluated as more internalized, less malleable, and more restrictive of individual ability to affect the phenotype, even though the purported cause in the experiential condition was whether one had been breastfed as an infant, which is clearly beyond the individual's ability to control. Interestingly, when individuals were not provided with a specific explanation for metabolic rate, people's weight-related beliefs and evaluations of

Table 1
Standardized regression coefficients (and SEs) indicating covariates, the effects of offering an experiential explanation (E), or an undefined explanation (Control) compared with a genetic explanation for fast metabolic rate.

	Beliefs about weight				Attributional styles (evaluation of the cause)				
	Control	Validity	Stability	Generalization	Locus	Malleability	Generalizability	Controllability	Stability
Intercept	.26 (.18)	.23 (.17)	–.26 (.18)	.38 (.18)*	–.03 (.18)	–.34 (.19)†	–.45 (18)*	–.33 (.19)†	–.21 (.19)
zAge	.14 (.08)	–.36 (.08)*	.14 (.08)†	–.10 (.08)	.14 (.08)†	.07 (.08)	0 (.08)	.12 (.09)	–.02 (.09)
Gender	–.51 (.18)*	0 (.17)	.05 (.18)	.07 (.18)	.48 (.17)*	–.02 (.18)	.30 (.18)	.20 (.19)	.37 (.19)*
zBMI	.04 (.08)	.04 (.08)	–.04 (.08)	.02 (.08)	.04 (.08)	–.01 (.08)	–.03 (.08)	.09 (.09)	.04 (.09)
E	**.42 (.21)***	**–.43 (.20)***	.34 (.21)	**–.64 (.21)***	**–.85 (.20)***	**.65 (.21)***	**.39 (.21)†**	**.42 (.22)†**	–.16 (.22)
Control	.01 (.20)	–.16 (.19)	.28 (.20)	**–.60 (.19)***	–.28 (.19)	**.42 (.20)***	**.36 (.20)†**	.15 (.21)	–.12 (.21)

Note: zAge, standardized ages; zBMI, standardized body mass index scores; E, offering an experiential explanation for high metabolism; Control, offering a non-specific explanation for high metabolism based on individual differences.
* p < .05.
† p < .10.

the cause often fell somewhere in between the genetic and experiential ratings, potentially indicating that both sorts of attributions may play a role in people's default causal beliefs.

Studies 1 and 2 indicated that beliefs related to control over one's weight correlate with endorsement of genetic attributions (Study 1) and are experimentally affected by exposure to such attributions (Study 2). Theoretical accounts and empirical findings converge to suggest that a decrease in perceived behavioral control reduces the likelihood of engaging in relevant weight-control behaviors (Ajzen, 1991, 2002; Armitage & Conner, 1999). To explore the potential relevance of causal attributions, we conducted a third study to test the effects of exposure to genetic and environmental explanations for obesity on actual behavior.

Study 3

Method

One hundred and sixty-two undergraduate students (124 women, 34 men, four undeclared) from a large Canadian university, ages 17–44 (M_{age} = 20.75, SD = 3.40), participated in a study entitled "Psychology and Food" in exchange for course credit. Participants were randomly assigned to one of three experimental conditions; in each condition they read an apparent newspaper article on food, which was actually written by the researchers. In one condition, the article presented a genetic explanation for obesity. In the second condition, the article presented a psychosocial explanation for obesity (suggesting that obesity is affected by social networks, as argued by Christakis & Fowler, 2007). In the third (control) condition, participants read an article about food, which made no mention of obesity or weight issues. Following the articles, participants rated the convincingness of the featured arguments. The articles, which served as manipulations, appear in Appendix 2.

To strengthen the purported goals of the study, participants completed a few additional questionnaires regarding their food preferences and eating habits. We then asked them to take part in a food-tasting task to evaluate the flavor of baked goods to be used in a future experiment. All participants indicated their willingness and were escorted to a different room, in which an oven was strongly emitting the scent of cookies. Participants sat in front of a large bowl of broken chocolate chip cookies and the experimenter left the room, instructing participants to taste and evaluate the cookies. The bowl was secretly weighed before and after the participants had an opportunity to sample the cookies and this difference in weight was used as the indicator of consumption.

