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Review

Stigma and the perpetuation of obesity

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ABSTRACT

Even as obesity rates reach new highs, the social stigmatization of obesity seems to be strengthening and globalizing. This review identifies at least four mechanisms by which a pervasive environment of fat stigma could reinforce high body weights or promote weight gain, ultimately driving population-level obesity. These are direct effects through behavior change because of feeling judged, and indirect effects of social network changes based on stigmatizing actions and decisions by others, psychosocial stress from feeling stigmatized, and the structural effects of discrimination. Importantly, women and children appear especially vulnerable to these mechanisms. The broader model provides an improved basis to investigate the role of stigma in driving the etiology of obesity, and explicates how individual, interpersonal, and structural dimensions of stigma are connected to variation in health outcomes, including across generations.

Obesity rates continue to rise. Yet, the stigma attached to obesity seems to be both strengthening and spreading globally (Brewis et al., 2011; Puhl et al., 2008). The experience of "being fat" is a miserable, chronic predicament (Link and Phelan, 2001) affecting millions. Reasons include: deep-seated and barely-questioned cultural norms that assign individual responsibility, failure, and blame to weight gain specifically (Brewis, 2011; Gimlin, 2007; Maddox et al., 1968; Rogge et al., 2004; Rothblum, 1992; Stafford and Scott, 1986); the increasing centrality of the physical body in the construction of social identities over the last half century (Bordo, 1993; Becker, 1995); difficulty in masking the stigmatized condition (Jones et al., 1984); negative messaging pervades almost every aspect of life, including media, family, work, school, and health care (Puhl and King, 2013; Puhl and Heuer, 2009; Puhl and Brownell, 2006); and damaging stigma-related mistreatment (such as bullying) can begin very early in life (Hayden-Wade et al., 2005; Lunner et al., 2000; Puhl et al., 2007; Sikorski et al., 2013). Very high rates of weight bias are recorded among medical, exercise science, and nutritional professionals, and prevention and treatment approaches to obesity continue to emphasize personal behavior change as the key to weight loss (Bombak, 2014; Foster et al., 2003; Harvey and Hill, 2001; Puhl and King, 2013; Puhl and Heuer, 2009; Saguy, 2013). Profoundly, obese people are as likely to believe, internalize, and project fat stigma as others (Bleich et al., 2013; Greener et al., 2010; Meana and Ricciardi, 2008; Schwartz et al., 2006).

Recently, Hatzenbuehler et al. proposed that stigma could act as a largely unrecognized, potentially powerful, driver of population-level health disparities (Hatzenbuehler et al., 2013; also Stuber et al., 2008). A recent prospective study of 6157 adults showed that those reporting weight-related discrimination were 2.5 times more likely to become obese and 3 times more likely to stay obese 4 years later (Sutin and Terracciano, 2013). A very wide array of research shows more generally that, once gained, weight is difficult to lose. Regardless of debates in the literature about exactly how difficult it is, the vast majority of adults who become obese stay that way (e.g., Kraschnewski et al., 2010). Together these observations suggest a fundamental and timely question that is the focus of this essay: *Might a pervasive environment of fat stigma and weight discrimination become literally embodied as perpetuated high body weights, significantly contributing to obesity rates? If so, by what means?*

This synthesis explicates candidate mechanisms by which stigma could underpin the perpetuation of high body weights, and outlines the theoretical and empirical bases of each below. To avoid an over-specified and hence fundamentally unusable model (Finegood et al., 2010), a full listing of evidence for possible links between stigma and weight gain/obesity is not the goal. Rather, the focus here is on highlighting and explaining factors that seem more central, more influential, better supported by current data, or more amenable to further study are highlighted. Women and girls report higher levels of fat stigma and weight discrimination (Puhl et al.,

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2008), so consideration of how gender could differently influence the various mechanisms is included. Further, the broader literature on the complex and inter-related causes of obesity beyond direct consideration of stigma is voluminous, and potentially relevant, but due to considerations of space is not included here: useful reviews in this regard include Spruijt-Metz (2011) and Brewis (2011).

