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Stress and Obesity

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Abstract

Many pathways connect stress and obesity, two highly prevalent problems facing society today. First, stress interferes with cognitive processes such as executive function and self-regulation. Second, stress can affect behavior by inducing overeating and consumption of foods that are high in calories, fat, or sugar; by decreasing physical activity; and by shortening sleep. Third, stress triggers physiological changes in the hypothalamic-pituitary-adrenal axis, reward processing in the brain, and possibly the gut microbiome. Finally, stress can stimulate production of biochemical hormones and peptides such as leptin, ghrelin, and neuropeptide Y. Obesity itself can be a stressful state due to the high prevalence of weight stigma. This article therefore traces the contribution of weight stigma to stress and obesogenic processes, ultimately describing a vicious cycle of stress to obesity to stigma to stress. Current obesity prevention efforts focus solely on eating and exercise; the evidence reviewed in this article points to stress as an important but currently overlooked public policy target.

Contents

INTRODUCTION

We live in a highly stressed society. National surveys conducted by the American Psychological Association (APA) show that a majority of US citizens reports moderate to high levels of stress (Am. Psychol. Assoc. 2012, 2017). At the same time, over one-third of US adults are in the obese category, meaning that they have a body mass index (BMI) greater than 30 (Hales et al. 2017). Could these phenomena be related? Obesity has many causes, including strong genetic origins (Bell et al. 2005), but could stress play at least a partial role? Based on the available scientific evidence, the conclusion of this review is that, yes, through multiple pathways, stress plays a role in the development and maintenance of obesity.

The idea that stress—that nebulous psychological experience—could determine a person's body mass may seem far-fetched. However, viewed through an evolutionary lens, it makes good sense that stress would affect metabolic processes. Our stress systems evolved to help us escape life-threatening situations, which, in ancestral times, often involved considerable metabolic effort. Our stress-response systems are responsible for releasing glucose into the bloodstream so that our muscles have the energy to flee (or fight, if that is the preference) predators and other physical threats (Lovallo & Thomas 2000). In modern times, however, most of the stressors we encounter are psychological (e.g., a fight with a spouse, hard times at work, worrying about money) rather than physical. Our bodies nonetheless still respond as if the stress is physical. This leaves us in a situation where the excess energy has nowhere to go and tends to ultimately be deposited as body fat (Björntorp 2001). Thus, stress and metabolic processes are intricately linked, and indeed, epidemiological studies show that stress and BMI are related (Block et al. 2009, Moore & Cunningham 2012), and a meta-analysis of longitudinal studies found a positive relationship between stress and weight gain (Wardle et al. 2011).

For the purposes of this article, stress is defined using Andrew Baum's comprehensive characterization: "a negative emotional experience accompanied by predictable biochemical, physiological, cognitive, and behavioral changes that are directed either toward altering the stressful event or accommodating to its effects" (Baum 1990, p. 653). Indeed, stress and obesity are linked through each of these types of changes, although more often toward the goal of accommodating to the effects of stress rather than of altering the actual stressful event. In other words, many of the pathways that lead from stress to obesity are attempts by an individual to cope with the negative emotional aspects of stress. These many pathways are summarized in **Figure 1**. Stress can be characterized as acute or chronic. As the main outcome in the model is obesity, which is by definition something that develops over time, this is a chronic stress model. However, repeated episodes of acute stressors can accumulate to cause obesity by repeatedly activating these pathways.

