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Can Raising Awareness about the Psychological Causes of Obesity  
Reduce Obesity Stigma?

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### **Abstract**

Obesity stigma largely remains a socially acceptable bias with harmful outcomes for its victims. While many accounts have been put forward to explain the bias, the role of obesity aetiology beliefs has received little scrutiny. The research examined the effect that beliefs about the psychological aetiology of obesity have on the expression of obesity stigma, and the mechanisms underpinning this effect. Participants (N = 463) were asked to evaluate a target person with obesity after reading one of three possible aetiologies: psychological, genetic or behavioural. The presentation of a psychological aetiology of obesity elicited less prejudice compared to behavioural causes but greater prejudice compared to genetic causes; observed differences were found to be a function of the agency ascribed to the target's obesity, and empathy expressed for the target. The findings highlight the impact that communicating obesity in terms of psychological causes can have for the expression of obesity stigma.

**Keywords:** obesity stigma; obesity aetiology; psychological causes; agency; empathy

Can Raising Awareness about the Psychological Causes of Obesity Reduce Obesity Stigma? Anti-fat bias, or obesity stigma, refers to the unfavourable judgment of people who are obese. As with other socially devalued groups, obesity stigma underpins the prejudicial and discriminatory experiences reported by obese people and reflects a general lack of concern, or empathy, for their welfare (Puhl & Heuer, 2009). Compared to other stigmatised groups, prejudice and discrimination towards people with obesity still largely remains socially acceptable (Latner, O'Brien, Durso, Brinkman & MacDonald, 2008). Obesity stigma is pervasive and evidenced across institutional contexts, including education (e.g., Lumeng, Forrest, Appugliese, Kaciroti, Corwyn, Bradley, 2010), employment (e.g., Baum & Ford, 2004), and healthcare (e.g., Teachman & Brownell, 2001). Obesity stigma, both public- and self-stigma, is also associated with various negative health outcomes amongst those that it affects, including depression, low self-esteem, maladaptive eating, exercise avoidance and disordered body image (e.g., Carr & Friedman, 2005; Farrow & Tarrant, 2009; Schvey, Puhl & Brownell, 2011).

Given its negative effects on victims, it is important to understand the conditions that make the expression of obesity stigma acceptable. Understanding this is likely to be critical to ongoing efforts to change people's attitudes towards people with obesity, particularly given the importance that the framing of the nature, causes, and consequences of obesity plays in public campaigns (see Barry, Gollust, McGinty & Niederdeppe, 2014). As a step towards this goal, the current research examined the impact of people's beliefs about the causes of obesity on their expression of obesity stigma. This focus builds on current understandings of the causes of obesity that emphasises a number of different determinants of (excessive) body weight, including some over which individuals exert little control and responsibility (e.g., genetics: Bouchard & Perusse, 1993).

Explanations for obesity stigma have centred on attribution theory (e.g., Crandall, D'Anello, Sakalli, Lazarus, Wiczorkowska Nejtardt & Feather, 2001; Weiner, Perry & Magnusson, 1983), and the idea that prejudice towards people with obesity evolves out of the belief that weight is personally controllable (Puhl & Brownell, 2003). This perspective attributes weight gain to individual, volitional, failures to eat a balanced diet and exercise regularly. However, rather than motivating people to adopt a healthy lifestyle, attributing excess weight to personal failure can undermine the motivation to lose weight (e.g., Brownell et al., 2010; Burnette, 2010; Pearl & Leibowitz, 2014), and is also antecedent to stigma (Crandall, 1994; Puhl & Heur, 2009; Teachman, Gapinski, Brownell, Rawlins & Jeyaram, 2003). Early research by Crandall (1994) showed that prejudice and discrimination towards people with obesity was higher when people focused on a behavioural compared to a genetic aetiology of obesity, and subsequent research has uncovered similar findings amongst both adults (e.g., Jeong, 2007; Persky & Eccleston, 2011; Teachman, et al., 2003) and children (e.g., Bell & Morgan, 2000). This suggests that the way in which the underpinning causes of obesity are communicated may play an important part in the expression of obesity stigma and thereby in interventions intended to mitigate obesity stigma. Although not directly tested empirically, perceiving a genetic aetiology for obesity may elicit less prejudice and discrimination by shifting attention away from personal responsibility-focused explanations for weight (Crandall, 1994; Teachman, et al., 2003).