1. Definitions

Fat stigma is the moral discrediting or "social death" (Yang et al., 2007) that people experience because of the negative social meanings attached to being overweight or obese. Common characterizations include laziness, non-compliance, lack of intelligence, a weak-will, dishonesty, and lack of self-control (Brewis, 2011). *Weight discrimination* is actual worse or unfair treatment resulting from that fat stigma, experienced both in everyday interactions with others (e.g., being assaulted, ignored, teased, or rejected) (Pescosolido et al., 2008) and as structural constraints (e.g., chairs that don't fit, worse treatment by healthcare professionals, or fewer career and educational opportunity). Just as people – even at the same weights – are differently exposed to the stigma or attendant discrimination of others, there is variation in the extent to which people notice, internalize, and ultimately *feel* and agree with stigma. The latter seems especially important to explaining vulnerabilities (Brewis and Wutich, 2012; Jacoby, 1994). By *embodied* (Krieger, 2005; Worthman and Costello, 2009) we mean the multiscalar mechanistic processes by which social, material, and institutional aspects of this felt fat stigma and weight discrimination become literally manifested in our biology as retained weight or additional weight gain.

2. Theorized mechanisms through which fat stigma can become embodied as gained or retained weight

2.1. Mechanism 1: direct behavioral change

If fat stigma negatively affects people's exercise, diet, and health-seeking, then it can lead to comparative weight gain or impede weight loss. The evidentiary basis for this mechanism is reasonably solid (Carels et al., 2009; Puhl and Heuer, 2010, 2009). Quantitative and qualitative studies of decision-making and behavior show those who feel judged by others because of their body size are less motivated to be physically active. For example, this can shape decisions to avoid exercising in public settings such as at school, gyms or swimming pools, or participate in organized sports (Lewis et al., 2011; Vartanian and Smyth, 2013; Wott and Carels, 2010). In a study of 111 US adults who considered themselves overweight, both reported experiences of weight stigma and own internalized anti-fat attitudes predicted avoidance of exercise (most especially at gyms), and less strenuous exercise (Vartanian and Novak, 2011).

Experimental and naturalistic studies provide parallel evidence to suggest stigma directly impacts people's decisions about how, when, and what to eat, including risky forms of dieting. In-laboratory exposure to stigmatizing materials like videos can trigger higher calorie consumption, especially in those who believe they are overweight (Major et al., 2014; Salvy et al., 2011; Schvey et al., 2011). Those who report on surveys that they feel stigmatized also report more comfort eating, bingeing, and extreme caloric restriction. If felt in childhood, this effect could shape diet choices for years afterward: one study of 2516 US adolescents showed that – controlling for body weight – boys who were teased were more likely to binge eat 5 years on, and girls were more likely to be frequent dieters (Haines et al., 2006).

Bingeing and caloric restriction are biologically relevant to weight gain because there is strong evidence they lead to counterproductive bounce-back cravings for high calorie and high fat foods when restriction stops, reshape eating behaviors to make long-term make weight gain more likely, contribute to weakening of hunger-satiety cues, and add up to weight cycling over time (Field et al., 2003; Lowe and Levine, 2005; Mann et al., 2007; Pietiläinen et al., 2012; Polivy and Herman, 1985; Puhl and Heuer, 2009; Saarni et al., 2006). For example, in a 10 year longitudinal study of dietary behavior and weight in 1902 adolescents, Neumark-Sztainer et al. (2012) found that greater levels of weight control behaviors (like eating less, skipping meals, and diet pills) at baseline and mid-study were associated with an increase of 4.63 body mass index (BMI) units over the decade, compared to 2.29 units in others. Animal and human studies alike show that weight cycling, especially in adolescence, can also reset basic metabolism, slowing resting energy expenditure across the lifespan (Brownell et al., 1986; Kayman et al., 1990; Leibel and Hirsch, 1984; Wadden et al., 1996).

