EATING BEHAVIOR IN RESPONSE TO ACUTE STRESS

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EATING BEHAVIOR IN RESPONSE TO ACUTE STRESS (Abstract): Obesity is a medical and social problem with a dramatically increasing prevalence. It is important to take action since childhood to prevent and treat obesity and metabolic syndrome. Infantile obesity affects all body systems starting in childhood and continuing to adulthood. Understanding the impact of stressors on weight status may be especially important for preventing obesity. The relationship between stress, eating behavior and obesity is not fully understood. However, there is evidence that stress causes disorders in hypothalamic-pituitary-adrenal (HPA) axis, system that regulates both stress and feeding responses. Also, the response is different depending on the type of stressors. Chronic stress, especially when people live in a palatable food environment, induces HPA stimulation, excess glucocorticoids, insulin resistance, which lead to inhibition of lipid mobilization, accumulation of triglyceride and retention of abdominal fat. Keywords: EATING BEHAVIOR, FOOD INTAKE, ACUTE STRESS, CORTISOL, OBESITY.

In modern society, stress is everywhere and affects people of different ages, including children. It is difficult to define stress because it is so different for each of us. We can say that it is the generalized, non-specific response of the body to any demand for change. Stress can trigger many diseases, including obesity and other eating disorders (1).

Obesity is a heterogeneous disease that is difficult to treat (2,3). It is associated with risk factors for cardiovascular diseases and accelerated atherosclerotic processes, including hypertension, atherogenic dyslipidemia, atherosclerosis and structural and functional cardiac changes (1). The association of stress with obesity was suggested by Cushing’s syndrome. Patients with this syndrome have upper body obesity, high levels of cortisol, atherosclerosis, glucose intolerance and hypertension (3).

Stress can be acute, short term or chronic, long-term. Stressors can be external (such as cold exposure, hunger, social stressors) or of internal cause (sleep deprivation) (4). When stress occurs, mechanisms are activated to maintain homeostasis. Homeostasis is the property of a system in which the variables are regulated so that internal conditions to remain stable and relatively constant. Homeostasis implies remaining stable by being the same, while allostasis is achieving stability through change. Allostasis is associated with
changes in food choice, appetite, energy storage and mobilization (4). This implies alteration in HPA axis hormones, autonomic nervous system, cytokines or a number of other systems. There is a close interaction between stress, allostatic, energy balance and eating patterns. Stress can influence human eating behavior in a bidirectional way (4).

**Stress response.** Allostasis is maintained in stress conditions by adaptive reactions originating in the central nervous system and in the periphery. Acute stress induces a fight-or-flight reaction with behavioral, autonomic and endocrinological changes. Heart rate and blood pressure are increased and energy spent on digestion and reproduction is minimized. Energy is saved for vital organs: brain, heart and muscle tissue to save life. So, in acute stress appetite and food intake are suppressed. However, there are situations of weight gain under stress. What determines direction of eating is not explained yet but HPA axis has an important role in both situation (4).

Stress response has two major components: the HPA axis and the autonomic nervous system (ANS) which encompasses the sympathetic and parasympathetic nervous system (fig. 1). Stress response depends on intensity, duration and type of stressor (5).

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**Fig. 1.** Stress response (modified after Torres et al.) with two major components: SAM and HPA axis

There are two antithetical responses to stress according to the stressor type:

- **Challenge stressor**, a demanding but controllable situation that activates the sympathetic-adrenomedullary (SAM) system, with fight/flight response, which shuts...
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For this type of stressor, the body has the resources to cope with.

- **Threat stressor**, for which the body does not have the resources to cope with or social stress (public embarrassment or failure, feeling defeated) activates HPA axis with cortisol release. Neurons of the paraventricular nucleus of the hypothalamus initiate stress response by corticotropin-releasing hormone (CRH) which modulates HPA axis. CRH stimulates the secretion of ACTH from the anterior pituitary. ACTH acts on the zona fasciculata of the adrenal cortex increasing cortisol secretion. Circulating cortisol has a negative feedback to the brain, limiting cortisol secretion, protecting the organism from prolonged cortisol exposure in chronic stress (6). It is known that cortisol stimulates hunger and feeding and visceral fat deposition. Glucocorticoids increase insulin secretion but has influence on orexigenic neuropeptide (NPY). Immune cells or adipocytes produces cytokines that may stimulate the HPA axis.

**Eating behavior as a consequence of body response to acute stress.**

To study the relationship between stress and eating behavior it is important to take into account the different types of stress. Stress can be acute (short term stressors) or chronic. Stressors can be physical (extreme temperatures, elevated sound levels, overillumination, surgery, trauma), physiological (pain, hemorrhagic shock, intense exercise), chemical (acid-base imbalance, hypoxia), psychological or emotional (anxiety, fear, sorrow) and social (low social status, giving a speech, meeting new people, discrimination) (3).