Results and discussion

A suspicion probe among participants indicated that 25 individuals did not believe that the second part of the study (i.e., the food tasting part) was independent of the first (i.e., the manipulations). We thus conducted an analysis of variance to assess whether these participants responded differently on the food tasting task and found that they did indeed differ on that critical variable, consuming significantly larger amount of cookies (M = 61.28, SD = 67.00) than the unsuspicious participants (M = 40.29, SD = 32.77; $F_{1,157}$ = 5.82, p = .02, d = .40). As suspicion regarding the study hypothesis and our deception introduces a host of demand characteristics, we removed these individuals from our analyses leaving a final sample of 137 participants.

The consumption of cookies was analyzed with a single factor analysis of variance. There was a significant effect of the article that participants read, $F_{2,131}$ = 4.25, p = .02, η_p^2 = .061. As Fig. 1 shows, Fisher LSD post-hoc comparisons, which have been recommended when comparing only three conditions (Seaman, Levin, & Serlin, 1991), indicated that participants in the genetic condition consumed

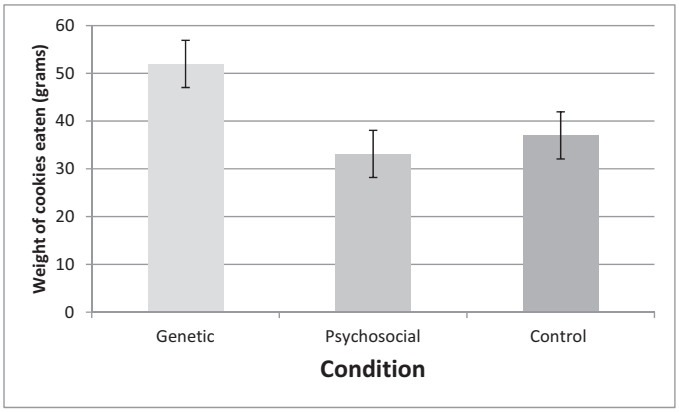

Fig. 1. Weight (grams) of cookies consumed following exposure to etiological claims for obesity.

significantly more cookies (M = 51.98, SD = 41.84) than participants in either the psychosocial condition (M = 33.14, SD = 22.91, p = .006, d = .56) or the control condition (M = 37.02, SD = 29.76, p = .03, d = .41). The latter two conditions did not significantly differ from each other (p = .56, d = .15). This pattern holds if we control for gender, age and self-reported BMI ($F_{2,126}$ = 2.98, p = .05, η_p^2 = .045). Fisher LSD comparisons for the latter analysis, adjusting for gender, age, and self-reported BMI, indicate that the genetic and social conditions are still significantly different (p = .02), but the genetic and control conditions are now only marginally significant (p = .07). The psychosocial and control conditions do not significantly differ in this latter analysis as well. When the more conservative Tukey post-hoc comparisons were conducted the significant difference between the genetic and social conditions is maintained (p = .02) and the difference between the genetic and control condition is marginally significant (p = .08; the social and control conditions are still not significantly different, p = .83).

To assess whether the experimental manipulations differed in their persuasiveness, an independent samples t-test was conducted on participants' evaluations of the convincingness of the arguments presented in the two experimental conditions. Arguments in the genetic condition (M = 4.62, SD = 1.27) were not perceived as more convincing than arguments in the psychosocial condition (M = 4.90, SD = 1.07; t(90) = –1.16, p = .25), undermining the alternative explanation that differences in the arguments' persuasiveness accounted for the difference in the amount of cookies consumed.