Stigma also affects health choices in ways that should affect weight retention or gain once people are already overweight or obese. Patients who feel judged or mistreated in clinical settings because of their weight status report avoiding preventive care altogether, or are more likely to "doctor shop" (Malterud and Ulriksen, 2011, 2010), ultimately resulting in less access to professional weight loss advice, support, and medical treatments (Amy et al., 2005; Olson et al., 1994; Puhl and Heuer, 2010). Alternatively, studies show that overweight and obese patients who internalize weight bias report more health care visits, although whether this results in better care is unclear (Hilbert et al., 2014). Another evidentiary counterpoint is that bariatric patients report that stigma related to their weight is a motivator in seeking surgery. However, it is often described in narratives as a last resort, and not based in positive feelings about the healthcare system (Meana and Ricciardi, 2008). Stigma also predicts lower sense of self-efficacy and lower self-esteem, and this mediates people's beliefs that their weight loss efforts will fail – and hence they are more likely to abandon or avoid them (Puhl et al., 2007; Salvy et al., 2011; Schvey et al., 2011; Seacat and Mickelson, 2009; Vartanian and Shaprow, 2008).

2.2. Mechanism 2: indirect effects of psychosocial stress

This mechanism rests on research showing that feeling stigmatized and being discriminated against are very stressful, and that stress leads to weight gain. There is a growing body of studies showing that chronic psychosocial stress predicts additional gain of adipose tissue over time, especially visceral, and most especially among those already overweight or obese (Dallman et al., 2003; De Vriendt et al., 2009; Epel et al., 2001; Kivimäki et al., 2006; Schvey et al., 2014; Torres and Nowson, 2007). For example, in a longitudinal study of 11,247 Australian adults, high perceived stress predicted a .20 kg/m^2 increase in BMI over low perceived stress in a 5 year period, taking into account diet and exercise behaviors (Harding et al., 2014). The best understood proximate mechanism is arousal of the hypothalamic–pituitary–adrenal (HPA) axis and sympathetic nervous system leading to chronic elevation of catecholamine and glucocorticoid (GC) pathways (Bose et al., 2009; Björntorp, 2001). Immediately, GCs inhibit HPA-axis activity but chronic activation is excitatory, encouraging sugar and fat-seeking then depositing excess calories as abdominal fat. Developing metabolic systems are most vulnerable to these effects, and this appears to have lifelong consequences for weight gain(Chrousos, 2009). Chronic

stress earlier in life also seems to canalize lifelong less-healthy eating behaviors (Dallman et al., 2005; Dallman et al., 2003).

Studies testing the physiological stress effects of weight stigma specifically on long term weight change are yet to be done. However, laboratory studies have shown that exposure to weight-stigmatizing materials does elicit stress responses. Watching stigmatizing videos immediately elevates cortisol reactivity (an acute stress marker) in obese and lean women alike (Schvey et al., 2014), and exposure to weight stigmatizing messages in media articles trigger more immediate snacking in women who consider themselves overweight compared to others (Major et al., 2014). A study of 938 midlife adults reported that those who experienced more weight discrimination had exacerbated levels of HbA1C (a non-diabetic measure suggesting lack of glycemic control) at a given waist-to-hip ratio (Tsenkova et al., 2011). The literature exploring how racial discrimination leads to embodied stress is by comparison substantive, shows strong effects (Gravlee, 2009; Schafer and Ferraro, 2011; Sellers et al., 2003; Stuber et al., 2008; Williams and Mohammed, 2009), and hence may serve as a proxy model. There is however just one study linking experience of racial discrimination specifically to weight gain: using national data for 1956 Asian Americans, Gee et al. (2008) reported that experience of racial discrimination was associated with both higher BMI and higher risk of obesity, controlling for individual experience of weight discrimination.

