Effect of Acute Stress on Taste Perception: In Relation with Baseline Anxiety Level and Body Weight

Esin Ileri-Gurel, Bilge Pehlivanoglu and Murat Dogan

Physiology Department, Faculty of Medicine, Hacettepe University, 06100 Sihhiye, Ankara, Turkey

Correspondence to be sent to: Esin Ileri-Gurel, Department of Physiology (39), Faculty of Medicine, Hacettepe University, 06100, Sihhiye, Ankara, Turkey. e-mail: eileri@hacettepe.edu.tr

Abstract

We aimed to determine the effect of acute stress on taste perception and its modulation in relation to body weight and baseline anxiety in this study. The anxiety of the participants, randomly allocated to stress (n = 35) or control (n = 16) groups, was assessed by State Trait Anxiety Inventory. Stroop color-word interference and cold pressor tests were applied as stress protocol. Glucose and salt taste detection thresholds were evaluated before and after the stress protocol in the stress group and corresponding times in the control group. Stress protocol increased heart rate and blood pressure as an indicator of stress system activation. Following stress glucose and salt thresholds decreased in the stress group, unchanged in the control group. Prestress salt thresholds were positively and decrements in salt thresholds were negatively correlated with trait anxiety scores of participants. The state anxiety levels of stress group positively correlated with the decrease in glucose thresholds. Waist-to-hip ratio was negatively correlated with prestress salt thresholds of the subjects. Our results revealed that thresholds for sweet and salty tastes are modulated during stressful conditions. Our data also demonstrated a relationship between taste perception and baseline anxiety levels of healthy individuals, which may be important to understand the appetite alterations in individuals under stressful conditions.

Key words: acute stress, anxiety, body weight, glucose threshold, taste perception, salt threshold

Introduction

Stress is the response of our body to emotional or physical threats and the stressors initiate a stress response that temporarily changes certain physiological functions in the body. These changes are associated with increased activity of the hypothalamic-pituitary-adrenal (HPA) axis that leads to the release of glucocorticoids, mainly cortisol, and sympathetic nervous system (SNS) where activation results with the release of noradrenaline and adrenaline (Axelrod and Reisine 1984). One of the physiological changes as a response to acute stress is altered food and energy intake (Oliver et al. 2000; Wardle et al. 2000; Zellner et al. 2006; Newman et al. 2007; Al'Absi et al. 2012). Both losing appetite (Charmandari et al. 2005; Adam and Epel 2007) and overeating (Gluck et al. 2004; García-Prieto et al. 2007; Newman et al. 2007) were reported in response to acute stress exposure depending on the type, duration and magnitude of the stressor.

Taste perception plays a major role in appetite and food choices but the effect of stress on taste sensitivity has not been investigated extensively in humans. Taste perception is determined by genetic, hormonal and metabolic factors. It can also be influenced by psychological conditions of the individual. It was demonstrated that the perceived duration of bitter, sour and sweet taste perceptions was shortened; the total amounts of bitter, sour and sweet tastes were reduced following mental stress (Nakagawa et al. 1996). Another study has reported that normal individuals who were exposed to stress were shown to have increased sensitivity to the bitter taste of saccharin (Dess and Edelheit 1998). There are also studies linking different affective states and anxiety to taste (DeMet et al. 1989; Miller and Naylor 1989; Al'Absi et al. 2012). Higher recognition thresholds for sucrose and altered suprathreshold measures of sucrose taste intensity and pleasantness were reported in depressed patients (Steiner et al. 1969; Amsterdam et al. 1987). A recent study pointed out the relation between acute stress and reduced sweet, sour and salty taste perception (Al'Absi et al. 2012). Taken these data together, it is apparent that taste perception is related with affect and stress response.

On the other hand, taste perception may influence eating behavior and hence body weight; and body weight changes may cause further modulations on taste perception (Woschnagg et al. 2002). Several studies have tried to elucidate the relationship between taste perception and obesity. Some of them failed to show this relationship, which mostly focused on sweet taste (Malcolm et al. 1980; Frijters and Rasmussen-Conrad 1982; Grinker et al. 1986; Anderson 1995). Whereas some other studies have shown a relationship between different taste thresholds and body weight (Bartoshuk et al. 2006; Simchen et al. 2006; Pasquet et al. 2007; Sartor et al. 2011). These limited and conflicting results led us to examine the relationship between body mass index (BMI) and taste perception as the second aim of this study.

On this background, we aimed to determine the effect of acute stress and body weight on sweet and salty taste detection thresholds and to investigate the correlation between baseline anxiety levels and perception of taste.

