



Stress-induced eating and the relaxation response as a potential antidote: A review and hypothesis



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ABSTRACT

There is an accumulating body of evidence to indicate that stress leads to the consumption of unhealthy, energy-dense, palatable food, potentially contributing to the alarming global prevalence of chronic diseases, including obesity. However, comparatively little research has been devoted to addressing how best to remedy this growing problem. We provide an overview of the influence of stress on dietary intake, and then explore the novel, yet simple, possibility that regular elicitation of the relaxation response may effectively reduce stress-induced eating via both physiological neuroendocrine and reward pathways and psychological pathways involving emotion regulation, and habitual coping. If shown to be effective, the regular practice of relaxation may provide a convenient, cost efficient, patient-centered therapeutic practice to assist in the prevention of unhealthy weight gain and other negative consequences of unhealthy food intake.

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1. Introduction

Stress-induced eating is characterised by an increased intake of energy-dense, highly palatable food, when faced with psychological stress (Gibson, 2012; McEwen, 2008). Indeed, numerous studies over the last 20 years have shown that stress leads to a change in eating behavior (Block, He, Zaslavsky, Ding, & Ayanian, 2009; Born et al., 2009; Dallman, 2010; Epel, Lapidus, McEwen, & Brownell, 2001; Kandiah, Yake, Jones, & Meyer, 2006). As a result, research, has served to highlight the prevalence of this problem (Diggin, Woods-Giscombe, & Waters, 2015; Mouchacca, Abbott, & Ball, 2013), delineate the underlying physiological and psychological drivers (Merali, Graitson, Mackay, & Kent, 2013; Pool, Delplanque, Coppin, & Sander, 2015; Rower, Maria Teresa, Tonantzin, & Pattussi, 2017), as well as attempt to identify those individuals most vulnerable to stress-induced eating (Darling, Fahrenkamp, Wilson, Karazsia and Sato, 2017; Neseliler et al., 2017; Rodrigues et al., 2017). However, potential solutions remain elusive.

The purpose of this narrative review is to explore the proposal that regular elicitation of the relaxation response, the very opposite of the stress response, may alleviate stress-induced eating. We begin by presenting the premise of our argument; followed by (a) a brief overview of the research pertaining to stress-induced eating, (b) coverage of the possible physiological and psychological drivers of stress-induced eating, and (c) a discussion of how relaxation techniques may influence the drivers of stress-induced eating, thus providing a simple and feasible, yet novel solution to dealing with the issue.

2. The stress response versus the relaxation response

Stress is commonly defined as a physiological and psychological state in which the demands upon an individual are perceived as outweighing the resources available to contend with them (Lazarus & Folkman, 1984). A stressor may be of a physiological or psychological nature, or simply the anticipation of such (McEwen, 2008). The acute physiological response to the stressor or the ‘flight or fight’ response sees that energy stores are mobilized and cardiovascular efforts are aimed at the delivery of essential nutrients to areas of high priority (McEwen, 2005). While the primary objective of this acute stress response is to ensure survival of the organism, unnecessary and/or chronic elicitation of the stress response (known as chronic stress) can have deleterious effects on the body (McEwen, 2008).

The relaxation response is the parasympathetic physiological opposite of the stress response. First coined by Herbert Benson (Benson, Greenwood, & Klemchuk, 1975), the relaxation response consists of four basic components including: 1) A mental focus: a repetitive sound, words or visual stimulus such as a symbol by which to minimize distraction. 2) A non-judgmental attitude: to allow the recognition and passing of thoughts. 3) Decreased muscular tone: the posture to be held during the practice should be relaxed. 4) A quiet environment: often with the eyes closed (Benson et al., 1975). It is important to note that we do not refer to ‘relaxation’ as engaging in pleasant activities that are popularly thought of as relaxing, such as occasional hobbies, watching television, socializing, or even massage. Nor do we consider relaxation to refer to all forms of mind-body practices, such as yoga, tai chi and meditation, as it cannot be assumed that all of these practices unequivocally elicit the relaxation response. For instance, Lumma, Kok, and Singer (2015) found that styles of meditation requiring relatively greater cognitive effort (such as focus on thoughts, or on the cultivation of positive feelings) were less relaxing (both psychologically and physiologically) than a meditation focused on the breath. Furthermore, for those mind-body practices that do elicit

the relaxation response, it is unclear whether it is this specific component of the practice that provides benefit, or the holistic effects of such activities on both the body and the mind.

Regardless, it is well established that relaxation reduces general stress (for example, Chelley, Evans, Fornes-Vives, Pérez, & Garcia-Banda, 2015). Indeed, the earliest studies that drew attention to relaxation as a potential healing modality were prompted by ‘hypometabolic’ changes seen in transcendental meditators. Such changes, distinct from those seen in sleep, included a decrease in oxygen consumption, carbon dioxide production, respiratory rate, and alterations in brainwave activity (Wallace, Benson, & Wilson, 1971). Other studies have reported reduced levels of stress hormones (such as cortisol) and central nervous system arousal in response to relaxation (Chelley et al., 2015; Dolbier & Rush, 2012; Jacobs, 2001), reduced anxiety and depression (Manzoni et al., 2009), in addition to heightening positive affect (Jain et al., 2007; Unger, Busse, & Yim, 2017). The proposition that elicitation of the relaxation response may also attenuate stress-induced eating is discussed in the following sections.

3. The problem: stress-induced eating

The phenomenon of stress-induced eating has been previously reviewed (Adam & Epel, 2007; Fink, 2016; Maniam & Morris, 2012; Rabasa, Dickson, Rabasa, & Dickson, 2016; Torres & Nowson, 2007). Indeed, numerous studies have demonstrated that *food choice* is markedly affected by stress (Dallman, 2010; Roberts, 2014). More specifically, preference for high fat-high sugar foods has been repeatedly documented (Epel et al., 2001; Macht, 2008; Newman, O’Connor, & Conner, 2007; Rutters, Nieuwenhuizen, Lemmens, Born, & Westerp-plantenga, 2009). In parallel, reductions in the intake of nutritious mealtime foods such as vegetables during times of stress has been reported (Ledoux et al., 2012; Mikolajczyk, El Ansari, & Maxwell, 2009; O’Connor, Jones, Conner, McMillan, & Ferguson, 2008; Unusan, 2006). Stress, therefore, may foster dietary habits that are in conflict with healthy eating guidelines, likely predisposing individuals to increased risk of chronic diseases, particularly the cluster of abnormalities associated with the metabolic syndrome (Mendoza, Drewnowski, & Christakis, 2007; Mikolajczyk et al., 2009). In addition, given excess intake by as little as 50–100 kcal/d can result in weight gain of clinical concern in the long-term (Mozaffarian, Hao, Rimm, Willett, & Hu, 2011), stress may be an important driver of poor dietary habits leading to weight gain, potentially contributing to the worldwide epidemic of obesity we face today (Jauch-Chara & Oltmanns, 2014; Sinha & Jastreboff, 2013). Of equal relevance, research also highlights the role of stress in the development of diagnosed conditions of uncontrolled eating such as binge-eating disorder and bulimia (Hilbert, Vögele, Tuschen-Caffier, & Hartmann, 2011; Smyth et al., 2007; Sulkowski, Dempsey, & Dempsey, 2011). Notwithstanding these issues, it should be acknowledged that there is significant inter-individual variation in the precise effect of stress on *total* energy intake (Wallis & Hetherington, 2009; Yeomans & Coughlan, 2009). Admittedly, the dietary response to stress can be subject to a vast array of physiological and psychological factors, including perception of stressor type, length, intensity, and the impact of environment (Adam & Epel, 2007). It is not our intention, however, to provide an extensive summary of the literature relating to stress-induced eating here, but rather to highlight a potential solution to this issue.