Experiences of weight stigma do predict greater risk of depression (Hilbert et al., 2014; Scott et al., 2008), and vice versa (Blaine, 2008; Luppino et al., 2010; Markowitz et al., 2008; Preiss et al., 2013). Explanations for the former focus on both stress activation of the neuroendocrine system and the use of antidepressants, especially SSRIs (Correll et al., 2009; Tsigos and Chrousos, 2002). Women and girls seem most at risk in both regards (Kalarchian and Marcus, 2012; Vazquez and Torres, 2012). Preliminary cross-sectional studies do suggest weight-related stigma can directly contribute to depression (Friedman et al., 2008; Katterman and Alverdy, 2007), and established trigger for lifetime risk for depression is bullying in childhood, to which girls again seem more vulnerable (Goldfield et al., 2010).

This indirect mechanism can intersect bi-directionally with direct behavior changes triggered by stigma (mechanism #1). For example, disordered eating behavior increases risk of depression (Gadalla and Piran, 2008) and depression symptoms predict eating disorders (Kalarchian and Marcus, 2012). The stereotype-threat literature from social psychology also straddles mechanisms #1 and #2, suggesting that apprehension ("stress") would be greatest when people fear their behaviors are reinforcing stereotypes about their group (such as eating high calorie foods or avoiding physical activity). The apprehension is greatest when the person endorses the stereotype and when they are primed in it, such as by experiencing weight discrimination. This threat then leads to behaviors like overeating consistent with the stereotype (Carels et al., 2013; Schmader et al., 2008; Shapiro and Neuberg, 2007).

2.3. Mechanism 3: indirect effects via changes in social relationships

This proposed mechanism recognizes that stigma could lead to weight gain if it changes the composition or quality of social networks in ways that then lead to different exercise or dietary behavior. There is a significant literature showing that peers directly shape diet and exercise and behavior (Sallis et al., 1987; Stuber et al., 2008), with closer friends often exerting greater influence (Barclay et al., 2013; De la Haye et al., 2013; De La Haye et al., 2011). Overall, compared to those for diet, peer influences on physical activity/sedentary behavior are more consistently described (McNeill et al., 2006), with greatest effects seen in adolescents and boys (reviewed by Sawka et al., 2013). This is a highly speculative mechanism, with likely multiple complicating bi-directional effects. For example, analysis of National Longitudinal Study of Adolescent Health (NLSAH) participants ($N = 1896$) has shown that adolescent networks influence exercise behavior, but high levels of physical activity also creates and reinforces friendships so that people with similar bodies converge within in social networks over time (Simpkins et al., 2013). A related study using NLSAH data has also shown that weight teasing itself can reinforce these processes in childhood, such that non-overweight adolescents were 30% more likely to select non-overweight friends, whereas overweight adolescents did not differentiate, leading to avoidance of overweight peers as a major shaper of social networks through time (Schaefer and Simpkins, 2014). While repeated rebuffs meant that overweight children turn to one another for friendship, the same study found no evidence, however, that overweight children have numerically fewer friends overall.

Studies have identified social rejection and isolation factors in psychosocial stress (Cohen and Wills, 1985), making those with more constricted social networks more likely to be depressed (De Wit et al., 2010). Reviews suggest that obese people have less social support within their networks overall, undermining weight-loss efforts (Markowitz et al., 2008). These provide a broader theoretical rationale to consider further bi-directionality within mechanism #3, at least among adults, and to consider significant iterative feedback effects with mechanism #1 and #2.

When it comes to shaping health generally, networks are both a source of stress and support (Umberson et al., 2010). Stigma's effect on networks likely relate to both, but would collectively tend to lead to obese people having proportionately more overweight or obese people in their networks. Networks might be more constricted and thus provide less support if others withdraw to avoid the taint of association("courtesy stigma") (Miller et al., 1990; Pryor et al., 2012; Stuber et al., 2008), or as people cope with stigma by choosing to withdrawing socially (Puhl and Brownell, 2003; Strauss and Pollack, 2003). These effects can start very early in childhood (Fletcher et al., 2011) and on balance it appears that obesity leads to social isolation, not vice versa (Apolloni et al., 2011). Stigma can also change network composition as people seek more and closer relationships with sympathetic others who share their plight and can provide understanding, acceptance, and emotional support (Brochu and Morrison, 2007; Carr and Friedman, 2006; Pescosolido et al., 2008).

