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Chewing after stress: Psychosocial stress influences chewing frequency, chewing efficacy, and appetite



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KEYWORDS

Hypothalamicpituitaryadrenocortical axis; Cortisol; Chewing frequency; Eating preference; Appetite

Summary

Background: Psychosocial stress is accompanied by an increase in the activity of the hypothalamic-pituitary-adrenocortical (HPA)-axis and by an increase in food intake. At present, no studies have been conducted to examine the impact of a potent laboratory stress test on the chewing frequency.

Methods: Thirty-one healthy participants (14 females, mean age 27.13) were compared after they had fulfilled the protocol of a standardized psychosocial stress test, the Trier Social Stress Test (TSST), and after a resting condition of silent reading in reference to their chewing frequency, chewing efficacy, food intake, and eating preferences. As part of the design free salivary cortisol levels and heart rate variability were measured repeatedly before and after the TSST and the resting condition.

Results: After the TSST, the participants exhibited a significantly higher mean chewing frequency than after the resting condition (F(2,60) = 3.600, p = .035, $\eta^2 = .107$). The testing condition had no influence on the amount of food intake. Following the psychosocial stress, however, the participants reported a significantly less general appetite (Z = -3.921, p < .001) and less of an appetite for eggs (Z = -2.023, p = .043) than after their resting condition. No correlation was found between the salivary cortisol response and the chewing frequency.

Conclusion: The results indicated that psychosocial stress is associated with an increase in chewing frequency, as measured with a sound-based apparatus, and with a decrease in appetite. © 2014 Elsevier Ltd. All rights reserved.

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1. Introduction

Based on the selfish brain theory, the brain occupies an important hierarchical position in the energy metabolism and behaves in a "selfish" manner by competing for energy resources with the body (Peters et al., 2004, 2011). The logistic supply chain (Slack et al., 2004) implies that information about the energy homeostasis of the body is transported to the brain by different mechanisms (brainpull-mechanisms; Peters et al., 2011) and hormones such as insulin, leptin, or cortisol (see for further details Peters et al., 2011). These hormones influence the expression of the neuropeptides in the hypothalamus (Burdakov et al., 2006; Gonzales et al., 2008). Stress leads to an activation of the hypothalamus-pituitary-adrenocortical (HPA) system and to a cortisol release (Dickerson and Kemeny, 2004), which changes the energy homeostasis in the brain (Peters et al., 2011). Both the selfish-brain-theory and the empirical evidence suggest that psychosocial stress and the stressinduced cortisol secretion may be responsible for a change in food intake (Stone and Brownell, 1994; Epel et al., 2001; George et al., 2010; Peters et al., 2011; Schulz and Laessle, 2012). Because emotional arousal and negative emotions triggered by stress are associated with an increase and also a decrease in food intake (Willenbring et al., 1986; Stone and Brownell, 1994), the mechanism and the predictors of stress-stimulated food intake are still under investigation.

First research concerning the effect of cortisol on food intake showed that the administration of cortisol to healthy individuals for 4 days leads to a significant increase in their food intake (Tataranni et al., 1996). Stress-induced cortisol secretion also played a role in food intake of healthy individuals (Epel et al., 2001). The amount of food intake and the increase in the cortisol level using a modified version of the Trier Social Stress Test (TSST) were analyzed. High cortisol reactors to stress consumed more food, especially sweets, in comparison to low reactors. On the resting day, the high reactors tended to eat less than the low reactors (Epel et al., 2001). The authors concluded that stress-induced cortisol secretion was perhaps a reason for the amount of food intake under stress. Nevertheless, in the above study, the effect of social-evaluative threat was attenuated as the evaluation committee remained behind a one-way mirror and was not present in the room (Epel et al., 2001; Dickerson and Kemeny, 2004).

Recent studies have shown that not only the cortisol level and the amount of food intake need to be evaluated but also the chewing frequency. In a chewing experiment without stress induction, the salivary cortisol levels could be reduced significantly by chewing (Tahara et al., 2007). In addition, the biofeedback of the chewing frequency/bite rate led to a decrease in food intake (Scisco et al., 2011). In this field of research, the methodological challenges lay in measuring chewing frequency and controlling meal size. Some researchers counted the bite rate manually while observing the participants through a one-way mirror (Gaul et al., 1975). The physical presence of an observer might have influenced eating and chewing behavior and manual counting may cause counting errors. Others used the electromyography of the mastication muscles or the kinematics of the lower jaw movements (sirognathography) (Woda et al., 2006) where the sensors limit mobility and sweat influences signal quality.

In the present study a newly-developed small sensor was implemented in order to specify the eating behavior in more detail (Päßler et al., 2012). This sensor enabled to record the sound sequences of food intake during meals and to specify the mastication frequency based on the chewing sounds. This sensor, a highly sensitive apparatus and standardized method, enables to record the sound sequences of food intake and to specify chewing frequency based on chewing sounds without an observer bias.

In order to understand the precise mechanism of stress on eating behavior, chewing frequency and food intake 31 participants were assessed under the influence of a standardized psychosocial stress test. The social evaluation and the lack of feedback served as components of stress induction using the original standardized Trier Social Stress Test (TSST; Kirschbaum et al., 1993). In this design salivary cortisol secretion, chewing frequency, chewing efficacy, heart rate variability and the type and amount of food intake were measured.

Previous results had proved that increased salivary cortisol levels after stress led to more food intake, especially of sweets (Epel et al., 2001). We postulated that individuals would eat more following the standardized TSST than following the resting condition. Since chewing contributes toward a decrease in stress perceptions and in cortisol concentration (Tahara et al., 2007), a high cortisol level, perhaps, may lead to an increased chewing frequency which in turn reduced stress and cortisol concentration. To conduct a study with this sensor our hypothesis is that using a standardized stressor would make it possible to determine if cortisol reactivity is a marker for an individual's vulnerability to stress-induced eating. The sensor would also allow us to identify who would eat more or less following psychosocial stress when controlling chewing frequency.

2. Materials and methods

2.1. Study participants

The study participants were recruited from January 2012 to September 2012 at the Technische Universität Dresden, Germany. The participants were healthy volunteers ranging from 18 to 65 years. A telephone screening interview based on the Structured Clinical Interview (SCID; Spitzer et al., 1990; Wittchen et al., 1990) for the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 2004) was conducted to ensure their psychological health and to exclude any kind of eating disorder and depression. To avoid any perceived selection bias in the recruitment process, habitual smoking (<10 cigarettes per day) and the use of oral contraceptives were allowed but controlled statistically. Female participants were tested only during their luteal phase. The participants reported their height and weight while those with a BMI >30 kg/m² were excluded. Finally, they were screened for their physical condition, medical history and were of medications that could influence the autonomic nervous system or the HPA axis.

All of the selected participants (n = 35), recruited through newspaper advertisements and the university students'

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