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Crystal L. Hoyt
University of Richmond, choyt@richmond.edu

Jeni L. Burnette

Lisa Auster-Gussman

Brenda Major

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The obesity stigma asymmetry model: The indirect and divergent effects of blame and
changeability beliefs on anti-fat prejudice

Crystal L. Hoyt

University of Richmond

Jeni L. Burnette

North Carolina State University

Lisa Auster-Gussman

University of Minnesota

Alison Blodorn & Brenda Major

University of California, Santa Barbara

Crystal L. Hoyt, Corresponding Author; Jepson School and Department of Psychology,
University of Richmond; 28 Westhampton Way, Jepson Hall, University of Richmond,
Richmond, Virginia, 23173; 804.287.6825; choyt@richmond.edu

Jeni L. Burnette, Department of Psychology, North Carolina State University;
Psychology department, 640 Poe Hall, Campus Box 7650, NCSU, Raleigh NC 27695-7650; 919
515 8260; jlburne5@ncsu.edu

Lisa Auster-Gussman, Department of Psychology, University of Minnesota; 75 E River
Road, Minneapolis, MN 55455; 612.625.2818; auste010@umn.edu

Alison Blodorn, Department of Psychological & Brain Sciences, University of California,
Santa Barbara, Santa Barbara, California, 93106; 805-893-7786; blodorn@psych.ucsb.edu

Brenda Major, Department of Psychological & Brain Sciences, University of California,
Santa Barbara, Santa Barbara, California, 93106; 805-893-7238; Brenda.major@psych.ucsb.edu

The obesity stigma asymmetry model: Indirect and divergent effects of blame and changeability beliefs on anti-fat prejudice

The American Medical Association (AMA) hoped that labeling obesity a disease would not only highlight the seriousness of the epidemic and elicit resources but also reduce stigma against obese individuals. In the current work, we tested the consequences of this decision for prejudice against obese individuals. In doing so, we highlighted the complicated link between messages stressing different etiologies of obesity and prejudice. More specifically, we conducted three experimental studies ($n_{\text{Study1}}=188$; $n_{\text{Study2}}=111$; $n_{\text{Study3}}=391$), randomly assigning participants to either an obesity is a disease message or a weight is changeable message. Our results indicated that messages focused on obesity as a disease, relative to those focused on the changeable nature of weight, both (a) decreased blame and via this mechanism, *decreased* anti-fat prejudice and (b) increased, or strengthened the belief in the unchangeable nature of weight and via this mechanism, *increased* anti-fat prejudice. We call these opposing effects the stigma asymmetry model. We conclude with theoretical and practical implications of this model.

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Weight-based stigmatization and prejudice are detrimental to the mental and physical health of those who experience the bias (Hunger & Major, 2015; Major, Eliezer, & Rieck, 2012; Puhl & Heuer, 2009). One powerful predictor of this stigma is beliefs about the etiology of obesity (Crandall & Reser, 2005). Although the source of these beliefs is multifaceted, public health messages from the medical community are influential (Puhl & Heuer, 2009). Therefore, the American Medical Association's (AMA's) decision to conceptualize obesity as a biomedical phenomenon has implications for weight-related beliefs and subsequently obesity stigma. The dominant theoretical perspective in social psychology is that biological explanations cast obese individuals as blameless sufferers of a disease, and thereby reduce negative attitudes (Crandall & Reser, 2005). However, other researchers, predominantly in psychopathology, provide evidence to the contrary: biological and genetic explanations for stigmatizing attributes fail to reduce negative attitudes (e.g., Haslam, 2011; Kvaale, Gottdiener, & Haslam, 2013) and may instead have mixed blessings in terms of both increasing and decreasing facets of stigma (Haslam & Kvaale, 2015). In the current research, we work to resolve the aforementioned conflicting theoretical perspectives by offering the stigma asymmetry model. According to this model, conceptualizing the etiology of stigmatizing traits as stemming from seemingly unalterable underpinnings has the capacity to both indirectly *decrease* and *increase* prejudice through two different and opposing psychological processes. We investigate this model in the context of obesity by studying the effects of labeling obesity a disease on prejudice towards obese individuals (e.g., Gailey, 2014; Garrey, 2013; Jamison, 2013;).

Obesity Stigma

Obese individuals are often the target of widespread prejudice and experience inequities in many domains, ranging from employment to education (Puhl & Heuer, 2009). Furthermore,

evidence is mounting that experiencing social-identity based prejudice or discrimination, including weight-based prejudice, contributes to adverse mental and physical health consequences (Hunger & Major, 2015; Major et al., 2012; Major, Mendes, & Dovidio, 2013; Puhl & Heuer, 2009, 2010). The obesity stigma that drives anti-fat prejudice and discrimination arises from several sources including the belief that obese individuals have an undesirable body that violates aesthetic norms (Farrell, 2011) and the belief that obese individuals have caused their excess weight through laziness and a lack of self-discipline, (Carr, Jaffe, & Friedman, 2008; Crandall & Martinez, 1996; Crandall & Schiffhauer, 1998). One powerful vehicle for influencing these beliefs about the nature of weight are messages focused on the etiologies of obesity, which appear in public health campaigns, in the news, and in the general media. These messages often communicate, explicitly or implicitly, a judgment about whether obese people should be held accountable for their excess weight (Puhl & Heuer, 2009). In particular, the AMA's decision to conceptualize obesity as a disease is likely to have implications for how people think about the nature of weight and their subsequent attitudes toward obese individuals.