Evidence from longitudinal analysis of social networks suggests that friends with higher or lower BMI tend to clump together socially through time (Christakis and Fowler, 2007; Hruschka et al., 2011). Christakis and Fowler suggest this is due to confluence or sharing of social norms (Christakis and Fowler, 2007). However, a recent study that interviewed overweight and non-overweight adult women and alters they nominated within their social networks found the observed clustering of obesity within social networks was unlikely due to shared norms (with norms measured in several ways). Rather, shared social behavior such as exercising is a better explanation for the observed convergence of body sizes (Hruschka et al., 2011). This fits with other recent evidence from studies of children's peer effects on diet and exercise that suggest exposure to peer behaviors might be much more important than exposure to norms (De la Haye et al., 2013, 2011).

2.4. Mechanism 4: indirect structural effects of discrimination

This mechanism proposes that the negative socioeconomic effects of weight discrimination can lead to additional weight gain.

There is clear evidence that weight bias negatively affects access and opportunity in almost all aspects of everyday life (Puhl and Heuer, 2009). Obese people report fewer training, work, educational and career opportunities (such as being promoted) than their non-obese peers. There is a well-documented "obesity wage penalty," especially for women (Baum and Ford, 2004; Cawley, 2004), meaning they are paid less even once other factors like job performance are taken into account. Being obese has also been shown to inhibit women's relative capacity to transition from welfare to work (Cawley and Danziger, 2005). Other relevant ripple effects of discriminatory practices include less access to the types of higher-wage, full-time jobs where high quality employer health insurance is provided, leading to higher personal health care costs and less preventive care (Puhl and Heuer, 2009).

Lower income is generally associated with increased obesity risk in the wealthier nations, and increasingly in middle-income countries (Brewis, 2011). US household food insecurity and reliance on federal food assistance programs predicts additional risk of obesity, most especially for women (Gibson, 2003) and girls (Leung et al., 2013; Schmeiser, 2009). Less wealth means fewer choices and options related to diet and exercise. Examples include: less money for expensive nutrient-dense foods (Drewnowski, 2009; Drewnowski and Darmon, 2005); less time to prepare home-cooked meals (Mancino and Newman, 2007); and less time and money to access pay-to-play sports or fitness facilities like gyms (Halpern, 2003).

Additional risk related to obesity can also be created spatially by downward mobility because relative wealth shapes where people live. There is consistent evidence of neighborhood clustering of higher BMI and obesity in lower income urban communities in higher income countries. Adults and children living in lower income neighborhoods are often structurally exposed to less favorable exercise environments (such as fewer safe places to walk less access to pleasant parks, and schools with fewer exercise amenities or after-school sports) and worse access to affordable healthy food (Black and Macinko, 2008; Cutts et al., 2009; Giles-Corti and Donovan, 2002; Gordon-Larsen et al., 2000; Holsten, 2008; Lovasi et al., 2009; Ohri-Vachaspati et al., 2013; Papas et al., 2007; Pearce et al., 2007; Sallis and Glanz, 2009; Sallis et al., 2007). Such neighborhoods also face structural barriers to health care that can influence weight, such easy access to primary care (Kirby and Kaneda, 2006; Lurie and Dubowitz, 2007). Findings demonstrating causally the effects of built environmental factors on weight-relevant behaviors tend to be stronger for physical activity than diet, but are overall mixed and often report small effect sizes (Black and Macinko, 2008; Sallis and Glanz, 2009). Studies explicitly linking weight discrimination, downward mobility, exposure to obesogenic environments, and longitudinal weight gain together are yet to be conducted.

