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Lay Theories of Obesity Predict Actual Body Mass

Brent McFerran[1] and Anirban Mukhopadhyay[2]
[1]Ross School of Business, University of Michigan, and [2]School of Business and Management, Hong Kong University of Science and Technology

Abstract
Obesity is a major public health problem, but despite much research into its causes, scientists have largely neglected to examine laypeople's personal beliefs about it. Such naive beliefs are important because they guide actual goal-directed behaviors. In a series of studies across five countries on three continents, we found that people mainly believed either that obesity is caused by a lack of exercise or that it is caused by a poor diet. Moreover, laypeople who indicted a lack of exercise were more likely to actually be overweight than were those who implicated a poor diet. This effect held even after controlling for several known correlates of body mass index (BMI), thereby explaining previously unexplained variance. We also experimentally demonstrated the mechanism underlying this effect: People who implicated insufficient exercise tended to consume more food than did those who indicted a poor diet. These results suggest that obesity has an important, pervasive, and hitherto overlooked psychological antecedent.

Keywords
health, food, individual differences, social cognition, obesity, lay theories, beliefs, self-regulation, diet

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More than two thirds of adults and one third of preschoolers in the United States are overweight or obese (Centers for Disease Control and Prevention, 2004), and similar rates exist in many developed countries (World Health Organization, 2006), despite the fact that weight loss and weight maintenance are among the most commonly held personal goals (Cloud, 2009; Kassirer & Angell, 1998). Millions of individuals attempt to regulate their weight but encounter advice that is often outright contradictory—some experts encourage greater exercise, some advocate reduced calorie intake, and others lay the blame on genetics. Given the amount of public discourse on this matter, it is probable that most laypeople have arrived at some conclusion for themselves about what generally causes people to become overweight. We term such beliefs peoples' personal *lay theories of obesity*, and in the research reported here, we investigated their existence and possible effects.

People hold lay theories (i.e., naive beliefs) about the causes and consequences of many phenomena (Ross & Nisbett, 1991; Wyer, 2004). Although these beliefs sometimes dovetail with scientific consensus and at other times do not, they nevertheless can exert profound and enduring influences on judgment and behavior (Dweck, 2000). For example, Robins and Pals (2002) tracked several hundred students as they progressed through college and found that lay theories about the causes of failure (i.e., whether it was attributed to a lack of intelligence or a lack of effort) predicted reactions of helplessness and drops in self-esteem and, eventually, grades. In this research, we examined similar naive beliefs about the causes of obesity (or weight gain more generally) and demonstrated that these beliefs have powerful and systematic influences on individuals' actual body masses and food consumption.

Dar Nimrod and Heine (2010) presented participants with research articles claiming that obesity is caused either by genetics or by social networks and found that those who read about the genetics account ate significantly more cookies. Though we do not disagree with this finding, we propose that people have other lay theories about the

Corresponding Author:
Brent McFerran, Ross School of Business, University of Michigan, 701 Tappan St., Ann Arbor, MI 48109-1234
E-mail: mcferran@umich.edu

causes of obesity that are more prevalent than theories involving genes or social networks. Individuals' food and exercise choices are influenced by the lay theories they hold (Burnette, 2010; Crum & Langer, 2007), and the stigma of obesity is rooted in the belief that individuals are largely responsible for their weights (Crandall, 1994; Puhl & Brownell, 2001). Indeed, people can make vast and relatively rapid changes to their diet and exercise patterns, but not to their genes or social networks. Therefore, we predicted that most people would implicate overconsumption of food, lack of exercise, or both as the most common causes of obesity.[1]

Beyond identifying different lay theories, we can also make predictions about their consequences. Research has shown that lay theories substantially influence goal-directed behaviors across domains as diverse as academics, relationships, and New Year's resolutions (Finkel, Burnette, & Scissors, 2007; Molden & Dweck, 2006; Mukhopadhyay & Johar, 2005). The use of lay theories, like other forms of implicitly held knowledge, can be triggered by features of the situational environment (Bargh, 1997; Wyer, 2004). In our context, this implies that lay theories of obesity should influence behaviors in situations relevant to weight loss or weight maintenance. For example, all else being equal, people who implicate overconsumption of food in obesity should be better able to resist a tempting slice of cake than should people who believe that obesity is caused by a lack of exercise. Consequently, we predicted that, compared with people who believe obesity is caused by a lack of exercise (*exercise theorists*), those who implicate overconsumption of food (*diet theorists*) should be more likely to approach a goal of weight loss or weight maintenance by consuming fewer calories.

