



Stigma and the etiology of depression among the obese: An agent-based exploration



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ABSTRACT

Background: Obesity and depression are comorbid more often than chance predicts. However, depression among the obese is more common in settings where obesity is less common. This suggests that body habitus norms and social stigmatization may play a role in the etiology of depression among the obese.

Methods: We developed an agent-based social network model to explore mechanisms by which deviance from normative body habitus may contribute to social isolation in the obese. At each of 240 simulated months (20 years), each agent updated its body mass index based on environmental, peer influence, and stochastic factors. At each month, each agent was subject to social ostracization and consequent depression if its body mass index deviated from that of its peers and the network-wide mean. We compared risk of depression as a function of obesity and obesity norms through simulations of a high-obesity context simulating the US state of Mississippi and a low-obesity context simulating the US state of Colorado, then explored the relationship between global obesogenic forces and agent-specific resistance to the forces.

Results: Over 1000 simulations in each context, 25 percent of obese agents in simulated Colorado were ever-depressed as compared to 21 percent in simulated Mississippi, although 10 percent overall were ever-depressed in both settings. High and low levels of resistance to obesogeneity prevented the most depression, whereas medium resistance levels were more depressogenic.

Conclusions: Social stigma and ostracization that occur as a consequence of deviance from body habitus norms may be a plausible mechanism by which weight stigma may influence depression in the obese. Public health interventions targeting individuals rather than obesogenic environments may modify body habitus norms with the unintended consequence of increasing stigma-based social isolation among those who remain obese.

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Obesity and depression are prominent public health concerns. Obesity is an important determinant of chronic disease morbidity and mortality (Bender, 1999; Gunnell, 1998; Wilson, 2002) whose prevalence has nearly tripled (to 34%) over the past 30 years (Ogden and Carroll, 2010). Lifetime depression prevalence has been estimated at 16% (Kessler et al., 2003), and may be increasing (Hidaka, 2012). World Health Organization projections suggest that depressive disorders will become the leading source of global disease burden by 2030 (Mathers et al., 2008). Depression is even more prevalent among the obese (Luppino et al., 2010; Ma and

Xiao, 2010) and the co-occurrence of obesity and depression may synergize to increase risk for sequelae of both conditions (Ladwig et al., 2006). Therefore, understanding the mechanisms linking obesity and depression (Pan et al., 2012) is vitally important to minimizing their negative consequences.

In part because body weight is widely considered a consequence of various personal choices around diet and physical activity (DeJong, 1980), obesity is highly stigmatized (Falkner et al., 1999; R. M. Puhl and Heuer, 2010). Conscious and unconscious comparisons of body size can result in body image dissatisfaction (Faith et al., 2002; Markowitz et al., 2008), discrimination, social distancing (Ali et al., 2012), and subsequent stigmatization and social ostracization (Jackson et al., 2000; Sikorski et al., 2015). One study using a nationally-representative sample reported that nearly 40% of adults with a body mass index (BMI) over 35 kg/m² perceived

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discrimination due to their body size (R. M. Puhl et al., 2008). Responses to weight-based stigma vary (R. Puhl and Brownell, 2003), but have been shown to include common risk factors for obesity and depression, including social isolation and anhedonia (Lewis et al., 2011), reduced self-esteem (Friedman et al., 2005), unhealthy food consumption (Major et al., 2014), and exercise avoidance (Vartanian and Shaprow, 2008). Thus, experiencing weight-based stigma may both induce depression and reinforce obesity.

The extent to which obese individuals experience weight-based stigma is likely shaped by the degree to which obesity is 'normal' in a particular context (Burke and Heiland, 2007). Indeed, this finding has been observed along both spatial and social axes. For example, one study using data from the World Health Survey demonstrated that the odds of depression conditional upon obesity were highest in Japan, the country with the lowest prevalence of obesity in the study (Scott et al., 2007). Data from the United States showed similar relations. Colorado and the District of Columbia (Washington, DC) were the states with the lowest self-reported obesity prevalence in 2010, whereas Mississippi and Louisiana had the highest prevalence (Centers for Disease Control and Prevention, 2015). Consistent with the stigma-based hypothesis, dissatisfaction with life among the obese was higher in Colorado and Washington, DC than in Mississippi or Louisiana. Across social axes, obesity is less common among Whites than among Blacks in the United States (Flegal et al., 2012) although depression is more common among obese Whites than among obese Blacks (Hicken et al., 2013; Rosen-Reynoso et al., 2011). Depression is also more common among the obese of high socioeconomic status than among the obese of low socioeconomic status, though obesity itself is more common among low socioeconomic status individuals (Moore et al., 1962; Ross, 1994; Stunkard et al., 2003).