4. The relaxation response – a potential antidote for stress-induced eating?

Stress has the potential to increase the intake of unhealthy

energy-dense foods, and relaxation is purported to be the physiological opposite to stress (Adam & Epel, 2007; Wallace et al., 1971). It is therefore reasonable to suggest that elicitation of the relaxation response may be protective in those susceptible to stress-induced eating. Yet, in stark contrast to the plethora of research devoted to stress-induced eating, the effect of the relaxation response on stress-induced eating has not been directly examined. Nonetheless, there is some evidence to suggest that relaxation can affect appetite. Pawlow, O'Neil, and Malcolm (2003), for example, reported reduced feelings of evening hunger after one week of daily home-based guided relaxation in the form of Abbreviated Progressive Muscular Relaxation (APMR) in individuals suffering from night-eating syndrome. Progressive Muscular Relaxation was also shown to reduce evening dietary intake amongst individuals with night-eating syndrome in a study by Vander Wal, Maraldo, Vercellone and Gagne, (2015). Meanwhile, others have observed reductions in emotional eating in obese emotional eaters compared with wait-list controls 3 months following a 3-week intervention period consisting of regular relaxation that incorporated PMR in conjunction with exposure to calming visual images (Manzoni et al., 2008, 2009). In another study of obese women, Christaki et al. (2013) compared an integrated stress reduction program consisting of dietary and stress management training (including PMR) with a control group that received dietary advice alone. Relaxation participants were required to maintain a twice daily home practice of relaxation for eight weeks. The eight week program resulted in greater weight loss in the relaxation group compared with the control group. The authors attributed the encouraging results to greater compliance with a dietary regime and higher restrained eating scores due to relaxation training, despite no change in perceived stress levels (Christaki et al., 2013). Also of relevance, based on the understanding that mindfulness practice may include some components of the relaxation response Benson et al. (1975), an emerging body of research supports a role for mindfulness in the treatment of disordered eating (Haynos, Forman, Butryn, & Lillis, 2016; Mason et al., 2016), and the intake of energy dense foods (Fisher, Lattimore, & Malinowski, 2016). For example, Jordan, Wang, Donatoni, and Meier (2014) demonstrated that a brief 15-min body scan led to 24% less energy intake in a sham taste testing of snack foods relative to a control group. However, as mentioned above, it is important to acknowledge that mindfulness and relaxation are not necessarily synonymous (Lumma et al., 2015).

Taken together, these findings suggest a potential role for relaxation in the regulation of food intake, although no research has specifically investigated whether relaxation can attenuate stress-induced eating. In this review, it is proposed that relaxation may provide a simple, cost-efficient, patient-centered approach to disrupt stress-induced eating at two critical points; 1) in ameliorating the stress response, itself, and/or 2) intervening at the stage at which the stress response leads to stress-induced eating. Given that stress may affect appetite through both physiological and psychological mechanisms, a discussion of how relaxation may play an equivalent opposing role in both respects follows.

5. Physiological mechanisms by which relaxation may attenuate stress-induced eating

Normal appetite (or the desire to eat) is determined by the integration of homeostatic (metabolic requirements of the body) and hedonic control (the body's drive for seeking reward and pleasure) (Begg & Woods, 2013). Research indicates that stress influences the impetus to eat; however, the precise mechanism by which homeostatic and hedonic control of appetite interact during stress is yet to be elucidated. It is evident, however, that a complex

interaction of appetite-related neuropeptides and stress hormones are involved, and relaxation may have the potential influence some of these.

5.1. Homeostatic determinants of appetite during stress: an interplay of appetite and stress hormones

5.1.1. Cortisol

The physiological stress response involves a coordinated neuroendocrinological cascade of events that involves a number of hormones that may influence subsequent eating. However, cortisol is most commonly implicated in stress-eating, with levels beginning to increase between 15 min and 60 min after the onset of an acute stressor (Sapolsky, Romero, & Munck, 2000). Experimental evidence for the stimulatory effect of cortisol on appetite has been found in humans, with peak cortisol release (in response to intravenously administered corticotropin-releasing hormone) significantly corresponding with increased ad libitum intake of snack foods in comparison with a placebo infusion (George, Khan, Briggs, & Abelson, 2010). Likewise, daily oral administration of 40 mg of cortisol over four days in healthy males resulted in a significant increase in total daily ad libitum energy intake, compared with a placebo group (Tataranni, Larson, Snitker, & Young, 1996). In relation to an acute stress-induced increase in cortisol, Epel et al. (2001) stratified participants according to a median division of their total cortisol release in response to a laboratory stressor. Those participants with a high cortisol response ate more energy dense foods compared with those participants exhibiting a low cortisol response to stress. In a separate study, participants exhibiting the highest cortisol response to a similar laboratory stressor were more likely to snack in response to daily life hassles compared with those individuals with a low cortisol response (Newman et al., 2007). In contrast, Appelhans, Pagoto, Peters, and Spring (2010) found that obese women with a higher cortisol response to a lab-induced stressor ate less, compared with a healthy weight control group in which dietary intake was unaltered by the magnitude of cortisol response. This inconsistency in past research may be related to variability in the individual sensitivity to stress, the ability to adapt, the acute versus chronic nature of the stressor, how this is reflected in the cortisol response and consequent dietary intake.

Although no studies have directly examined whether relaxation can reduce stress-induced eating, there is evidence in the literature that relaxation can attenuate the cortisol response to stress. Pawlow et al. (2003), found that a 20-min session of guided relaxation (APMR) administered before and after a week of daily home-based reduced salivary cortisol levels and subjective reports of stress and anxiety compared with a control group. Chellew et al. (2015) also reported reductions in cortisol levels following a series of five 45-min sessions of PMR held over a week. Similar findings are reported by others (Krajewski et al., 2011; Pawlow & Jones, 2005). However, the implications of these changes for eating behavior remain to be determined.