Given current medical knowledge, these differences in approaches to weight loss should have downstream consequences for actual body masses. Although some research has implicated sedentary lifestyles (Blair & Brodney, 1999) or genetics (Comuzzie & Allison, 1998) as causes of obesity, the strongest scientific evidence points to increased consumption of food and drink (Jakicic, Marcus, Gallagher, Napolitano, & Lang, 2003; Ledikwe, Ello-Martin, & Rolls, 2005; Pontzer et al., 2012). On average, Americans eat approximately 200 more calories a day now than they did in 1980 (Centers for Disease Control and Prevention, 2004), and portion sizes are directly linked to obesity (Young & Nestle, 2002). Meanwhile, exercise rates have either increased or remained stagnant over this period (Cloud, 2009; Westerterp & Speakman, 2008), and the human genome cannot have morphed enough in so short a time to fully explain the increased obesity rates. While it is theoretically possible to counteract increased caloric intake with additional exercise, people both underestimate how many calories they consume and overestimate how many they burn while exercising (Lichtman et al., 1992; Wansink, 2006; but see Crum, Corbin, Brownell, & Salovey, 2011; Harris, 1990; and Tomiyama & Mann, 2011, for alternatives to a calorie-balance model of obesity). To summarize, a recent *Journal of the American Medical Association* editorial definitively emphasized the importance of diet over genes or exercise in causing obesity:

> Clearly, environmental causes for obesity are far more influential than genes. . . . Obesity results from overnutrition and the primary therapeutic target is preventing or reversing overeating . . . Exercise is associated with weight loss but its duration or intensity has minor effects on weight loss relative to diet. (Livingston & Zylke, 2012, pp. 971–972)

In sum, because lay theories guide actual eating behavior, and because overeating is largely responsible for weight gain, a person's lay theory of obesity—ascribing it to diet versus exercise—should predict his or her actual body mass. Formally, we predicted that, all else being equal, exercise theorists should have higher body mass indexes (BMIs) than should diet theorists. Put differently, exercise theorists, compared with diet theorists, believe that diet has a lower marginal impact on weight and, hence, should eat more calories and consequently be heavier (because their additional exercise burns fewer calories than the additional consumption adds). In what follows, we report results from six studies in which we tested these hypotheses.

Study 1

A series of pretests (for details, see Pretest 1 and Pretest 2 in the Supplemental Material available online) supported the following premises: (a) that laypeople commonly ascribe obesity to diet, exercise, and genetics but very rarely ascribe it to other factors; and (b) that laypeople are less likely than physicians to believe that diet is the primary cause of obesity.

As a first test of our hypothesis that lay theories predict actual body mass, we asked 301 South Koreans drawn from a nationally representative sample (54% female, 46% male; mean age = 32.61 years, age range 15–68) to indicate what they believed was the primary cause of obesity: eating too much, not exercising enough, or genetics (the item and all possible responses were presented in Korean). A separate study revealed that these effects were robust to several alternate phrasings—see Supplementary Data: Alternative Phrasings (Study 1) in the Supplemental Material. Participants also reported their height and weight (which were later converted into BMI; $M = 22.25$, $SD = 3.34$).

