Acute Psychosocial Stress Effects on Food Consumption

Subjects: Physiology

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The most central physiological stress response is the activation of the hypothalamic-pituitary-adrenal (HPA) axis, which begins with the release of corticotrophin-releasing factor (CRF) from the hypothalamus. The physiological stress response can be dampened by eating, as the secretion of adrenocorticotropic hormone (ACTH) is reduced following consumption of food and the activation of the HPA axis is thereby attenuated.

Keywords: food pleasure ; food reward ; stress ; sensory science ; appetite ; eating ; diet ; pleasure scale

1. Introduction

The German sociologist Hartmut Rosa has described the paradox of how modern work life has been organized [1]. On one hand, modern work life seeks to accommodate higher levels of individual freedom, and on the other hand, it has been associated with increased levels of perceived stress, and health issues are registered as a result of working under these conditions [1][2][3][4]. Rosa describes this paradox as a result of a phenomenon that he has termed ‘social acceleration’ [1][2]. Social acceleration is a theoretical expression of how the pace of all parts of life constantly speed up, leaving people with a feeling of not being able to keep pace, feeling insufficient, and as a result, some may experience increased stress or burnout. Prolonged exposure to stressful conditions has been linked to a long and varied list of clinically defined diseases. These links are mediated by both the neuroendocrine systems, alterations of health behavior and affective regulation, which potentially can result in diseases such as depression, cardiovascular diseases, diabetes, infectious diseases and neurodegenerative diseases [5][6][7][8][9].

2. Physiological Mechanisms of the Endocrine System Affected by Stress

The most central physiological stress response is the activation of the hypothalamic-pituitary-adrenal (HPA) axis, which begins with the release of corticotrophin-releasing factor (CRF) from the hypothalamus [10][11][12][13][14][15][16][17]. CRF stimulates the release of adrenocorticotropic hormone (ACTH) from the anterior pituitary gland. ACTH then circulates through the bloodstream to the adrenal cortex, where it stimulates secretion of glucocorticoids, such as cortisol. Glucocorticoids (GC) can promote eating and weight gain in two ways. First, GC in rodent models has been shown to stimulate eating, especially highly palatable foods, on its own [14][18]. Secondly, GC stimulates insulin secretion, and the two hormones can then further act synergistically to promote food intake and visceral fat accumulation, as GCs functions to increase general food-associated drives, while insulin influences preferences for which specific types of food are consumed [14][16][19]. Additionally, cortisol will bind to GC receptors and thereby activate lipoprotein lipase, which may increase triglyceride accumulation in fat tissue, especially in the abdominal region. In the presence of insulin, cortisol will also concurrently inhibit the lipid-mobilizing system, which can also result in further fat accumulation. The HPA axis is thus not only the manager of the stress response but is also highly intertwined with the endocrine regulation of appetite, as the hypothalamus is a critical region for regulation of both food intake and energy balance and the stress circuit [10][11][14][16][20]. Furthermore, stress also activates the autonomic nervous system (ANS), which may result in heightened sympathetic-nervous-system activity and subsequent release of adrenaline and norepinephrine. It is especially these hormones that activate the allostatic stress response, and in synergy with the glucocorticoids starts a series of adaptive processes that can alter the structure and function of a variety of cells and tissues [8][21]. Furthermore, ongoing high levels of experienced stress can also result in overactivity of the sympathetic nervous system, which is related to insulin resistance [16]. The ANS may therefore in part be responsible for the higher prevalence of cardiovascular diseases, metabolic diseases and changes of the immune system that are seen among patients suffering from stress [8][9][21][22].

3. Stress-Induced Eating

There are two different, yet interacting, pathways of the physiological response to acute stress that can affect food intake. The first is the activation of the HPA axis, with subsequent stimulation of the secretion of glucocorticoids (including cortisol) as described above. The other pathway is that of the sympathetic nervous system, which leads to increases in arousal parameters such as secretion of adrenalin, elevated blood pressure and a diversion of blood flow from the gastrointestinal tract towards the skeletal muscles and brain [16][20]. This reaction is also known as the 'fight-or-flight' response and will most often lead to a decrease rather than an increase in food consumption. Nevertheless, research has shown that if the stressor is perceived as ego-threatening, i.e., as a threat to self-esteem or understanding of social self, cortisol will be released, and thereby stimulation of appetite and food intake will follow [20][23].

Multiple studies have shown that being stressed, both by acute stressful events as well as during longer periods due to interpersonal and work-related daily hassles, can cause non-homeostatic hunger, as food intake can dampen the physiological and behavioral stress responses [12][14][19][24]. The physiological stress response can be dampened by eating, as the secretion of ACTH is reduced following consumption of food and the activation of the HPA axis is thereby attenuated [16][18][19]. The relationship between stress and food intake can therefore be characterized as bidirectional, as stress and negative effects can alter eating behavior by increasing intake of especially highly palatable foods via activation of the HPA axis. Conversely, food consumption can alter mood by attenuating the stress response via the endocrine system and hedonic effects of the food. In particular, the hedonic experience of eating has been proposed to serve a special role in the effect of stress-induced eating, as eating can activate neural substrates, such as dopamine, similarly to drug abuse [5][16][20][25][26]. Dopamine is a neurotransmitter that codes for pleasure and enhances the desire for food, while it also functions to deactivate the HPA axis activity [16][27][28]. The pleasure one experiences from food may therefore be one of the main reasons for the comforting effect of food when feeling stressed. Thereby, eating as a means to dampen the physiological and behavioral stress response, the reward pathways are concurrently stimulated, which can potentially lead to neurobiological adaptations that promote the compulsive nature of overeating in a way that resembles drug abuse [20]. GCs are furthermore involved in the regulation of memory, specifically, those memories that are consolidated of emotionally arousing experiences, such as stress [18][19][29]. Thus, as stress promotes secretion of GCs, which may lead to intake of highly palatable foods, the GCs at the same time can facilitate an association between the indulgence of these 'comforting' foods and a subsequent positive effect on stress and mood [14][16][20][30].

4. Food Reward and Its Subcomponents

In addiction research, there is a sharp distinction between 'liking' and 'wanting' as two key components in food reward [25][26][31]. Liking, or consummatory pleasure, is linked to the hedonic reaction to a food, and can be detected by behavioral or neural signals in hedonic hotspots in the brain. Wanting, or incentive salience, on the other hand, relates to the motivation for a reward, typically triggered by a reward-related cue [25][26][32]. 'Wanting' is generated by large dopamine-related mesolimbic brain systems, and will ordinarily occur together with 'liking' and learning. However, as the 'liking' and 'wanting' systems are separate from each other, both mechanisms can also occur independently from the other [25][26][33][34][35]. A well-known mechanism from drug addiction is the case of experiencing compulsive levels of 'wanting' without 'liking', as a consequence of sensitization of the reward pathway [19][25][35]. It is hypothesized that this mechanism is what may be predominant in the case of food addiction also [26][27][33][34][36]. The link between the stress response, stress-related eating and behavioral changes thus seems to be closely related to the reward pathway, as well as changes in the mesolimbic system due to increased dopamine secretion or dopamine sensitization [16][26][35][37][38].

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