5.1.2. Insulin, ghrelin, and leptin

Other hormones that may play a role in mediating the relationship between stress and energy intake include insulin, ghrelin and leptin. Although insulin is considered an anorexigenic hormone (Könner, Klöckener, & Brüning, 2009), in tandem with high levels of cortisol, and the presence of energy dense palatable food, insulin may promote consumption, thus acting to palliate the chronic hypothalamic-pituitary axis (HPA) activation associated with stress (Dallman, 2010). This may occur at the cost of increased risk of abdominal obesity and associated metabolic imbalance (Dallman, 2010). In support of this, Epel et al. (2004) found that in a

group of 131 university students, self-reported stress-hyperphagics had higher cortisol and insulin profiles compared with their hypophagic counterparts during periods of high academic stress.

Meanwhile, ghrelin exerts its orexigenic effect through both homeostatic and hedonic pathways, and is known to rise during stress (Rouach et al., 2007). Rouach et al. (2007) undertook one of the first studies in humans to show that the commonly used laboratory stressor, the Trier Social Stress Test, increased the concentration of both cortisol and ghrelin. However, the rise in ghrelin seen by Rouach et al. (2007) was not strongly correlated with the self-reported compulsion to eat, although self-reported measures may not necessarily represent true behavior when actually in the physical presence of palatable food (Adams, Greenway, & Brantley, 2011). This stress-induced rise in ghrelin has been confirmed by others (Jaremka et al., 2014; Monteleone et al., 2012), while some studies have not reported significant alterations in ghrelin with stress (Macedo & Diez-Garcia, 2014; Raspopow, Abizaid, Matheson, & Anisman, 2010). Studies specifically addressing the response of leptin and energy intake to an acute stressor indicate great variation in leptin reactivity, with those individuals with a lower leptin response having greater subsequent food intake. For instance, Appelhans (2010) observed an inverse relationship between stress-induced intake and leptin levels following an acute mental stressor, independent of BMI. A later study by Tomiyama et al. (2012) specifically highlighted increased intake of high energy food being related to lower plasma leptin after exposure to an acute lab-stressor, also independent of BMI.

While there is evidence in the literature that relaxation can attenuate the response of cortisol to stress, no research to date has examined the effect of eliciting the relaxation response on the circulating concentrations of insulin, ghrelin, and leptin, which also appear to have a role in stress-induced eating. Future research is needed to address this issue.

5.2. Hedonic influences on appetite during stress

In addition to the above-mentioned pathways through which stress may influence dietary intake, it is acknowledged that the hedonic reward system may also play a significant role in determining dietary intake in response to stress (Yau & Potenza, 2013). For instance, a Brazilian study reported that 77% of women suffering from stress reported having sweet cravings (defined as “a strong desire to eat sweet foods over the last 3 months”), compared with only 31% in individuals assessed as not stressed (Macedo & Diez-Garcia, 2014). Others claim that acute stress can manifest in reduced sensitivity to the perception of sweetness (Al'absi, Nakajima, Hooker, Wittmers, & Cragin, 2012), while Luckett et al. (2015) showed that chronically stressed individuals found the look and taste of low-calorie chips less acceptable compared with less stressed individuals. This finding was in agreement with Born et al. (2009), who found that acutely stressed participants sought more richness of taste compared with their control counterparts. Thus, stress may alter the reward activation system such that increased dietary intake is necessary to obtain the usual reward (Born et al., 2009).

The manner in which reward, stress, and appetite interact is unclear, although the appetite-related hormones thus far discussed may play a role. For instance, the anorexigenic function of insulin may in part be achieved by reducing the rewarding value of food, as evidenced by insulin receptors in the limbic system (Davis, Choi, & Benoit, 2010). In regard to insulin's effect on reward pathways during stress, Jastreboff et al. (2013) found that insulin resistance in a group of obese women was positively correlated with activation in reward centers in the brain after exposure to personalized stressful scenarios and palatable food prompts. The authors

suggested that insulin sensitivity may play a significant role in motivating the intake of palatable food when stressed.

Similarly, researchers focusing on the hedonic influence of ghrelin postulate that eating palatable food may ameliorate the stress response via reward pathways that involve the neurotransmitters serotonin and dopamine (Malik, McGlone, Bedrossian, & Dagher, 2008). Dopamine acts to create the motivation (known as ‘incentive salience’) to obtain what is desired (such as highly palatable food) (Berridge & Robinson, 2003). Accordingly, during stress, dopamine release motivates the search for distraction and palatable food, or heightened alertness to unhealthy food cues (Morris, Beilharz, Maniam, Reichelt, & Westbrook, 2015). In support of this notion, intravenous injection of ghrelin in humans undergoing magnetic resonance imaging resulted in activity in brain regions associated with reward (Malik et al., 2008). More specifically, injection of ghrelin into reward areas has been shown to increase dopamine release in rodents, as well as increase subsequent dietary intake (Abizaid et al., 2006). Leptin, on the other hand, has been shown to reduce dopamine action, the hedonic appeal of food, and subsequent urges to eat in rodents (Hommel et al., 2006). However, Burghardt et al. (2012) found that leptin levels were positively associated with dopamine release in reward areas after exposure to a laboratory pain stressor in healthy men and women. The apparent inconsistency demonstrated in this study may reflect the specific changes seen under the influence of stress, and particularly, a physical stress. Alternatively, it may illustrate the diverse action of leptin on functionally distinct groups of dopamine neurons, yet to be identified (Opland, Leininger, & Myers, 2010).

Research related to stress-eating and reward is still in its infancy. However, evidence indicating that reward areas of the brain are also affected by relaxation (Jastreboff et al., 2011), suggests the intriguing notion that relaxation may provide a counteracting stimulus for dopamine, thus overriding the need to stress-induce eat. In other research, relaxation meditation (which may also induce the relaxation response) (Esch, Fricchione, & Stefano, 2003) has been shown to produce a 65% increase in dopamine release (a key player of reward) in the ventral tegmental area of the brain (Kjaer et al., 2002). Future research is needed to explore the possibility of whether relaxation-mediated increases in dopamine release can influence stress-induced eating.

5.3. Interrelationships between physiological mechanisms

In summary, each of the hormones discussed have both a metabolic and hedonic role. As to which component is dominant during times of stress remains unknown. Although research has identified apparent independent roles of cortisol, insulin, ghrelin and leptin (amongst a large number of other polypeptides mediating appetite), this oversimplifies the complexity of the stress-appetite system. It is probably more correct to state that each hormone is subject to the effect of the other components in the system, with varying sensitivity and responses depending on the individual and whether acute or chronic stress is at play. Stress-induced eating can thus be described as a neurobiological interplay of energy homeostasis and brain reward mechanisms falling prey to a maladapted stress response system in an environment that offers symptomatic relief by way of highly palatable, readily available processed food (Jauch-Chara & Oltmanns, 2014). If the stress response is the original impetus from which stress-induced eating results, intuitively, the effects of relaxation may offer a means by which to reduce stress, and the concomitant desire to stress-induce eat. As to how these physiological mechanisms manifest in behavior when stressed, or when relaxed, leads to a consideration of the psychological aspect of the stress response.