The current research extended the above focus to consider the stigma consequences of holding beliefs centred on the psychological underpinnings of obesity. Weight gain is known to be brought about both by behavioural factors (e.g., overeating), and genetic factors, including both single gene mutations (e.g., leptin (LEP) and melanocortin-4 receptor (MC4R)) and common gene variations (e.g., FTO gene; for an overview see Bray & Bouchard, 2014). Less readily acknowledged are psychological factors, although these

clearly also contribute to weight gain. Binge Eating Disorder (BED), which has a prevalence of 30% amongst obese people who seek weight management treatment (de Zwaan, 2001), is understood to have a largely psychological aetiology (Fairburn & Wilson, 1995) and is classified as a psychological disorder in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013). BED is treated most effectively through psychological interventions (Vocks et al., 2010). Similarly, depression is closely linked to obesity (Luppino et al., 2010) and the maladaptive eating behaviours that contribute to excess weight gain (e.g., Goldschmidt et al., 2014; Skinner, Haines, Austin & Field, 2012).

Whether beliefs about the psychological underpinnings of weight status structure the expression of obesity stigma is unknown. On one hand, it might be expected that a focus on the psychological aetiology of obesity will make obesity stigma expressions more likely because of the potential “double” stigma associated with being both obese *and* suffering from psychological ill-health. Conversely, focusing on the psychological aetiology of obesity may inhibit the expression of weight stigma by bringing to mind factors over which individuals have little control (as is the case for mental health stigma: see Corrigan, 2000). By testing these competing predictions, the current study aimed to provide a fuller understanding of how the three different aetiology beliefs—behavioural, genetic and psychological—affect the expression of obesity stigma. Our priority in assessing these three aetiologies was not upon professional beliefs, but rather lay understandings about the aetiology of obesity. Even though a wealth of scientific evidence has indicated that obesity is the result of a complex interaction of behavioural, genetic, and psychological factors (e.g., Llewellyn & Wardle, 2015), information about the causes of obesity provided by healthcare services, such as the NHS (2016) and BUPA (2016), tend to present determinants as distinct rather than interactive (for an exception see the ‘Obesity System Map’ published by Public Health

England (PHE, 2016)) - it is the impact of such oversimplified presentations of obesity on the expression of stigma which are the focus of this research.

We also explored two potential mechanisms (mediators) underpinning these effects: perceptions of personal control (or agency), and empathy. Peoples' psychological states are known to be shaped by external factors over which they have little control. For example, a range of environmental factors exert a significant impact upon psychological health outcomes (e.g., Clark, Myron, Stansfeld & Candy, 2007). Accordingly, knowledge that an individual's obesity has psychological underpinnings might trigger a focus on controllability beliefs that inhibit stigma expression. That is, the individual with obesity may be perceived as lacking personal agency, or control, over their condition. This would be in line with findings from research that has examined how beliefs and/or information about the causes of obesity (and other health conditions) affects stigma expressions. Such research has consistently shown that an emphasis on uncontrollable external determinants is associated with more accepting and favourable attitudes toward people with obesity, as well as policies aimed at addressing the condition (e.g., Niederdeppe, Shapiro, Kim, Bartolo, Porticella, 2015; Pearl & Leibowitz, 2014; Young, Hinnant & Leshner, 2015).

Experiencing empathy for a member of a stigmatised group is associated with low levels of expressed prejudice towards that group (Batson et al., 1996, 1997; Campbell & Babrow, 2004; McKeever, 2015), including in the context of obesity (e.g., Kushner, Zeiss, Finglass & Yelen, 2014). Accordingly, perceiving obesity as having a psychological aetiology may yield an empathetic response because it increases understanding of the psychological underpinnings and challenges associated with obesity and its management. Based on the above, we predicted that high(er) perceived levels of agency and lower empathy would be associated with stronger expressions of obesity stigma.

## Method

### Sample and Design

Ethical approval for the research was provided by the Psychology Research Ethics Committee at the University of Exeter, United Kingdom (ref: 2012/553). Participants (N = 463; 304 females) were recruited using Crowdfunder (www.crowdfunder.com), a web-based crowdsourcing platform for the recruitment of participants in research studies (for an overview of the validity and reliability of data collected using crowdsourcing see Buhrmester, Kwang & Gosling, 2011; Mason & Suri, 2011). The survey was presented in Qualtrics (www.qualtrics.com), an online survey creation and data collection platform. All participants were recruited from the U.S.