**Animal research.** In animal studies all variables (type and intensity of the stressors, food offered) can be well controlled. Acute stress can be induced using stressors such as cold exposure, tail pinch, hunger, physical restraint, inescapable foot shock, exposure to a socially dominant member of the same species (6,7). Rats can be used for studying the effects of acute stress in a controlled laboratory setting with accurate evaluation of food intake. Stressors of different intensity can be used (6,7). Also, another factor is the type of food offered: commercial food for rodents (rat chow) or highly palatable food (sweetened milk). In rats, stress can lead in some cases to increases but mainly to decreases in food intake (6,7). The severity of stress is also very important. A severe stress is associated with a decrease in food intake. Moderate and mild stressors also lead to a decrease in food intake but increase the ingestion of highly palatable food (5,6,7).

According to the intensity and type of stressors the effect was either a decrease in food intake in rats exposed to acute physical stress (ex. pinch tail), or increased food intake after exposure to social stress (exposure to a socially dominant member of the same species, resident-intruder paradigm or social defeat), especially when offered a calorically dense diet (6). The last situation reproduces the modern society with numerous stressors, especially social stressors, and exposure to energy dense, highly palatable food.

Most studies highlight that stress reduces food intake in rodents, unless palatable food is given during the stress period. Also, administration of glucocorticoids increases food intake. Adrenalectomy reduces food intake, while subsequent corticosterone replacement normalizes it (3).

**Human studies.** Human studies confirm ingestion of palatable foods in response to stressful situations. Association between stress, comfort food and obesity is
In humans it is difficult to differentiate the emotions because it could be intricate (6). Human studies show that stress decreases food intake in 30% of cases while most of subjects, 70%, increase their food intake with preference for palatable food (7,8). In modern society, a palatable food environment, it is more likely for people to increase food intake in stressful periods. Also, they will choose high calorie foods to cope with stress.

Stress reactivity has been studied in people with eating disorders (bulimia, anorexia and binge eating disorder - BED). Subjects with BED had either greater basal cortisol or greater cortisol reactivity (6,9).

Stress can be induced in laboratory conditions with the Trier Social Stress Test (TSST). After inducing stress with TSST subjects with high cortisol levels were likely to eat more calories, especially calorically dense food. On the control day there were no difference between high and low cortisol reactor groups (6). Another study compared a similar threat stressor to a positive challenge stressor (identical tasks but with positive feedback from the audience). The “threat” condition stimulated greater food intake, particularly of highly palatable food than the “challenge” condition (10,11). The relationship between cortisol and food intake in humans may also involve the effects of glucocorticoids on Neuropeptide Y (NPY), CRH, leptin as well as opioid and endocannabinoid signaling (12,13). Through multiple mechanisms, excess glucocorticoids determine insulin resistance. Even though cortisol causes hyperinsulinemia, this high insulin level does not affect plasma glucose concentration because of insulin resistance.

Stressors stimulate HPA axis and interfere with the reward system. Under stress conditions the mesolimbic dopaminergic system is activated and increased CRF secretion is stimulated. Food and drugs of abuse can stimulate similar pathways in the nervous system, including the dopaminergic and opioidergic systems. As well as drugs, hyper palatable foods alter CRF, GC and noradrenergic activity and increase sensitization of reward pathways which influence preference for addictive substance and hyper palatable foods and increases craving and intake. Repeated stimulation of the dopaminergic reward pathways may trigger neurobiological adaptations that may promote progressively compulsive behavior (4, 16).

Repeated stimulation of the reward pathways through highly palatable food may lead to neurobiological adaptations that determine overeating characterized by frequent drive to initiate eating. Opioid release increases palatable food intake and palatable food sustains opioid release. When stress becomes chronic, highly palatable food may appear to be “addictive” because the body is using food for coping with stress (4, 13, 14).

CONCLUSIONS

Our analysis of the literature studies revealed that the impact of stress depends on the intensity and type of stressors: acute or chronic. It involves multiple pathways, molecules and receptors. An important role is played by HPA axis which interferes with the reward systems. Repeated stimulation of the reward pathways through highly palatable food leads to overeating because of the neurobiological adaptations. Also, palatable food can lead to opioid release which in its turn increases the palatable food intake. Palatable foods can activate the brain reward system, so repeated stimu-
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lation of the reward pathways through highly palatable food may lead to overeating with frequent drive to initiate eating. Moreover, the exogenous factors (nutritional) have a pro-obesity action by increasing the caloric intake determining energy imbalance. Additional studies are needed to understand the complex relationship between stress, alterations in HPA activity and obesity because in humans it is difficult to differentiate the emotions because it could be intricate.

REFERENCES