THE EFFECTS OF CAUSAL BELIEFS ON THE STIGMATIZATION OF OBESITY

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ABSTRACT OF THE THESIS

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Weight disorders and overeating are increasingly being labeled as addictions. It is important to identify and understand the consequences of this label for the stigmatization of obese individuals, the treatments to which they are assigned, and the anticipated outcomes of those treatments. This study was designed to determine whether causal beliefs about the etiology of obesity affect participants' attitudes toward obese individuals. It also examined participants' beliefs about prognoses and appropriate treatments. In a 2x3 between-subjects design, undergraduate students from Rutgers University (*N*=374) were assigned randomly to one of six conditions. Participants read a scenario about either an obese woman or an obese woman with binge eating disorder (BED) followed by an account of the cause of her obesity as a psychological disorder, a biological addiction, or a disorder of ambiguous origins (Cause). Participants then completed a battery of questionnaires designed to assess stigma and beliefs about the person and her treatment and prognosis. The Cause manipulation check revealed no difference between groups and there were no significant differences between the Cause conditions on any of the dependent measures. Participants in the obesity with BED condition rated obese persons as less attractive and more to blame for their weight, and indicated that they desired more social distance from obese persons compared with participants in the non binge eating condition. Participants also judged obese persons

with binge eating disorder as having a more severe illness, to be more likely to drop out of treatment, and rated their illness as less curable. The demonstration of the importance of obese persons' behavior (binge-eating) has important implications for understanding the stigmatization of this disorder. Future work should examine treatment attributions and prognostic beliefs of mental and physical health professionals, and of obese individuals with and without binge eating disorder. Efforts at stigma reduction should target binge eating as well as obesity.

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I am honored to acknowledge the people who helped in the design, implementation, and composition of this work. G. Terence Wilson, Ph.D., my mentor, was a source of constant support and encouragement and was instrumental in the creation of this study. I would like to thank the other members of my committee, Richard J. Contrada, Ph.D. and Robert Karlin, Ph.D. Thank you, Dr. Contrada, for your unremitting support, time, and feedback. Thank you, Dr. Karlin, for your input on the research design, your help with recruitment, and your statistical support. I would also like to thank Dorian Hunter-Reel, an early collaborator in this project who shared her ideas, time, and resources. Thank you to the research assistants who diligently worked on this project. Finally, thank you to Willis Taylor and my family, whose love and patience give me the strength to persevere.

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Introduction

Obesity and Stigma

Obesity is recognized as a global epidemic by the World Health Organization (WHO) and affects approximately 32.9% of the adult population in the United States (Ogden, Yanovski, Carroll, & Flegal, 2007). Obesity is not included in the Diagnostic and Statistical Manual of Mental Disorders IV-TR (DSM), but generally accepted clinical characteristics include: elevated fat mass (Body Mass Index over 30), elevated risks for physical and psychological disorders, and poor body image (e.g., Friedman & Brownell, 2002; Friedman, Reichmann, Costanzo, Zelli, Ashmore, & Mustante, 2005; Schwartz & Brownell, 2005). Although not all obese persons have an eating disorder, binge eating is a common clinical feature among persons who are obese and who seek treatment for weight loss.

There are numerous physical and social consequences for obese individuals. One of the more salient negative social effects is the stigma and subsequent discrimination that these individuals face. The stigma concept derives its origins from the Greek language and refers to the physical marks used to expose disgrace (Katz, 1981). Goffman defined stigma as an attribute that "is deeply discrediting" (Goffman, E., 1963; Katz, 1981, p.2). Crocker, Major, and Steele (1998) more recently defined stigma as "a devalued social identity across most social contexts" (King, Hebl, & Heatherton, 2005, p. 109). Aspects of this devaluation include: discrimination, negative judgments, economic disadvantages, and negative stereotypes. Although the field of stigma and obesity is relatively new, the pervasiveness of obesity stigma is well documented in the literature. In a general study of stigma, obese persons reported teasing, harassment, slurs, insults,

negative judgments and assumptions, and perceived discrimination at home, in the work place, among strangers, and among friends (Cossrow, Jeffery, & McGuire, 2001). Puhl, Andreyeva, and Brownell (2008) estimated that the prevalence of weight/height discrimination ranges from 5% among men and 10% among women; when BMI increased above 35, discrimination increased to 40%. The authors found that women were more discriminated against than men, as were younger individuals with a high BMI (particularly women). The discrimination reported in this study often was experienced through interpersonal relationships; people who were discriminated against felt less respected and perceived as inferior. Andreyeva, Puhl, and Brownell (2008) estimated that weight discrimination among Americans has increased from 7% in 1995-1996 to 12% in 2004-2006; the authors attributed this increase to the rise in obesity prevalence.

The effects of stigma in the workplace include weight bias in hiring, negative perception of obese employees, and unjust disciplinary actions (Fikkan & Rothblum, 2005). In a review of the literature, Roehling (1999) found discrimination against overweight employees at the level of hire, promotion, compensation, and discharge. In studies where participants were asked to assess job applicants or workers, they were less likely to want to work with or to promote obese persons (Brink, 1988; Jasper & Klassen, 1990). Stigmatization toward obese individuals also exists in health care settings (Fabricatore, Wadden, & Foster, 2005). A recent study surveyed primary care physicians about their attitudes toward obese patients. Fifty percent of the respondents reported that obese patients were awkward, unattractive, ugly, and noncompliant. To a lesser extent, physicians rated obese individuals as lazy and sloppy (Foster et al., 2003). Health practitioners also demonstrate implicit biases against obese individuals. Hebl and Zu

(2001) evaluated the effects of different vignettes describing patients as obese versus normal weight. Physicians recommended the normal weight client for psychotherapy more than the obese client, and they rated obese persons as more annoying, having less self discipline, and not benefitting from treatment. Schwartz, Chambliss, Brownell, Blair, and Billington (2003), using the Implicit Association Test (IAT), found that health professionals attending an obesity conference associated the word "fat" with the words "lazy", "stupid", "bad", and "worthless." The findings regarding patients' perceived stigma have been variable. Rand and MacGregor (1990) reported that 80% of obese patients felt that they had been treated disrespectfully by doctors. In a separate study, Anderson and Wadden (2004) found that only 13.5% reported feeling this way.

Stigma also affects children and adolescents. Cramer and Steinwert (1998) found that children as young as 3 years old displayed stigmatizing attitudes, rating chubby figures as "mean." Kraig and Keel (2001) found that children ages 7-9 rated figures of chubby children more negatively on descriptions such as "gets teased", "lonely", "lazy", and "ugly." The study of adolescents and stigma has focused on the school environment and the effects of teasing (Neumark-Sztainer & Eisenberg, 2005). Adolescents who are teased on account of their weight reported that they are bothered by this teasing. Teased teens were more likely to attempt to control their weight by taking diet pills and laxatives, by skipping meals, and by binge eating (Neumark-Sztainer, Falkner, Story, Perry, Hannan, & Mulert, 2002).

The consequences of stigma for obese individuals are manifold and may include an increased risk of depression, economic hardship, isolation, social withdrawal, and overeating (Puhl & Brownell, 2003). Obese persons who are stigmatized have negative

body image and poorer psychosocial functioning (Annis, Cash, & Hrabosky, 2004). Also, stigma may increase the incidence of binge eating in obese individuals (Puhl & Brownell, 2003). A potential source of this stigma may be the causal beliefs that people hold about the etiology of obesity. Such causal beliefs may not only affect the stigmatization of obese individuals, but also may affect their clinical prognoses, treatment expectations, treatments received, and treatment adherence.

Causal Beliefs

The causal beliefs associated with mental illness may influence mental health practitioners, patients, and the general public. Such beliefs may be integral to aspects of treatment and recovery, such as treatment choice, patient prognosis, illness perception, and the stigma associated with the illness. The results of such attitudes may lead to discrimination and exacerbate illness (Walker & Read, 2002). Patients' conceptualization, or mental representations, of their own illness (particularly whether that illness is chronic or acute) affects post-treatment stress and indicates poor prognosis (Rabin, Leventhal, Goodin, 2004). In a recent review, Leventhal, Weinman, Leventhal, and Philips (2008) expounded upon the effects of illness representations (which include causal beliefs). Illness representations may affect treatment adherence, treatment initiation, and handling of illness symptoms and side effects (e.g., depression). Leventhal et al. (2008) suggested that patients' understandings of their own illness must be an integral aspect to effective and efficient behavioral interventions to manage chronic illness.