Sample size was determined on the basis of two planned analyses. First, using G\*Power (Faul, Erdfelder, & Buchner, 2007), it was estimated that a sample size of 112 participants would be required to achieve 0.8 power for the planned MANOVA. Second, following suggestions by Fritz and MacKinnon (2007), it was estimated that a sample size of between 396 and 462 participants would be required to achieve 0.8 power in case the magnitudes of the relationships in the planned mediation models were small ( $\alpha = 0.14 - \beta = 0.14$ ). Responses from 500 participants were originally collected, but responses from 37 participants were excluded due to being incomplete (> 10%). No stopping rule was applied to the collection of data.

Participants were resident in 49 U.S. states and were aged between 18-57 years (mean age = 39 years, SD = 13 years). Most participants were college or university educated (59%); 13% were current students. Participants' average Body Mass Index (BMI), calculated on the basis of their self-reported height and weight, was 27 (SD = 7; range 16 - 38)<sup>1</sup>.

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<sup>1</sup> According to the WHO and the Centers for Disease Control and Prevention (CDC), a BMI between 25 and 29.9 classifies as pre-obesity (overweight), and a BMI larger than or equal to 30 classifies as obesity.

Participants were randomly assigned to one of three experimental conditions that varied according to the stated aetiology of a target individual's obesity (psychological, genetic, or behavioural aetiology). All participants were first shown a photograph of a man with obesity (the target), referred to as John, along with the following text:

*“This is a picture of John. He is 55 years old and works as a research scientist at a pharmaceutical company. John has a Body Mass Index (BMI) of 39. This means that he classifies as having obesity.”*

Participants in the “psychological aetiology” condition (n = 155) were told that his obesity was caused by a psychological eating disorder that had developed due to a range of traumatic events that had taken place in his life; participants in the “genetic aetiology” condition (n = 151) were told that John's obesity was caused by a genetic condition that meant that his metabolic rate was lower than that of a person who does not suffer from the condition; and participants in the “behavioural aetiology” condition (n = 157) were told that his obesity was caused by eating too much food high in fat and sugar, and not engaging in enough physical activity<sup>2</sup>.

The photograph depicting “John” in the vignette was of an actual patient awaiting bariatric surgery, and was provided by Commonwealth Surgical Associates (MA, United States) with the approval of the patient.

### **Outcome Measures**

Two measures assessed the expression of stigma towards the target. First, participants completed an adapted version of the short form of the *Fat Phobia Scale* (FPS; Bacon, Scheltema & Robinson, 2001). The scale consists of 14 traits and corresponding antonyms

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<sup>2</sup> The vignettes can be requested by contacting the authors.



intended to describe people with obesity, and participants were asked to indicate their beliefs (e.g., “*attractive*” vs. “*unattractive*”; “*active*” vs. “*inactive*”; “*secure*” vs. “*insecure*”). The scale is devised to assess attitudes towards people with obesity in general but was adapted to allow participants to indicate the extent to which they believed that the traits described the target. Participants indicated their responses on 7-point Likert scales: higher scores indicate greater fat phobia (or stigma expression). Reflecting previous research (Bacon et al., 2001;  $\alpha = .87-91$ ), the FPS was highly reliable in the current study ( $\alpha = .89$ ).

The second measure of stigma expression was a *Stereotype* checklist based upon the Stereotype Content Model (SCM; Fiske, Cuddy, Glick, & Xu, 2002). This included seven traits concerning the perceived warmth of the target (“*well-intentioned*”, “*sincere*”, “*trustworthy*”, “*friendly*”, “*good-natured*”, “*tolerant*” and “*warm*”), and seven traits pertaining to the perceived competence of the target (“*intelligent*”, “*skilful*”, “*efficient*”, “*capable*”, “*confident*”, “*self-disciplined*” and “*competent*”). Participants indicated the extent to which they believed that the traits described the target using 7-point Likert scales anchored by “*Not at all*” and “*Very much*”. Higher scores indicated lower levels of competence and warmth, i.e., more negative stereotypes (or stigma expression). Similar lists of traits have commonly been used in social psychological research and have high reliability (e.g., Yzerbyt, Provost & Corneille, 2005;  $\alpha = .87-91$ ;  $\alpha = .87-91$ ). The Cronbach’s alpha for the stereotypes was .95 in the current study.

