Supplemental Material – Refereed (SOM-R)

Lay Theories of Obesity Predict Actual Body Mass

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Pretest 1

A research assistant blind to our hypothesis approached 548 people (53% female) in public areas (e.g., waiting at subway stations) in Hong Kong and introduced herself as a university student conducting a survey for a class project. She asked them (in Chinese), "What do you believe is the main cause of obesity?" All respondents were alone. The RA recorded whether the first responses implicated poor diet, lack of exercise, or something else, and then terminated the interview. The RA also recorded the respondent's gender and location of the interview, which did not affect responses. Responses supported our hypothesis, as nearly 90% spontaneously implicated diet or exercise; fewer than 10% implicated other causes, including genetics. Poor diet was invoked most often (66.4%), followed by lack of exercise (22.9%). Since this was a face-to-face interview, respondents were not asked their weights.

Pretest 2

We compared 100 members of an online panel (53% female, mean age = 34, range 18-61) to a sample of 40 family physicians at a leading US university hospital. Participants first indicated their supposed main cause of obesity using the forced choice question. They then estimated the relative contribution of each of the three potential causes, by allocating 100 points amongst the options such that higher numbers indicated greater culpability. Results again indicated that lay people do not hold the three lay theories with equal frequency $\chi^2(2) = 29.12$, p

<. 001, $\varphi = 2.91$. Equal proportions of respondents implicated diet (44%) and exercise (48%; $\chi^2(1) = 0.1$, $p > .75$), but the genetics lay theory was far less prevalent (8%). A very similar pattern was found on the points-allocation measure, with the strongest beliefs for diet (M = 44.06, SD = 15.36), and exercise (M = 38.97, SD = 15.58), and relatively less for genetics (M = 17.06, SD = 12.51). Both diet $t(99) = 11.57$, $p < .001$, $d = 1.16$ and exercise $t(99) = 9.24$, $p < .001$, $d = .93$ were rated as contributing more to obesity than genetics.

As expected, the physicians differed significantly from the online sample $\chi^2(2) = 11.58$, $p < .001$, $\varphi = .98$. Twenty-nine respondents chose the diet lay theory while only eight chose exercise $\chi^2(1) = 11.92$, $p < .001$, $\varphi = 1.88$, one chose genetics, and two attributed equal weights across factors. The physicians also allocated significantly more points to diet (M = 51.17, SD = 14.54) than to either lack of exercise (M = 31.92, SD = 11.85; $t(39) = 5.10$, $p < .001$, $d = .81$), or genetics (M = 16.92, SD = 11.52; $t(39) = 9.25$, $p < .001$, $d = 1.47$), indicating that their beliefs were substantially more aligned with scientific agreement, and differed from lay people (see figure S1). These results confirm that considerable variation exists in both beliefs about what causes obesity, as well as the strength with which these beliefs are held.

Insert figure S1 here

Supplementary Data: Alternative Phrasings (Study 1)

We acknowledge the numerous ways the potential causes, especially diet, can be phrased. For example, “eating too much food”, could potentially be perceived differently than “eating inappropriately”, “poor diet”, or “consuming too many calories”, among others. While we

believe that our forced choice paradigm made it clear to participants that we were asking them about the relative intake vs. expenditure of calories, we conducted a separate pretest. In addition to the phrasing used in this study, we also added three additional between subjects conditions where we substituted in each of the alternative diet phrasings above and asked 205 participants from the Amazon.com Mechanical Turk panel (Paolacci, Chandler, & Ipeirotis, 2010) to indicate the primary cause of obesity. Results showed that no matter how we phrased the options, the proportions allocated to the three options were identical, $\chi^2(6) = 2.12$, $p = .91$. The same analysis was conducted for the points-allocation as well as the single-item measure used in subsequent studies. Again, no differences were found on either, $\chi^2(6) = 1.25$, $p = .97$ and $F(1, 201) = .13$, $p = .94$, respectively.

Measures of Known Correlates of Obesity (Study 2)

Education level (assessed as a continuous measure: 1 = never been to school, 2 = primary school, 3 = middle school, 4 = high school, 5 = community college/diploma program, 6 = bachelors, 7 = masters, 8 = PhD), hours of sleep per night, self-reported stress, anxiety, and depression (each 1 = very low, 5 = very high, $\alpha = .77$), having any of the following conditions: underactive thyroid (hypothyroidism), Cushing's syndrome, or polycystic ovarian syndrome (1 = yes, 2 = no), and taking any known weight-affecting medications (corticosteroids, antidepressants, or seizure medicines; 1 = yes, 2 = no; see Gutiérrez-Fisac et al., 2002; NIH, 2011).