Whereas there are various causal explanations about the etiology of mental illnesses, the biological-pharmacological and the psychological-cognitive-behavioral

approaches prevail (Lam, Salkovskis, & Warwick, 2005). Bio-psychosocial accounts of mental illnesses exist; however, practitioners, the public, patients, and even the media tend to think in the dichotomous terms of the biological versus psychological causes of the disorder (Lam et al, 2005). The implications of biological versus psychological causal beliefs might affect stigma and thereby influence patient treatment and prognosis.

The medicalized-biological casual account for mental illness is widely supported and has deep roots. The aim of this causal account is "to define mental illness as an illness like any other" (Walker & Read, 2002). Biological causation of mental illness is endorsed by the United States National Institute of Mental Health (NIMH), the National Alliance for the Mentally Ill (NAMI), pharmaceutical companies, and insurance companies, for reasons as varying as the desire to reduce patient stigma to that of making a profit or to reducing health care costs (see Walker & Read, 2002). Proponents of the biological model of mental illness believe that this approach reduces stigma, as it portrays the patients as the "victims" of a biologically based disorder, something for which they must seek medical treatment (Wahl, 1999). There is evidence that patients may prefer this model. Gammell and Stoppard (1999) found that depressed women preferred to view their illness as biologically based because it felt more legitimate. However, this study found these same women had lower self-efficacy in believing that they could help themselves. The authors concluded that while women may prefer a biological explanation, they may be less likely to seek treatment and more likely to view their disease as something that only can be helped via pharmacotherapy, which may not be the best line of treatment (Dimidjian et al., 2006; Gammell & Stoppard, 1999).

Other proponents of the biological model, such as NAMI, believe that a psychosocial model of mental illness will only further stigmatize the sufferers. NAMI fears that describing mental illness as something other than biological may place blame on both the individual and the individual's family. They further argued that if an individual is unable to overcome illness, it will be seen as a character flaw and weakness (Goldstein & Rosselli, 2003). In support of this argument, one study demonstrated that people were less prone to blame individuals for task failures if they viewed their depression as biologically based (Mehta & Farina, 1997). It has also been demonstrated that more stigma is attached to people with controllable versus uncontrollable disabilities (Jones, Farina, Hastorf, Markus, Miller, & Scott, 1984). However, there is contradictory evidence as to the benefits of the biological model. In fact, a biological model may do little for, if not worsen, the attitudes and stigma associated with mental illness.

Studies have assessed public preferences for causal models and there is a great concurrence in the literature: People prefer a psychosocial explanation (e.g. Wahl, 1987; Angermeyer and Matschinger, 1996; & Read and Harre, 2001). Other studies have investigated the effects of causal beliefs on stigma. In New Zealand, researchers found that people harbored more negative attitudes towards "mental patients" if they believe in a biological model of mental illness (Read & Harre, 2001). A similar study found that when people hold biological causal beliefs, they are likely to see patients as more dangerous and unpredictable, and they try to avoid contact with them (Read & Law, 1999). Psychological casual explanations may influence how the affected person feels about their own illness and its course. Fisher and Farina (1979) found that a social learning explanation resulted in persons making a greater effort at change. Additionally, a

psychological model may support a view that recovery is possible, as opposed to a more "incurable" biological model (Phelan, 2002).

Goldstein and Rosselli (2003) examined the effects of causal beliefs about patients with depression. College students were surveyed to determine their views on the etiology of depression and their subsequent reports on treatment preference, empowerment, and stigma. A factor analysis revealed three etiological causes: psychological, biological, and environmental. Participants who viewed depression as biological rated the patients as having greater empowerment, less stigma, and recommended psychotherapy treatment. Participants who viewed depression as psychological rated patients as having greater self-efficacy, but they also reported greater stigma. Participants who viewed depression as environmentally-based reported that patients were more to blame and more violent than less-depressed persons. However, participants in the environmental condition desired less social distance from depressed patients.

One study investigated the effects of manipulated causal beliefs on a person with psychotic symptoms (Walker & Read, 2002). Participants' attitudes toward the person with psychotic symptoms were assessed both prior to and after viewing a video in which the person displayed psychotic symptoms. The conditions included a biogenetic condition, a psychosocial condition, and a combined, biogenetic/psychosocial, condition. Participants in the psychosocial condition displayed a small improvement in their attitudes about the individual in the video, but this was not significant. Participants who had greater contact with mental health service demonstrated less bias. The authors

cautioned against acceptance of a biological model and suggested that exposure to mental health may be useful in destigmatizing mental illness.

Research has investigated the effects of manipulating causal beliefs and assessing participants' reaction to a patient's illness, treatment, and prognosis. Recently, Lam and Salkovskis (2007) explored the effects of causal explanations on persons' perceptions of a patient with panic disorder. In this investigation, participants diagnosed with either anxiety or depressive disorders were presented with the clinical background of a person with panic disorder and viewed a video of the patient describing a panic episode. In each condition, participants received different information about the etiology of the disorder: biological, psychological, or ambiguous. Participants were then asked to evaluate the person with panic disorder on a variety of dimensions. Results showed that the target person with panic was viewed as having a worse prognosis after treatment if the panic was ambiguous or biological. If the panic was biological, by contrast, the person with panic was deemed to need longer treatment and rated as posing a higher risk of danger to self and others. There were no differences in need for help, motivation for change, or level of disability. The results of this study indicate that labeling panic disorder as a biologically based illness may lead people to believe that prognosis is worse, treatment needs to be longer, and that they are more likely to harm themselves and others.

In a similar study, Lam et al., (2005) assessed the effects of biological versus psychological causal explanations with participants from a community sample. The authors concluded that psychological causal beliefs may reduce stigma, whereas biological beliefs may either increase or have a neutral effect on stigma. These conclusions are based on participants' reports that patients' with psychologically-based

illness were significantly more likely to be cured, and significantly less likely to hurt themselves, require professional help or hospitalization, and to be labeled as disabling.

In summary, the current information on causal beliefs is mixed. Goldstein and Rosselli (2003) found that labeling depression as a psychological disorder increased stigma. However, the psychological label also increased perceived self-efficacy. Lam and Salkovskis (2007) reported psychological labels increased ratings for better patient prognosis. The research demonstrates beneficial effects for varying explanations. However, the literature does underscore the point that endorsing a purely biological model may not be best for patients. The way practitioners, patients, and the public perceive the causes of mental illness may affect treatment, perceived prognosis, and potential stigmas attached to the person.

Causal Beliefs and Obesity

The study of obesity, causal beliefs, and stigma is grounded in the relationship between causal beliefs and the perception of control over obesity (Puhl & Brownell, 2003). Crandall (1994) proposed that the more controllable people believe obesity to be, the more they will hold obese persons responsible for their weight; the more responsible people deem obese individuals, the more those individuals will be stigmatized. More biological accounts of obesity may result in less perceived responsibility by comparison with behavioral/psychological explanations of obesity. In a review of the origins of obesity stigma, Puhl and Brownell (2003) list "perception of causality and controllability" as one of the "core components underlying the stigma of obese individuals (p. 216)".

Previous research has attempted to reduce stigma toward obese individuals by comparing different etiological models and by attempting to influence the controllability or blameworthiness that is often attributed to obese individuals (Crandall, 1994). Lewis, Cash, Jacobi and Bubb-Lewis (1997) sought to determine whether providing a biogenetic explanation of obesity would lesson stigma. Their study included a biogenetic condition, a behavioral strategy condition (describing strategies of eating, nutrition, and exercise), and a control condition about memory. Participants in the behavioral strategy condition were more likely to blame obese persons for their weight and, although the biogenetic condition yielded the least amount of bias on all scales, the only significant finding was blame. The authors concluded that there was limited evidence to indicate that explaining obesity as biogenetic would have an effect on stigma, but that this may be due to the brevity of the experimental condition.