### **Mediators**

*Agency Attributions* were measured using three items adapted from Crandall (1994) and Vartanian and Fardouly (2013): “*To what extent do you believe that John’s obesity is caused by factors that John can control or factors outside of John’s control?*”; “*How much control do you think John has over his obesity?*”; “*How responsible do you think John is for*

*his obesity?*”. Participants indicated their responses on 7-point Likert scales, with higher scores ascribing lower levels of agency to the target ( $\alpha = .81$ ).

*Empathy* for the target was assessed using six items derived from the Communication Emotional Response Scale (CERS; Batson, O’Quin, Fultz, Vanderplas, & Isen, 1983). Participants were asked to indicate the extent to which learning about the target’s condition evoked the following feelings: “*sympathy*”, “*soft-heartedness*”, “*warmth*”, “*compassion*”, “*tenderness*” and “*moved*”. Responses were recorded on 7-point Likert scales anchored by “*Not at all*” and “*Very much*”, with higher scores indicating more empathy for the target. The CERS has been shown to have high internal consistency (Batson et al., 1983;  $\alpha = .79 - .91$ ); Cronbach’s alpha in the current study was .95.

### **Covariates**

Two measures of *General Weight Bias* were included as covariates in the study in order to control for general (as opposed to target-specific) weight-bias between the participants in the three different conditions. The measures were the *Beliefs About Obese Persons Scale* (BAOPS; Allison, Basile & Yuker, 1991;  $\alpha = .69$ ) and the *Anti-Fat Attitudes Scale* (AFAS; Morrison & O’Connor, 1999;  $\alpha = .82$ ). No significant differences could be found in general weight-bias, as measured by the BAOPS and AFAS, between the three conditions (Omnibus MANOVA:  $F(4, 919) = 2.21, p = .107, \eta^2 = .01$ ; BAOPS:  $F(2, 461) = 2.05, p = .131, \eta^2 = .01$ ; AFAS:  $F(2, 461) = .39, p < .250, \eta^2 = .00$ ).

## **Results**

### **Descriptive Statistics and Correlations**

Table 1 reports descriptive statistics and correlations for all scales. Chi-square and ANOVA tests indicated no significant differences in the socio-demographic characteristics (i.e., age,

gender, and educational status) or BMI between the participants allocated to the respective conditions ( $p > .05$ ).

--- Insert Table 1 about here ---

Stigma expression (both fat phobia and negative stereotypes) was significantly correlated with ascriptions of lower agency and higher empathy. While fat phobia was positively correlated with general weight bias, as measured by the BAOPS and AFAS, the relationship between stereotypes and general weight bias was mixed: its correlation with BAOPS, which measures the controllability of obesity, was non-significant, but its correlation with AFAS, which measures negative attitudes towards people with obesity, was positive and significant. This pattern of results indicates that weight bias is more strongly correlated with fat phobia than with stereotypes. Similarly, it should be noted that agency attributions were more strongly correlated with general weight bias compared to (the lack of) empathy. Finally, Table 1 shows that higher participant BMI was significantly correlated with less fat phobia, weaker ascriptions of agency, lower general weight bias, and more empathy.

### **Main Analysis**

The main analysis was conducted in two steps. First, a MANCOVA was performed to examine whether stigma expression, and agency attribution, and empathy differed between the three aetiology conditions (behavioural versus genetic versus psychological). Second, indirect effects analyses using bootstrapping examined whether the effects of aetiology on the expression of stigma could be explained by agency attributions and empathy. Age, gender, education, general weight bias and BMI were entered as covariates in both steps. All analyses were conducted in SPSS v.22.

**MANCOVA.** The omnibus MANCOVA was significant for condition ( $Box M = 44.53, p < .01; F(8, 896) = 20.29, p < .001, n_p^2 = .15$ ). Between-subjects contrasts indicated significant differences for every scale as a function of aetiology (*Fat Phobia*:  $F(2, 458) = 36.39, p < .001, n_p^2 = .14$ ; *Stereotypes*:  $F(2, 458) = 13.89, p < .001, n_p^2 = .06$ ; *Agency*:  $F(2, 458) = 74.51, p < .001, n_p^2 = .25$ ; *Empathy*:  $F(2, 458) = 25.78, p < .001, n_p^2 = .10$ ).