This study provides the first direct evidence for a behavioral effect of exposure to genetic explanations for obesity. Whereas a scientific psychosocial explanation for obesity did not significantly affect people's eating behaviors (compared with the control condition), exposure to scientific claims that emphasized a genetic etiology for obesity provided indication of an increase in the consumption of unhealthy food.

General discussion

Clarke (1995) has argued that testing for susceptibility genes may divert people's attention away from modifiable lifestyle and environmental factors. The present findings support this argument, indicating that a perceived genetic etiology for obesity is associated with a belief that obese people cannot control their weight. Additionally, individuals appear to perceive more control over one's weight and less determinism when one's metabolic rate is attributed to an experiential cause rather than a genetic cause. Furthermore, exposure to genetic explanations for obesity led to less restricted eating behaviors.

Interestingly, some research suggests that receiving personal feedback on whether one is a carrier of a genetic variant associated with obesity may serve to *reduce* the deterministic perceptions linking genetic etiology to obesity (Harvey-Berino et al., 2001). Specifically, that study found that participants who were truthfully informed that they carried an obesity-linked polymorphism stated that they believed that losing weight was *more* under an individual's control than did participants who were informed that they were not carriers of this specific variant. These results suggest some rationalizations on the part of people who were identified as carriers of an obesity-linked polymorphism, rationalizations that were perhaps motivated responses to deal with the threatening information that their obesity may have a genetic component. In contrast, the present studies demonstrated that people's encounters with genetic arguments for obesity led them to report and act as though the existence of links between genes and obesity made one's obesity risk an issue beyond one's personal control. Future research that compares and contrasts messages communicating public genetic etiological claims and personal genomic/genetic feedback is greatly needed.

The present findings are concordant with Dar-Nimrod and Heine's (2011) arguments that many people perceive genes to be the ultimate source for one's essence and defining characteristics, and that encounters with genetic etiological arguments prime essentialist thinking. When people encounter such genetic attributions for some characteristic, they come to view that characteristic as beyond an individual's control. Adding to nascent research on behavioral consequences of exposure to genetic attributions for various phenomena (e.g., Beauchamp et al., 2011; Chao, Chen, Roisman, & Hong, 2007; Dar-Nimrod & Heine, 2006; Moè & Pazzaglia, 2010), the findings in the present studies direct our attention to some unintended, but worrisome, consequences of the intersection between genetic research reports and public reactions. The current findings that point to a reduction in attempts to regulate eating behavior following exposure to genetic attribution for obesity complement other recent work (Hoyt, Burnette, & Auster-Gussman, in press), which indicated that presenting participants with the message that obesity is a disease undermines weight-loss self-regulatory processes. This overlap further illustrates some of the shared process and consequences of medicalization and geneticization (e.g., Árnason & Hjörleifsson, 2007; Perez, 2014; Shostak, Conrad, & Horwitz, 2008).

As genetic research steadily grows and appears to be one of the more enthusiastically featured subjects covered by science writers in the popular media (e.g., Conrad, 1999; Nelkin & Lindee, 1995), it is not surprising to find a sizable shift in perceived causes of obesity among the general public (Singer et al., 1998). As the present studies' findings suggest, this change in causal attributions may actually contribute to overeating, although additional data are recommended to gain more confidence in this effect. As the focus on genetic explanations for common human phenomena is not predicted to abate, there is a growing need for researchers and the media to be mindful in considering the social and behavioral implications of genetic etiological communications. Research into the manners in which such explanations can be communicated without detrimental effects is a worthy future goal.

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Appendices

Appendix 1 High metabolism vignettes as used in Study 2

Identical for all conditions

Twenty-five-year-old Jeremy Cooper is training to be a chef at his local Culinary Arts Institute. One of the policies of the school is that students must be educated on the importance of food and physical health as part of their curriculum. As part of their unit on metabolism, they must learn about what metabolism is, and what causes rates of metabolism to be faster or slower.

As part of this learning process, it is the goal of the administration for students to know their own metabolism so that they are more aware of the food that they create and the ingredients that they will use.