Crandall (1994) attempted to alter participants' beliefs about the causes of obesity by persuading them that weight is due to physiology. Participants were either in a persuade "Sailing and Weight Control" condition, where obesity was described in terms of physiology, or a "Sailing and Stress" condition. Participants in the persuade condition scored lower on willpower (reporting that obese persons have more willpower) and lower on dislike (reporting less dislike toward obese persons). The results from Crandall (1994) and from Lewis et al. (1997) demonstrated that individual attitudes toward obese individuals may be manipulated by causal beliefs and that a biological, genetic, or more physiological cause may reduce such stigma. However, neither of these studies provided adequate control groups and without comparing other etiological causes (explicitly and

not via strategy, for example discussing weight regulation and exercise only) it is difficult to be certain that a biological explanation reduces stigma.

DeJong (1980) found that participants who were told that obesity was a glandular disorder displayed differences in reported beliefs from those participants given no causal information. The latter participants evaluated an obese person less favorably and reported that they liked the obese person less. Teachman, Gapinski, Brownell, Rawlins, and Jeyaram (2003) provided different causal explanations for obesity and subsequently tested implicit Anti-Fat bias (via IAT). Out of the three conditions in this study (control-no prime, genetic prime, and behavioral prime), participants who were told that obesity was mainly due to overeating and lack of exercise (behavioral prime) demonstrated higher implicit and explicit bias compared to the control condition. The authors reported a marginally significant difference between the genetic and behavior prime groups. Interestingly, participants in the genetics prime group did not demonstrate lower bias than the control group, indicating that this explanation may neither worsen nor improve stigma toward obese individuals. Such findings have been obtained in research on other areas of mental health (e.g., Lam et al., 2005). A recent study investigated stigma and obesity in the context of customer service (King, Shapiro, Hebl, Singletary, & Turner, 2006). This group used a model based on justification-suppression model (JSM) (Crandall & Eshleman, 2003), which states that justified stigma (e.g., controllability of weight in obesity) is more likely to be expressed. The authors found that when they removed the justification for bias by having an obese confederate explain that he/she was physically active and on a diet, subsequent discrimination (from store clerks) decreased.

Researchers are now investigating the relationship between causal beliefs of obesity and treatment. In a recently published report from the United Kingdom, investigators explored the relationship between causal beliefs of obesity among general practitioners (GPs) and lay persons and solutions/treatments for obesity (Ogden & Flanagan, 2008). Similar to the work conducted by Lam and Salkovskis (2007), this literature is concerned with the impact that these beliefs have on mental health services provided and received. Interestingly, GPs reported believing that obesity is more behavioral, structural, social, and psychological than biological, as opposed to lay persons who endorsed a biological model. The authors found that GP's causal beliefs mapped to the treatments they believed suitable; they did not find this relationship among lay persons. The authors proposed that the disconnection between more medicalized solutions to obesity and GP's disbelief of obesity fitting into a medical model may contribute to the low effectiveness of obesity care. Also, the noted difference between GP's and lay person's beliefs demonstrates a "mismatch" that may contribute to failure of treatments.

Current Study

The current study was designed to expand upon the extant literature on causal beliefs, patient prognosis, and stigmatization of obesity. We aimed to assess the effects of various causal explanations (Cause) on participants' beliefs about treatment, prognosis, and associated stigma for a person who is described as obese with BED, and a person described as obese without BED (Target). We included the binge eating versus non binge eating variation (presence of BED) because this is a clinical feature of obesity that may underlie stigmatization, particularly as binge eating is defined by being out of control.

This feature has not been a target of interest in previous work. Also, we adjust the biological model to describe obesity as a biological addiction, as this is currently widely portrayed by the media and endorsed by lay public and is gaining increasing support in more basic research (e.g., Avena, Rada, & Hoebel, 2008). We included a control condition, modeled after Lam and Salkovskis (2007), wherein obesity was described as having ambiguous origins. Research on the effects of causal beliefs in most mental disorders has provided evidence that a biological model may do little to reduce stigma. However, the obesity literature would seem to promote this explanation, as it adjusts people's negative attributions about the causes and maintenance of obesity. We hypothesized that the psychological condition would yield the least amount of bias and the best prognosis for recovery, as we expected people to have more negative attributions to food addiction and would therefore render a worse prognosis. We expected more stigma toward obesity with presence of BED than toward obesity without BED, as people may blame obese persons more if they are engaging in binge eating behavior. We expected that binge eating and the label of biological addiction will yield the most stigma and worse prognosis, as this includes both the negative attributes held toward a food addiction and binge eating behavior.

Methods

Participants

Participants were recruited from the undergraduate subject pool at Rutgers University and from students enrolled in a quantitative methods course. A total of 374 students, 154 men (41.2%) and 220 women (58.8%), participated. Participants were self-identified as Caucasian (50%), African American (9.6%), Hispanic/Latino (7.5%), Asian

American (25.9%), Multicultural (6.4%), and Native American (.5%). Our main inclusion criteria were that participants be 18 years of age or older and have hearing and vision within normal range. Students from the undergraduate psychology subject pool received course credit for their participation. Students from the quantitative methods course received extra credit for their participation.

Design

This study was a 2 (Obesity without Binge Eating; Obesity with Binge Eating) x 3 (Biological Addition, Psychological, Ambiguous) design. Our main outcome measures were level of stigmatization of obesity and the target patient's perceived prognosis. Secondary predictors of interest included the effects of gender, history of eating disorder or other psychological disorder, and race.

Materials

Scenarios

Obesity without Binge Eating Disorder (Part I)

Susan, age 37, has three children, Bill and Steve aged 7 and 11 and Cathy aged 5. Until she had her third child, Susan, who is 5'5" tall, was a healthy weight. But after Cathy was born Susan never lost the 40 pounds of "baby weight" as she had after her other pregnancies; instead, she gained even more weight. So instead of weighing 125, as she had since late adolescence, she has weighed about 185 ever since, and instead of a size 8, she wears a size 18 to 20. Susan has tried a number of diets, from grapefruit to Atkins to Weight Watchers. These generally work in the short run (she can lose 5-10 lbs), but as soon as she goes back to eating relatively normally she winds up back at about 185. So she feels helpless to take off the weight. Exercising is much harder as well. She used to love to play tennis, but no longer. Finally, since she gained the weight, her husband seems much less interested in sex. While he says the change is due to aging, Susan thinks it is because the weight makes her unattractive to him. Part of the problem is the kind of foods her kids and husband demand and the foods she ends up eating. These include pastas, sugary cereals, cookies, and other unhealthful and fattening foods. None of the children is overweight, nor is her husband, who also loves pasta. So, Susan, who does all the cooking, is constantly surrounded by fattening food. It is very hard never to give in.

She also tends to give in more under times of stress and when she is upset. This is also when her diets tend to fail.

Obesity without Binge Eating Disorder-Biological Addiction Explanation (Part II)

There is now evidence to suggest that particular parts of the brain are involved in the development of obesity and that obesity is caused by a physical addiction to food. Research indicates that food is an addiction just like cigarette smoking and may be impacted by genetic factors. Like smoking, there are substances in high caloric food that satisfy craving. We know something about cravings; the way to stop them is to never satisfy the craving (it will only get worse). But, food cravings must be reinforced because you must eat something. Also, while eating unhealthfully looks and even feels like voluntary behaviour, it occurs when stress makes the brain even more demanding of calories.

Obesity without Binge Eating Disorder-Psychological Explanation (Part II)

There is now evidence to suggest that obesity may be the result of some psychological processes relating to strict dieting and emotions. Research indicates that obesity involves patients' acting in a vicious circle with dieting and unhealthful eating, where the more they restrict overall food intake and certain types of food, the more likely they are to overeat. This cycle doesn't allow them to eat healthfully, gain control of their eating and potentially to lose weight. This cycle further decreases self-esteem. Obese individuals who eat unhealthfully may often have relationship and emotional difficulties that cause them to eat unhealthfully and stay in this negative cycle.

Obesity without Binge Eating Disorder-Ambiguous (Part II)

Research suggests that the causes of obesity are not yet entirely clear.