Design and Procedure of Study 3

The survey was identical to study 2, with two exceptions. First, we included several

additional control variables: socio-economic status both currently and as a youth, since food choices are shaped early in life, (αs = .86 and .67, respectively; Griskevicius et al., 2011), home location (what best describes where you live? 1 = urban area, 2 = suburban area, 3 = smaller city or town, 4 = rural area), current pregnancy (1 = yes, 2 = no), employed outside of the home (1 = yes, 2 = no), smoker (1 = yes, 2 = no), self-reported overall health (1 = poor, 2 = fair, 3 = good, 4 = very good, 5 = excellent), interest in nutrition ("my personal interest in nutrition is…" 1 = very low, 5 = very high) and the single-item self esteem scale (1 = not very true of me, 5 = very true of me; Robins, Hendin, & Trzesniewski, 2001). Second, to ensure that our results were not driven by the response mode of the points-allocation task, we also measured lay theories using the item: "Obesity is caused more by…?" (1 = eating too much, 7 = not exercising enough). A single item has the added benefit of being able to freely correlate with other potentially related constructs, so we can assess its discriminant validity.

Distinctiveness of single-item measure

A separate study (N = 73) confirmed that this single-item measure was a distinct construct from several other psychological constructs. It was uncorrelated with appearance self-esteem (Heatherton & Polivy, 1991; r = -.02), restrained eating orientation (Herman & Polivy, 1980; r = -.07), body esteem (Franzoi & Shields, 1984; r = .10), hyperopia (Haws & Poynor, 2008; r = -.06), generalized self-efficacy (Schwarzer & Jerusalem, 1995; r = -.10), impulsivity (Puri, 1996; r = .15), lay theory of self-control limited-unlimited (r = .12), or fixed-malleable (Mukhopadhyay & Johar, 2005; r = -.07), lay theory of emotion transience (Labroo & Mukhopadhyay, 2009; r = -.18), and gender (r = -.07) (all ps >.10). For additional support, we submitted the above measures (except gender) to an exploratory factor analysis. Results revealed a five-factor solution that explained 71% of the variance. Importantly, our lay theory item loaded

on its own factor (.90), did not load on any other of the factors (all below .08) and no other constructs cross-loaded on the lay theory factor (all below .36 and loading much higher on another factor). These results provide evidence regarding the discriminant validity of lay theories of obesity from other potentially related constructs. Not only does our measure share little common variance with these other measures, in this sample, none were significantly correlated with it.

Results

A hierarchical regression with the points-allocation measure and only the study 2 control variables replicated the study 2 results. Again, the diet coefficient was significant, $\beta = -.15$, $t = 2.05$, $p = .04$. We then substituted the bipolar measure (diet vs. exercise lay theory) instead of the point allocation, and the results held, $\beta = .11$, $t = 1.99$, $p < .05$. Next, we repeated the analyses adding in the additional control variables measured in this study. The results still held on both the points allocation, $\beta = -.18$, $t = 2.50$, $p = .01$, and single item measure, $\beta = -.13$, $t = 2.37$, $p = .02$ – both explained variance in BMI above and beyond all the control variables, with parameters larger than most (see tables 2 and 3). A difference score (diet – exercise) also resulted in a significant coefficient ($\beta = -.14$, $t = 2.48$, $p = .02$).

Alternative Explanations and Reverse Causality

While we control for many potential variables that could explain the relationship we find between lay theories and BMI, we cannot definitively claim a cause and effect relationship between these two variables. However, much research has shown that lay theories are an antecedent to actual behavior, both inside and outside of the domain of food consumption. Indeed, we also demonstrate a causal effect of lay theories of obesity on food consumption

(study 6). Many studies have shown that increasing food intake is the primary cause of obesity (Livingston & Zylke, 2012). Still our central thesis linking lay theories and actual BMI relies on cross-sectional data, and reverse causation is a potential concern. We believe this is unlikely given what we state above, but this limitation should be noted.