Pairwise comparisons (Bonferroni) indicated that participants in the psychological aetiology condition expressed greater stigma, stronger ascriptions of agency and less empathy towards the target compared to participants allocated to the genetic aetiology condition. The same pattern of results was found for the comparison between the genetic and behavioural aetiology conditions, with participants allocated to the behavioural aetiology condition stigmatising the target more. The comparison between the psychological and behavioural aetiology conditions indicated that participants in the psychological aetiology condition ascribed the target less agency for his obesity and reported more empathy for him. Figure 1 presents the estimated marginal means and standard errors from the MANCOVA; Table 2 displays the condition mean differences. General weight-bias, as measured by the BAOPS ( $F(4, 447) = 21.67, p < .001, n_p^2 = .16$ ) and AFAS ( $F(4, 447) = 14.99, p < .001, n_p^2 = .12$ ), but not age ( $F(4, 447) = 0.53, p = .99, n_p^2 = .00$ ), gender ( $F(4, 447) = 1.99, p = .10, n_p^2 = .02$ ), education ( $F(4, 447) = .94, p = .44, n_p^2 = .01$ ), and BMI ( $F(4, 447) = 1.03, p = .39, n_p^2 = .01$ ), were found to be significant covariates in the model.

--- Insert Figure 1 and Table 2 about here ---

**Indirect effects analyses.** These analyses were performed using MEDIATE (Hayes & Preacher, 2014). Only one condition can serve as a reference condition in MEDIATE (i.e., baseline comparison condition against which other conditions are compared), and so two sets

of analyses were performed. The psychological aetiology condition was entered as the reference condition in the first set, enabling comparison between this and the conditions in which the target's obesity was attributed to a genetic and behavioural aetiology. The behavioural aetiology condition was then entered as the reference condition in the second, enabling a comparison between the genetic and behavioural aetiology conditions. Within each analysis set, four models were specified and tested: two models included target-specific fat phobia as the dependent variable and agency attributions (Model 1) and empathy (Model 2) as mediating variables, and two models included stereotypes as the dependent variable and agency attributions (Model 3) and empathy (Model 4) as mediating variables. Age, gender, education, general weight bias and BMI were entered as covariates. All models were tested using 5000 bootstrap resamples and 99% confidence intervals. Table 3 summarises the results.

--- Insert Table 3 about here ---

The results in part mirror the pattern of results from the MANCOVA, showing that participants assigned to the psychological and behavioural aetiology conditions expressed greater stigma towards the target than those assigned to the genetic aetiology condition. Likewise, the difference in the expression of stigma towards the target between the psychological and behavioural aetiology conditions was not statistically significant.

Differences in agency attributions and empathy between the three conditions accounted for differences in the expression of stigma in all models; that is, the indirect effects were significant in all models, which can be inferred from the finding that zero did not fall between the upper and lower levels of the 99% confidence intervals for the indirect effects in any of the models (see Table 3). In other words, compared to participants who learned that

the target's obesity had a psychological and behavioural aetiology, participants presented with a genetic aetiology for the target's obesity formed a more favourable impression of him to the extent that they empathised with him more and ascribed him less agency. The same pattern of effects and associations was found for the comparison of the psychological with the behavioural aetiology condition. Participants ascribed the target less agency and expressed greater empathy towards him when his obesity was attributed to a psychological compared behavioural aetiology, and these differences were in turn associated with significantly weaker expressions of stigma towards the target.

Finally, with the exception of one model (genetic vs behavioural aetiology), the suppression of the direct effects indicated that empathy accounted for more variance in the expression of stigma than agency attributions. The models also explained greater variance in target-specific fat phobia attitudes compared to stereotypes as indicated by the adjusted R-squared values in the respective models.

### **Discussion**

The findings from this study indicate that aetiology beliefs play an important part in the expression of obesity stigma. When participants learned that a target individual's obesity had a psychological cause, they stigmatised that individual more (reported more fat bias and negative stereotypes) compared to when they learned that his obesity had a genetic aetiology. Similarly, participants expressed greater stigma towards the individual when his obesity was attributed to a behavioural compared to a genetic aetiology. Indirect effects analyses revealed that participants presented with a psychological compared to a behavioural aetiology for the target individual's obesity stigmatised the target less to the extent that they also ascribed him less agency and expressed greater empathy towards him.