6. Psychological mechanisms by which relaxation may attenuate stress-induced eating

The psychological effects of stress may lead to cognitive, emotional, and behavioral consequences that may impact food choice, either in isolation or through interaction (Kandiah et al., 2006). They may be deliberate and conscious attempts to comfort oneself, or unconscious and driven more by habit for reduction of negative affect. Here, we review some of the findings from research on psychological factors associated with stress-induced food consumption and consider where relaxation may play a role in moderating these responses.

6.1. Stress, cognition and behavior

Consistent with the strength model of self-control (Muraven & Baumeister, 2000), coping in the face of stress, along with the required regulation of aversive thoughts, emotions, and behaviors, draws on one's finite ability to maintain self-control (Muraven & Baumeister, 2000). Thus, high stress impairs planned behavior and can lead to more automatic and unconscious actions to seek highly palatable food that is easy to access. Cognitive consequences of stress include an inability to focus, ruminative thinking (the dwelling on one's thoughts), and thinking the worst of a situation (Gianferante et al., 2014), all of which may compromise one's ability to make an informed decision regarding food choice (Dallman, 2010). In support of this notion, Kandiah et al. (2006) found that academic stress in female college students was associated with an increase in appetite coupled with less care for healthy dietary practices. While 80% of the participants believed they made healthy choices normally, only 33% did so during times of stress imposed by personal, environmental, and academic pressure. Likewise, Sims et al. (2008) found that perceived stress was associated with less ordered meal planning and eating in response to emotional cues in a group of African American men and women.

6.2. Stress and emotion regulation

Stress leads to a range of negative emotions, and eating may be used, both consciously and unconsciously, to down-regulate negative affect. Increased intake of energy-dense foods in response to negative mood states may be related, in part, to the presence of a negative feedback system between mood and food. For example, Macht and Mueller (2007) showed that consumption of palatable chocolate alleviated laboratory induced-negative mood compared with eating less palatable chocolate or eating nothing. The effect was seen instantly but lasted only a few minutes, thus potentially promoting overeating in order to prolong the desirable effect (Macht & Mueller, 2007). The consumption of foods that are perceived as personally enjoyable can be seen, therefore, as a means to avert the negative feelings associated with stress, giving rise to the term 'comfort foods' (Dallman, Pecoraro, & la Fleur, 2005). More recently, however, Wagner, Ahlstrom, Redden, Vickers, and Mann (2014) published findings contrary to those of Macht and Mueller (2007), serving to illustrate that mood is yet another variable further complicating our understanding of stress-induced eating.

While the remedial effect of palatable food consumption during stress is the most commonly stated cause of stress-eating, an interesting alternate hypothesis is presented by Pool et al. (2015). They suggest that stress may actually reduce the enjoyment associated with high energy food (thereby encouraging greater intake to attain the same pleasurable experience), promoting increased awareness of surrounding stimuli (palatable food or food cues), and increased motivation to access high energy foods. This behavior is

not motivated by wanting to reduce the initial stressor (as is the motivation purported by physiologists). Rather, stress may exhaust the ability of the individual to employ goal-driven behavior, leading one to succumb to habit-driven behavior, potentially leading to mindless eating (Neal, Wood, & Drolet, 2013; Pool et al., 2015).

6.3. Stress, coping style and drive to eat

A plausible unifying theory that may explain stress-induced eating, whether it be conscious planned behavior or an automatic response, is that proposed by Heatherton and Baumeister (1991). These researchers proposed that a stressful task can heighten one's awareness of his/her inadequacies or inability to cope. The resulting aversive state would then prompt the seeking out of an escape/avoidance, or relief from the external environment, in the form of palatable food intake. Thus, stress-induced eating according to this theory allows for a state of reduced self-awareness (Heatherton & Baumeister, 1991). Accordingly, the coping style (involving cognitive and subsequent behavioral responses to stress) of the individual may be an important moderator of the relationship between stress and unhealthy eating (Raspopow et al., 2010). Broadly categorized, individuals may utilize a coping response that involves a problem solving approach (aiming to manage the stressor), an emotion-focused approach (eating to regulate the emotional reaction to the stressor), or an avoidance focused approach (turning to food as a distraction or to seek distance from the stressor) (Raspopow et al., 2010). Expectancies related to reinforcement from eating may also constitute a mechanism for stress-induced eating. In other words, an individual's drive to eat in response to stress may be impelled by the expectation that such behavior will lead to escape, the ability to cope, provide comfort, or reward, and alleviate negative affect (Combs, Smith, & Simmons, 2011). In support of this mechanism, numerous studies have shown that expectancies for reinforcement from eating predicts disordered eating behavior (e.g., Fischer, Settles, Collins, Gunn, & Smith, 2012; Smith, Simmons, Flory, Anus, & Hill, 2007).

6.4. Characteristics of the stressor and individual stress appraisal

In addition to individual attributes that may influence the association between stress and eating, characteristics of the stress stimuli itself are an important consideration. Indeed, the type of stress (psychological versus physical, for instance) can produce varying effects on appetite with more psychological stressors leading to overeating in contrast with physical stressors (O'Connor et al., 2008). Furthermore, the nature of the psychological stressor and whether an ego-threat is involved may also be of relevance, with the latter tending to result in increased dietary intake versus those of traumatic origin leading to a reduction in energy intake (Jaremka et al., 2014). As previously discussed, the length of exposure to a stressor (acute versus chronic) is also an important factor leading to variation in the stress response (Dallman et al., 2003).

Derived from theories of stress appraisal, more recently, literature has emerged that questions the harmful effect of stress itself and posits that it may not be stress, per se, that leads to negative health consequences, but rather one's *perception* of the likely negative consequences (Crum, Salovey, and Achor, 2013; Lazarus & Folkman, 1984). This has been identified as one's 'stress mindset' which is one's perception of how harmful or beneficial stress is. Hence, re-appraising stress in a positive way – by focusing on the components of the stress response that allow an individual to survive in the wake of a challenge (increased arousal, sharpness, improved immunity, intellectual growth) may offset the potentially harmful effects of stress on the body by not only influencing

behavioral outcomes, but by also influencing hormonal effects leading to health and wellbeing (Crum et al., 2013). Whether this influences the relationship between stress and subsequent energy intake remains to be elucidated.

6.5. Effect of the relaxation response on psychological pathways for stress-induced eating

Amongst the limited collection of studies that have explored the influence of relaxation on appetite, the previously mentioned study by Manzoni et al. (2008) compared groups of obese emotional eaters receiving (a) PMR combined with calming visual imagery, (b) a relaxation recording with imagined calming scenarios, and (c) control (neutral) conditions. The relaxation sessions were administered nine times over three weeks and supplemented by a home-based relaxation practice. These researchers reported a significant reduction in resting heart rate (within single relaxation sessions), and reduced symptoms of anxiety and depression post intervention in both relaxation groups. A three month follow-up revealed that participants also reported significantly less emotion-induced eating in contrast with controls, together with an increase in self-efficacy in eating control (a measure of their coping ability when faced with a challenging situation) (Manzoni et al., 2009).