Obesity with Binge Eating Disorder (Part I)

Susan, aged 37, has been obese for years; this has had considerable negative impact on her life. She often has episodes where she binge eats. During these episodes, she eats large quantities of food and feels totally out of control with her eating. A typical binge for Susan may include a couple pints of ice cream, half a box of Oreo cookies and pastries. Prior to binge episodes, she experiences a desire to eat foods that she had been trying to "cut out" of her diet, such as ice cream. Often she craves these foods and is upset with herself for not having better control. Her thoughts are that she is worthless and will never be able to eat normally or to lose weight. Once Susan begins eating the foods she was thinking about, she can't stop. At first she feels comforted and relieved by the food, but this is only temporary. Soon she is full to point of discomfort and feels disgusted with her behavior. Her latest binge episode happened in her car after she left the supermarket last

Sunday. She felt upset before she went, having just had a fight with her husband about her weight. When she was at the store she passed by the ice cream and candy isles several times before she filled her cart. She had not eaten these foods for 2 days. She began to feel an intense anticipation that is typical for her before the episode, looking forward to eating her favorite sweets and forgetting the fight with her husband. Susan returned to her car with the food and binged. She felt extremely guilty afterwards; she felt fat; and she felt worse about the fight with her husband. She vowed to never eat these things again.

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Obesity with Binge Eating Disorder-Ambiguous Explanation (Part II)

Research suggests that the causes of obesity are not yet entirely clear.

Measurements

Patient Attitudes Questionnaire (PAQ)

This questionnaire previously was modified by Lam and Salkovskis (2007) and is based on the clinical assessment questionnaire (CAQ), used to measure the clinical assessments of clinicians. The authors' adaptation included clarifying the language to be less technical and more appropriate for non-clinicians and they reported good test-retest

reliability (r = 0.86) for the previous version and for their modified version (r = 0.82). For the current study, the questionnaire was adapted to inquire about a patient who is obese. The current study utilized 24 items, 23 of which we submitted to factor analysis to construct subscales. Items are rated on a 0-100 point visual analogue scale, where scores of 0 or 100 convey extremely positive or negative judgments, depending on the item. Items assess treatment matching and prognosis for the described obese individual. Item 24 asks how long treatment is necessary (in days). This questionnaire asks questions specifically in regard to the woman in the scenarios.

Antifat Attitudes Test (AFAT)

The AFAT is a 47-item scale, with each item rated on a 5-point Likert scale ranging from "Strongly disagree" to "Strongly agree" (Lewis et al., 1997). Several items are reverse-keyed and higher total scores indicate greater bias. The AFAT has three internally consistent factors for both males and females (Cronbach's α = .77-.95) that are not correlated with social desirability: Social/Character Disparagement (15 items), Physical/Romantic Unattractiveness (10 items), and Weight Control/Blame (9 items). The scales have demonstrated discriminant validity. This questionnaire assesses stigmatization of obesity in general, not specifically in regard to the woman in the scenarios.

Universal Measure of Bias of Fat (UMBFAT)

The UMBFAT is a 20-item scale, with each item rated on a 7-point Likert scale ranging from "Strongly agree" to "Strongly disagree" (Later, O'Brian, Durso, Brinkman & MacDonald, in press). The UMBFAT displays good internal consistency (Cronbach's α = .87) and a clear 4 factor structure that includes: negative judgment (4 items), distance

(4 items), attraction (4 items) and equal rights (4 items). The UMBFAT demonstrates convergent validity, as it correlates highly with two established measures of bias (*r*=.50-.81*)*. The strengths of this scale are its universal applicability to other areas of stigmatization and its neutrality with regard to social desirability. As with the AFAT, this questionnaire assesses stigmatization of obesity in general, not specifically in regard to the woman in the scenarios.

Anti-Fat Attitudes scale (AFA)

The AFA is a 13-item scale, with each item rated on a 9-point Likert scale ("Very strongly disagree" to "Very strongly agree") and with higher scores indicating greater bias (Crandall, 1994). The AFA consists of 3 internally consistent subscales: Dislike (Cronbach's α =.84; 7 items), Fear of Fat (Cronbach's α = .79; 3 items) and Willpower (Cronbach's α = .66; 3 items). Dislike and Willpower are correlated (*r* = .43), but Fear of Fat is not correlated with either of the other two factors. This scale demonstrates convergent validity with other measures of stigma (see Latner et al., in press). As with the previous measures of stigmatization, this questionnaire assesses stigmatization of obesity in general, not specifically in regard to the woman in the scenarios.

Demographics Questionnaire

A brief demographics questionnaire was created for this study. Participants were asked to report: date of birth, race/ethnicity, gender, highest level of education, parents' highest level of education, family household income, height, weight, primary language and parents' country of birth.

Manipulation Check

A brief manipulation check was created for the purpose of this study. It consisted of 7 items rated on a 5-point Likert scale, ranging from "Definitely disagree" to "Definitely agree". For the three main questions aimed to assess the effectiveness of the manipulation, participants were asked whether they agreed that obesity is a biological addiction, a psychological disorder, or ambiguous. Participants also were asked if they agreed with the account the read in the experiment.

Procedure

Participants signed up for the experiment either online, by email, or in class. Participation involved one group session that lasted approximately 30 minutes. Group size depended on the number of participants who signed up for a given session and ranged from approximately 2 to 40. When all participants had arrived, procedures for the experiment were described to the group by a member of the research staff. Packets were divided into "Male" and "Female" piles and were pre-ordered prior to the experiment to randomize participants into one of the six experimental conditions. Participants retrieved the appropriate packets, which included a consent form, one of the six scripts (see materials section) and all the questionnaires. Participants were asked to read carefully and complete the packets. At the end of the experiment, participants returned their packets and received a copy of the consent form and a debriefing form that described the nature and purpose of the experiment.

Statistical Analytic Plan

A principal components extraction method with Varimax rotations was utilized to determine an appropriate factor structure for the PAQ. We utilized Analysis of Variance

(ANOVA) to determine the effects of the Cause, presence of BED, and gender on the dependent variables of stigma and patient prognosis. We also investigated the effects of race and history of mental health treatment.

Results

Data Reduction/Factor Structure

Table 1 reports the results of the principal component extraction for the PAQ. Six factors had Eigenvalues > 1 and accounted for 59.46% of the variance (See Table 1). Items with factor loading above .40 were included. The highest loading item for each factor correlated significantly with the other items on the factor (p <.05). Items 1-6 defined Factor 1, which is best described as "Illness Severity". Factor 2, labeled "Treatment Appropriate," was defined by items 17 and 21-23. Factor 3, labeled "Relapse," included items 11-12 and 7-9. Items 14-17 defined Factor 4, which was best labeled as "Availability to Change". Factor 5, labeled "Dropout," included items 10-13. The last factor was labeled "Curable" and was made up of items 18-20.

There were two items that loaded onto multiple factors. Item 17 loaded onto Factor 2 (Treatment) and Factor 4 (Availability to Change). It is appropriate for this item to be included on both factors as it assesses treatment fit which involves both treatment and readiness to change. Item 11 loaded on Factor 3 (Relapse) and Factor 5 (Dropout). This item explicitly assesses the likelihood of the client dropping out of psychotherapy treatment. However, as this item loaded highly on the relapse factor, and dropout may be predictive of relapse, we included this item on both factors.

Manipulation Check

A one-way Analysis of Variance (ANOVA) was utilized to check the effectiveness of the manipulation. There were no differences between the levels of Cause (psychological, biological addiction and ambiguous) on the following questions: "I agree that obesity is a biological addiction," F (2, 361) = .43, p = NS; "I agree that obesity has ambiguous origins," F (1, 361) = 2.34, p = NS; and "I agree that obesity is psychological," F (1, 361) = 1.35, p = NS, indicating that our manipulation was ineffective. [1]

Comparison of Groups

Effects on measurements of stigma

An Analysis of Variance (ANOVA) was conducted to determine the effects of condition and gender on stigmatization of obesity and on the target's perceived prognosis. In a 3x2x2 ANOVA we tested the effects of Cause, presence of BED, and gender on the three measurements of Stigma (See Tables 2-4). There were no significant main effects of Cause. The analysis revealed multiple significant main effects of presence of BED. There was a significant main effect on Attractiveness, F (1, 362) = 3.73, p = .05, such that participants in the non binge eating condition rated obese persons as more attractive (M = 4.86, SD =1.16) than participants in the binge eating condition (M = 5.06, SD = 1.05). There was a significant main effect on Distance, F (1, 362) = 4.34, $p <$ .05, such that participants in the non binge eating condition were more comfortable being close to obese persons (M=2.38, SD= 1.05) than participants in the binge eating condition

[1] The manipulation check revealed no significant differences between groups. We therefore cannot interpret the results for Cause. However, we include this in our analysis as it was in the original design of the study and we report the findings in the comparison of groups section.