We found that the three focal lay theories were not equally held, $\chi^2(2, N = 254) = 74.94$, $p < .001$, $\varphi = 4.70$. Similar proportions of respondents—together, more than 90% of respondents overall—believed that the primary cause of obesity was diet (50.4%) or exercise (41.3%), $\chi^2(1, N = 233) = 2.27$, $p > .13$; only 8.3% implicated genes. An analysis of covariance on BMI with age and gender entered as covariates showed that different lay theories were associated with different BMIs, $F(2, 249) = 5.27$, $p = .006$, $\eta_p^2 = .04$. As expected, diet theorists had significantly lower BMIs ($M = 21.55$, $SD = 2.82$) than did exercise theorists ($M = 23.10$, $SD = 3.80$), $F(1, 249) = 12.34$, $p = .001$, $d = 0.46$ (the mean for genetics theorists was 22.21, $SD = 3.01$). These results, as well as those from all subsequent studies, held without the specified covariates. Examined another way, the data showed that of the participants who met the medical criterion for being overweight as defined by the National Institutes of Health (BMI > 25), nearly twice as many were exercise theorists ($n = 30$) as diet theorists ($n = 17$), despite there being more diet theorists in the sample overall.

This study provided initial evidence supporting three important propositions. First, there is no single prevalent belief that individuals hold about the cause of obesity. Second, diet and exercise are far more likely than genetics to be reported as primary causes of obesity. Finally, these beliefs do matter: Diet theorists were systematically thinner than exercise theorists. However, our paradigm did not allow people to indicate the strength of their belief in each lay theory—someone who believes that poor diet is 60% responsible for obesity, with other factors also contributing, may be different from someone who believes it is 100% responsible. In Study 2, we investigated this nuance while controlling for several known correlates of BMI.

Study 2

We asked 84 U.S. residents from an online panel (63% female, 37% male; mean age = 38.39 years, $SD = 13.12$, age range 20–82) to indicate the relative strength with which they believed in each lay theory by allocating 100 points among the three lay theories (with more points indicating greater culpability) and to report their height and weight (converted into BMI; $M = 25.58$, $SD = 5.79$) and report on several other factors known to affect weight: level of education, hours of sleep per night (a measure of sleep deprivation), stress, presence or absence of various medical conditions, and use of various medications—see Measures of Known Correlates of Obesity (Study 2) in the Supplemental Material.

As expected, there was a significant negative correlation between strength of diet lay theory and BMI ($r = -.23$, $p < .04$). Reflecting the reciprocal relationship between diet and exercise theories, results revealed a corresponding positive correlation between exercise theory and BMI ($r = .25$, $p < .03$) and a negative correlation between the two lay theories ($r = -.53$, $p < .001$). The correlation between genetics lay theory and BMI was not significant ($r = -.01$, $p > .96$). Replicating our findings from Study 1, results revealed that participants who were overweight or obese (BMI > 25; $n = 38$)[2] were less likely than normal-weight participants ($n = 38$) to indict diet as the cause of obesity (i.e., they allocated fewer points to diet in the lay-theory measure; normal-weight participants: $M = 50.95$, $SD = 15.23$; overweight or obese participants: $M = 41.58$, $SD = 13.51$), and this difference was significant, $t(78) = 2.90$, $p < .01$, $d = 0.65$.

To test for robustness, we ran a hierarchical regression analysis with the control measures entered in the first step, the genetics lay theory entered in the second, and the diet lay theory entered in the final step (exercise was treated as the holdout variable; see Table 1). This allowed us to examine if our diet theory measure predicted significant variance in BMI above and beyond the control variables. Supporting our conjecture, results revealed that, compared with the genetics lay theory, the diet lay theory was again significantly associated with a lower BMI, controlling for the associated factors, $\beta = -0.30$, $p = .02$. We also created a difference score by subtracting points allocated to diet from those allocated to exercise. The results remained significant when we used this measure, $\beta = -0.27$, $p = .02$. As a further test, we coded whether each participant exceeded the criterion for being overweight and conducted a binary logistic regression. Again, diet theorists were significantly less likely than exercise theorists to be overweight, even after controlling for the other known predictors of obesity, Wald = 7.22, $p < .01$. Given that diet and exercise theories were strongly negatively correlated, treating diet as the holdout variable resulted in identical but opposite results. Entering all the variables in the same step did not change the results.