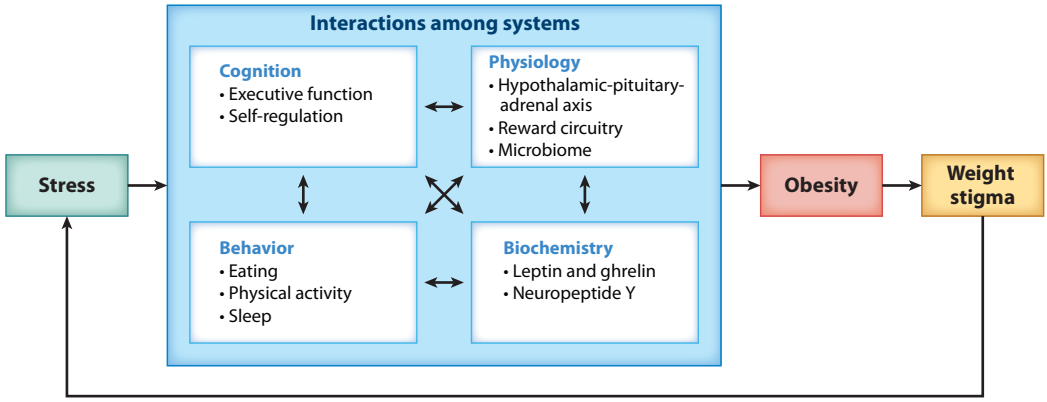

Figure 1

Pathways that connect stress to obesity. The components in the blue box are theorized to interact with one another, although existing studies do not test all possible interactions. Weight stigma is characterized as a stressful state, thus creating a positive feedback loop.

The pathways that connect stress to obesity are organized in the model and this article according to the four categories of changes resulting from stress in Baum's characterization—cognition, behavior, physiology, and biochemistry. Importantly, the factors identified in the model affect one another. For example, self-regulation is necessary to control food intake. The relationships can also be bidirectional. For example, lack of sleep can hinder physical activity, and lack of physical activity can disrupt sleep. Empirical evidence for these interactions is noted when available, but an overarching point to keep in mind is that the true state of affairs is likely one in which many of these factors simultaneously affect many others in an emergent process—something that is difficult, if not impossible, to capture within a single study.

One often overlooked aspect of stress and obesity is that obesity in itself can be a stressful state. Throughout most of the world, strong stigma against heavier bodies exists (Brewis et al. 2011). Weight stigma is defined as the sum of prejudice, discrimination, and negative attitudes aimed at those perceived as overweight (Tomiyama 2014) and is highly prevalent (Spahlholz et al. 2016). Some studies report that weight stigma is even more common than stigma based on other social identities, such as race and ethnicity or gender, in certain domains (e.g., interpersonal mistreatment) (Puhl et al. 2008). Experiencing weight stigma can induce stress, and thus, this review also addresses this feedback pathway linking obesity to weight stigma and back to stress.

The following sections review evidence for each of the pathways depicted in **Figure 1**. As this review covers multiple systems spanning the mind, behavior, and body, it focuses, by necessity, on seminal studies, reviews, and meta-analyses, and studies with the most rigorous designs or those that offer particularly strong insight. Nonhuman animal studies are described when human data are lacking or impossible to obtain.

STRESS TO OBESITY

Cognition

Self-regulation is important for controlling one's own behavior and is relevant in this case because self-regulation is required to enact obesity-preventing behaviors such as eating and physical activity (Heatherton 2011). Stress, however, can undermine self-regulatory cognitive processes such as executive functioning (Pechtel & Pizzagalli 2011) and interferes with the brain areas responsible for self-regulation (Liston et al. 2009).

In one illustrative study, Evans and colleagues (2012) used a delay of gratification task wherein children (244 9-year-olds) were told that they could have a medium-sized plate of candy now or a large plate later. Children who had experienced more cumulative life stressors were more likely to choose the large plate of candy, and those children, in turn, had higher BMI growth 3 years later.

Stress can also inhibit cognitive emotion regulation (Raio et al. 2013), thus rendering individuals even more susceptible to emotional processes that can trigger unhealthy eating (see the next section). Thus, stress can lead to obesity by hindering the cognitive processes required for self-regulation.

Behavior

Failures in self-regulation can feed into the deleterious health behaviors discussed in the following sections. However, stress can also promote these behaviors in the absence of an explicit self-regulation goal.