We initially anticipated that highlighting the psychological causes of an individual's obesity (e.g., trauma and depression) might encourage a more marked expression of stigma relative to other aetiologies through a "double stigma" effect, highlighting both the health condition and its psychological causes. However, presentation of a psychological aetiology was actually associated with weaker perceptions of target agency and greater empathy, which in turn were associated with the expression of less stigma towards the target compared to being told that his obesity was attributable to behavioural factors (e.g., dietary and exercise behaviour). This suggests that, rather than leading to "double stigma", emphasising the psychological underpinnings of obesity can have a prophylactic effect on the expression of obesity stigma.

The study also extends existing understanding of obesity stigma by demonstrating the mechanisms by which aetiology beliefs affect its expression. The ascription of agency to the target for his condition, and reduced empathy for him, explained the stigmatisation of the target. This mediation pattern compares with previous literature into mental health and obesity and similarly indicates that the attribution of control and lack of empathy are each antecedents of stigma expression (e.g., Corrigan, 2000; Kushner, Zeiss, Finglass & Yelen, 2014; Niederdeppe, Shapiro, Kim, Bartolo, Porticella, 2015; Pearl & Leibowitz, 2014; Young, Hinnant & Leshner, 2015). In the current study, the expression of empathy exhibited more explanatory power than the ascription of agency.

In the face of surging obesity rates worldwide and corresponding increases in resources and efforts assigned to manage the epidemic (see Wang, McPherson, Marsh, Gortmaker & Brown, 2011), a multi-faceted understanding of the determinants of obesity is beginning to emerge. Given the pervasiveness of obesity stigma, it seems particularly pertinent to examine whether focusing people's attention on different underlying causes of obesity impacts on their attitudes towards people with obesity. Previous research has

highlighted the importance that perceived behavioural agency plays in the expression of obesity stigma; the current study shows that perceptions of other underpinnings of obesity also have a marked effect on stigma expressions.

Future research should address how more complex understandings and belief systems about obesity aetiology impact on stigma expressions. This study operationalised the three aetiologies of obesity as being separate from one another, but obesity often has a more complex aetiology, involving the interaction of different factors (see Llewellyn & Wardle, 2015). An appropriate next step for research, therefore, may be to build on existing research that examines how beliefs and information about the complexities of obesity aetiology affects people's stigma expressions (e.g., Niederdeppe et al., 2015; Niederdeppe, Shapiro, Kim, Bartolo, & Porticella, 2014), including a focus on contextual influences (e.g., socioeconomic status and the food environment: see Donaghue, 2014; Morland & Evenson, 2009).

Experiencing stigma is disempowering for people with obesity, has negative consequences for psychological health and can perpetuate weight gain. As well as developing ways in which people with obesity can overcome and respond more positively to their stigma experiences, it is important to explore ways in which the expression of obesity stigma can be reduced. Beliefs about the causes of obesity are modifiable: harnessing this import may offer new ways of challenging stigma in the future.



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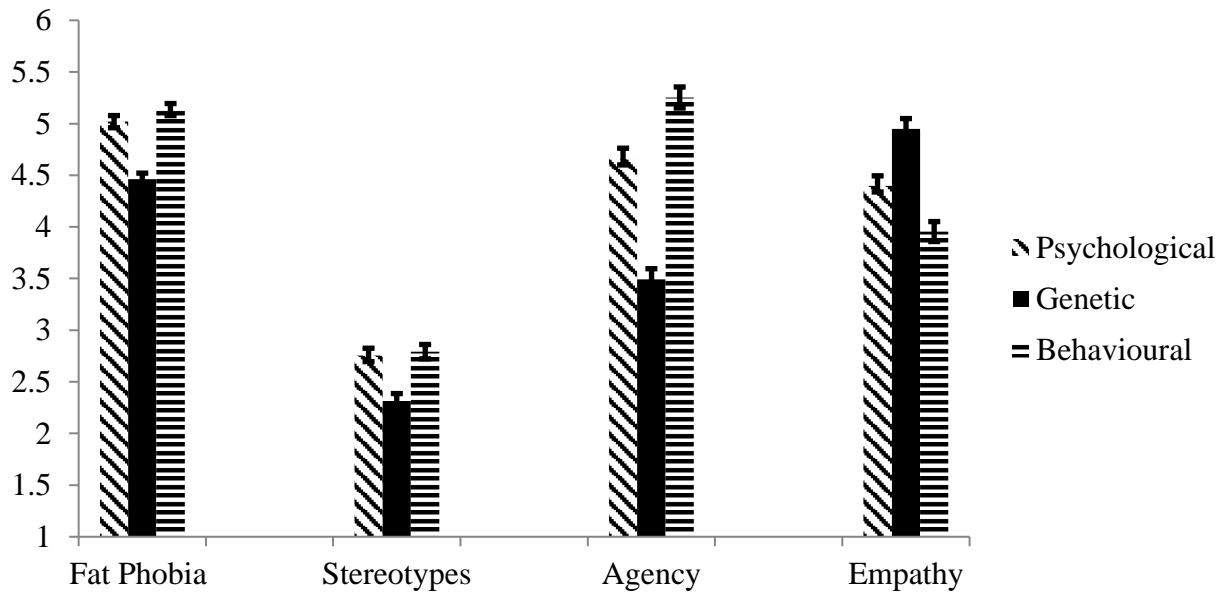