The Stigma Asymmetry Model: The Role of Blame and Changeability Beliefs

We argue that conceptualizing the etiology of obesity as a disease and therefore as stemming from genetic, or seemingly fixed, underpinnings, has the capacity to both indirectly *decrease* and *increase* anti-fat prejudice through two disparate psychological processes. First, drawing on attribution theory, labeling a stigmatized attribute as genetic can *decrease* prejudice by reducing the blame placed on stigmatized individuals for their condition (Crandall & Reser, 2005). Indeed, the relationship between attributions of controllability (seeing overweight people as responsible for their weight) and prejudice against overweight people is “one of the best-established relations in the study of attitudes toward fat people” (p.83, Crandall & Reser, 2005).

According to attribution theory (Weiner, 1985), by reducing blame, messages suggesting weight is static, versus changeable, should reduce prejudice (e.g., Weiner, Perry, & Magnusson, 1988).

Second, we suggest that messages indicating weight is static, versus changeable, will also indirectly *increase* anti-fat prejudice by strengthening beliefs about the fixed nature of obesity and therefore suggest that obese individuals have an unchanging essence. Attributing human conditions such as obesity to genetics can lead people to consider those conditions as immutable and discrete (Dar-Nimrod & Heine, 2011). In making this argument, we draw from the implicit theory perspective, which distinguishes between the belief that human attributes are changeable, called *incremental theories*, and the belief that human attributes are fixed, called *entity theories* (Dweck, 1999). The implicit theory literature has linked entity theories to both essentialist thinking and to greater prejudice (e.g., Haslam et al., 2006; Haslam, Rothschild, & Ernst, 2002; Rydell, Hugenberg, Ray, & Mackie, 2007). When characteristics that are deemed fixed are also devalued, such as with obesity, entity beliefs can serve to exacerbate stigma, in part, by imbuing the stigmatized person with an inherent ‘differentness’ that is deemed both serious and persistent. We propose that messages suggesting weight stems from invariable underpinnings will strengthen entity beliefs of weight, which entrench the undesirable characteristics of obesity in the very nature of obese individuals, thereby promoting anti-fat prejudice (Haslam, Bastian, Bain, & Kashima, 2006; Hegarty & Golden, 2008; Rothbart & Taylor, 1992).

The stigma asymmetry model, which highlights the way in which disease-focused messages may both decrease and increase stigma, can help explain why obesity-bias interventions designed to decrease controllability beliefs have little to no effect on anti-fat attitudes (Daníelsdóttir, O'Brien, & Ciao, 2010; Lee, Ata, & Brannick, 2014). Similarly, it can help explain why although biogenetic explanations of mental disorders typically decrease the

extent to which people blame those with mental illness for their problems, they are *not* directly associated with decreases in discrimination or stigmatization (Deacon & Baird, 2009; Kvaale et al., 2013; Kvaale, Haslam, & Gottdiener, 2013; Lebowitz, & Ahn, 2014; Schomerus, Schwahn, Holzinger, Corrigan, Grabe, Carta, & Angermeyer, 2012). A similar model, termed the mixed-blessings model, argues that biogenetic explanations of mental disorders serve to both reduce one component of stigma (blame) and enhance three other facets of stigma related to psychological essentialism: increased prognostic pessimism, avoidance, and perceived dangerousness (Haslam & Kvaale, 2015). The mixed-blessings model is more narrowly focused than our asymmetry model; it is concentrated squarely on the implications of biogenetic explanations for stigma toward people with mental disorders (e.g., perceptions of danger and unpredictability). The stigma asymmetry model offers a broader framework for understanding the nuanced link between perceptions of controllability and stigma by drawing upon the rich theoretical perspectives offered in the attribution and implicit theory literature.

Overview of Current Work

In the current research, we empirically tested the stigma asymmetry model within the context of obesity (see Figure 1). Specifically, we proposed and tested a model wherein conceptualizing obesity as a biologically-driven disease simultaneously reduces blame and strengthens beliefs about the fixed nature of weight, thus serving to *indirectly* both decrease and intensify weight-based prejudice. We tested these predictions using an experimental paradigm wherein we randomly assigned participants to read a message explaining why obesity is now categorized as a disease to increase beliefs in the fixed nature of weight or a message focused on increasing beliefs about the changeable nature of weight. Considering the emerging call for replications (e.g., Pashler & Wagenmakers, 2012), we sought to demonstrate the robustness of

the effect by replicating the findings across three studies using three different types of messages about the changeable nature of weight and two fixed messages describing obesity as a disease. Finally, such an approach provided an opportunity to conduct a continuous cumulating meta-analysis that combines the data from the three studies in order to “index the degree of confidence we have that a bona fide phenomenon is being investigated” (p. 334, Braver, Thoemmes, & Rosenthal, 2014).