Control condition

The administration just wanted them to be aware of the variability in metabolism rates in the people that they will eventually have to cook for. Thus, the students should be wary of the types of ingredients they use – not everyone has the high metabolic rate to facilitate burning away of calories from fats quickly.

Jeremy thought about his own metabolism and, through chatting with his other classmates, decided that he may have relatively higher metabolic rate compared to others, but he isn't quite sure. He understands, though, that he still needs to be aware of others' dietary needs.

Genetic condition

The administration hopes to achieve this by discussing a cause of variability in metabolic rates, borrowing from a new trend in the culinary field – molecular gastronomy, which is the study of how to make use of physical and chemical reactions in cooking. The exception is that, for this unit, the administration wants to shift the focus from the food to the students, focusing on the biochemical interactions between their bodies and the food they eat. This will be accomplished by taking advantage of the cheap genetic testing technology that has emerged on many genetic testing websites. After students obtain their results, the instructor will depict the distribution of the relevant gene among the students. The administration hopes that this will allow students to have a broader appreciation of the variability in metabolism in the population, as shown by the distribution of the gene.

Jeremy's results show that he does, in fact, have the KLF15 gene, a gene that has been associated with faster metabolism, especially of fats, according to a recent journal article by researchers at the Case Western University (Haldar et al., 2012). In other words, this gene is associated with the ability to more quickly burn off calories from fats, with strong implications for keeping off excess weight and a lower prevalence of cardiovascular disease.

Experiential condition

The administration hopes to achieve this by discussing a cause of variability in metabolic rates. They brought in Dr. Guy Putet, a professor of pediatrics from the French Paediatric Society. Speaking about a journal article that he published in 2011, he lectured the students about the key role that having been breastfed as an infant plays in affecting metabolic rates in adulthood. He suggests that there is something about the nurturing that occurs during the nursing of an infant that sets the initial conditions for developing his or her metabolism. Specifically, having been breastfed is associated with higher metabolism rates than having been bottle-fed. In other words, having been breastfed is associated with the ability to more quickly burn off calories from fats, with strong implications for keeping off excess weight and a lower prevalence of cardiovascular disease. Dr. Putet encouraged each student to consider their own upbringing, and depicted the distribution of the frequency of breastfeeding among students during infancy. The administration hopes that this will allow students to have a broader appreciation of the variability in metabolism in the population, as shown by the distribution of other students' experiences.

Jeremy considered his own upbringing, and brought up the fact that he was breastfed as a child.

Appendix 2 Bogus media reports used as manipulations in Study 3

Instructions (prefaced all conditions)

In this study we are trying to assess several media reports for use in an upcoming study.

In the following section we would like to explore how people evaluate media reports related to food. Please answer the questions in regard to this article as detailed and as truthfully as possible to help us evaluate the best article for future use in our studies.

Genetic condition

Over the course of the past century, modern science has made extensive progress in eradicating infectious diseases such as polio and tuberculosis, and continues to try to find treatments for diseases such as cancer and HIV with an expected cure for them before the end of the century. Currently one of the most pressing health concerns is that of obesity. It has been called an "escalating pandemic" that continues to spread across the globe at a frightening pace. Obesity cannot be easily dismissed as it carries significant health risks for anyone affected by it.

Over the past decades, many solutions have been proposed to obesity. Weight loss is a big business and has grown to be a multimillion dollar industry. It largely depends on the public's belief that obesity and associated conditions are a direct result of poor diet and exercise. This would explain why new research indicating genes to be the primary cause of obesity has received little media attention and continues to be overshadowed by outdated claims attributing the major cause of obesity to a modern lifestyle rather than genes. Unfortunately this also affects public health policy, resulting in the promotion of ineffectual solutions to the problem.