Eating. The slogan for Mars Inc.'s chocolate candy bar, "Milky Way: Comfort in every bar," describes the phenomenon in which individuals eat to comfort their stress-related negative emotions. Individuals can eat more or eat differently under stress, with most gravitating toward palatable foods that are high in sugar, fat, and calories (Adam & Epel 2007, Torres & Nowson 2007). The behavior of stress-induced eating is highly prevalent, with 39% of US adults in an APA survey reporting that they either overeat or eat unhealthy foods in response to stress (Am. Psychol. Assoc. 2012). Other survey studies support a general pattern whereby individuals engage in unhealthy eating post-stress (Torres & Nowson 2007). For instance, a study of workplace stress showed that employees reported higher energy, saturated fat, and sugar intake during high-workload periods (Wardle et al. 2000). Experimental and daily diary studies support these survey data, showing that, for example, people randomly assigned to unsolvable (versus solvable) anagrams ate more high-sugar, high-fat food (Zellner et al. 2006), and that daily hassles were associated with increased high-fat and high-sugar snack consumption (O'Connor et al. 2008).

Furthermore, humans are not the only animals to engage in stress-induced eating. For example, rats exposed to numerous kinds of stress engage in stress-induced eating of lard and sucrose (Pecoraro et al. 2004). Stress-induced eating has also been observed in captive common marmosets (Johnson et al. 1996). The fact that this behavior is conserved across species indicates that the behavior is likely important for humans, as well (Dallman et al. 2005). In sum, stress-induced eating is a major pathway through which stress begets obesity, whether that manifests as eating more or eating more unhealthily.

Physical activity. Stress can disrupt activity patterns, whether by decreasing physical activity or by increasing sedentary behavior. In other words, people can volitionally exercise less due to stress and simultaneously or independently spend more time being sedentary. Of the two, the evidence is stronger for decreased physical activity. A survey of over 12,000 participants found that higher stress was related to less frequent exercise (Ng & Jeffery 2003), and similarly, a longitudinal study of almost 1,400 women showed 3-year prospective relationships between higher perceived stress and lower leisure-time physical activity (Mouchacca et al. 2013). This latter study did not find associations between higher stress and more sedentary behaviors like sitting time or television viewing (or, in fact, higher palatable food intake).

A very comprehensive review divided 168 studies testing the link between stress and physical activity by the different types of stressors that the studies examined (Stults-Kolehmainen & Sinha 2014). Focusing on the 55 longitudinal studies that they identified, the authors found that a majority of studies supported an association whereby greater stress was tied to less physical activity. This was

true across objective stressors such as examination stress or caregiving stress, as well as subjective stressors. There were, however, several null studies (although those had weaker designs), and in some cases, stress predicted more physical activity. The authors speculated that those findings may have reflected the tendency for some individuals to use exercise as a means to cope with stress. Nonetheless, the preponderance of evidence indicates that stress disrupts physical activity, thus leading to a higher likelihood of obesity.

Sleep duration. Stress is a known disrupter of sleep (Åkerstedt et al. 2007). Sleep, in particular shorter sleep duration, is tied to a higher likelihood of obesity (Ogilvie & Patel 2017). A systematic review of 31 cross-sectional and five longitudinal studies found that shorter sleep duration was independently associated with and predictive of higher weight, higher likelihood of obesity, and other adiposity markers (Patel & Hu 2008). A meta-analysis quantified the relationship between sleep duration and BMI, finding that the pooled odds of obesity were 1.55 with shorter sleep duration. Viewing the data another way, the decrease in BMI was −0.35 for every hour increase in sleep (Cappuccio et al. 2008). Another review of this literature concurred with these general conclusions but noted that there are some studies that find a U-shaped relationship between sleep duration and weight, with long sleepers also having higher body weight (Marshall et al. 2008).

According to a model by Patel & Hu (2008) based on findings from experimental sleep deprivation studies, four mechanisms tie poor sleep to higher weight. The first is a direct physiological pathway, with shorter sleep decreasing thermogenesis, thereby decreasing one's energy expenditure (Shaw 2005). Shorter sleep can also promote fatigue, further contributing to decreased energy expenditure via reduced physical activity and increased sedentary behavior (Dinges et al. 1997). Shorter sleep also makes people report being hungrier (Spiegel et al. 2004b), particularly for high-fat and -carbohydrate foods. This effect appears to be mediated by leptin and ghrelin (Spiegel et al. 2004a,b), which are appetite regulating hormones covered in more detail below. Finally, those who sleep for a shorter period of time are, by definition, awake longer, meaning that they have more time to eat, although this pathway has not found much empirical support (Patel & Hu 2008). Thus, many pathways tie short sleep duration to obesity, but research is needed that ties stress to short sleep duration to obesity in a single study.