Study 1

Method

Participants and procedure. We recruited two hundred and four participants from a StudyResponse panel¹ (<http://www.studyresponse.net/index.htm>). The StudyResponse project is a nonprofit academic service that facilitates online data collection for social scientists. This online panel of volunteers from a variety of occupations, races/ethnicities, and ages provide high quality data. Across all studies, we restricted participation to people residing in the United States. Sixteen participants, 8 in each condition, did not pass the article attention check (see below) resulting in a final sample size of one hundred eighty-eight people (47% female; 51% male; 3 people did not indicate) with an average age of 42.43 years ($SD = 11.27$). Using the BMI categories based on the National Institutes of Health (NIH) cutoff for weight categories, 3.4% were underweight, 44.1% of participants were categorized as normal weight, 22.3% as overweight, and 30.2% as obese.

We randomly assigned participants to either the *obesity is a disease* or the *weight is*

¹ Across studies we used a rule of thumb and sought 75-100 participants per condition. In Study 1 (2 conditions) we contracted for 200 participants with StudyResponse. In Study 2 (2 conditions) we paid for 200 participants on MTurk; after three days of no responses we stopped the survey and did not look at data before making that decision. In Study 3, in order to recruit approximately 100 participants per condition, we incorporated a stop-rule of 500 M-turk participants.

changeable condition. In all studies, data were collected using Qualtrics online survey software and random assignment to condition was accomplished with the randomizer function.

Participants in the *disease condition* read a New York Times article (Pollack, 2013) discussing the decision of the AMA to categorize obesity as a disease (Hoyt, Burnette & Auster-Gussman, 2014). The author summarizes some of the major benefits of this decision such as compensation for obesity related drugs, surgery and counseling as well as some of the drawbacks. Participants in the *weight is changeable condition* read a *Psychology Today* type article about the capability humans have to change their weight (Burnette, 2010). After reading their respective article, participants responded to the article attention check items. We used these items to screen out participants who did not read the article.² Next, participants completed the implicit theory of weight scale followed by the weight blame and bias scales presented in random order. Finally, participants completed demographic questions including questions on their height and weight in order to compute BMI³.

Measures

Article attention-check and manipulation check items. Following the article, we asked participants to summarize the theme of the article in one sentence and rate the understandability of the article on a 5-point scale ranging from very comprehensible (1) to very incomprehensible

² We excluded participants who failed to answer this question as well as those who wrote an answer that was not at all related to the article. We included any participant who answered this question with something related to the article. Thus, we included participants who answered this question with “obesity”, but excluded participants who wrote unintelligible combinations of letters or unrelated answers such as “no” or “good.”

³ Across the first two studies we also administered the Attitudes Toward Obese Persons scale, and in Study 2 we also administered the Weight Self-stigma Questionnaire. In Study 3, we also administered self-regulation questions. These measures are not included in this paper as they do not assess bias or prejudice and thus are not directly relevant to the research questions.

(5). For a manipulation check, participants indicated how the article they read described weight on a 5-point scale from fixed and unchangeable (1) to malleable and changeable (5).

Blame attributions. We assessed the extent to which participants blame overweight people for their excess weight by using two well-validated scales: the eight-item Beliefs About Obese Persons scale (BAOP; Allison, Basile, & Yunker, 1991) and the three-item willpower subscale of the Anti-fat Attitudes (AFA) Questionnaire (Crandall, 1994). We combined all items into a single 11-item measure of blame that demonstrated good reliability and better reliability than the individual scales alone. A sample item included “Fat people tend to be fat pretty much through their own fault.” Participants responded on 6-point scales, ranging from strongly disagree to strongly agree. Higher numbers indicate greater levels of blame (see Table 1 for reliabilities).

Implicit theories of weight (ITW). We used the previously established valid and reliable scale of implicit theories of weight (ITW; $\alpha = .82$ in studies 1-2; Burnette, 2010). The ITW consists of six items, with three entity-worded items and three incremental-worded items. A sample item included “You have a certain body weight, and you can’t really do much to change it.” Participants responded to each item on a 7-point scale (strongly disagree to strongly agree). We recoded items such that higher numbers represent agreement with an entity theory of bodyweight.

