SHORT COMMUNICATION

The relationship between stress and obesity

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ABSTRACT

Stress and obesity, two of society's most pervasive diseases, are linked by a variety of mechanisms. For starters, stress disrupts cognitive functions such as executive function and self-control. Second, stress can alter behaviour by causing overeating and the ingestion of high-calorie, high-fat, or high-sugar foods, as well as reducing physical activity and shortening sleep. Third, stress affects the hypothalamic-pituitary-adrenal axis, reward processing in the brain, and possibly the gut microbiota, causing physiological changes. Finally, biological hormones and peptides including leptin, ghrelin, and neuropeptide Y can be stimulated by stress. Due to the great prevalence of weight stigma, obesity can be a difficult condition in and of itself. As a result, this article explores the role of weight stigma in stress and obesogenic processes, culminating in a vicious cycle of stress to obesity to stigma to stress. Currently, obesity prevention efforts are mainly focused on diet and exercise; however, the research discussed in this article suggests that stress is an essential but currently underappreciated public policy priority.

Key Words: *Stress; Obesity; Eating, Physical activity; Cortisol; Reward; Stigmaa*

INTRODUCTION

We live in a society that is tremendously pressured. The American Psychological Association (APA) conducted national surveys that revealed that the majority of Americans experience moderate to high levels of stress. At the same time, more than a third of individuals in the United States are obese, defined as having a BMI of greater than 30. Could these occurrences be linked? Obesity has a variety of factors, including deep genetic roots, but may stress play a role? The conclusion of this review is that, yes, stress plays a role in the development and maintenance of obesity through many routes, based on the existing scientific data. It may seem far-fetched that stress—that vague psychological experience—could influence a person's body mass. Stress, on the other hand, makes sense from an evolutionary perspective because it affects metabolic processes. Our stress mechanisms evolved to aid humans in escaping life-threatening circumstances, which typically required a lot of metabolic work in the past. Our stress-response mechanisms are in charge of releasing glucose into the bloodstream so that our muscles have enough energy to flee (or fight) predators and other physical threats. In today's world, however, most of the stressors we face are psychological rather than physical (e.g., a dispute with a spouse, difficult times at work, financial worries). Regardless, our bodies react as though the stress is tangible. As a result, we find ourselves in a scenario where surplus energy has nowhere to go and is eventually released. Stress and metabolic processes are therefore intricately intertwined; in fact, epidemiological studies suggest a correlation between stress and BMI, and a meta-analysis of longitudinal studies discovered a positive relationship between stress and weight gain.

For the purposes of this article, stress is defined as "a negative emotional experience accompanied by predictable biochemical, physiological, cognitive, and behavioural changes that are directed either toward altering the stressful event or accommodating to its effects," according to Andrew Baum's comprehensive definition. Indeed, each of these sorts of modifications is associated with stress and obesity, though more commonly with the purpose of adjusting to the effects of stress rather than changing the stressful event itself. To put it another way, many of the pathways that lead from stress to obesity are attempts by an organism to overcome a challenge. Acute and chronic stresses are two different types of stress [1-2]. This is a chronic stress model because the major outcome is obesity, which is by definition something that accumulates over time. However, by persistently activating these pathways, repeated experiences of acute stresses can lead to obesity. The four types of alterations resulting from stress in Baum's characterization—cognition, behaviour, physiology, and biochemistry—are used to structure the pathways that connect stress to obesity in the model and this article. Importantly, the model's elements interact with one another. Self-control is required to manage food consumption, for example. Bidirectional linkages are also possible [3-4] .Physical exercise can be hampered by a lack of sleep, and sleep can be disrupted by a lack of physical activity. When empirical evidence for these interactions is available, it is highlighted, but the true state of affairs is likely to be one in which several of these elements interact with many others in an emergent process—something that is difficult, if not impossible, to capture in a single research. Obesity can be stressful in and of itself, which is an often ignored element of stress and obesity.