Study 3

We next tested whether the results from a U.S. sample replicated in a nationally representative sample from France. In Study 3, 303 French participants (43% female, 57% male; mean age = 42.44 years, $SD = 13.62$; mean BMI = 24.86, $SD = 4.83$) completed the same survey used in Study 2 (translated into French) with measures of several additional control variables added: current and childhood socioeconomic status, the extent to which their home location was urban or rural, pregnancy status, employment status, self-reported overall health, interest in nutrition, tobacco use, and self-esteem. Again, there was a significant relationship between strength of belief

Table 1. Results of Linear Regression Predicting Body Mass Index (Study 2)

Predictor	Step 1	Step 2	Step 3
Age	0.016	0.014	0.033
Gender (1 = male, 2 = female)	−0.018	−0.032	0.000
Education	−0.201*	−0.231**	−0.258**
Presence of medical conditions known to affect weight (1 = present, 2 = absent)	0.061	0.059	0.092
Use of medications known to affect weight (1 = use, 2 = no use)	−0.306**	−0.328***	−0.303**
Hours of sleep per night	−0.152	−0.163	−0.172
Self-reported stress, anxiety, and depression	0.102	0.095	0.039
Belief in genetics lay theory	—	0.117	−0.042
Belief in diet lay theory	—	—	−0.302**
R^2	.15	.17	.23
ΔR^2		.02	.06**
F	$F(7, 72) = 1.88$*	$F(8, 71) = 1.77$*	$F(9, 70) = 1.27$**

Note: Standardized regression coefficients are presented.
*$p < .10$. **$p < .05$. ***$p < .01$.

in the diet lay theory and BMI ($r = -.13$, $p = .03$). Further, overweight participants ($n = 130$) allocated fewer points to the diet theory ($M = 41.54$, $SD = 16.22$) than did participants at a normal weight ($n = 172$; $M = 46.51$, $SD = 18.00$), $t(300) = 2.48$, $p < .01$, $d = 0.29$ (see Fig. 1).

To ensure that our results were not driven by the response mode of the points-allocation task, we also measured endorsement of lay theories by using an additional, bipolar item after all the covariates were assessed: "Obesity is caused more by . . . ?" Responses were made on a scale from 1 (*eating too much*) to 7 (*not exercising enough*; see Design and Procedure of Study 3 in the Supplemental Material for details about robustness and validity checks). A hierarchical regression with the points-allocation measure including all control variables replicated the previous results, $\beta_{diet} = -0.18$, $p = .01$ (see Table 2). The patterns also held with the single-item bipolar measure, $\beta = -0.13$, $p = .02$, as well as the difference score, $\beta = -0.14$, $p = .02$. Both measures explained variance in BMI above and beyond all the control variables, with parameters larger than most.

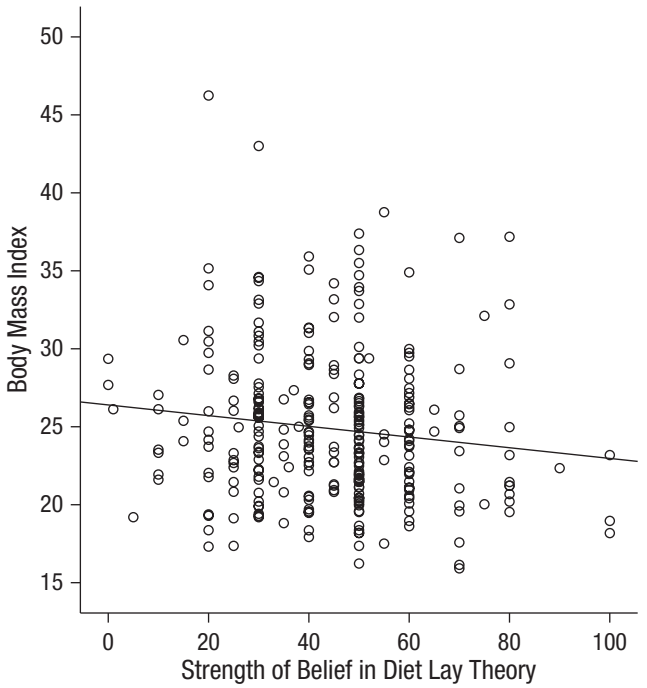

Fig. 1. Results from Study 3: body mass index (BMI) as a function of strength of belief that diet (as opposed to exercise or genetics) is the cause of obesity.