Anti-fat prejudice. We assessed anti-fat prejudice using two well-validated measures of fat bias. The 7-item dislike subscale of the Anti-fat Attitudes (AFA) scale measures antipathy toward obese individuals. It includes items such as “Although some fat people are surely smart, in general, I think they tend not to be quite as bright as normal weight people” (Crandall, 1994). We also included the 20-item Universal Measure of Fat Bias (UMB-FAT), which has been

shown to be less responsive to response bias than other scales including the AFA scale (Latner, O'Brien, Durso, Brinkman, & MacDonald, 2008). The UMB-FAT has four subscales: negative judgment, distance, attraction, and equal rights. Participants responded to items from both scales using a 6-point scale (strongly disagree to strongly agree) with coding such that higher numbers representing greater anti-fat bias. Because the results are the same for both measures and because the measures are highly correlated, we standardized and combined the AFA and the UMB-FAT into one anti-fat prejudice assessment.

Results

See Table 1 for scale means, standard deviations, intercorrelations, and reliabilities across all three studies. As a manipulation check, we conducted a univariate ANOVA showing that participants in the disease condition indicated that the article described weight as more fixed and unchangeable ($M=3.25$; $SD=.98$) than those in the changeable condition ($M=4.01$; $SD=1.28$; $F(1, 185)= 20.64$, $p<.001$; $\eta^2=.10$).

We next examined the blame/entity predictions stemming from the stigma asymmetry model. Controlling for participant BMI, we conducted a multivariate ANOVA on blame, implicit theories and anti-fat prejudice. We report analyses controlling for BMI (although results are similar without it) as it often predicts the constructs of interest⁴. This analysis revealed that those in the disease condition both reported lower levels of blame ($M=4.13$; $SD=.62$) and endorsed an entity theory of weight ($M=2.64$; $SD=.91$) significantly more than those in the changeable condition {blame: $M=4.38$; $SD=.71$; $F(1, 176)= 5.64$, $p=.019$; $\eta^2=.03$); entity theory: ($M=2.26$;

⁴ BMI data were missing from 3 participants in Study 1, 9 participants in Study 2, and 7 participants in Study 3.

$SD=.98$; $F(1, 176)= 6.08$, $p=.015$; $\eta^2=.03$). There was no direct effect of condition on anti-fat prejudice ($p = .99$; disease: $M=-.00$, $SD=.86$; changeable: $M=.04$, $SD=.94$).

Indirect effect analyses. To test the predictions that the weight messages have significant, and oppositional, indirect effects on anti-fat prejudice by decreasing blame attributions and increasing entity theories of weight, we conducted indirect effect analyses using Hayes' PROCESS macro model 4. Controlling for BMI, we conducted an indirect effect analysis with condition (1=disease; 0=changeable) predicting anti-fat bias through blame and implicit theories (Hayes, 2013). Analyses revealed two significant indirect effects of message condition on anti-fat prejudice with 95% confidence intervals: through blame (indirect effect=-.08; CI=-.177,-.018) and implicit theories (indirect effect=.16, CI=.047,.290). There was no direct effect of condition on prejudice ($p=.545$). The direction of the effects indicated that relative to the changeable message, the disease message predicted less blame ($B=-.24$, $p=.019$) and stronger entity views of weight ($B=.35$, $p=.015$). In turn, less blame predicted less prejudice ($B=.35$, $p<.001$), whereas stronger entity views of weight predicted greater prejudice ($B=.45$, $p<.001$).

Study 1 provided initial evidence for our asymmetry model. In Study 2, we sought to replicate this effect using a different message to promote beliefs in the changeable nature of weight. Specifically, we presented a message focused on why obesity should *not* be considered a disease. Instead of directly presenting evidence of the changeable nature of weight, as in the Study 1 article, this article outlined the importance of behavior change in modifying obesity rates.

Study 2

Method

Participants and procedure. One hundred seventeen participants were recruited from

Amazon's Mechanical Turk to take part in Study 2. Mechanical Turk is an internet marketplace where employers pay workers to complete small tasks. Researchers use it to recruit a diverse online sample and research has shown that it can be a source of high quality data (Buhrmester, Kwang, & Gosling, 2011; Casler, Bickel, & Hackett, 2013). Six participants were screened out based on their failure to respond properly to the article attention check (two in the disease condition, 4 in the not a disease condition), leaving a final sample of 111 participants (50% female; 49% male; 2 people did not indicate) with a mean age of 35.37 years ($SD = 13.24$). Using NIH categorization, 4.6% were underweight, 46.3% were normal weight, 30.6% were overweight, and 18.5% were obese.

We randomly assigned participants to the *disease condition*, where they read the same *New York Times* article used in Study 1, or the *not a disease* condition where they read an article of similar length describing why obesity is *not* a disease (Hoyt et al., 2014). This latter article included selected paragraphs from articles published in Forbes Magazine (Kabat, 2013) and on the Fox news website (Ablow, 2013). In this article, the authors compared obesity to smoking in order to highlight the importance of personal choice in the negative health outcomes such as diabetes and heart disease that result from obesity. After reading their respective article, participants responded to all of the same measures as in Study 1 including the attention and manipulation check items.

Results

See Table 1 for scale means, standard deviations, intercorrelations, and reliabilities. A univariate ANOVA conducted on the manipulation check item showed that participants in the disease condition indicated that the article described weight as more fixed and unchangeable

($M=3.31$; $SD=1.10$) than those in the not disease condition ($M=4.21$; $SD=1.23$; $F(1,109)=16.61$, $p<.001$; $\eta^2=.13$).