More generally, there is a substantial and diverse body of evidence that living in poverty and dealing with everyday resource shortages and conflicts is also chronically stressful (Sapolsky, 2004; Weaver and Hadley, 2009; Wutich and Brewis, 2014). Thus this downward mobility likely intersects in important ways with mechanism #2. For example, the correlation between food insecurity and food stamp use and weight gain is suggested to be partially explained by underlying psychosocial stress related to food insecurity itself (Jilcott et al., 2011; Jones and Frongillo, 2006). These are the same poverty contexts in which stress-generating social inequalities tend to be amplified, suggesting complex interactions with mechanism #2. Importantly for further research on these structural contexts, the stigma of obesity likely intersects with many other stigmas, such as those related to race, place, or disease status (Hatzenbuehler et al., 2013). Such layering of stigmas (Mill et al., 2009) likely exacerbates even further the stress, suffering, lost opportunities, downward mobility, and social injustices that can become embodied as obesity through the same or similar mechanisms.

2.5. Potential intergenerational effects

There is good theoretical rationale, but very limited empirical evidence, to propose possible inter-generational weight gain or weight retention effects of these mechanisms, especially from stress (mechanism #2) and poverty (mechanism #4). There is growing interest in human biology in understanding how maternal physiological condition shapes fetal neuroendocrine systems in ways that make obesity more likely into the next generation. Gestational diabetes in particular leads to an over-feeding of the fetus, so that the child is then born larger with altered glucose/insulin metabolism (Barker, 1997; Godfrey et al., 2010; Kuzawa, 2010; Kuzawa et al., 2007). Prospective studies establishing causation in humans are few, however one seminal study comparing children born to the same mothers before versus after the mother underwent bariatric surgery and subsequent weight loss showed a 3-fold decrease in prevalence of severe obesity in the children and numerous metabolic improvements, including lower insulin sensitivity (Smith et al., 2009).

Animal (especially rodent) models provide more robust evidence, showing stress plus poor nutrition *in utero* can lead to weight-inducing developmental changes such as in hypothalamic connectivity that changes feeding and satiation cues, HPA-axis dysfunctions with lifetime changes in glucocorticoid levels, and with complex interactions with inflammatory processes (Zambrano and Nathanielsz, 2013; Sominsky and Spencer, 2014). Longitudinal studies with humans are few, but one shows that children of mothers who had a major stressful life event when pregnant displayed weight-associated effects on HPA-axis regulation well into their lives compared to controls (Entringer et al., 2009). Proposed proximate mechanisms for such effects in humans include: maternal stress hormones directly crossing the placental barrier; release of placental hormones into fetal circulation due to maternal stress; and maternal stress effects on placental blood flow resulting in fetal restriction and release of fetal glucocorticoids (Entringer et al., 2009; Huizink et al., 2004; Wadhwa, 1998). One additional epigenetic mechanism is DNA methylation, which both silences and turn on genes, and a recent study with mice has shown that early life stress alters methylation and this then shapes later depression-like behavior (Franklin et al., 2010). Animal model work also shows that paternal stress can modify epigenetic settings and be passed onto offspring via sperm, influencing the development of fetal neuroendocrine systems (Morgan and Bale, 2011).

3. Conclusion

This review suggests there is good theoretical rationale to suggest embodied weight-related stigma and discrimination, a social phenomenon, may contribute to weight retention and gain at the individual level through multiple – probably reinforcing – mechanisms operating at multiple scales. Even if each of the specified mechanisms related to stigma and discrimination only weakly predict weight-gain or weight-retention, multiple weak mechanisms interacting in a larger biosocial system could none-the-less act collectively to increase population-level obesity. And, because exposure to weight-related stigma and discrimination tends to magnify as people gain more weight (Puhl and Brownell, 2006), there is also a concerning possibility that systemic approaches to understanding obesity might even reveal powerful self-reinforcing cycles that can act to amplify both emotional suffering and weight

gain within and across generations. This provides an impetus for specific future studies focused on modeling the emergent properties of interactions and feedback loops across and between these identified mechanisms (e.g., Finegood and Cawley, 2011), and with due consideration of possible intergenerational effects.

This review also highlights that in relation to the identified mechanisms it is women, adolescents, and children that appear more vulnerable to most, providing the practical suggestion that addressing how overweight and obese children experience and feel stigma is a particularly useful place to focus in obesity prevention and intervention efforts.

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