Study 4

One concern was that our question format in our first three studies may itself have primed an endorsement of the diet and exercise lay theories by respondents, for whom these particular beliefs might otherwise not be salient. To address this issue, we recruited 251 U.S. residents from an online panel (64% female, 36% male) to participate in a fourth study.

We first had participants respond to an open-ended question: "In general, what do you think is the primary factor responsible for people being overweight? (in at most one sentence)." Participants' computer screens presented an empty box in which responses could be typed. Next, participants entered their gender and their height and weight (converted into BMI; $M = 26.22$, $SD = 6.11$). Two coders blind to our hypotheses coded each open-ended response as indicating belief in a lay theory of obesity based on diet (e.g., "They eat too much food"), exercise (e.g., "The lack of exercise people get"), both diet and exercise (e.g., "Eating and exercise habits"),

Table 2. Results of Linear Regression Predicting Body Mass Index (Study 3)

Predictor	Step 1	Step 2	Step 3
Age	0.103	0.103	0.116*
Gender (1 = male, 2 = female)	−0.232***	−0.232***	−0.206***
Education	−0.188***	−0.188***	−0.174***
Presence of medical conditions known to affect weight (1 = present, 2 = absent)	−0.031	−0.031	−0.026
Use of medications known to affect weight (1 = use, 2 = no use)	0.021	0.021	0.026
Hours of sleep per night	0.020	0.020	0.027
Self-reported stress, anxiety, and depression	0.032	0.032	0.055
Current socioeconomic status	−0.096	−0.096	−0.091
Childhood socioeconomic status	0.062	0.062	0.059
Home location	0.043	0.043	0.054
Current pregnancy (1 = yes, 2 = no)	0.065	0.065	0.060
Employment outside of home (1 = yes, 2 = no)	−0.025	−0.025	−0.021
Tobacco usage (1 = yes, 2 = no)	0.145**	0.145**	0.148***
Self-reported quality of overall health	−0.199***	−0.199**	−0.221***
Interest in nutrition	0.011	0.011	−0.028
Self-esteem	0.096*	0.096*	0.106*
Belief in genetics lay theory	—	−0.001	−0.120
Belief in diet lay theory	—	—	−0.184**
R^2	.23	.23	.25
ΔR^2		.00	.02**
F	$F(16, 273) = 5.04$***	$F(17, 272) = 4.72$***	$F(18, 271) = 4.90$***

Note: Standardized regression coefficients are presented.
*$p < .10$. **$p < .05$. ***$p < .01$.

genetics (e.g., "Hereditary"), or some other cause (inter-coder agreement = 90%; disagreements were resolved by discussion).

We observed the same patterns seen in our previous studies in spontaneously evoked lay theories of obesity. Diet alone was invoked most often (49.0%), followed by both diet and exercise (20.3%) and, finally, lack of exercise (15.1%). Hence, diet and exercise lay theories accounted for nearly 85% of all responses. Importantly, comparing respondents who spontaneously cited only diet with those who cited only exercise revealed a convergent pattern of results: Exercise theorists again had higher BMIs ($M = 27.73$, $SD = 6.27$) than did diet theorists ($M = 25.53$, $SD = 5.51$), $F(1, 155) = 4.22$, $p = .04$, $d = 0.37$ (see Fig. 2), a pattern of results that reassured us that the effect observed in previous studies was not caused by the question format or response priming. Interestingly, the mean BMI of respondents who cited both diet and exercise as causes of obesity was 26.45 ($SD = 5.86$), which is approximately the midpoint between the means for the diet theorists and the exercise theorists.