Next, controlling for participant BMI, we conducted a multivariate ANOVA on blame, implicit theories, and anti-fat prejudice. This analysis revealed that those in the disease condition reported lower levels of blame ($M=4.09$; $SD=.74$) than those in the not disease condition ($M=4.50$; $SD=.74$; $F(1,105)=6.72$, $p=.011$; $\eta^2=.06$) and endorsed an entity theory of weight significantly more ($M=2.54$; $SD=1.04$) than those in the not disease condition ($M=2.13$; $SD=.76$; $F(1,105)=4.48$, $p=.037$; $\eta^2=.04$). There was no direct effect of condition on the measure of prejudice ($p=.82$; disease: $M=-.01$, $SD=.90$; not disease: $M=-.01$, $SD=.88$).

Indirect effect analyses. Once again, we conducted indirect effect analyses using PROCESS macro model 4. Controlling for BMI, with condition (1=disease; 0=not disease) predicting anti-fat prejudice through both blame and entity theories, analyses revealed two significant indirect effects with 95% confidence intervals: there was an indirect effect of message condition on prejudice through lower blame attributions (indirect effect=-.10; CI=-.256,-.020) and stronger entity theories (indirect effect=.08; CI=.004,.255). That is, relative to the changeable message, the disease message predicted less blame ($B=-.36$, $p=.011$) and stronger entity views of weight ($B=.36$, $p=.037$). In turn, lower levels of blame predicted less prejudice ($B=.29$, $p=.036$), whereas stronger entity views of weight predicted greater prejudice ($B=.22$, $p=.053$). Thus, the disease message decreased prejudice through lower blame attributions, but increased prejudice through stronger entity theories. There was no direct effect of message on prejudice ($p=.71$).

In Study 2, using a different message focused on the changeable nature of weight, we once again found support for the stigma asymmetry model. In Study 3, we sought to replicate the

findings a third time using different articles. Specifically, we included four messages in Study 3: two that describe weight as changeable (the same article in Study 2 and a novel one) and two that present weight as fixed (the same disease article used in the first two studies and a novel one).

Study 3

Method

Participants and procedure. Participants in Study 3 included three hundred and ninety-one individuals recruited from M-Turk (60% female; 40% male; $M_{\text{age}} = 34.60$, $SD_{\text{age}} = 13.66$; 2% underweight, 39.4% normal weight, 28.6% overweight, and 28.1% obese with 1.8% missing).⁵ We randomly assigned participants to read one of four articles. Participants in the *disease condition* ($n=89$) read the same *New York Times* article used in the first three studies. Participants ($n=100$) in the *focused disease condition* read a modified version of this article; this article only introduced the decision made by the AMA without discussing any of the costs of the classification or the disagreement within the medical community about the classification. Participants ($n=101$) in the *not a disease condition* read the same article used in Study 2. Finally, participants ($n=101$) in the *diet and exercise message condition* read a fabricated article, designed to be the counterpart of the *New York Times* disease article, stating that the AMA recognized that obesity is due to unhealthy behaviors such as poor diet and insufficient exercise.

After reading their respective article, we asked participants to summarize the theme of the article in one sentence⁶. Next, participants completed the same implicit theory measure from Studies 1 and 2 and slightly shorter measures of blame attributions and anti-fat prejudice. Lastly,

⁵ These participants were in a study designed to explore the impact of weight-related media messages on stigma as well as self-regulation and self-perceptions. The self-oriented measures are not included in this paper as they are not directly relevant to the research questions; results hold when controlling for these additional assessments.

⁶ A total of 527 participants agreed to consent, 115 of these were excluded because they failed to answer at least 75% of the survey, and an additional 21 were excluded because their summary of the article indicated that they had not read the article.

they completed a final manipulation check and demographic questions including questions on their height and weight in order to compute BMI. We describe the additional or altered measures included in Study 3 below.

Measures

Manipulation check. At the end of the survey, we included five items to measure the extent to which participants supported that obesity is a lifestyle choice rather than a disease. One item failed to load leaving a four-item manipulation check (e.g., “obesity is a disease” and “obesity is not a disease but a consequence of a chosen lifestyle”) rated on a 6-point agreement scale and coded such that higher numbers represent more agreement with obesity being a disease ($\alpha=.76$).

Blame attributions. In line with best practices for opt-in online panel research, we sought to minimize respondent fatigue by slightly decreasing the length of our survey (Ben-Nun, 2008). In this study, we did not administer the 3-item willpower subscale of the AFA, thus, the blame measure consisted of the eight-item BAOP scale (Allison et al., 1991). However, the full eight-item scale had extremely low reliability due to the two reverse-scored items (see van Sonderen, Sanderman, and Coyne, 2013, for a discussion of the problems associated with reverse worded items). Removing those two items resulted in a highly reliable 6-item scale ($\alpha=.85$). Higher numbers indicate greater levels of blame.