Physiology

Stress reliably causes activation of multiple physiological systems. Of these, the three pathways that are most pertinent to obesity are discussed next.

Hypothalamic-pituitary-adrenal axis activation. When an individual perceives stress, a physiological cascade occurs in the hypothalamic-pituitary-adrenal (HPA) axis (Lovallo & Thomas 2000). Corticotropin-releasing factor (CRF) is sent from the hypothalamus to the pituitary gland, which sends a signal to the adrenal glands via adrenocorticotropic hormone. The adrenal glands, which sit atop the kidneys, then secrete the hormone cortisol. Although cortisol has many jobs in the body, it is critical to the discussion of stress and obesity because it triggers processes that lead to weight gain.

First, cortisol promotes eating. The strongest evidence for this comes from studies that administer synthetic glucocorticoids to participants and then measure their eating. In one such study, Tataranni and colleagues (1996) demonstrated that participants randomly assigned to ingest cortisol versus placebo ate more food. A seminal study by Epel and colleagues (2001) supports this notion in the context of naturally occurring levels of cortisol, finding that participants experiencing a standardized laboratory stressor who had higher cortisol ate more food than those with lower cortisol. Cortisol also promotes eating by reducing the brain's sensitivity to leptin (Jéquier 2002),

regulating neuropeptide Y (NPY) stimulation (Sinha & Jastreboff 2013), and potentiating reward pathways, as discussed in more detail below.

Second, cortisol directly promotes fat deposition, particularly in the abdominal region. This is readily evident in Cushing's disease, in which individuals have congenitally high levels of cortisol. A hallmark symptom of Cushing's disease is abdominal obesity, which resolves when cortisol is medically reduced to normal levels (Björntorp 2001, Shibli-Rahhal et al. 2006). Diurnal salivary and 24-h urine cortisol levels predict higher BMI and abdominal obesity (Fraser et al. 1999, Rosmond et al. 1998). Indeed, the link between cortisol levels and abdominal obesity is so consistently observed that abdominal obesity has been suggested as a marker for long-term cortisol levels (Björntorp & Rosmond 2000). Abdominal obesity is a particularly toxic pattern of fat deposition and predicts poor metabolic and cardiovascular health in longitudinal studies (Després et al. 2001).

In sum, cortisol—the end product of stress-responsive HPA axis activation—can promote obesity in two ways. First, it stimulates eating on its own, both through other hormones and peptides and through its effects on the brain and reward processing. Second, cortisol can directly promote fat deposition, particularly in the abdominal region.

Reward processing. Stress makes people susceptible to substances that, via reward processing, motivate individuals to overconsume them (Sinha 2008). Foods high in sugar, fat, and calories are examples of such substances. In fact, highly palatable foods like sugar have addictive potential, indexed by signs of tolerance, withdrawal, and neurochemical changes in the nucleus accumbens, sometimes to an even greater extent than cocaine in nonhuman animal models, as shown in the landmark work by Bart Hoebel (for a discussion of this work, see Avena 2010). A hallmark symptom of addictive-like behavior is wanting (i.e., motivation or desire for a substance) and intake of a substance in the absence of liking (i.e., enjoyment in consuming a substance; Berridge et al. 2010), and acute stress increases both food wanting and food intake in humans (Lemmens et al. 2011).

Dopamine is a neurotransmitter that codes for pleasure and enhances the desire for food (Volkow et al. 2013). Stress independently triggers dopamine release, as reliably shown in nonhuman animals (Dallman 2010) and in a few human studies (Pruessner et al. 2004, Wand et al. 2007), and this dopamine release has further downstream effects on food seeking and eating (Dallman 2010).