Further evidence of a beneficial effect of relaxation on self-efficacy for healthy eating comes from a study by Katzer et al. (2008), in which a ten-week relaxation intervention that included PMR, was followed by eight months of group-support for overweight/obese women and compared with non-relaxation controls. After 12 months, effect sizes for reduction in symptoms of depression, improvement in stress reduction, reporting of medical symptoms and self-efficacy for healthy eating were greatest for the relaxation group compared with the non-relaxation control group. A 24-month follow-up of the same cohort found that only the relaxation group participants (compared to non-relaxation controls) continued to maintain reduced levels of depression, reduced rate of suffering from general medical symptoms, together with improved self-efficacy for healthy eating (Hawley et al., 2008). Similarly, Christaki et al. (2013) provided evidence of the beneficial effect of the addition of PMR to a weight loss intervention, by promoting healthier eating, higher restrained eating, and resultant weight loss, though perceived stress remained unchanged (Mendoza et al., 2007). Together, these findings suggest that relaxation may improve behavioral and psychological resources such that individuals can better deal with a perceived stressor instead of resorting to food (Manzoni et al., 2009).

In support of the notion that relaxation may be of therapeutic benefit in stress-induced eating, studies that have examined the effect of mindfulness, which may have a component of the relaxation response (Benson et al., 1975), purport that the mechanism by which mindfulness may reduce stress is by purposefully and non-judgmentally paying attention to the present moment (Kabat-Zinn, 2013) by detaching from the source of stress. If the psychological effect of relaxation is similar, this creation of distance may lead to less reactive or habitual behavior in relation to food intake. Additionally, the cultivation of awareness of bodily sensations and cues can arise from a relaxation response, especially when practiced in the context of mindfulness training. The focus on sensations within the body cultivated by relaxation techniques may draw one's attention to interoceptive awareness and the distinction between metabolic hunger and hedonic hunger. Beyond this, mindful relaxation may influence how one acts on feelings of hunger, or cravings for palatable food. For instance mindfulness has been associated with decreased intake of palatable food (Mason et al., 2016). Further, as illustrated by Marchiori and Papies (2014), mindful body scanning led to reduced propensity to satisfy hunger

with energy dense cookies compared with 'non-mindful' controls. However, to what degree the relaxation component of mindfulness may influence stress-induced eating remains to be determined.

7. An integrative summary

Taken together, research thus far suggests a potential role for relaxation in the regulation of food intake, although no research has specifically investigated whether relaxation can attenuate stress-induced eating. Nonetheless, there are several mechanisms through which such an effect may operate. Given that stress may affect appetite through both physiological and psychological mechanisms, relaxation may play an equivalent opposing role in both respects. Physiologically, relaxation may reduce the activation of the HPA and the subsequent hormonal response resulting in a decrease in cortisol. Furthermore, the role of insulin, ghrelin, and leptin in stress-induced eating leads us to question whether these appetite-related peptides may be favorably influenced by relaxation and therefore reduce eating due to stress – an area yet to be investigated. In addition, relaxation may act as an alternative form of 'reward', displacing the neuropeptide-induced dopamine release that may promote hedonic overeating. From a psychological perspective, regular practice of the relaxation response may influence the appraisal of, and ability to cope with, a stressor, thereby weakening the cognitive component of the stress process that may lead to overeating, in addition to facilitating less impulsive eating typically promoted by stressful states.

8. Conclusion

Research indicates that stress is associated with the consumption of energy-dense palatable food driven by a complex interaction of physiology and psychology, fettered by our desire for reward. The relaxation response involves a mental focus, a non-judgmental attitude, and relaxed body, and thus is the physiological and psychological opposite of stress. This raises the question of whether it may play a role in attenuating stress-induced eating, by dampening the neuro-endocrinological response, and raising the awareness of one's psychological and bodily states. Future research is needed to investigate if the relaxation response alone can reduce stress eating, both in the laboratory and when practiced regularly in the long-term. Furthermore, given that it is commonly believed that the relaxation response may be achieved using an array of mind-body practices that are becoming more widely available, such as meditation, yoga, PMR and breathing exercises that train ability to focus, examination of which specific aspects of mind body practices, including components of the relaxation response, are effective at targeting stress-induced eating is needed. If shown to be effective, regular relaxation practice may provide a convenient, patient-centered, cost and time efficient intervention that could be implemented in a broad range of population groups to enhance the health and wellbeing of our community.

Conflicts of interest

None.