In our final two studies, we aimed to investigate the mechanism underlying the results observed in our four previous studies and thereby test causality. People who believe that exercise, as opposed to other factors, causes

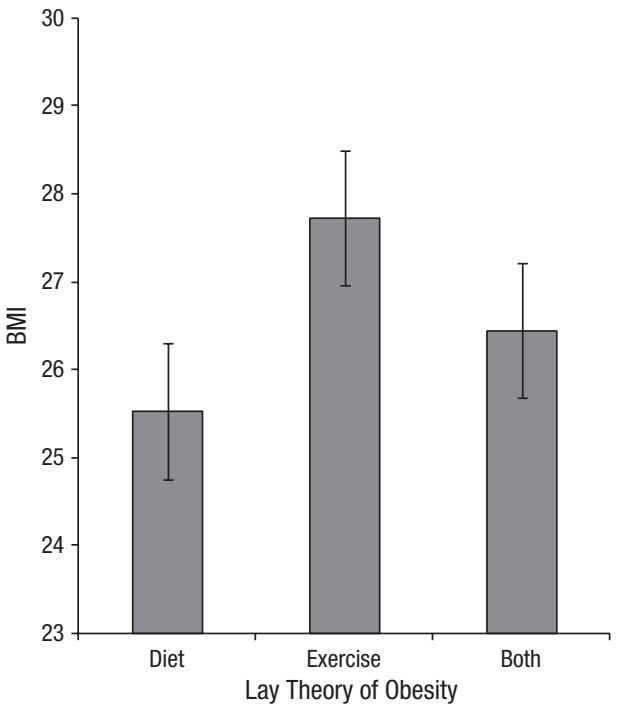

Fig. 2. Results from Study 4: body mass index (BMI) as a function of belief about the cause of obesity.

obesity are presumably less concerned with regulating their caloric intake (relative to using other methods of weight loss or maintenance) and, therefore, should consume more calories. We tested this proposition in Studies 5 and 6.

Study 5

Participants were 172 University of British Columbia undergraduates (58% female, 42% male) who came into the lab to participate in a set of studies in return for course credit. Upon arrival, all participants were given an opaque cup containing seven individually wrapped chocolates "to snack on" while they completed their surveys. They were told that the chocolates were left over from a prior experiment and that they were free to eat however many they liked. After completing several other studies (all unrelated to food), participants completed the same single-item measure of lay theory of obesity used in Study 4. After each participant had left, we counted the number of chocolates remaining in his or her cup.

The sample mean on the lay-theory measure (M = 4.20, SD = 1.68) did not differ statistically from the midpoint of the scale, $t(171) = 1.54$, $p > .12$, and responses ranged over the entire scale, which suggested again that people do not uniformly believe that overconsumption (or insufficient exercise) is the overwhelming cause of obesity. Regression analysis controlling for age and gender showed that greater belief that a lack of exercise (as opposed to a poor diet) caused obesity led to the consumption of more chocolates, $\beta = 0.18$, $p = .01$. Estimating means 1 standard deviation above and below the mean for lay theory showed that participants who primarily implicated exercise ate more chocolates than did those who implicated overconsumption (Ms = 3.37 vs. 2.67). Given that we measured lay theories at the end of the experimental session but that consumption took place throughout the experiment, it is unlikely that mere measurement (Morwitz, Johnson, & Schmittlein, 1993) can explain this effect. However, this study relied on correlation, so in the next study, we tested the relationship experimentally.

Study 6

Participants were 93 undergraduates at the Hong Kong University of Science and Technology (52% female, 48% male) who came to the lab to complete several studies in return for course credit. The questionnaire for this study, which contained the lay-theory manipulation (diet vs. exercise vs. control; between subjects), was the first in the set. Other studies were unrelated to this research. Upon arrival, all participants were directed to individual workstations, each with a computer monitor and a paper bowl containing nine individually wrapped chocolates. The same cover story was used to explain the food as in Study 5.