In turn, eating highly palatable foods further affects reward processing to augment the stress-eating cycle. Changes in CRF, glucocorticoids including cortisol, and norepinephrine sensitize reward centers in the brain such as the nucleus accumbens and dorsal striatum, which increases the drive to eat these foods (Cottone et al. 2009, Sinha & Jastreboff 2013). Through negative reinforcement (Adam & Epel 2007), these foods can serve to provide relief from both stressful states and the hedonic withdrawal that occurs as a result of long-term exposure to cortisol (Dallman et al. 2003) and can then be ingrained as reward-driven habits (Schwabe & Wolf 2009). Thus, stress, reward, and highly palatable foods form a positive feedback loop, captured by Sinha & Jastreboff (2013) in their sensitized feedforward process model.

Microbiome. In a fascinating emerging area of research, it is becoming evident that the microbes in the gastrointestinal tract (collectively referred to as the gut microbiome) are both responsive to and generative of psychological states such as stress. The nonhuman animal literature has now amassed a solid body of evidence showing that the gut microbiome is affected by stress, whether acute (Galley et al. 2014), chronic (Moussaoui et al. 2017), or even prenatal (Golubeva et al. 2015). In turn, and more eerily, the gut microbiome can affect how we feel and behave. This happens through afferent (body to brain) neural signaling through the vagus nerve, generation of endocrine

hormones and neurotransmitters, and immune signaling (see Foster et al. 2017). Different compositions of gut microbiota can affect how stress responsive an organism is, and thus, one way that the gut microbiome could affect obesity is by increasing stress and stress-related physiological activation. For example, transferring gut microbiota from stress-prone mice to normal mice induces anxiety-related behaviors, and doing the opposite—transferring gut microbiota from normal mice to stress-prone mice—induces reduction in anxiety (Bercik et al. 2011). The gut microbiome is also capable of increasing the activity of the HPA axis (Tilders et al. 1994).

Relevant to stress and obesity, the gut microbiome can also influence eating behavior through several mechanisms. Much of this evidence comes from models that use germ-free mice, which are born and raised in sterile conditions and thus without microbiota. For instance, germ-free mice have more sweet and fat taste receptors than regular mice (Duca et al. 2012, Swartz et al. 2012). Microbes can also manufacture ghrelin and NPY, as well as peptides that act like these appetite-regulating hormones (Fetissov et al. 2008). Germ-free mice, in contrast, have lower levels of satiety hormones like leptin (Duca et al. 2012).

In addition to eating behavior, the gut microbiome also directly influences body weight. In the mouse model, for example, raising genetically obesity-prone mice in a germ-free environment results in lean mice (Bäckhed et al. 2004). Interestingly, this result was independent of food consumption and, rather, due to intestinal changes that led directly to larger fat cells. In a human twin study, researchers found that the twin without obesity had a more diverse gut microbiome than the twin with obesity (Turnbaugh et al. 2009).

Although the literature on the gut microbiome and obesity in nonhuman animal models is substantial and growing rapidly, there is currently only a small literature on humans. In human intervention studies, there is some evidence that changing the gut microbiome through probiotics such as *Lactobacillus* or *Bifidobacterium* affects weight by promoting weight and abdominal fat loss and by reducing weight gain (Alcock et al. 2014), even when energy intake and exercise are held constant (Kadooka et al. 2010).

What is missing from even the nonhuman animal literature is evidence that ties the entire chain together—that is, no study has shown that the specific gut microbiome changes resulting from stress are, in turn, directly responsible for changes in eating behavior and weight gain. While this is an exciting area of research, an important further caveat is that very strong evidence in animal models has not always translated to humans (Kelly et al. 2017).

Biochemistry

In addition to activating physiological systems, stress can also modulate levels of biochemical substances that are relevant to weight and obesity. Leptin, ghrelin, and neuropeptide Y are discussed in the following sections. However, other appetite-stimulating peptides, such as Agouti-related peptide and propriomelanocortin, are also implicated in stress, but the literature on these peptides is much smaller (Maniam & Morris 2012) and is thus not covered in this review.