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Authors contribution

TM, JD, EE, and KG contributed to the conception, design,

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References

- Abizaid, A., Liu, Z.-W., Andrews, Z. B., Shanabrough, M., Borok, E., Elsworth, J. D., et al. (2006). Ghrelin modulates the activity and synaptic input organization of midbrain dopamine neurons while promoting appetite. *Journal of Clinical Investigation*, *116*, 3229–3239.
- Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. *Physiology & Behavior*, *91*, 449–458.
- Adams, C. E., Greenway, F. L., & Brantley, P. J. (2011). Lifestyle factors and ghrelin: Critical review and implications for weight loss maintenance. *Obesity Reviews*, *12*, e211–e218.
- Al'absi, M., Nakajima, M., Hooker, S., Wittmers, L., & Cragin, T. (2012). Exposure to acute stress is associated with attenuated sweet taste. *Psychophysiology*, *49*, 96–103.
- Appelhans, B. M. (2010). Circulating leptin moderates the effect of stress on snack intake independent of body mass. *Eating Behaviors*, *11*, 152–155.
- Appelhans, B. M., Pagoto, S. L., Peters, E. N., & Spring, B. J. (2010). HPA axis response to stress predicts short-term snack intake in obese women. *Appetite*, *54*, 217–220.
- Begg, D. P., & Woods, S. C. (2013). The endocrinology of food intake. *Nature Reviews Endocrinology*, *9*, 584.
- Benson, H., Greenwood, M. M., & Klemchuk, H. (1975). The relaxation response: Psychophysiological aspects and clinical applications. *The International Journal of Psychiatry in Medicine*, *6*, 87–98.
- Berridge, K. C., & Robinson, T. E. (2003). Parsing reward. *Trends in Neurosciences*, *26*, 507–513.
- Block, J. P., He, Y., Zaslavsky, A. M., Ding, L., & Ayanian, J. Z. (2009). Psychosocial stress and change in weight among US adults. *American Journal of Epidemiology*, *170*, 181–192.
- Born, J. M., Lemmens, S. G. T., Rutters, F., Nieuwenhuizen, A. G., Formisano, E., Goebel, R., et al. (2009). Acute stress and food-related reward activation in the brain during food choice during eating in the absence of hunger. *International Journal of Obesity*, *34*, 172.
- Burghardt, P., Love, T., Stohler, C., Hodgkinson, C., Shen, P.-H., Enoch, M.-A., et al. (2012). Leptin regulates dopamine responses to sustained stress in humans. *Journal of Neuroscience*, *32*, 15369–15376.
- Chellew, K., Evans, P., Fornes-Vives, J., Pérez, G., & Garcia-Banda, G. (2015). The effect of progressive muscle relaxation on daily cortisol secretion. *Stress*, *18*, 538–544.
- Christaki, E., Kokkinos, A., Costarelli, V., Alexopoulos, E. C., Chrousos, G. P., & Darviri, C. (2013). Stress management can facilitate weight loss in Greek overweight and obese women: A pilot study. *Journal of Human Nutrition & Dietetics*, *26*, 132–139.
- Combs, J. L., Smith, G. T., & Simmons, J. R. (2011). Distinctions between two expectancies in the prediction of maladaptive eating behavior. *Personality and Individual Differences*, *50*, 25–30.
- Crum, A. J., Salovey, P., & Achor, S. (2013). Rethinking stress: The role of mindsets in determining the stress response. *Journal of Personality and Social Psychology*, *104*, 716–733.
- Dallman, M. F. (2010). Stress-induced obesity and the emotional nervous system. *Trends in Endocrinology & Metabolism*, *21*, 159–165.
- Dallman, M. F., Pecoraro, N., Akana, S. F., la Fleur, S. E., Gomez, F., Houshyar, H., ... Manalo, S. (2003). Chronic stress and obesity: A new view of "comfort food". *Proceedings of the National Academy of Sciences of the United States of America*, *100*(20), 11696–11701. <http://doi.org/10.1073/pnas.1934666100>.
- Dallman, M. F., Pecoraro, N. C., & la Fleur, S. E. (2005). Chronic stress and comfort foods: Self-medication and abdominal obesity. *Brain, Behavior, and Immunity*, *19*, 275–280.
- Darling, K. E., Fahrenkamp, A. J., Wilson, S. M., Karaszia, B. T., & Sato, A. F. (2017). Does social support buffer the association between stress eating and weight gain during the transition to college? differences by gender. *Behavior Modification*, *41*, 368–381.
- Davis, J. F., Choi, D. L., & Benoit, S. C. (2010). Insulin, leptin and reward. *Trends in Endocrinology & Metabolism*, *21*, 68–74.
- Diggins, A., Woods-Giscombe, C., & Waters, S. (2015). The association of perceived stress, contextualized stress, and emotional eating with body mass index in college-aged Black women. *Eating Behaviors*, *19*, 188–192.
- Dolbier, C. L., & Rush, T. E. (2012). Efficacy of abbreviated progressive muscle relaxation in a high-stress college sample. *International Journal of Stress Management*, *19*, 48–68.
- Epel, E., Jimenez, S., Brownell, K., Stroud, L., Stoney, C., & Niaura, R. (2004). Are stress eaters at risk for the metabolic syndrome? *Annals of the New York Academy of Sciences*, *1032*, 208–210.
- Epel, E., Lapidus, R., McEwen, B., & Brownell, K. (2001). Stress may add bite to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology*, *26*, 37–49.
- Esch, T., Fricchione, G. L., & Stefano, G. B. (2003). The therapeutic use of the relaxation response in stress-related diseases. *Medical Science Monitor*, *9*, RA23–RA34.
- Fink, G. (2016). *Stress: Concepts, cognition, emotion, and behavior handbook of stress diet and stress: Interactions with emotions and behavior*.
- Fischer, S., Settles, R., Collins, B., Gunn, R., & Smith, G. T. (2012). The role of negative urgency and expectancies in problem drinking and disordered eating: Testing a model of comorbidity in pathological and at-risk samples. *Psychology of Addictive Behaviors*, *26*, 112–123.