Anti-fat prejudice. We assessed anti-fat prejudice using a shortened eleven-item scale composed of items from the two well-validated measures of fat bias used in the first two studies, the AFA and the UMB-FAT.

Results

See Table 1 for scale means, standard deviations, intercorrelations, and reliabilities. We

created a variable with two conditions by combining the two disease conditions (disease condition and focused disease condition), coded as 1 and combining the two weight is changeable conditions (not-a-disease condition and diet and exercise condition), coded as 0 because of the theoretical interest in changeable messages compared to more static disease-related messages. In support of this approach, we first conducted an ANOVA with all four conditions predicting blame and implicit theories of weight. First, there was a significant overall effect on blame, $F(1, 386)=6.36, p<.001; \eta^2=.05$). In Fisher LSD post-hoc analyses, participants in the two changeable conditions (i.e., not a disease, $M=4.74; SD=.78$; diet and exercise, $M=4.70; SD=.84$) reported higher levels of blame than participants in the two disease conditions (disease, $M=4.22; SD=1.09$; focused disease, $M=4.49; SD=.91$). The difference between the diet and exercise message and the focused disease message was marginally significant; both changeable messages are significantly different from the original disease message. We also found a significant overall effect, $F(1, 387)= 4.36, p=.005; \eta^2=.03$) on implicit theories of weight. Fisher LSD post-hoc analyses show that participants in the two changeable conditions (i.e., not a disease, $M=1.99; SD=.84$; or diet and exercise, $M=2.04; SD=.84$) reported a significantly weaker entity theory of weight than participants in the two disease conditions (disease, $M=2.38; SD=.93$; focused disease, $M=2.27; SD=.90$). And, for both blame and implicit theories there was no statistically significant difference between the two fixed messages (disease vs. focused) or the two changeable messages (not a disease vs. diet and exercise). Thus, for subsequent analyses we focused on the disease conditions compared to the changeable conditions. See Table 2 for effect sizes for comparisons between conditions predicting blame and entity theorizing using a continuous cumulating meta-analysis (CCMA; see Braver, et al., 2014).

A univariate ANOVA on the manipulation check item revealed that participants in the

disease conditions reported greater support for obesity being a disease rather than a lifestyle choice ($M=2.89$, $SD=1.08$) than participants in the changeable conditions ($M=2.28$, $SD=.00$); $F(1, 388)=33.47$, $p<.001$; $\eta^2=.08$.

Next, controlling for participant BMI, we conducted a multivariate ANOVA on blame, theories and anti-fat prejudice. This analysis revealed participants in the disease conditions reported lower levels of blame ($M=4.36$; $SD=1.01$) than those in the changeable conditions ($M=4.72$; $SD=.81$; $F(1, 381)=13.69$, $p<.001$; $\eta^2=.04$) and endorsed a stronger entity theory of weight ($M=2.32$; $SD=.90$) than those in the changeable conditions ($M=2.01$; $SD=.84$; $F(1, 381)=10.72$, $p=.001$; $\eta^2=.03$). There was no direct effect of condition on prejudice ($p=.70$; disease: $M=2.05$, $SD=1.07$; changeable: $M=2.12$, $SD=1.23$).

Indirect effect analyses. Finally, we tested the predictions that the weight messages have significant, and oppositional, indirect effects on anti-fat prejudice through both blame and theories. Controlling for BMI, we conducted an indirect effect analysis with condition predicting anti-fat bias through blame and entity theories. Analyses revealed an indirect effect of message condition on anti-fat prejudice (95% CI) through blame (indirect effect $=-.14$; CI $=-.232, -.067$) and entity theories (indirect effect $=.07$; CI $=.016, .131$). There was no direct effect of messages on prejudice ($p=.79$). The direction of the paths indicated that relative to changeable messages, the disease messages predicted less blame ($B=-.34$, $p<.001$) and stronger entity theories of weight ($B=.29$, $p=.001$). In turn, lower levels of blame predicted less prejudice ($B=.41$, $p<.001$), whereas stronger entity theories predicted greater anti-fat prejudice ($B=.23$, $p=.001$).

CCMA Analyses

We additionally conducted a continuous cumulating meta-analysis (Braver et al., 2014) of the three studies in order to investigate and compare the mean effect sizes associated with

differences between disease and changeable messages. We meta-analyzed these effect sizes, shown in Table 3, to examine the differences between the conditions across all studies. The bottom row of Table 3 shows the mean effect size, weighted by sample size. Disease messages, relative to changeable messages led to less blame, $d = -.41$, but more entity theorizing, $d = .38$ —both moderate effect sizes. It is not surprising that the effect is not large given the subtlety of the manipulation; indeed, it is impressive that merely reading a brief article can reliably and meaningfully (a) impact the extent to which participants blame obese people for their weight and (b) shift people's beliefs about the nature of weight. Thus, across the three studies, disease messages have the potential, at least indirectly, to significantly both decrease anti-fat prejudice via a decrease in blame but also to increase it via a strengthening of entity theorizing. Understanding these mechanisms can help us tailor messages that decrease blame without increasing fixed beliefs about the nature of weight.