Leptin and ghrelin. Although it is considerably less established compared to the literature on stress and cortisol, a small amount of evidence indicates that the hormones leptin and ghrelin may respond to stress. These hormones govern appetite in opposite ways. As evidenced by exogenous administration studies, leptin suppresses appetite, and ghrelin stimulates it (Schwartz et al. 2000). Ghrelin also heightens food reward processes (Dickson et al. 2011, Perello & Zigman 2012). Thus, these two hormones represent important regulators of appetite and, over time, weight gain.

Rather than contributing to obesity, leptin in the context of stress may be an individual difference factor preventing weight gain. One study directly examined the role of leptin in the context

of stress-induced eating (Tomiyama et al. 2012). Participants were repeatedly sampled for leptin while undergoing a standardized laboratory stressor and were offered an array of high- and low-fat sweet and salty foods. Those that reacted to the stressor with the most leptin ate the smallest amount of high-fat sweet foods. Although baseline levels of leptin did not predict eating post-stress in this study, a similar study found that individuals with higher daily leptin levels ate less food post-stress (Appelhans 2010).

However, an alternative perspective on leptin could be formulated given that individuals with obesity tend to be leptin resistant—that is, they actually have higher levels of leptin than lower-BMI individuals, but the leptin signal is not being heard due to fewer leptin receptors in the body (Mantzoros 1999). Stress could, therefore, beget obesity by increasing leptin repeatedly, contributing to leptin resistance and obesity over time. This theory is speculative and needs much more empirical support, but there is some evidence that stress increases leptin for all individuals (Brydon et al. 2008), rather than only for some, as in the two studies discussed above (Appelhans 2010, Tomiyama et al. 2012). For example, participants in a study by Brydon and colleagues (2008) underwent two laboratory stressors and, on average, leptin increased across the entire study sample from pre- to post-stress.

Compared to leptin, ghrelin represents a more straightforward path from stress to obesity. For example, Raspopow and colleagues (2010) randomly assigned individuals to either a modified Trier Social Stress Test (a gold-standard laboratory stress paradigm; see Kirschbaum et al. 1993) or control and found increases in ghrelin levels in the stress condition. Post-stress, participants were provided with food, which led to a decline in ghrelin in nonemotional eaters but not in emotional eaters. However, this study did not observe greater food intake related to ghrelin levels, and thus, any conclusions about ghrelin's role in stress and obesity are still preliminary.

In sum, the literature tying stress to obesity is messy in the case of leptin and sparse in the case of ghrelin. However, there is a preponderance of evidence that both are responsive to stress, and as both are such important appetite regulators, researchers should pursue further work in this area.

Neuropeptide Y. NPY is a peptide that stimulates hunger (Tatemoto et al. 1982). It also stimulates lipogenesis (Park et al. 2014), or the conversion of sugars like glucose into fat. The discussion of NPY requires two caveats. First, most of the evidence for the role of NPY in stress and obesity comes from nonhuman animal models. Second, although NPY is shown in **Figure 1** along with the other biochemical pathways, and although chronic stress directly stimulates NPY secretion in nonhuman animal models (Jeanrenaud & Rohner-Jeanrenaud 2000, Zhang et al. 2014), the role of NPY in the relationship between stress and obesity may be better characterized as that of a modulator. That is, NPY, in concert with chronic stress, promotes greater adiposity (Kuo et al. 2007).

Human evidence for the modulatory role of NPY is found in studies like that of Kim et al. (2016), who found that having any one of several NPY single-nucleotide polymorphisms interacted with stress to predict adiposity in almost 1,500 participants. Aschbacher and colleagues (2014) found that women with higher levels of chronic stress had more NPY, which, in turn, magnified the relationship between highly palatable food consumption and abdominal obesity. Overall, animal and human studies point to NPY's ability to augment the effect of stress on obesity, but this area requires much more research.

OBESITY TO STRESS

We live in a society that stigmatizes individuals of higher body weight. One common but often overlooked aspect of obesity is how stressful it is to experience such weight stigma. Defined as the collective prejudice, discrimination, and negative attitudes aimed at those perceived as having

too heavy a body (Tomiyama 2014), weight stigma is pervasive. Weight stigma has been documented in every corner of society, including media, employment, healthcare, and interpersonal and educational settings (Puhl & Heuer 2009).