(*M*= 2.57, *SD*=1.02). There was a significant main effect on Blame, F (1, 362) = 6.63, p < .05, with participants in the binge eating condition more likely to blame obese persons for their weight (*M* = 2.79, *SD* = 0.68) than participants in the non binge eating condition (*M* = 2.62, *SD* = 0.66)[2] (See Figure 1).

Gender had several main effects on stigma, which included: Social Disparagement, F (1, 362) = 17.82, p < .05, Blame, F (1, 362) = 4.26, p < .05, and Physical and Romantic Unattractiveness, F (1, 362) = 19.51, p < .05. In each case, men held more stigmatizing beliefs (*M* = 1.95, *SD*=0.53; *M* = 2.70 *SD*= 0.71; *M* =2.95, *SD*= 0.61, respectively) than women (*M* = 1.73, *SD*=0.48; *M* = 2.64, *SD* = 0.65; *M* = 2.65, *SD* = 0.64, respectively). Men and women differed significantly on ratings of Attractiveness F (1, 362) = 13.83, p < .05, with men rating obese persons as less attractive (*M*=5.22, *SD* = 1.11) than did women (*M*= 4.78, *SD* = 1.08). There also were significant gender effects on Distance, F (1, 362) = 4.81, p < .05, with men preferring more distance (*M*= 2.62, *SD* = 1.03) than women (*M*= 2.37, *SD* = 1.04), and on Negative Judgment, F (1, 362) = 7.56, p < .05, with men displaying more negative judgments (*M*= 2.65, *SD* = 1.10) than women (*M* = 2.34, *SD* = 1.02). We found main effects of gender on the Fear of Fat and Will Power subscales. Women gave higher ratings on Fear of Fat, F (1, 362) = 58.55, p < .05, (*M*=6.28, *SD*=2.19, versus men (*M*=4.39, *SD* = 2.52). For Willpower, F (1, 362) = 5.48, p

[2] Please see Figure 1 for a pictorial explanation. Here, it appears as though the main differences are between the psychological and biological condition versus the ambiguous condition for obesity with BED. However, the relevant interaction was not significant and not interpretable as we are unable to understand any effects of Cause because the manipulation was unsuccessful.

< .05, men reported that obese persons have less willower (M = 5.94, SD =1.70) than did women reported (*M* =5.54, *SD* =1.58).

There was a significant two-way interaction between the presence of BED and gender on Equal Rights *F* (2, 362) = 5.89, *p* < .05. Men in the binge eating condition perceived less need for equal rights (*M* =3.53, *SD* = 1.58) than men in the non binge eating condition (*M* = 2.83, *SD* = 1.71), whereas women in these two conditions did not differ (See Figure 1).

The effects on the patient's perceived prognosis

A 3x2x2 ANOVA was conducted on the PAQ (see Tables 5-7). There was significant main effect of Cause on the target's prognosis. Presence of BED had a main effect on Severity of Illness, *F* (1, 362) = 153.35, *p* < .05, with participants in the binge eating condition viewing the target's obesity as more severe (*M* = 55.09, *SD* = 14.06) than did participants in the non binge eating condition (*M* = 35.74, *SD* = 15.40). Presence of BED had a main effect on Treatment Appropriate, *F* (1, 362) = 15.53, *p* < .05, Drop Out, *F* (1, 362) = 4.23, *p* < .05, and Curability, *F* (1, 362) = 12.33, *p* < .05. Participants in the binge eating condition recommended treatment more, indicated a higher drop-out rate, and saw obesity as less curable, (*M* = 64.71, *SD* = 14.29; *M* = 50.73, *SD* = 14.69 ; *M* = 54.86 , *SD* = 16.47, respectively), compared to participants in non binge eating condition, (*M* = 57.83, *SD* = 17.63; *M* = 47.15, *SD* = 15.60; *M* = 60.81 , *SD* = 14.8 , respectively).

Gender had a main effect on Curability, *F* (1, 362) = 13.86, *p* < .05, such that men reported that obesity was less curable, (*M* = 54. 26, *SD* = 16.99) than woman (M = 60.39, *SD* = 14.63). There was an interaction effect between gender and cause on Severity of

Illness, F (1, 362) = 3.67, $p < .05$ (See Figure 3). In the biological cause condition, men rated the target's obesity as less severe (M= 40.10, SD = 18.43) than women (M = 48.45, SD = 17.31). In the ambiguous cause condition, men again rated obesity as less severe (M = 43.94, SD = 7.60) than women (M = 46. 90, SD = 12.25). In the psychological cause condition this relationship reversed and men rated the target's obesity as more severe (M= 46. 62, SD = 14.26) than the women (M = 44.15, SD = 17.32).[3]

Secondary findings

An ANOVA was conducted to determine group effects on specific treatment matching. Presence of BED had a main effect on recommending psychotherapy, F (1, 361) = 15.74, $p < .05$ and combined treatment, F (1, 361) = 11.10, $p < .05$. Participants in the binge eating condition recommended psychotherapy (M = 76.41, SD= 17.90) and combined treatment of psychotherapy and drugs (M = 67.12, SD = 23.16) more than participants in the non binge eating condition (M = 67.41 SD= 23.11; M= 57.99, SD = 25.91, respectively). Women recommended psychotherapy treatment (M = 73.68 SD = 19.83) more than men (M = 69.22, SD = 22.76), yielding a main effect for gender, F (1, 361) = 4.47, $p < .05$.

An ANOVA was conducted to determine the effects of race. Race had a main effect on Attractiveness, F (1, 368) = 2.95, p = .05, where white participants (n = 187) reported that obese persons were less attractive (M = 5.16, SD = 1.05) compared to the report of Hispanic/Latinos (n = 28; M = 4.53, SD = 1.09). ANOVA was utilized to test the effects of previous treatment for obesity, an eating disorder or a general psychological

[3] As we are not able to interpret the Cause condition, this interaction is likely a chance finding and better characterized as an effect of gender, which resulted had a main effect on Severity of Illness, F (1, 362) = 4.09, $p < .05$.

disorder. Previous treatment of an eating disorder (n= 15), which included Bulimia Nervosa (BN), Anorexia Nervosa (AN) and BED, had a significant main effect on Fear of Fat, F (1, 371) = 14.43, $p < .05$, such that participants who have been treated for an eating disorder reported a significantly greater fear of gaining weight (M = 7.87, SD = 1.11) compared to participants who have not been treated for an eating disorder (n = 358; M = 5.40, SD = 2.51). Previous treatment of a psychological disorder, unspecified (n = 35), also had a significant main effect on Fear of Fat, F (1, 371) = 8.01, $p < .05$, such that participants previously treated for a psychological disorder reported a greater fear of gaining weight (M = 6.23 , SD = 2.44) than participants who have not been treated for a psychological disorder (n = 338; M = 5.38, SD = 2.49). Previous treatment of obesity had a main effect on illness severity, F (1, 362) = 4.19, $p < .05$, such that participants who have been treated for obesity rated it as less severe (M = 27.92, SD = 10.66) than participants who have not been treated for obesity (M = 45.88, SD = 17.49).

Discussion

The manipulation check revealed no significant differences between our groups, either indicating that our manipulation was unsuccessful or that our manipulation check was inadequate. Based on this finding, we were not able to interpret the lack of effects of Cause on stigma or the target's prognosis. The method of manipulation used in this study may not have been sufficiently robust. Participants only were required to read a brief account of the causes of obesity or obesity with BED. Lewis and colleagues (1997) conducted a similar experiment. They wrote that "brief exposure to information…may be insufficient to alter preconvictions and prejudices…" (pp. 304-305). Stigma toward obesity is strong; and it is one of the only prejudices that remains socially acceptable (see

Latner, in press; Puhl & Brownell, 2003). A stronger manipulation or a better check of the manipulation may be necessary. Among the studies that did find differences due to causal beliefs, some of the more robust manipulations included reading entire articles about the causes of obesity (Teachman et al, 2003) and watching videos (Lam & Salkovskis, 2007).