In 2003, Jeffrey Birkner conducted a study, which found that after a 4-week maintenance diet controlling for caloric intake, fat cells had shrunk in size and the participants' weight had decreased. However, no matter how dedicated the participants were to maintaining said weight loss, they all regained. Birkner conducted further tests and came to a startling conclusion: if a person loses large amounts of weight, his/her body appears to show signs of starvation, with an 80% decrease in metabolism, in comparison to a naturally thin individual. These individuals also presented with signs of semi-starvation neurosis: dreaming and fantasizing about food, breaking the diet, hoarding and hiding large amounts of food and binge eating. These findings led to a study by Polly Clifton who set out to force a 25% weight gain in her participants. This proved to be surprisingly difficult as the participants' metabolism increased by 50% once they had grown heavier. After the end of the forced weight gain period, the participants had no trouble losing the weight again.

Other studies examining data from the Danish Registry of Adoptees and the Swedish Twin Registry found that childhood family environment alone has little or no effect on an individual's weight. It appears that 70% of variation in people's weight is accounted for by inheritance, meaning that it is more strongly inherited than conditions such as mental illness, breast cancer and heart disease. These findings are supported by research conducted in recent years, implicating GATA-2 and GATA-3 Fat Genes in the development of fat tissue. Furthermore, in 2003 Kent Lloyd found that individuals born with two copies of a particular variant of the FTO gene have a 70% higher risk of obesity than those with the low-risk variants. Even if only one copy of the high-risk FTO variant is inherited, an individual faces a 30% increased risk of obesity.

Diane Liebling suggests that for each person there is a comfortable weight range that the body gravitates to, and going much above or much below natural weight range is extremely difficult. This would suggest that individuals attempting to lose weight are fighting a losing battle unless they are committed to exist in a permanent state of starvation, which cannot be considered to be appealing or healthy. In coming years, new genetic research may help us to further understand the effects of Fat Genes such as Gene Insig2, which regulates fatty acid and cholesterol synthesis, and maybe will finally bring us closer to a cure for obesity. Until then, billions of dollars will be spent on false hopes, as exercise and healthy diet will not be able to overwrite genetics.

Psychosocial condition

Over the course of the past century, modern science has made extensive progress in eradicating infectious diseases such as polio and tuberculosis, and continues to try to find treatments for diseases such as cancer and HIV, with an expected cure for them before the end of the century. Currently one of the most pressing health concerns is that of obesity. It has been called an "escalating pandemic" that continues to spread across the developed world at a frightening pace. Obesity cannot be easily dismissed as it carries significant health risks for anyone affected by it.

In order to find a permanent solution to the problem of obesity, researchers have focused their efforts on understanding the factors involved in the gaining and retaining of excess amounts of weight. In his study of East African baboons, Frederick Nordmann found that weight is more closely associated with the energy expended while foraging for food, rather than with the baboon's overall caloric intake. He proposes that before the industrial revolution, humans used to live an energy-intensive lifestyle which offsets any potential weight gain; however, in the twenty-first century, a human-created situation of abundance coupled with significantly decreased energy expenditure is having disastrous consequences. Recent surveys indicate that more than half of the adults living in developed countries are insufficiently active, resulting in fewer calories burned than consumed and putting individuals at significant risk for diabetes and other obesity-related health problems. This trend is not only limited to developed countries either, as a study by Carolyn Burkner found in 2005. Many urban areas of developing countries experience similar problems, as the introduction of television and 'remote everything', coupled with a lack of infrastructure, has significantly decreased the number of adults who are sufficiently active. Cities, such as São Paulo, report that a staggering 70% of their population is inactive. One can conclude that this truly is a worldwide problem that urgently needs to be addressed.

In 2006, a promising research study conducted by Paul Wazniek found that obesity can actually spread from person to person, much like a virus. A detailed analysis of a social network containing more than 10,000 individuals revealed that people were most likely to be obese when a friend became obese. However, while a friend's obesity increased one's own likelihood of becoming obese by 57%, there was no effect when a neighbor gained or lost weight. This influence even remained if the friend lived hundreds of miles away, as the deciding factor was not physical proximity but rather the strength of the close personal bond between the individuals. This was reflected in the finding that best friends experienced a 171% increased chance of becoming obese if one of them was obese. No such effects were found for family members or one's spouse. One explanation put forth by Wazniek suggests that friends affect each others' perception of "fatness". This spreading process, or social contagion, could help to explain why countries such as the United States have experienced such rapid increases in obesity in recent years.