- Fisher, N., Lattimore, P., & Malinowski, P. (2016). Attention with a mindful attitude attenuates subjective appetitive reactions and food intake following food-cue exposure. *Appetite*, *99*, 10–16.
- George, S. A., Khan, S., Briggs, H., & Abelson, J. L. (2010). CRH-stimulated cortisol release and food intake in healthy, non-obese adults. *Psychoneuroendocrinology*, *35*, 607–612.
- Gianferante, D., Thoma, M. V., Hanlin, L., Chen, X., Breines, J. G., Zoccola, P. M., et al. (2014). Post-stress rumination predicts HPA axis responses to repeated acute stress. *Psychoneuroendocrinology*, *49*, 244–252.
- Gibson, E. L. (2012). The psychobiology of comfort eating: Implications for neuropharmacological interventions. *Behavioural Pharmacology*, *23*, 442–460.
- Hawley, G., Horwath, C., Gray, A., Bradshaw, A., Katzer, L., Joyce, J., et al. (2008). Sustainability of health and lifestyle improvements following a non-dieting randomised trial in overweight women. *Preventive Medicine*, *47*, 593–599.
- Haynos, A. F., Forman, E. M., Butryn, M. L., & Lillis, J. (2016). *Mindfulness and acceptance for treating eating disorders and weight concerns evidence-based interventions*. Oakland: The Context Press Mindfulness and Acceptance Practica Series Context Press.
- Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin*, *110*, 86–108.
- Hilbert, A., Vögele, C., Tuschen-Caffier, B., & Hartmann, A. S. (2011). Psychophysiological responses to idiosyncratic stress in bulimia nervosa and binge eating disorder. *Physiology & Behavior*, *104*, 770–777.
- Hommel, J. D., Trinko, R., Sears, R. M., Georgescu, D., Liu, Z.-W., Gao, X.-B., et al. (2006). Leptin receptor signaling in midbrain dopamine neurons regulates feeding. *Neuron*, *51*, 801–810.
- Jacobs, G. D. (2001). The physiology of mind–body interactions: The stress response and the relaxation response. *The Journal of Alternative and Complementary Medicine*, *7*, 83–92.
- Jain, S., Shapiro, S., Swanick, S., Roesch, S., Mills, P., Bell, I., et al. (2007). A randomized controlled trial of mindfulness meditation versus relaxation training: Effects on distress, positive states of mind, rumination, and distraction. *Ann. Behav. Med.*, *33*, 11–21.
- Jaremka, L. M., Belury, M. A., Andridge, R. R., Malarkey, W. B., Glaser, R., Christian, L., et al. (2014). Interpersonal stressors predict ghrelin and leptin levels in women. *Psychoneuroendocrinology*, *48*, 178–188.
- Jastreboff, A. M., Potenza, M. N., Lacadie, C., Hong, K. A., Sherwin, R. S., & Sinha, R. (2011). Body mass index, metabolic factors, and striatal activation during stressful and neutral-relaxing States: An fMRI study. *Neuropsychopharmacology*, *36*, 627–637.
- Jastreboff, A. M., Sinha, R., Lacadie, C., Small, D. M., Sherwin, R. S., & Potenza, M. N. (2013). Neural correlates of stress- and food cue-induced food craving in obesity: Association with insulin levels. *Diabetes Care*, *36*, 394–402.
- Jauch-Chara, K., & Oltmanns, K. M. (2014). Obesity – a neuropsychological disease? Systematic review and neuropsychological model. *Progress in Neurobiology*, *114*, 84–101.
- Jordan, C. H., Wang, W., Donatoni, L., & Meier, B. P. (2014). Mindful eating: Trait and state mindfulness predict healthier eating behavior. *Personality and Individual Differences*, *68*, 107–111.
- Kabat-Zinn, J. (2013). *Full catastrophe living*. New York: Bantam Books.
- Kandiah, J., Yake, M., Jones, J., & Meyer, M. (2006). Stress influences appetite and comfort food preferences in college women. *Nutrition Science*, *26*, 118–123.
- Katzer, L., Bradshaw, A. J., Horwath, C. C., Gray, A. R., O'Brien, S., & Joyce, J. (2008). Evaluation of a "nondiets" stress reduction program for overweight women: A randomized trial. *American Journal of Health Promotion*, *22*, 264–274.
- Kjaer, T. W., Bertelsen, C., Piccini, P., Brooks, D., Alving, J., & Lou, H. C. (2002). Increased dopamine tone during meditation-induced change of consciousness. *Cognitive Brain Research*, *13*, 255–259.
- Köner, A. C., Klöckener, T., & Brüning, J. C. (2009). Control of energy homeostasis by insulin and leptin: Targeting the arcuate nucleus and beyond. *Physiology & Behavior*, *97*, 632–638.
- Krajewski, J., Sauerland, M., Wieland, R., Krajewski, J., Sauerland, M., & Wieland, R. (2011). Relaxation-induced cortisol changes within lunch breaks – an experimental longitudinal worksite field study. *Journal of Occupational and Organizational Psychology*, *84*, 382–394.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer Publishing Company.
- Ledoux, T. A., Mama, S. K., O'Connor, D. P., Adamus, H., Fraser, M. L., & Lee, R. E. (2012). Home availability and the impact of weekly stressful events are associated with fruit and vegetable intake among African American and Hispanic/Latina women. *Journal of Obesity*, *2012*. <http://dx.doi.org/10.1155/2012/737891>. Article ID 737891, 10 pages.
- Luckett, C. R., Oswald, C. G., Wilson, M. K. M., Pinto de Carvalho Alves, M., Sullivan, L. B., Ferreira Floriano, G., et al. (2015). Chronic stress decreases liking and satisfaction of low-calorie chips. *Food Research International*, *76*(Part 2), 277–282.
- Lumma, A.-L., Kok, B. E., & Singer, T. (2015). Is meditation always relaxing? Investigating heart rate, heart rate variability, experienced effort and likeability during training of three types of meditation. *International Journal of Psychophysiology*, *97*, 38–45.