Facing such ubiquitous stigma, heavier individuals are at risk for experiencing stress and its resulting cascade of negative consequences. Dickerson & Kemeny's (2004) social self-preservation model stipulates that, of the myriad stressors that we encounter throughout our lives, it is those that are social and evaluative in nature that most engage the HPA axis. Given our strongly antifat culture, weight stigma can be described by both of these characterizations. Thus, obesity can elicit feelings of social–evaluative threat and trigger HPA-mediated obesogenic processes (Tomiyama 2014). Another theoretical perspective is that of Hunger and colleagues (2015), who use the term weight-based social identity threat to capture the psychological experience of holding a socially devalued identity. This type of threat, they theorize, triggers emotional, physiological, and behavioral consequences, many of which cause weight gain (Hunger et al. 2015, Major et al. 2018).

Indeed, a now substantial body of evidence shows support for the stressful and obesogenic nature of weight stigma. Studies randomly assigning participants to experience weight stigma or not have found that weight stigma elicits stress and negative emotions (Major et al. 2012, Schvey et al. 2014). The weight stigma manipulations used were giving a speech about being a good dating partner, with the weight stigma group videotaped and the control group audiotaped (Major et al. 2012), and watching a video of stigmatizing television and movie clips (Schvey et al. 2014). Thus, empirical data support the theoretical assertion that weight stigma is stressful.

In terms of the model in **Figure 1**, weight stigma has also been shown to affect the different pathways leading from stress to obesity. In terms of cognitive processes, the study by Major et al. (2012) described above found that those in the weight stigma condition had lower executive function, as indexed by performance on the Stroop task. In a different study, Major and colleagues (2014) randomly assigned participants to read mock *New York Times* articles either titled "Lose Weight or Lose Your Job" in the stigma condition or "Quit Smoking or Lose Your Job" in the control condition. They found that experiencing weight stigma caused lower diet self-efficacy than was found for the controls (Major et al. 2014). Major et al.'s study, as well as a different study by Schvey et al. (2011) also using the stigmatizing video clip paradigm, found that participants randomly assigned to the weight stigma condition had higher calorie consumption compared to controls, particularly in those who were or perceived themselves to be overweight. Furthermore, survey studies find a link between weight stigma and binge eating (Durso et al. 2012, Neumark-Sztainer et al. 2002, Wott & Carels 2010), as well as counterproductive dieting behaviors (Haines 2006, Neumark-Sztainer et al. 2002). Lastly, there is survey evidence that links weight stigma to exercise avoidance or decreased physical activity (Vartanian & Shaprow 2008, Wott & Carels 2010).

In terms of physiological pathways, weight stigma has been shown to affect cortisol reactivity (Himmelstein et al. 2015, Schvey et al. 2014). In Himmelstein et al.'s (2015) study, participants in the weight stigma condition were excluded from a group shopping activity because they were not the "right size and shape" to fit into the clothes. Compared to controls, weight stigma participants had higher cortisol levels after exposure to the manipulation, particularly if they self-identified as being heavy. Interestingly, many of those who self-identified as heavy were not overweight or obese by BMI standards, indicating that weight stigma processes can affect even those who do not have objectively high BMI.

Whether weight stigma affects sleep duration remains to be seen. Moreover, with the exception of the HPA axis, researchers have not examined physiological and biochemical processes involved in weight stigma. These areas would be fruitful pursuits for future weight stigma science.

In addition to the social self-preservation model (Dickerson & Kemeny 2004) and weight-based social identity threat perspectives (Hunger et al. 2015) discussed above, another interesting theoretical aspect of weight stigma is that it need not even be consciously perceived to trigger obesogenic processes (Brewis 2011, Major et al. 2018). Through discrimination in employment and education, heavier individuals can be tracked into lower socioeconomic status (SES) positions. Weight discrimination has been documented at all parts of the employment cycle, from hiring to promotion to firing (Roehling et al. 2007), and heavier individuals, particularly women, are subject to discrimination in the education sphere. For example, parents provide less financial support for their higher- than for their lower-BMI daughters (Crandall 1995), and heavier women are less likely to go to or graduate from college (Cohen et al. 2013). Lower SES is linked to greater likelihood of obesity (Wang & Beydoun 2007), likely through multiple pathways. For example, lower-SES individuals and the neighborhoods in which they live have less access to healthy foods and more access to unhealthy foods (Carroll-Scott et al. 2013). Lower-SES neighborhoods also have fewer safe options for physical activity (Carroll-Scott et al. 2013). Finally, low SES is itself a stressful state (Cohen et al. 2006).