There were multiple main effects of the presence of BED on stigma. This effect was pervasive across the measures, indicating a consistent structure where presence of BED resulted in increased stigma. Participants in the obesity with BED condition found obese persons less attractive, blamed them more for their weight, and they desired more social distance from obese persons. The more justification participants had for their stigma (binge eating), the more stigma they revealed. The findings support Crandall's (1994) theoretical model of attribution of controllability and blame as a potential underlying cause of stigma toward obese individuals. It also supports his more recent model (Crandall & Eshleman, 2003), which states that if there is justification for stigmatization, it is more likely to be displayed. Participants reacted with less stigmatizing beliefs toward obesity alone than if BED was present. Participants blamed obese persons less, found them more attractive and did not report needing as much social distance if BED was absent. In this case, a cue to their blameworthiness for being obese (binge eating) was absent and therefore less stigma was displayed. It would seem that obesity, without binge eating disorder (BED) is more socially acceptable than obesity alone. Puhl and Brownell (2006) found that persons who were obese with BED did not feel more stigmatized than obese individuals without BED. It may be that persons with

obesity and BED do not feel the differential effects of this stigma, but that individuals do have discrepant attitudes toward those who are obese with BED.

There were also main effects of gender on stigma, where men demonstrated greater negative attitudes and stigma toward obesity than women. This is seen consistently in the literature (e.g., Crandall, 1994; Lewis et al., 1997). There was also one main effect for race, where self described white participants evaluated obese persons as less attractive. This result has been demonstrated before among college students (Latner, Stunkard and Wilson, 2005).

The presence of BED had multiple main effects on the prognosis for the target. Participants rated obesity with BED as harder to cure, more severe, and that the target was more likely to drop out of treatment. The findings suggest a better prognosis may be dependent on whether a person has BED and is in control of his/her diet. This is interesting, as there do exist effective treatments for BED; although often these may result in negligible weight loss, the binge eating is often improved (e.g., Wilfley et al., 2002). In a sense, the BED aspect of obesity may more curable, contrary to what participants in this study reported. It will be interesting to study whether these effects are shown for mental health practitioners and obese individuals. It is well documented that physicians and mental health practitioners have stigmatizing beliefs toward obese individuals (e.g. Schwartz et al., 2003; Davis-Coelho, Waltz, & Davis-Coelho, 2000). The research on effects of this stigma on patient care has yielded mixed results. One study found that younger psychologists expected lower effort from fat clients and gave a poorer prognosis and female psychologists predicted poorer prognosis for fat clients (Davis-Coelho et al., 2000). Another study that assessed stigma and health practitioners

found no differences between practitioner's reports between an obese client and a non-obese client on prognosis, expected duration of treatment or motivation (Agell & Rothblum, 1991). Among mental health practitioners, stigma may be differentiated between patients with BED and without BED.

In the previously discussed study by Ogden and Flanagan (2008), the authors argued that lack of successful treatment for obesity may arise because practitioners hold a "coherent" but non medical model of obesity's causes and treatments and that there is a disconnection between patients' and practitioners' beliefs about appropriate treatments. This disconnection may result in less treatment adherence and motivation, but would not account for total treatment failure. It is possible that treatment may be influenced by the clinical characteristics displayed by obese individuals (binge eating versus not), and the stigmas and the prognostic beliefs these characteristics carry. We investigated the effects of the conditions on specific treatment matching to cognitive behavioral therapy (CBT), drug treatment, or a combination of CBT and drug treatment. Women recommended psychotherapy more than men. There also were main effects for binge eating versus non binge eating. Participants in the binge eating group recommended psychotherapy or a combination of psychotherapy and drug treatment more than drug treatment alone. Although Ogden and Flannigan (2008) found no consistencies for lay persons between their causal belief and treatment matching, there is a preference of undergraduates in this study to match binge eating appropriately with psychotherapy or a combined treatment.

The results of this study provide further support for existing theoretical models of stigma in obesity that posit that, if people attribute controllability and blame to a person's weight, more stigmatization will occur. It also supports the just world hypothesis

according to which people experience appropriate outcomes and therefore the obese are overweight because they overeat (see Puhl & Brownell, 2003). However, the current study explored a previously unexamined factor, namely whether obesity with BED is more stigmatizing than obesity alone. The results indicate that signaling whether someone engages in binge eating or not affects the stigmatizing and prognostic beliefs of others. This is an interesting finding, as whether someone has an "addiction" to food or they are in a never ending cycle of binge eating and dieting, people look down upon it. Again, we are unable to interpret the lack of finding for cause in this study, but binge eating certainly appears important in the stigmatization of obesity. It may be the binge eating behavior that begets stigma. Participants in the obesity with BED condition not only demonstrated greater stigma, but also endorsed poorer prognoses for obese persons. Obese persons with BED may be at greater risk for stigmatization and receiving compromised treatment due to these biases.

There were limitations to this study. As previously noted, the intervention for the causal manipulations was brief and ineffective; this may be due to the brief scenarios presented or to participants' previously held beliefs. However, the BED condition was relayed in the same medium and was effective. This study was limited to an undergraduate population. Also, we did not check for social desirability. Many of our items were theoretically robust to this construct, but future research should include this check. There may also have been demand characteristics, such that participants understood the purpose of this study and responded accordingly.

Future research should investigate the effects of labeling obesity a biological addiction. Considering that this is such a pervasive public and medical view, the effects

on stigma and treatment should be further investigated. This study attempted to do so, but our causal conditions were not strong enough to elicit differences. Research should continue to investigate effects of the presence of BED on mental health practitioners' attitudes and practices. It will be just as important to investigate these effects on people who are obese, with and without BED. Importantly research must continue to investigate ways to reduce and counteract this stigma. Education and exposure to binge eating may be a new component for existing methods for stigma reduction.

Table 1

Factor Loadings for Individual Items on the PAQ

	Factors					
	Illness Severity	**Treatment Appropriate**	**Relapse**	**Availability to Change**	**Dropout**	**Curability**
Item						
1	**.76**	.01	-.10	.13	.22	.11
2	**.76**	.01	-.14	.14	.28	-.02
3	**.72**	,13	.19	-.06	-.15	-.03
4	**.75**	.20	.07	.10	-.03	-.08
5	**.76**	.10	.07	-.07	-.04	-.10
6	**.62**	.02	.10	-.04	.12	.16
7	.03	-.01	**.48**	.13	.1	-.03
8	.10	.12	**.84**	.02	.10	-.19
9	.03	-.22	**.78**	.01	.19	-.11
10	.16	-.08	.17	-.24	**.60**	.03
11	.12	.11	**.44**	-.13	**.68**	.07
12	.02	-.10	**.41**	-.21	**.68**	.00
13	.06	.04	-.12	.17	**.66**	-.22
14	-.01	-.16	-.02	**.66**	-.14	.09
15	.07	.03	-.10	**.69**	-.02	.15
16	.07	.39	.21	**.64**	.15	.10
17	.10	**.44**	-.03	**.64**	-.04	.12
18	.03	.07	-.04	.13	-.00	**.76**
19	.01	.06	-.15	.18	-.06	**.77**
20	-.15	-.05	-.07	-.00	-.07	**.67**
21	.07	**.86**	-.12	.13	.01	-.03
22	.26	**.47**	-.24	.15	-.02	.19
23	.24	**.83**	-.12	.13	.01	-.03
Variance Explained	**14.89%**	**24.38%**	**33.79%**	**42.68%**	**51.37%**	**59.46%**

Table 2
Means (and Standard Deviations) of Stigma Measures for Each Cause Condition