- Macedo, D. M., & Diez-Garcia, R. W. (2014). Sweet craving and ghrelin and leptin levels in women during stress. *Appetite*, *80*, 264–270.
- Macht, M. (2008). How emotions affect eating: A five-way model. *Appetite*, *50*, 1–11.
- Macht, M., & Mueller, J. (2007). Immediate effects of chocolate on experimentally induced mood states. *Appetite*, *49*, 667–674.
- Malik, S., McGlone, F., Bedrossian, D., & Dagher, A. (2008). Ghrelin modulates brain activity in areas that control appetitive behavior. *Cell Metabolism*, *7*, 400–409.
- Maniam, J., & Morris, M. J. (2012). The link between stress and feeding behaviour. *Neuropharmacology*, *63*, 97–110.
- Manzoni, G. M., Gorini, A., Preziosa, A., Pagnini, F., Castelnuovo, G., Molinari, E., et al. (2008). New technologies and relaxation: An explorative study on obese patients with emotional eating. *Journal of Cybertherapy and Rehabilitation*, *1*, 182–193.
- Manzoni, G. M., Pagnini, F., Gorini, A., Preziosa, A., Castelnuovo, G., Molinari, E., et al. (2009). Can relaxation training reduce emotional eating in women with obesity? An exploratory study with 3 months of follow-up. *Journal of the American Dietetic Association*, *109*, 1427–1432.
- Marchiori, D., & Papias, E. K. (2014). A brief mindfulness intervention reduces unhealthy eating when hungry, but not the portion size effect. *Appetite*, *75*, 40–45.
- Mason, A. E., Epel, E. S., Aschbacher, K., Lustig, R. H., Acree, M., Kristeller, J., et al. (2016). Reduced reward-driven eating accounts for the impact of a mindfulness-based diet and exercise intervention on weight loss: Data from the SHINE randomized controlled trial. *Appetite*, *100*, 86–93.
- McEwen, B. S. (2005). Stressed or stressed out: What is the difference? *Journal of Psychiatry & Neuroscience*, *30*, 315–318.
- McEwen, B. S. (2008). Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology*, *583*, 174–185.
- Mendoza, J. A., Drewnowski, A., & Christakis, D. A. (2007). Dietary energy density is associated with obesity and the metabolic syndrome in U.S. adults. *Diabetes Care*, *30*, 974.
- Merali, Z., Graitson, S., Mackay, J., & Kent, P. (2013). Stress and eating: A dual role for bombesin-like peptides. *Frontiers in Neuroscience*, *7*.
- Mikolajczyk, R. T., El Ansari, W., & Maxwell, A. E. (2009). Food consumption frequency and perceived stress and depressive symptoms among students in three European countries. *Nutrition Journal*, *8*, 31.
- Monteleone, P., Tortorella, A., Scognamiglio, P., Serino, I., Monteleone, A. M., & Maj, M. (2012). The acute salivary ghrelin response to a psychosocial stress is enhanced in symptomatic patients with bulimia nervosa: A pilot study. *Neuropsychobiology*, *66*, 230–236.
- Morris, M. J., Beilharz, J. E., Maniam, J., Reichelt, A. C., & Westbrook, R. F. (2015). Why is obesity such a problem in the 21st century? The intersection of palatable food, cues and reward pathways, stress, and cognition. *Neuroscience & Biobehavioral Reviews*, *58*, 36–45.
- Mouchacca, J., Abbott, G. R., & Ball, K. (2013). Associations between psychological stress, eating, physical activity, sedentary behaviours and body weight among women: A longitudinal study. *BMC Public Health*, *13*, 1–11.
- Mozaffarian, D., Hao, T., Rimm, E. B., Willett, W. C., & Hu, F. B. (2011). Changes in diet and lifestyle and long-term weight gain in women and men. *The New England Journal of Medicine*, *364*, 2392–2404.
- Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin*, *126*, 247–259.
- Neal, D. T., Wood, W., & Drolet, A. (2013). How do people adhere to goals when willpower is low? The profits (and pitfalls) of strong habits. *Journal of Personality and Social Psychology*, *104*, 959–975.
- Neseliler, S., Tannenbaum, B., Zaccchia, M., Larcher, K., Coulter, K., Lamarche, M., et al. (2017). Academic stress and personality interact to increase the neural response to high-calorie food cues. *Appetite*, *116*, 306–314.
- Newman, E., O'Connor, D. B., & Conner, M. (2007). Daily hassles and eating behaviour: The role of cortisol reactivity status. *Psychoneuroendocrinology*, *32*, 125–132.
- O'Connor, D. B., Jones, F., Conner, M., McMillan, B., & Ferguson, E. (2008). Effects of daily hassles and eating style on eating behavior. *Health Psychology*, *27*, S20–S31.
- Opland, D. M., Leininger, G. M., & Myers, J. M. G. (2010). Modulation of the mesolimbic dopamine system by leptin. *Brain Research*, *1350*, 65–70.
- Pawlow, L., & Jones, G. (2005). The impact of abbreviated progressive muscle relaxation on salivary cortisol and salivary immunoglobulin A (sIgA). *Applied Psychophysiology and Biofeedback*, *30*, 375–387.
- Pawlow, L. A., O'Neil, P. M., & Malcolm, R. J. (2003). Night eating syndrome: Effects of brief relaxation training on stress, mood, hunger, and eating patterns. *International Journal of Obesity*, *27*, 970–978.
- Pool, E., Delplanque, S., Coppin, G., & Sander, D. (2015). Is comfort food really comforting? Mechanisms underlying stress-induced eating. *Food Research International*, *76*(Part 2), 207–215.
- Rabasa, C., Dickson, S. L., Rabasa, C., & Dickson, S. L. (2016). Impact of stress on metabolism and energy balance. *Current Opinion in Behavioral Sciences*, *9*, 71–77.
- Raspopow, K., Abizaid, A., Matheson, K., & Anisman, H. (2010). Psychosocial stressor effects on cortisol and ghrelin in emotional and non-emotional eaters: Influence of anger and shame. *Hormones and Behavior*, *58*, 677–684.
- Roberts, C. J. C. I. C. T. N. (2014). Increases in weight during chronic stress are partially associated with a switch in food choice towards increased carbohydrate and saturated fat intake. *European Eating Disorders Review*, *22*, 77–82.
- Rodrigues, D. M., Reis, R. S., Dalle Molle, R., Machado, T. D., Mucellini, A. B., Bortoluzzi, A., et al. (2017). Decreased comfort food intake and allostatic load in adolescents carrying the A3669G variant of the glucocorticoid receptor gene. *Appetite*, *116*, 21–28.
- Rouach, V., Bloch, M., Rosenberg, N., Gilad, S., Limor, R., Stern, N., et al. (2007). The acute ghrelin response to a psychological stress challenge does not predict the post-stress urge to eat. *Psychoneuroendocrinology*, *32*, 693–702.
- Rower, H. B., Maria Teresa, A. O., Tonantzin, R. G., & Pattussi, M. P. (2017). The role of emotional states in fruit and vegetable consumption in Brazilian adults. *Ciência & Saúde Coletiva*, *22*, 489–498.
- Rutters, F., Nieuwenhuizen, A. G., Lemmens, S. G. T., Born, J. M., & Westerterp-plantenga, M. S. (2009). Acute stress-related changes in eating in the absence of hunger. *Obesity*, *17*, 72–77.
- Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*, *21*, 55.
- Sims, R., Gordon, S., Garcia, W., Clark, E., Monye, D., Callender, C., et al. (2008). Perceived stress and eating behaviors in a community-based sample of African Americans. *Eating Behaviors*, *9*, 137–142.
- Sinha, R., & Jastreboff, A. M. (2013). Stress as a common risk factor for obesity and addiction. *Biological Psychiatry*, *73*, 827–835.
- Smith, G. T., Simmons, J. R., Flory, K., Annun, A. M., & Hill, K. K. (2007). Thinness and eating expectancies predict subsequent binge-eating and purging behavior among adolescent girls. *Journal of Abnormal Psychology*, *116*, 188–197.
- Smyth, J. M., Wonderlich, S. A., Heron, K. E., Sliwinski, M. J., Crosby, R. D., Mitchell, J. E., et al. (2007). Daily and momentary mood and stress are associated with binge eating and vomiting in bulimia nervosa patients in the natural environment. *Journal of Consulting and Clinical Psychology*, *75*, 629–638.
- Sulkowski, M. L., Dempsey, J., & Dempsey, A. G. (2011). Effects of stress and coping on binge eating in female college students. *Eating Behaviors*, *12*, 188–191.
- Tataranni, P., Larson, D., Snitker, S., & Young, J. (1996). Effects of glucocorticoids on energy metabolism and food intake in humans. *American Journal of Physiology*, *34*, E317.
- Tomiya, A. J., Schamarek, I., Lustig, R. H., Kirschbaum, C., Puterman, E., Havel, P. J., et al. (2012). Leptin concentrations in response to acute stress predict subsequent intake of comfort foods. *Physiology & Behavior*, *107*, 34.
- Torres, S. J., & Nowson, C. A. (2007). Relationship between stress, eating behavior, and obesity. *Nutrition*, *23*, 887–894.
- Unger, C. A., Busse, D., & Yim, I. S. (2017). The effect of guided relaxation on cortisol and affect: Stress reactivity as a moderator. *Journal of Health Psychology*, *22*, 29–38.
- Unusan, N. (2006). Linkage between stress and fruit and vegetable intake among university students: An empirical analysis on Turkish students. *Nutrition Research*, *26*, 385–390.
- Vander Wal, J. S., Maraldo, T. M., Vercellone, A. C., & Gagne, D. A. (2015). Education, progressive muscle relaxation therapy, and exercise for the treatment of night eating syndrome. A pilot study. *Appetite*, *89*, 136–144.
- Wagner, H. S., Ahlstrom, B., Redden, J. P., Vickers, Z., & Mann, T. (2014). The myth of comfort food. *Health Psychology*, *33*, 1552–1557.
- Wallace, R. K., Benson, H., & Wilson, A. F. (1971). A wakeful hypometabolic physiologic state. *The American Journal of Physiology*, *221*, 795.
- Wallis, D. J., & Hetherington, M. M. (2009). Emotions and eating. Self-reported and experimentally induced changes in food intake under stress. *Appetite*, *52*, 355–362.
- Yau, Y. H. C., & Potenza, M. N. (2013). Stress and eating behaviors. *Minerva endocrinologica*, *38*, 255–267.
- Yeomans, M. R., & Coughlan, E. (2009). Mood-induced eating. Interactive effects of restraint and tendency to overeat. *Appetite*, *52*, 290–298.