Given the extent of the evidence reviewed above, it is not surprising that studies find that weight stigma predicts future weight gain and likelihood of obesity. This has been replicated in multiple national longitudinal studies (Jackson et al. 2014, Sutin & Terracciano 2013) and in data from adolescents documenting the emergence of obesity, as well (Hunger & Tomiyama 2014).

Thus, the evidence shows that a vicious cycle may be at play, whereby stress begets obesity, which begets stress, and so on. This positive feedback loop may be one reason why obesity rates remain high in the United States (Hales et al. 2017). Moreover, the corpus of evidence supporting weight stigma's obesogenic effects has important implications for public health and public policy. Some prominent health policy scholars and medical ethicists have lobbied for increasing weight stigma to motivate individuals to lose weight (e.g., Callahan 2013). Based on the available data, it is clear that this would be a counterproductive policy direction.

CONCLUSIONS

Stress and obesity are connected through multiple interacting pathways that span cognition, behavior, physiology, and biochemistry. Research in this area is important because it targets two major challenges, stress and obesity, that are highly prevalent in society today. This review has described ten pathways. Which among these is the ripest for intervention?

Intervention efforts are already underway that target stress-induced eating (e.g., Katterman et al. 2014). For example, Daubenmeier and colleagues (2011) tested a 4-month mindfulness-based stress eating intervention and found that reductions in perceived stress and cortisol were associated with lower visceral obesity in the treatment group. However, stress-induced eating may be difficult to eliminate because food is accessible and eating is pleasurable. Indeed, in the Daubenmeier et al. study, the intervention itself did not appear to be effective, as there was no main effect of intervention on the primary endpoint of abdominal obesity.

Moreover, stress-induced eating may be difficult to eradicate because it seems to actually work—that is, engaging in stress-induced eating has been shown to reduce physiological stress at every step of the HPA axis and to reduce behavioral indications of stress in rodent models (Dallman et al. 2005). Some human data also indicate that stress-induced eating functions to dampen psychological (Finch & Tomiyama 2015) and physiological stress (Tomiyama et al. 2011, Tryon et al. 2013, van Strien et al. 2013). Thus, the limited efficacy of current stress-induced eating interventions, paired with the apparent importance of this behavior as a stress-dampening coping strategy, indicate that, perhaps, eating poses a challenging intervention target.

What, then, should be targeted? Although biomedical researchers are targeting physiological and biochemical pathways to eliminate obesity, it is difficult to imagine using pharmaceutical interventions to specifically cut short the negative effects of stress. That leaves physical activity and sleep, and both are amenable to intervention (Conn et al. 2011, Irwin et al. 2017).

However, if the model pictured in **Figure 1** is truly a causal model, then the most logical intervention target would be stress itself. Particularly because of the interrelated nature of the many pathways, targeting stress as the upstream causal factor would be efficacious and efficient. Doing so could also ameliorate the stress resulting from weight stigma, interfering with the feedback loop of stress to obesity to stigma to stress.

Intervening on stress seems like a straightforward conclusion based on the discussion provided in this article, but it is unconventional from a public health policy standpoint. Current public policy efforts to prevent and treat obesity focus mainly on decreasing energy intake and increasing energy expenditure (Segal et al. 2017). Of course, stress is not the only factor involved in obesity—many genetic and environmental causes contribute to it. However, combatting stress is currently not even part of the conversation for obesity prevention and treatment. Given the high prevalence of obesity and associated metabolic disease (Am. Heart Assoc. 2016), we are in need of new approaches. The many interacting pathways from stress to obesity make stress a promising target to prevent obesity and improve health.

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