In order to find a permanent solution to this healthcare crisis, we need to direct our attention to the environmental causes of obesity. While Wazniek's study helps to explain why obesity spreads at such an alarming rate, it also proposes a solution to the problem. In order to prevent the further spread of obesity, we must prevent the 'normalizing' of fatness. Note that this is not to be mistaken as embracing the unrealistic physical ideals presented to us by the popular media. In addition, one should target not the individual but rather groups of friends when promoting physical activity and the abandonment of the comforts of a sedentary lifestyle. If obesity can spread like a social contagion, so can its antidote.

Control condition

Native peoples from different parts of North America have used a wide range of agricultural techniques. Perhaps the best known is the interplanting of corn, beans, and squash together – a trio often referred to as the "three sisters." In a three sisters planting, the three partners benefit one another. Corn provides support for beans. Beans, like other legumes, have bacteria living on their roots that help them absorb nitrogen from the air and convert it to a form that plants can use. (Corn, which requires a lot of nitrogen to grow, benefits most.) The large, prickly squash leaves shade the soil, preventing weed growth, and deter animal pests.

The "three sisters" were important sources of food. In fact, corn was very important to the survival of the first English colonists during their first winters in Northeastern America. The survival of the early colonists depended on what corn they could beg, borrow or steal from the native peoples plus what they were able to grow under their guidance.

It is believed that corn dates back even further than the inhabitancy of native people. Corn's origin is believed to be in the Mexican plateau or the highlands of Guatemala. Fossil pollen grains of corn have been found in drill cores of lake sediment beneath Mexico City. These sediments could be 80,000 years old or more. When Jacques Cartier visited the village of Hochelaga (now Montreal) in 1535, he noted the extensive corn fields growing in all directions. There are records of Champlain finding corn growing in the area of Georgian Bay in 1615. Archaeological studies have found that corn was grown near Campbellville, Ontario before 1200 AD.

Corn belongs to the grass family. Theory suggests that at one time, each individual kernel was covered by its own floral parts similar to the kernels of oats and barley, and that the cob readily broke down into small segments. It is believed that this has allowed corn as a species to survive. The husk and cob as we know them today were gradually developed from wild varieties by the native population.

Native groups developed the major classes of corn that we recognize today; the types being sweet, popping, flint, flour and dent corns. Flint corn tends to have a larger grain with relatively little flour tissue in the endosperm. Flour corn is soft, floury and breaks apart readily. Dent corn is a cross between flint and flour corn and can be used for the purpose of making bread or used for wet grinding or for making hominy. Dent corn usually produces higher grain yields and to this day dominates production in North America and much of the rest of the world.

From its original use as a food for man, corn now yields over 100 by-products to the industry. A few of the by-products are dyes, paints, oilcloth, oil for soaps, syrups, starches, size and glaze, corn gum (used as a rubber substitute), vegetable substitutes for lard and butter, corn cellulose in press boards and insulating materials and various chemicals.

The livestock industry continues to be the largest user of corn grown. In Ontario, approximately 60% of the crop harvested is fed to livestock. Approximately 30% is used for industrial and commercial usage which leaves about 10% for the export market.

Research continues to find new uses for corn. Environmentally friendly products are one such area. Corn as a renewable resource can be beneficial in making products such as ethanol fuel, ethanol windshield washer fluid, CMA (calcium magnesium acetate) road de-icer, and other degradable products made from corn starch.

Corn is as important today to mankind as it was in the beginning to native peoples. According to Indian legend, corn was of divine origin – “it was the food of the gods that created the earth.”