Discussion

In this research, we found support for the obesity stigma asymmetry model, which offers a new framework for understanding the nuanced link between messages about the etiologies of obesity, obesity-related beliefs, and prejudice toward obese individuals. More specifically, messages that simultaneously promote the idea that obese people are not responsible for and do not have the ability to lose their excess weight, such as labeling obesity a disease, encourage *both* the view that obese individuals are not to blame for their obesity and that they have a negative and unchanging essence. Although allies of the AMA's decision hoped that categorizing obesity as a disease would help reduce obesity stigma (Garrey, 2013; Jamison, 2013), we elucidate how such a message, through opposing mechanisms, does not have a direct effect on anti-fat attitudes. Rather, across three experimental studies, we demonstrated that the message that obesity is a

disease, relative a message that weight is changeable both (a) decreased blame and via this mechanism, decreased anti-fat prejudice and (b) increased entity beliefs and via this mechanism, increased anti-fat prejudice.

The current research makes theoretical and practical contributions to our understanding of obesity prejudice and stigma. The dominant perspective on anti-fat attitudes focuses on the role of blame in negative attitudes toward the obese (Crandall & Reser, 2005). However, the findings in the current work suggest that this perspective is crucial but deficient without also taking into account the effect of implicit theories of weight. We also make important contributions to the stigma literature beyond obesity; by drawing on attribution theory and an implicit theory perspective we offer a model that can be extended to other stigmatizing attributes. Understanding these underlying psychological mechanisms can equip us to tailor public health messages in such a way to maximize the prejudice reducing processes and minimize the prejudice enhancing processes. Moreover, these findings lend empirical support for the mixed-blessings model in the domain of mental health, which has yet to be empirically tested.

Additional work is needed to build on the model offered in the current work. For example, future inquiry can investigate how the asymmetry model extends to other stigmatizing attributes, beyond weight, that can be conceptualized as stemming from genetic underpinnings (e.g., mental disorders; Lebowitz, 2014). More specifically, the stigmatizing characteristics that are likely most prone to this asymmetry effect are those that cross two of the three primary types of stigmas proposed by the esteemed stigma theorist Goffman (1963): “blemishes of individual character” (which is related to blame) and “abominations of the body” (which is related to entity theorizing). Additionally, building on the current work as well as the theoretical foundations laid out in the mixed blessing framework (Haslam & Kvaale, 2015), further empirical work is needed

to better understand the processes underlying *whether* and *when* people shift their focus from exculpating people for their stigmatized identity to viewing it as informative of people's very nature.

In conclusion, our work suggests that in examining all messages conveying information about the etiology of obesity, it is critical to understand the underlying psychological processes that increase or decrease stigma, not just the overall effect of a given message on stigma. A nuanced understanding of these processes has implications for reducing stigma and prejudice against obese individuals, which can have psychological, social, economic, and health benefits (Hunger & Major, 2015; Puhl & Heuer, 2009). Our findings suggest that messages designed to simultaneously decrease the blame placed on obese individuals, while also encouraging a belief in the changeable nature of weight (i.e., incremental theory) might be most effective in reducing anti-fat prejudice. Such messages are likely to promote healthy and successful weight management by decreasing levels of weight stigma (Major et al., 2012) while retaining the self-regulatory benefits of an incremental theory of weight (Burnette, O'Boyle, VanEpps, Pollack, & Finkel, 2013). We hope these initial findings foster future empirical inquiry into which messages and interventions are most effective at reducing obesity stigma.

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Table 1: Scale means, standard deviations, intercorrelations, and scale reliabilities

Dependent Variable	M	SD	1	2	3	4	5
Study 1							
1. Implicit theories	2.46	.96	.86				
2. Blame	4.23	.68	-.40***	.76			
3. UMB-Fat	2.81	.78	.21**	.08	.89		
4. AFA_Dislike	2.45	1.23	.40***	.11	.63***	.92	
5. Anti-fat prejudice	.00	.90	.34***	.10	.90***	.90***	na
6. BMI (range: 16-49)	27.18	6.35	.15*	-.04	-.29***	.01	-.16*
Study 2							
1. Implicit theories	2.31	.93	.88				
2. Blame	4.31	.76	-.59***	.86			
3. UMB-Fat	3.04	.91	-.01	.26**	.92		
4. AFA_Dislike	2.36	1.08	.05	.08	.71***	.89	
5. Anti-fat prejudice	.00	.92	.02	.18	.92***	.92***	na
6. BMI (range: 16-55)	26.04	6.66	.31***	-.31***	-.24*	-.17	-.23*
Study 3							
1. Implicit theories	2.16	.89	.88				
2. Blame	4.54	.92	-.37***	.85			
3. Anti-fat prejudice	2.09	1.17	.05	.27***	.96		
4. BMI (range: 17-58)	27.73	7.01	.10*	-.13*	-.15**		

Note: Cronbach's alphas are along the diagonal; Na: This measure combines the two anti-fat prejudice scales (UMB-Fat and AFA_Dislike).