	Biological (n=124)	Psychological (n=121)	Ambiguous (n=129)
UMFAT			
Attractiveness	4.91 (1.11)	5.10 (1.09)	4.88 (1.13)
Social Distance	2.49 (1.04)	2.49 (1.07)	2.44 (1.01)
Equal Rights	2.77 (1.53)	2.77 (1.53)	2.82 (1.59)
Negative Judgment	2.55 (1.12)	2.39 (1.08)	2.47 (.99)
AFAT			
Social Disparagemei	1.85 (.54)	1.80 (.48)	1.81 (.48)
Romantic/Physical Unattractiveness	2.77 (.66)	2.81 (.68)	2.75 (.59)
Weight Control/Blai	2.73 (.74)	2.70 (.70)	2.69 (.60)
AFA			
Dislike	2.80 (1.26)	2.72 (1.33)	2.74 (1.56)
Willpower	5.72 (1.70)	5.79 (1.59)	5.62 (1.64)
Fear of Fat	5.78 (2.36)	5.21 (2.65)	5.51 (2.52)

Table 3
Means (and Standard Deviations) of Stigma Measures for Presence of BED

	Binge Eating (n=185)	Non Binge Eating (n = 189)
UMFAT		
Attractiveness	5.06 (1.05)	4.86 (1.16)
Social Distance	2.57 (1.02)	2.38 (1.05)
Equal Rights	2.91 (1.52)	2.66 (1.56)
Negative Judgment	2.58 (1.06)	2.36 (1.06)
AFAT		
Social Disparagement	1.84 (.49)	1.80 (.52)
Romantic/Physical Unattractiveness	2.80 (.63)	2.75 (.65)
Weight Control/Blame	2.79 (.68)	2.62 (.66)
AFA		
Dislike	2.82 (1.30)	2.68 (1.41)
Willpower	5.85 (1.59)	5.56 (1.68)
Fear of Fat	5.53 (2.46)	5.47 (2.57)

Table 4
Means (and Standard Deviations) on Measures of Stigma for Gender

	Men (n=154)	Women (n=220)
UMFAT		
Attractiveness	5.22 (1.11)	4.78 (1.08)
Social Distance	2.62 (1.03)	2.37 (1.04)
Equal Rights	3.18 (1.68)	2.51 (1.39)
Negative Judgment	2.65 (1.10)	2.34 (1.02)
AFAT		
Social Disparagement	1.95 (.53)	1.7 (.46)
Romantic/Physical Unattractiveness	2.95 (.61)	2.65 (.64)
Weight Control/Blame	2.80 (.71)	2.64 (.65)
AFA		
Dislike	2.88 (1.48)	2.66 (1.26)
Willpower	5.94 (1.70)	5.55 (1.58)
Fear of Fat	4.39 (2.52)	6.28 (2.19)

Table 5
Means (and Standard Deviations) on Measures of Prognosis for Each Cause Condition

	Biological (n = 124)	**Psychological (n= 121)**	**Ambiguous (n =129)**
Factors			
Illness Severity	45.01 (18.18)	45.19 (16.09)	45.71 (18.59)
Treatment Appropriate	59.88 (16.81)	61.65 (16.90)	62.13 (15.57)
Relapse	47.18 (15.99)	46.48 (14.36)	47.38 (13.52)
Availability to Change	58.18 (12.78)	58.18 (13.61)	56.50 (12.58)
Dropout	48.31 (16.64)	48.22 (14.77)	50.17 (14.29)
Curable	56.67 (15.08)	58.15 (17.38)	58.76 (15.28)
	Biological (n = 123)	**Psychological (n= 121)**	**Ambiguous (n =129)**
Individual Items			
Psychotherapy Treatment	68.78 (21.86)	73.57 (21.90)	73.18 (19.56)
Drug Treatment	41.79 (22.03)	42.64 (24.38)	44.34 (22.87)
Combined Treatment	60.89 (24.70)	62.48 (26.59)	64.03 (23.77)

Table 6

Means (and Standard Deviations) on Measures of Prognosis for presence of BED

	Binge Eating (n = 185)	**Non Binge Eating (n = 189)**
Factors		
Illness Severity	55.09 (14.06)	35.74 (15.40)
Treatment Appropriate	64.71 (14.29)	57.83 (17.63)
Relapse	47.63 (14.47)	46.43 (14.77)
Availability to Change	57.04 (12.47)	58.13 (12.47)
Dropout	50.73 (14.69)	47.15 (15.60)
Curable	54.87 (16.47)	60. 81 (14.80)
	Binge Eating (n = 184)	**Non Binge Eating (n = 189)**
Individual Items		
Psychotherapy Treatment	76.41 (17.90)	67.41 (23.11)
Drug Treatment	45.38 (22.81)	40.58 (23.14)
Combined Treatment	67.12 (23.16)	57.99 (25.91)

Table 7

Means (and Standard Deviations) on Measures of Prognosis for Gender

	Male (n= 154)	**Female (n = 220)**
Factors		
Illness Severity	43.56 (16.97)	46.54 (18.02)
Treatment Appropriate	60.98 (16.99)	6.41 (16.41)
Relapse	48.51 (14.78)	45. 98 (14.44)
Availability to Change	57.24 (12.65)	57.86 (12.23)
Dropout	50.19 (15.07)	48.03 (15.33)
Curable	54.26 (16.99)	60. 39 (15.91)
	Male (n= 154)	**Female (n = 220)**
Individual Items		
Psychotherapy Treatment	69.22 (22.76)	73. 68 (19.83)
Drug Treatment	43.79 (24.03)	42.36 (22.41)
Combined Treatment	63.40 (25.00)	61.86 (25.00)