Socially mediated mechanistic hypotheses can be difficult to investigate using traditional epidemiologic approaches, because feedback processes wherein a subject's exposure status may affect the perceptions of other subjects, which in turn may affect subject's exposure status, violate the stable unit treatment value assumption necessary for causal inference (Kaufman et al., 2003; Rubin, 1980). Agent-based models offer complementary evidence, owing to their ability to incorporate bi-directional, dynamic feedback operating at multiple levels of influence (El-Sayed et al., 2012; Galea et al., 2010; Zhang et al., 2015). This simulation-based approach enables discovery of large-scale "emergent properties", such as shared perception of the body size considered normal, that can emerge from small-scale interactions (Axelrod, 1997; Bonabeau, 2002).

We developed an agent-based social network model to investigate whether context-dependent body habitus norms could explain higher prevalence of depression among the obese in low-obesity contexts. Our exploration first tested the norming mechanism in two simulated contexts (representing the US states of Mississippi and Colorado, which had the highest and lowest obesity prevalence in the US, respectively, in 2011's BRFSS) and second explored how this mechanism might lead to unintended increases in depression incidence as a result of interventions that increase resistance to obesogenic environmental forces among individuals.

1. Methods

1.1. Model

1.1.1. Network structure

We developed an agent-based social network model to interrogate and illustrate the hypothesized mechanism by which body habitus norms, conceptualized as the mean of peers' body sizes, and weight-based stigma due to positive deviance from those norms may shape depression in the obese. In constructing agent-

based models, an investigator must define the characteristics of interest in a population, analogous to a regression modeler's choice of covariates. While increased detail may help the model to resemble the real-world, excessive detail may obscure emergent phenomena (Bonabeau, 2002; Macy and Willer, 2002). Real-world social networks, with inter-relationships of social class, race, gender, and age, and perceptions of what is normal varying within subgroups defined in part by these demographic characteristics, are highly complex. To avoid allowing the modelling assumptions necessary to simulate this complexity to obscure the emergence of stigma patterns owing to norm violation, we simulated a simplified population wherein agents were not assigned an age, sex, or race.

In this model, each agent represents one individual and is embedded within a social network that contains all agents. Within the network, agents are connected to other agents if there is potential for social influence between them (e.g. because they are friends). All potential social influence is bi-directional and equal, meaning that if agent A is a peer of (i.e. connected to and thus potentially influenced by) agent B, agent B is a peer of agent A. For the main analysis, we used a small-world (Watts-Strogatz) network topology, which has been observed to approximate real-world social network topologies (Watts and Strogatz, 1998). Agents started with 10 network peers each. Fig. 1A displays one simulated network at baseline.

1.1.2. Population obesity

The promulgation of obesity in the model was a function of three factors: 1) environmental obesogenic influence consistent across the simulated neighborhood, representing the combined impact of the food environment (Holsten, 2009), lack of access to safe physical spaces (Burdette et al., 2006), an unattractive and unsafe environment (Lovasi et al., 2009), and shared social norms around food and exercise (Kemper et al., 1994); and 2) peer influence, representing the specific influences of other agents (Christakis and Fowler, 2007; Yakusheva et al., 2011, 2014), and 3) depression status, representing the influence of clinical depression on obesity over time (Kivimäki et al., 2009; Pan et al., 2012). Consistent with the finding that exposure to unhealthy food appears to be differentially obesogenic between individuals (Bader et al., 2013; Li et al., 2009), some agents were randomly selected at baseline to be resistant to environmental obesogenic influence such that obesogeneity had no influence on their BMI values. Conceptually, this resistance represents differences in genetic composition, health literacy, social support, the ability to marshal resources against a harmful influence, and other mechanisms that shape inter-individual response to obesogenic exposures described above (Egger and Swinburn, 1997; Link and Phelan, 1995). The number of resistant agents varied across simulations to represent differences in the population dynamics of obesity. We initially set peer influence to 0.034 BMI points/average peer BMI points over 9 months based on natural experiment evidence that was unlikely to be subject to confounding and homophily artifacts (Yakusheva et al., 2014). The study from which we parameterized reported only peer influence on weight controlling for height; for simplicity, we assumed the scale of influence would be the same on BMI, though we also tested alternate peer influence parameterizations in sensitivity analyses as described below and detailed in the online supplement. Depression caused a 0.12 unit increase in BMI for each month an agent was depressed, reflecting a mean elevation of about 0.7 BMI units among agents who became depressed compared to those who did not (Pan et al., 2012).

At baseline, some agents were randomly selected to be obese. The BMI values of obese agents were chosen randomly from a normal distribution with mean 35.0 kg/m², standard deviation 2.0, and minimum 30 kg/m², while non-obese agents' BMI values were

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