We attempted to address two rival explanations that would produce the relationship we found, but would have causality reversed. The first alternate account is that heavier people, many of whom have tried to lose weight by reducing their caloric intake with little success, ultimately conclude that the cause must be their lack of exercise. This explanation does not seem to match our data for two reasons. First, lay theories of obesity do not correlate with the dietary restraint scale, which would be expected if this were true. Second, heavy people who have tried to diet and failed should be *equally* (or even more likely) to blame genetics than exercise (or indeed just say "I don't know"). The fact that they overwhelmingly cite exercise – in both aided and unaided recall – argues against this, as does the fact that the genetics theory does not correlate with BMI, even though it would be the least self-damning theory to adopt if another option failed them.

The second alternative explanation is rooted in cognitive dissonance (e.g., Festinger, 1957). According to this possible explanation, an exercise theory may be adopted post-hoc by someone who is overweight or obese. This could be done in order to reduce the discomfort of thinking of her/himself as being responsible for being his or her weight. This could be a self-esteem preservation tactic. In other words, an exercise theory may be less self-damning than a diet theory.

We tested this account in several ways. First, we re-analyzed the Study 3 (French) data where we had BMI, lay theory, and self-esteem data. If heavy people "switch" their (reported) lay theory to preserve their self-esteem, then self-esteem should correlate more strongly with lay

theory for high (vs. low) BMI individuals. We tested this empirically, and found no support for it in our data. For normal weight individuals (n = 173), the relationship between self-esteem and lay theory was very low (r = .06, p = .44), and identical in magnitude to those overweight or obese (n = 130, r = .06, p = .49). Still, such an argument relies on correlation, so we conducted three additional experiments to rule this account out.

In the first study, 102 US-based participants from Mechanical Turk (29% female, mean age = 28.27, SD = 8.91) were randomly assigned to one of three conditions, where they read either "Imagine you gained weight because of consuming more calories from food" (diet condition), "Imagine you gained weight because of burning fewer calories from exercise" (exercise condition), or "Imagine you gained weight" (control condition). This was followed by three self-conscious emotions (guilt, shame, embarrassment; rated 1 = not at all, 7 = very much), as well as three measures assessing how they would feel about themselves in such a scenario (1 = bad, immoral, unethical, 7 = good, moral, ethical), and a single item measure of self esteem (1= very low, 7 = very high). If an exercise theory is less self-damning, thus motivating cognitive dissonance, we would expect participants in the exercise condition to have different scores on some or all of these measures. Such a finding would cast doubt on our claim that lay theories are likely to precede behavior (actions that would cause weight gain). However, there were no statistically significant differences between the conditions on any of these measures.

As people may have trouble imagining themselves gaining weight personally, or may be reluctant to admit their true feelings in a first person scenario, we also employed a common technique of having participants rate, in the third person, how another individual would feel if (s)he gained weight. In a second experiment, also conducted on Mechanical Turk (N = 107, 38% female, mean age = 28.12, SD = 9.17), we changed the scenario from the first to the third person

perspective. The diet condition read, “Leslie has been consuming more calories than usual because of increased food consumption. Leslie is now obese.” The exercise condition read, “Leslie has been burning fewer calories than usual because of less exercise”. The control condition read only, “Leslie is now obese”. The manipulation was followed by the same dependent measures as the previous study, framed in the third person (e.g., “Leslie feels…”). We replicated the null findings.

As a robustness test, and given we are making claims based on a null finding, we ran a variant of this study a third time using the same population (N = 94, 39% female, mean age = 26.37, SD = 7.89), replacing only the statement “Leslie is now obese” with the more subtle “Leslie has recently gained weight”. Results fully replicated with no significant between condition differences. Given we observe a consistent null pattern, we conclude it is an unlikely possible alternative account.

Still, we cannot definitively claim a cause and effect relationship between lay theory and actual BMI. For example, in our study where lay theories were manipulated directly (study 6), participants who ate more during the experiment could have exercised more later. However, measuring actual calories burned over time is virtually impossible. Instruments like pedometers cannot accurately assess all the calories one burns (basal metabolic rates, which account for the most calories burned, vary with recent exercise history). It is also unclear what the “right” length of time after the study would be before assessing BMI. If we assessed exercise for the rest of the day, and those who ate more did not exercise more, that would still not be convincing evidence, even though it would support our hypothesis. Instead of exercising more, they could shave those additional calories off a later meal, or start a new exercise regimen subsequently. Even with a very long time horizon of both diet and exercise data, we would still have to rely on correlation,

since a prime may not have continuing effects outside of the laboratory for months. However, based on all the above, while our largely cross-sectional data prohibit us from making a true causal claim; the causal direction we propose seems more plausible than the reverse.

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Figure S1. Lay theories of obesity held by physicians vs. laypeople (Pretest 2)