Figure 1. Estimated Marginal Means and Standard Errors as a Function of Condition

Table 1. Means, Standard Deviations and Correlations

|  | <u>Scales</u>     |                    |                |                |                |                |                 |
|--|-------------------|--------------------|----------------|----------------|----------------|----------------|-----------------|
|  | <i>Fat Phobia</i> | <i>Stereotypes</i> | <i>Agency</i>  | <i>Empathy</i> | <i>BAOP</i>    | <i>AFA</i>     | <i>BMI</i>      |
|  | <u>M (SD)</u>     | <u>M (SD)</u>      | <u>M (SD)</u>  | <u>M (SD)</u>  | <u>M (SD)</u>  | <u>M (SD)</u>  | <u>M (SD)</u>   |
| <u>Samples</u>                           |                   |                    |                |                |                |                |                 |
| <i>All Participants</i>                  | 4.88<br>(.87)     | 2.62<br>(.90)      | 4.48<br>(1.53) | 4.42<br>(1.73) | 4.71<br>(.92)  | 4.18<br>(1.25) | 26.62<br>(6.83) |
| <i>Psychological Aetiology Condition</i> | 5.03<br>(.76)     | 2.74<br>(.89)      | 4.67<br>(1.34) | 4.40<br>(1.29) | 4.74<br>(.85)  | 4.14<br>(1.19) | 25.76<br>(5.65) |
| <i>Behavioural Aetiology Condition</i>   | 5.17<br>(.84)     | 2.78<br>(.91)      | 5.27<br>(1.08) | 3.98<br>(1.35) | 4.79<br>(.88)  | 4.17<br>(1.34) | 26.43<br>(7.05) |
| <i>Genetic Aetiology Condition</i>       | 4.44<br>(.81)     | 2.35<br>(.85)      | 3.46<br>(1.55) | 4.91<br>(1.23) | 4.58<br>(1.02) | 4.26<br>(1.21) | 27.69<br>(7.57) |
| <u>Scales</u>                            | <u>r</u>          | <u>r</u>           | <u>r</u>       | <u>r</u>       | <u>r</u>       | <u>r</u>       | <u>r</u>        |
| <i>Fat Phobia</i>                        |                   | .36***             | .51***         | -.38***        | .40***         | .30***         | -.13***         |
| <i>Stereotypes</i>                       |                   |                    | .26***         | -.51***        | .04            | .16***         | -.09            |
| <i>Agency</i>                            |                   |                    |                | -.37***        | .27***         | .24***         | .17***          |
| <i>Empathy</i>                           |                   |                    |                |                | -.03           | .31***         | .17***          |
| <i>BAOP</i>                              |                   |                    |                |                |                | .42***         | -.09            |
| <i>AFAS</i>                              |                   |                    |                |                |                |                | .24***          |
| <i>BMI</i>                               |                   |                    |                |                |                |                |                 |