Figure 1

Blame as a function of presence of BED

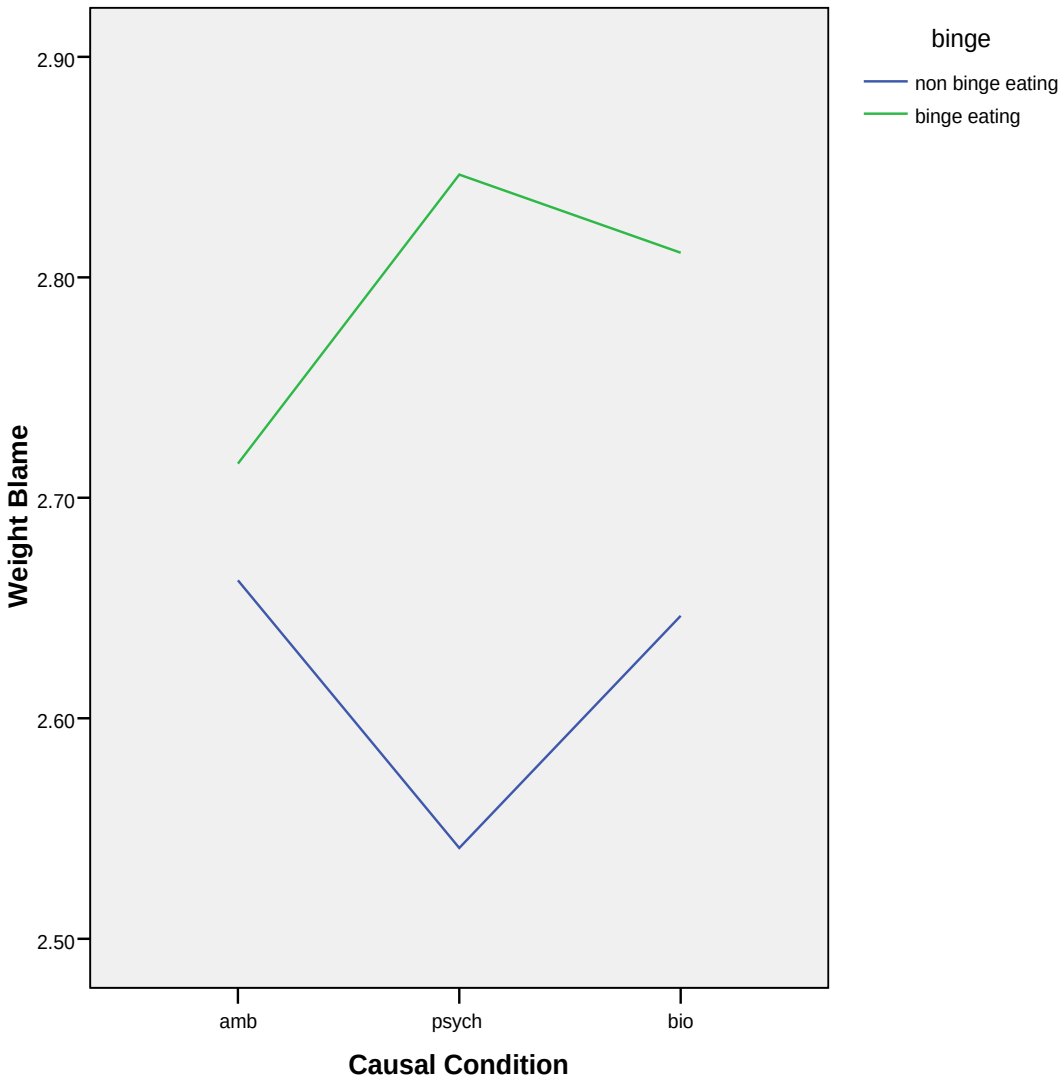


Figure 2

Equal rights of obese individuals as a function of gender and presence of BED

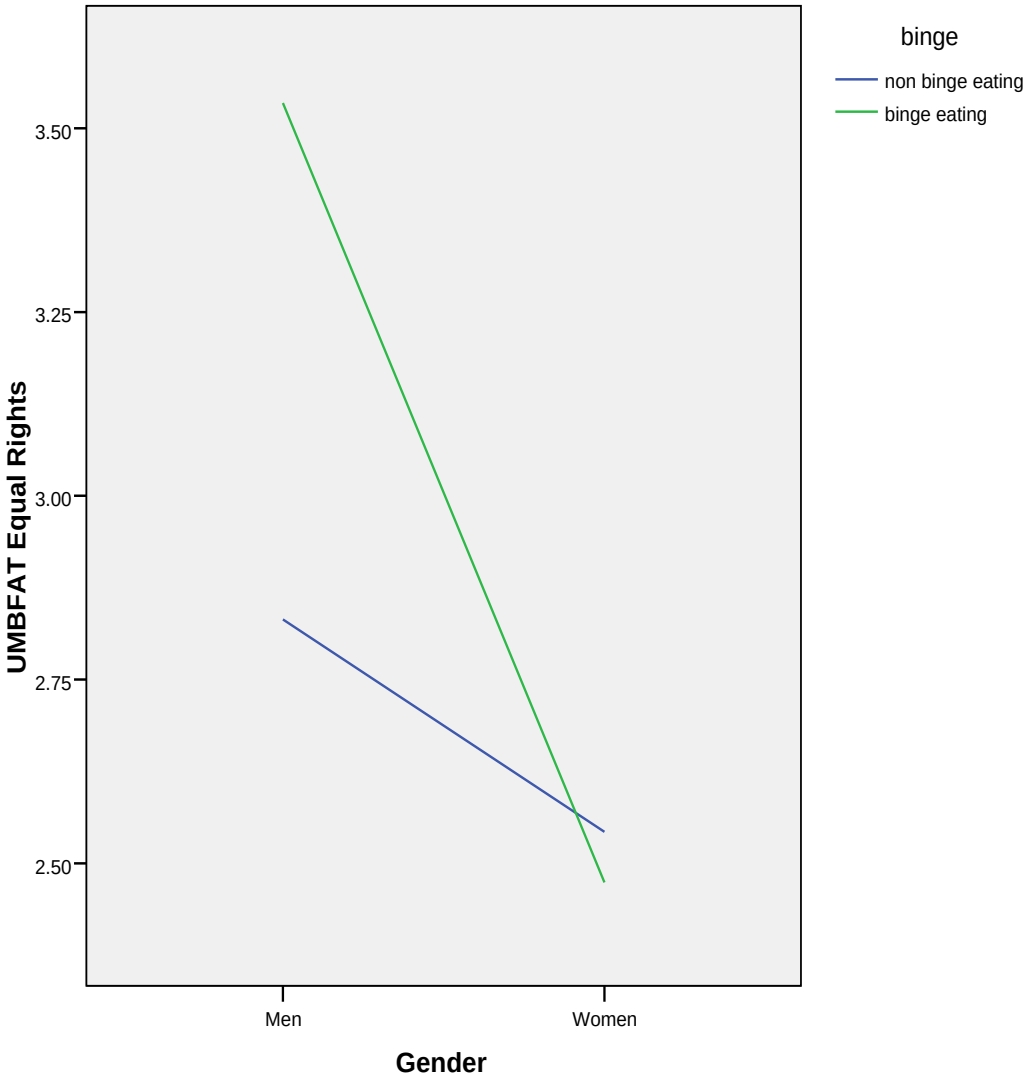
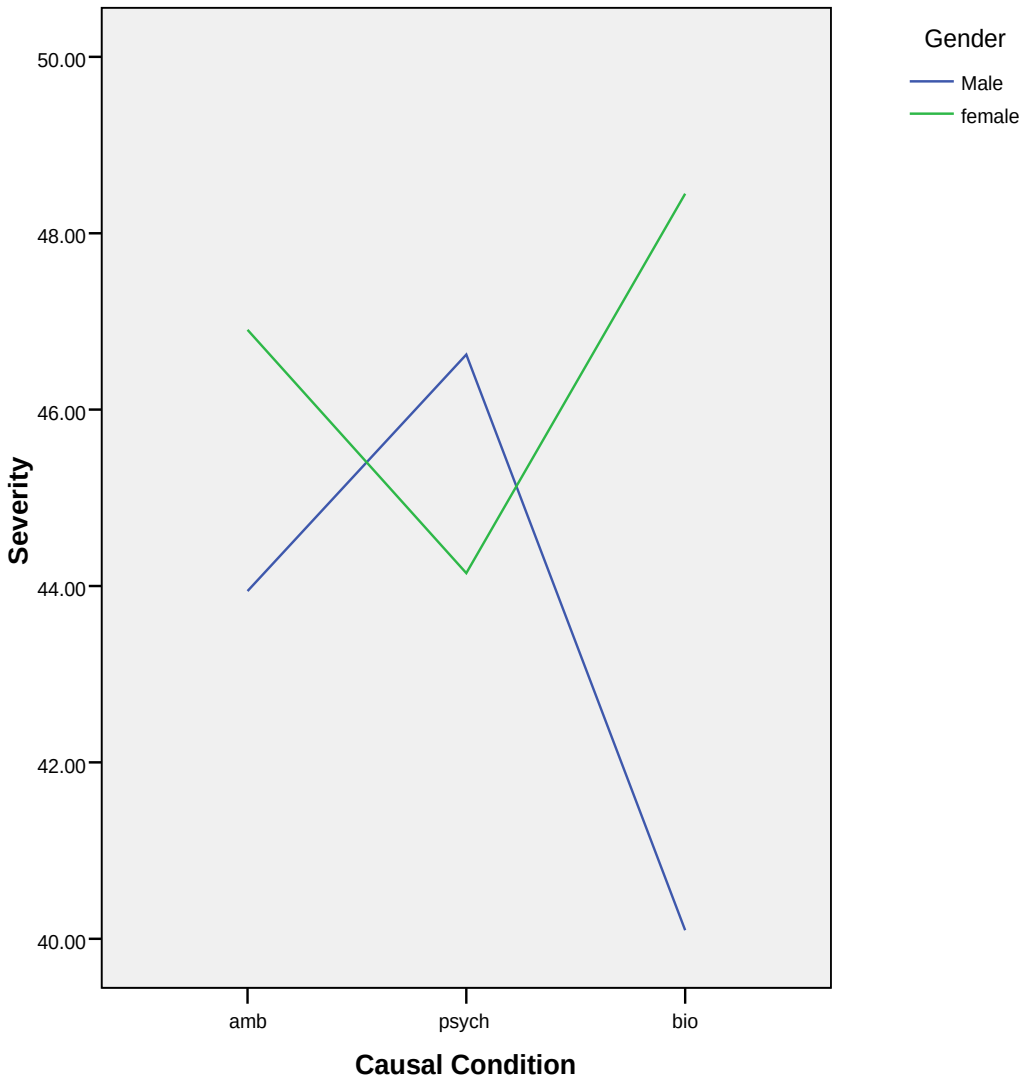


Figure 3

Illness severity as a function of gender and cause



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