The lay-theory manipulation exactly followed that used in past research involving the manipulation of (other) lay theories (e.g., Nussbaum & Dweck, 2008). It was presented as a study titled "Understanding Scientific Research"; participants were tasked with reading a passage that described (fictitious) research. Depending on condition, the passage stated either that obesity is caused almost entirely by overeating and poor diet or by sedentary habits and insufficient exercise. In the control condition, the passage was adapted from a real *Psychological Science* abstract about fingerprinting (Tangen, Thompson, & McCarthy, 2011). After reading the passage, participants in the experimental conditions summarized its message in one sentence and responded to an item asking what it implicated as the primary cause of obesity; responses were made on a scale from 1 (*diet*) to 7 (*exercise*). All participants then rated how convincing the passage was, using a scale from 1 (*not at all*) to 7 (*very*). The time taken to read the passage was recorded unobtrusively by the computer.

Manipulation checks confirmed that the lay-theory manipulation was effective: Participants who read the exercise-related passage had significantly higher scores on the lay-theory measure (M = 5.77, SD = 1.59) than did participants who read the diet-related passage (M = 1.83, SD = 1.23), $F(1, 60) = 113.04$, $p < .001$, $d = 2.77$. There were no differences in how convincing participants found the passages ($p > .57$; mean responses in all conditions were above the scale midpoint) or in reading times ($p > .30$).

To test our hypothesis, we conducted an analysis of variance on the number of chocolates consumed, which revealed that participants who had been primed with an exercise theory of obesity ate more (M = 3.74, SD = 2.80) than did those primed with a diet theory (M = 2.45, SD = 2.08), $F(1, 90) = 4.31$, $p = .04$, $d = 0.52$, whereas participants who read the control passage ate a middling quantity (M = 3.32, SD = 2.40; see Fig. 3).[3] This study provides causal evidence that lay theories of obesity influence food consumption.

General Discussion

Across multiple studies, we found the first evidence that people generally have two different lay theories about what causes obesity, and that these beliefs impact people's actual likelihood of being overweight, findings that explain previously unexplained variance in BMI. Consistent with research on goal-directed behavior, our studies showed that people's lay theories about obesity impacted their actual food choices. We observed these

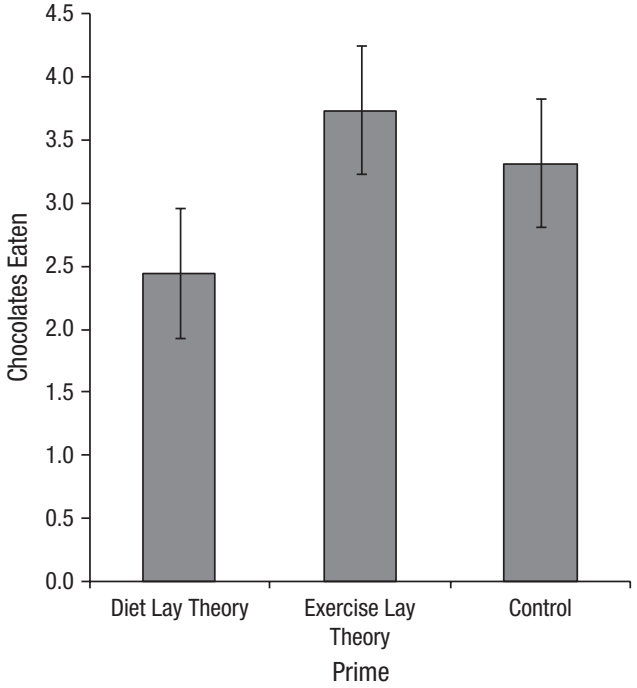

Fig. 3. Results from Study 6: mean number of chocolates consumed as a function of primed lay theory of obesity.

effects in samples of participants from three continents, and our replication of the basic relationship using multiple measures provides confidence in its validity and robustness.