Table 2. Bonferroni Pairwise Comparisons

| <i>Scale</i> | <i>Condition (Aetiology)<br/>Comparison</i> | <i>Mean<br/>Difference</i> | <i>99% Confidence Interval</i> |                        |
|--------------|---|----------------------------|--------------------------------|------------------------|
|              |   |                            | <i>Lower<br/>Bound</i>         | <i>Upper<br/>Bound</i> |
| Fat Phobia   | Psychological vs. Genetic                   | -.56, $p < .001$           | .308                           | .807                   |
|              | Psychological vs.<br>Behavioural            | .12, $p > .250$            | -.361                          | .128                   |
|              | Genetic vs. Behavioural                     | .67, $p < .001$            | -.922                          | -.427                  |
| Stereotypes  | Psychological vs. Genetic                   | -.44, $p < .001$           | .143                           | .737                   |
|              | Psychological vs.<br>Behavioural            | .04, $p > .250$            | -.328                          | .254                   |
|              | Genetic vs. Behavioural                     | .48, $p < .001$            | -.772                          | -.182                  |
| Agency       | Psychological vs. Genetic                   | -1.17, $p < .001$          | .733                           | 1.603                  |
|              | Psychological vs.<br>Behavioural            | .59, $p < .001$            | -1.021                         | -.168                  |
|              | Genetic vs. Behavioural                     | 1.76, $p < .001$           | -2.194                         | -1.330                 |
| Empathy      | Psychological vs. Genetic                   | .55, $p < .001$            | -.966                          | -.966                  |
|              | Psychological vs.<br>Behavioural            | -.44, $p < .001$           | .038                           | .847                   |
|              | Genetic vs. Behavioural                     | -1.00, $p < .001$          | .586                           | 1.405                  |

Table 3. Indirect Effects of Condition upon Fat Phobia (FPS) and Stereotypes via Agency and Empathy

| <u>Condition Comparison</u>     | <u>Mediating Variable</u>           | <u>Effects</u>    |  | <u>Dependent Variable</u>                             |   |  |
|---------------------------------|-------------------------------------|-------------------|--|---|---|--|
|                                 |                                     | <i>Fat Phobia</i> | <i>Stereotypes</i>                                     |   |   |  |
| Psychological<br>vs.<br>Genetic | Agency                              | Direct            | $\beta = -.56, p < .001$                               | $\beta = -.44, p < .001$                              |   |  |
|                                 |                                     | Indirect          | $\beta = -.21$<br>LLCI = $-.328$<br>ULCI = $-.117$     | $\beta = -.13$<br>LLCI = $-.246$<br>ULCI = $-.038$    |   |  |
|                                 | Empathy                             | Direct            | $\beta = -.56, p < .001$                               | $\beta = -.44, p < .001$                              |   |  |
|                                 |                                     | Indirect          | $\beta = -.10$<br>LLCI = $-.180$<br>ULCI = $-.032$     | $\beta = .18$<br>LLCI = $.064$<br>ULCI = $.318$       |   |  |
|                                 | Psychological<br>vs.<br>Behavioural | Agency            | Direct   | $\beta = .12, p = .160$                               | $\beta = .04, p > .250$                         |  |
|                                 |                                     |                   | Indirect   | $\beta = .11$<br>LLCI = $.040$<br>ULCI = $.191$       | $\beta = .07$<br>LLCI = $.015$<br>ULCI = $.143$ |  |
| Empathy                         |                                     | Direct            | $\beta = .12, p = .160$                                | $\beta = .04, p > .250$                               |   |  |
|                                 |                                     | Indirect          | $\beta = .08$<br>LLCI = $.013$<br>ULCI = $.152$        | $\beta = .14$<br>LLCI = $.025$<br>ULCI = $.027$       |   |  |
| Genetic<br>vs.<br>Behavioural   | Agency                              | Direct            | $\beta = .67, p < .001$                                | $\beta = .48, p < .001$                               |   |  |
|                                 |                                     | Indirect          | $\beta = .09$<br>LLCI = $.030$<br>ULCI = $.180$        | $\beta = .19$<br>LLCI = $.054$<br>ULCI = $.359$       |   |  |
|                                 | Empathy                             | Direct            | $\beta = .67, p < .001$                                | $\beta = .48, p < .001$                               |   |  |
|                                 |                                     | Indirect          | $\beta = .17$<br>LLCI = $.088$<br>ULCI = $.279$        | $\beta = .32$<br>LLCI = $.187$<br>ULCI = $.064$       |   |  |
| Total Effects Model             |                                     |                   | $R^2_{adj} = .29$<br>$F(7,450) = 24.42,$<br>$p < .001$ | $R^2_{adj} = .09$<br>$F(7,450) = 6.56,$<br>$p < .001$ |   |  |

ULCI = Lower Level Confidence Interval

UCLI = Upper Level Confidence Interval

\*  $p < .05$ . \*\*  $p < .01$  \*\*\*  $p < .001$