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There is a severe stigma against bigger bodies in most parts of the world. Weight stigma, which is defined as the totality of prejudice, discrimination, and unfavourable attitudes directed at persons who are thought to be overweight, is quite common. In some sectors, according to some studies, weight stigma is even more widespread than stigma based on other social characteristics, such as race and ethnicity or gender (e.g., interpersonal mistreatment). Because weight stigma can cause stress, this review also looks at the feedback loop that connects obesity to weight stigma and then back to stress. Self-regulation is necessary for managing one's own behaviour, and it's important in this situation because obesity-prevention habits like eating and physical activity demand it. Stress, on the other hand, can interfere with self-regulatory cognitive processes such as executive functioning and undermine self-regulatory cognitive processes. Evans and colleagues (2012) employed a delay of gratification test in which children were given the option of eating a medium-sized dish of candy right now or a large plate later [5]. Children who had been exposed to more cumulative life stressors were more likely to suffer, making them more vulnerable to emotional processes that might lead to unhealthy eating. These children, in turn, had higher BMI increases. After three years. Self-regulation failures can contribute to the harmful health behaviours outlined in the following sections. Stress, on the other hand, can increase these actions even if there is no stated self-regulation goal. "Milky Way: comfort in every bar," the slogan for Mars Inc.'s chocolate candy bar, reflects the phenomena in which people eat to relieve stress-related negative emotions. Under stress, people can eat more or eat differently, with the majority opting for pleasant foods heavy in sugar, fat, and calories. Stress-induced eating is common, with 39% of US people indicating that they either overeat or eat unhealthy meals in response to stress in an APA poll. Furthermore, humans are not the only creatures who eat when they are stressed. Rats exposed to various types of stress, for example, participate in stress-induced lard and sucrose consumption. In captive common marmosets, stress-induced feeding has also been seen. The fact that this behaviour is preserved across species suggests that it is likely beneficial to humans as well. To summarise, stress-induced eating is a fundamental process through which stress leads to obesity, whether that obesity presents itself as eating more or eating more unhealthily. Stress can cause activity patterns to be disrupted, either by reducing physical activity or increasing sedentary behaviour. In other words, people can voluntarily exercise less as a result of stress while independently spending more time inactive. The evidence for lower risk is stronger of the two. Higher stress was linked to less frequent exercise in a survey of over 12,000 people, while longitudinal research of nearly 1,400 women demonstrated 3-year prospective connections between higher felt stress and reduced leisure-time physical activity [6-7]. Higher stress levels were not linked to greater sedentary habits such as sitting or watching television in this study (or, in fact, higher palatable food intake). A thorough evaluation categorises 168 studies that looked at the link between stress and physical activity into the many types of stressors studied. The authors concluded that the majority of the 55 longitudinal studies they investigated indicated a link between increased stress and decreased physical activity. Stress is a well-known sleep disruptor. Obesity is linked to a lack of sleep, particularly shorter sleep duration. Shorter sleep duration was independently linked with and predictive of higher weight increased chance of obesity, and other adiposity indicators, according to a comprehensive evaluation of 31 cross-sectional and five longitudinal investigations [8]. A meta-analysis evaluating the connection between sleep length and BMI found that shorter sleep duration was associated with a 1.55 increased risk of obesity. In another way of looking at the statistics, for every hour more of sleep, the BMI decreased by 0.35.

Another review of the literature agreed with these general conclusions but noted that some studies found a U-shaped relationship between sleep duration and weight, with some studies finding a U-shaped relationship between sleep duration and weight, with some studies finding a U-shaped relationship between sleep duration and weight, with some studies finding a U-shaped relationship between sleep. Stress can affect the quantities of biological compounds linked to weight and obesity, in addition to activating physiological systems. The following sections go over leptin, ghrelin, and neuropeptide Y. Other appetite-stimulating peptides, such as Agouti-related peptide and proopiomelanocortin, have been linked to stress, but the body of research on these peptides is significantly smaller. We live in a society that stigmatises those who are overweight. One facet of obesity that is sometimes ignored is how difficult it is to be judged by one's weight. Defining prejudice, discrimination, and unfavourable sentiments directed at people who are thought to be overweight. The stigma associated with being overweight is widespread. Weight stigma has been recorded in all aspects of society, including the media, the workplace, healthcare, and interpersonal and educational contexts [9].

CONCLUSION

Stress-induced eating is already the subject of intervention initiatives. Daubenmeier and colleagues (2011), for example, investigated 4-month mindfulness-based stress eating intervention and discovered that decreases in perceived stress and cortisol were linked to lower visceral adiposity in the treatment group. However, because food is readily available and eating is pleasurable, stress-induced eating may be difficult to quit. Indeed, the intervention did not appear to be helpful in the Daubenmeier et al. study, as there was no major effect of the intervention on the primary endpoint of abdominal obesity. Furthermore, stress-induced eating may be difficult to eliminate because it appears to work—that is, it has been proven to reduce physiological stress at every level of the HPA axis as well as behavioural stress.

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