* = $p < .05$; ** = $p < .01$; *** = $p < .001$

Table 2

Effect sizes representing the standardized difference between means in each sample/message.

	Blame	Entity Theorizing
	<i>d</i> (<i>p</i>)	<i>d</i> (<i>p</i>)
Study 3: Disease vs. Not a Disease	-.56(.000)	.41 (.005)
Study 3: Disease vs. Behavior Change	-.52 (.001)	.38 (.011)
Study 3: Disease Focused vs. Not a Disease	-.29(.039)	.32 (.025)
Study 3: Disease Focused vs. Behavior Change	-.25(.078)	.29 (.044)
Pooled Cohen's <i>d</i> Overall Disease vs. Changeable Messages	-.40(.000)	.35 (.000)
95% CI	-.54, -.26	.21, .49
Homogeneity tests <i>Q</i> ; <i>I</i> ²	3.42, ns; 12	.46, ns; 0

Note. Positive effect sizes indicate that the first named group reported more than the second group. Negative effects sizes indicate the first name group reported less than the second group.

Table 3

Effect sizes representing the standardized difference between means in each sample/message.

	Blame	Entity Theorizing
	<i>d</i> (<i>p</i>)	<i>d</i> (<i>p</i>)
Study 1: Disease vs. Changeable	-.37 (.013)	.40(.008)
Study 2: Disease vs. Not a Disease	-.55 (.005)	.40(.021)
Study 3: Disease/Dis. focused vs. Not Disease/Behavior Change	-.39(.000)	.36(.001)
Pooled Cohen's <i>d</i> for Overall Disease vs. Changeable Messages	-.41 (.000)	.38 (.000)
95% CI	-.57, -.26	.23, .54
Homogeneity tests <i>Q</i> ; <i>I</i> ²	.61, ns; 0	.20, ns; 0

Note. Positive effect sizes indicate that the first named group reported more than the second group. Negative effects sizes indicate the first name group reported less than the second group.

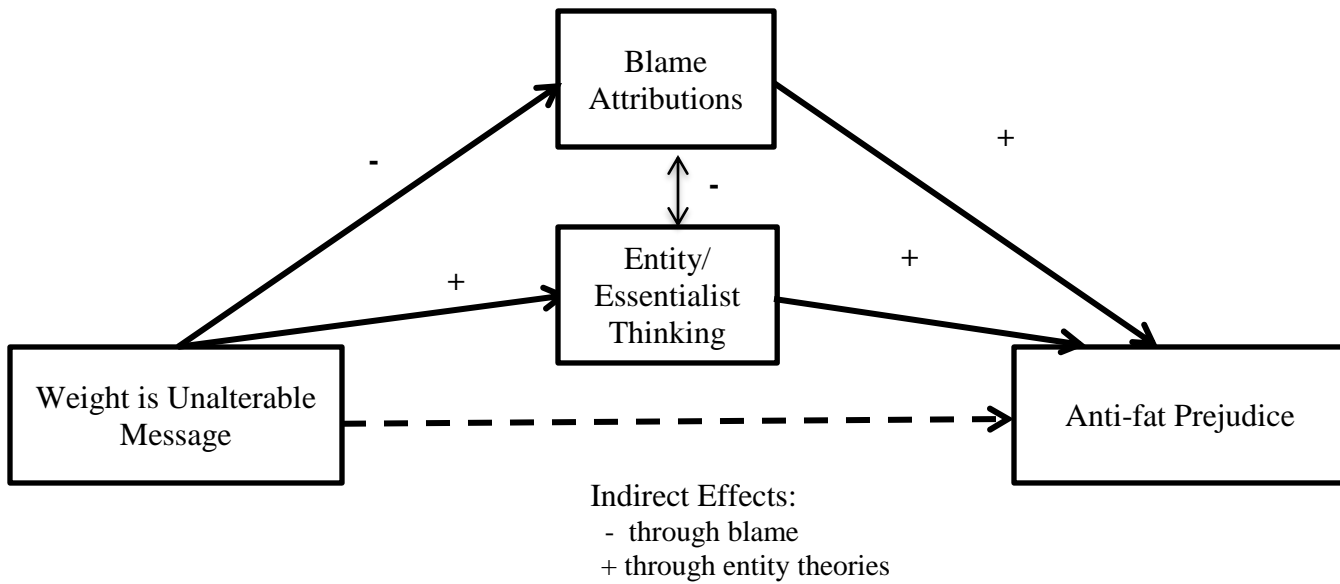


Figure 1. Theoretical representation of the obesity stigma asymmetry model. Indirectly, messages suggesting that weight is unalterable serve to both diminish and intensify weight-based prejudice.