Causality

Although we controlled for many potential variables that could explain the effect of lay theories on BMI, we cannot definitively claim that our results reveal a cause-and-effect relationship. Much research has shown that lay theories influence actual behavior, both in the domain of food consumption and otherwise (Labroo & Mukhopadhyay, 2009; Molden & Dweck, 2006), and that increased food intake is the primary cause of obesity (Livingston & Zylke, 2012). We, too, demonstrated a causal effect of lay theories of obesity on food consumption (Study 6), but we do not know whether participants' net caloric impact (and thereby their BMI, at least eventually) changed because of the increased consumption. Unfortunately, it is hard to conclusively answer this question, because participants may proactively modify their behaviors outside the lab on the basis of what they did during an experiment. Even with a long-term time horizon of data on both diet and exercise, we would have to rely on correlation because a prime may not have effects that continue outside of the laboratory for months.

Our thesis linking lay theories and BMI relies on cross-sectional data, and reverse causation is a potential concern. We cannot conclusively reject the possibilities that people's BMI affects which theory they find more plausible or that an overweight person may adopt an exercise lay theory to reduce cognitive dissonance, although we conducted a number of additional studies to address these possibilities (see Alternative Explanations and Reverse Causality in the Supplemental Material). Our largely cross-sectional data prohibit us from making a strong causal claim, but the causal direction we propose seems more plausible than the reverse.

Conclusion and implications

Individuals hold lay theories about various phenomena. Our research highlights the fact that these mere beliefs can influence the actual likelihood that a phenomenon—in this case, being overweight—will occur. Obesity is an important health concern, and much research has studied its demographic, social, and medical correlates. However, the health of the body often depends on the health of the mind, and our research is among the first to identify a true psychological antecedent of obesity—one that has an effect over and above that of the other major correlates.

The medical community has come to a fairly decisive conclusion about the relative effectiveness of changing one's diet versus exercising, and about the small role genetics plays in obesity. Our studies provided a unique empirical test of these propositions. The fact that people who believe that diet causes obesity are less likely to be overweight supports the medical conclusion that the most effective path to reducing obesity is indeed dietary change. Again, this is not to say that exercise does not help reduce weight—it does, especially if it is not accompanied by an increase in caloric intake. Our finding is simply that people who strongly believe that insufficient exercise, rather than poor diet, is the primary cause of obesity tend to have higher body masses.

Could simply informing people that eating too much is the main cause of weight gain impede the obesity epidemic? Our results from Study 6 appeared to suggest that it could. People's beliefs can change in response to coordinated public health campaigns from trusted sources; hence, our research supports initiatives such as the Small Plate Movement (www.smallplatemovement.org), which focus attention on consumption quantities. Our research demonstrates that people do have different beliefs about the causes of obesity, and it highlights the importance of acknowledging these disparate beliefs and understanding their implications.

Author Contributions

B. McFerran and A. Mukhopadhyay developed the study concept, designed the studies, collected and analyzed the data, and drafted and revised the manuscript. Both authors approved the

final version of the manuscript for submission. The order of authors is alphabetical.

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Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information may be found at http://pss.sagepub.com/content/by/supplemental-data

Notes

1. Other factors known to affect body mass (e.g., sleep patterns, smoking) are not themselves primary causes of obesity but influence weight gain or loss via their influences on eating and exercise habits. We controlled for virtually all major known correlates of BMI across our studies.
2. Criteria for classification as overweight (BMI > 25) and obese (BMI > 30) are arbitrary cut points from the medical literature, but research has suggested that health risks are more pronounced for people in the latter category (Campos, Saguy, Ernsberger, Oliver, & Gaesser, 2006; Must et al., 1999). We did not have the statistical power to differentiate between these groups.
3. Demand checks revealed that 5 participants (1 in the exercise condition and 2 in each of the other two conditions) connected the lay-theory manipulation with the dependent variable. Omitting these participants strengthened the results (M_{exercise} = 3.83, M_{diet} = 2.35, M_{control} = 3.34).

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