Materials and methods

Participants

Volunteers responding to a public announcement in the University participated to the study. They have given their informed consent and the experimental protocol designed in compliance with the Declaration of Helsinki was also approved by Ethical Commission of Hacettepe University. The participants were healthy, nonsmoking volunteers having no history of any systemic disease and use of medication. Exclusion criteria were chronic illness, history of eating disorders and current dieting, high State Trait Anxiety Inventory (STAI) scores as indicated below. All participants completed a self-administered questionnaire (STAI) before the experimental protocol.

General characteristics of the groups are given in Table 1. There were 35 participants (19 females and 16 males) in the stress group and 16 participants (9 females and 7 males) in the control group. Ages of both groups ranged from 18 to 21 years. BMI of the stress group was $23 \pm 0.4 \, \text{kg m}^{-2}$, with three overweight participants (two women and one man). BMI of the control group was 22 ± 0.7 and only one of the

Table 1   General characteristics of the participants

	Control group (n = 16)	Stress group (n = 35)
Age (years)	20 ± 0.2	19 ± 0.2
BMI (kg m^{-2})	22 ± 0.7	23 ± 0.4
WHR	0.76 ± 0.02	0.78 ± 0.01
State anxiety score	28 ± 1.3	32 ± 1.2
Trait anxiety score	37 ± 2.0	38 ± 1.0

Values are given as mean ± SEM.

participants was overweight. Mean age and BMI of the genders were comparable in both groups, but the waist-to-hip ratio (WHR) of males was higher than females ($P < 0.01$). The participants scored within the normal range of STAI (40 for STAI-S and STAI-T) were included in the study.

Taste perception test

Each tastant (glucose and sodium chloride) was diluted with distilled water that was also used for rinsing mouth after each solution. The solutions (Table 2) were stored at +4 °C and before the taste perception test they were allowed to reach room temperature. Plastic cups were used to present the solutions and each cup was marked with a code, by which both the participant and the tester were blinded to the content. The taste perception test was done between 11.30 am and 13.00 pm in a fasted state on weekdays. The participants were asked to have a light breakfast on the test day and refrain from eating, drinking, except for water, and exercise after breakfast until the experimental protocol.

The taste perception test was a stair-case method modified from Cornsweet (1962). Taste detection thresholds for glucose and sodium chloride (NaCl) were determined by this method. Each participant was informed about the set of taste categories he or she could be faced with (such as salty and sweet) but the test was carried out in blind conditions (the order of presentation of the solutions was not known by the participants). Two series of solutions were presented in a random order. The solutions were offered in increasing concentrations. The participants took the solutions from the cups with a 10-mL plastic spoon. The solutions were swirled in the mouth to allow it to reach all parts of the mouth cavity and not swallowed. The mouth was washed with distilled water after tasting each solution and there were 30 s intervals between successive solutions. Once the taste of

Table 2   Concentrations of taste perception test solutions

Bottle no.	NaCl concentration		Glucose MH concentration	
	g L^{-1}	mol L^{-1}	g L^{-1}	mol L^{-1}
1	0.22	0.004	0.77	0.004
2	0.45	0.008	1.55	0.008
3	0.9	0.015	3.1	0.015
4	1.82	0.030	6.2	0.030
5	3.65	0.060	12.37	0.060
6	7.3	0.120	24.75	0.120
7	14.6	0.250	49.5	0.250
8	29.2	0.5	99	0.5
9	58.4	1	198	1

Glucose MH = glucose monohydrate

two successive concentrations was recognized correctly, the subject was given the previous unrecognized concentration. This up-and-down procedure was performed twice until the taste of two increasing stimuli was named correctly. Actual detection threshold was recorded as the lowest concentration recognized.

Stress test

After the first session of the taste perception test, the participants, randomly allocated to the stress group, were exposed to the stress protocol. The participants of the control group sat quietly for 20 min without being exposed to the stress protocol until the second session of taste perception test. A sphygmomanometer cuff was placed in dominant arms of the participants to measure blood pressure (BP). Electrocardiogram electrodes were placed on right and left arms and left leg to monitor heart rate (HR). After instrumentation, participants of the stress group were instructed about the test and the stress protocol was started.

Stress protocol comprised a "Stroop color-word interference test" followed by "cold pressor test." It has been demonstrated previously that this protocol is effective to elicit psychological stress with reliable adrenocortical and cardiovascular responses, has test–retest reliability and is not gender-specific (Pehlivanoglu et al. 2005; Pehlivanoglu et al. 2012). The computerized version of the Stroop color-word interference test was composed of three parts each lasting 3 min. In the first part, participants were required to indicate the color of the word independently of the meaning of the written word. Participants were instructed to choose the color of each word as quickly and as accurately as possible while ignoring the written color names. The second part required the participants to indicate the written color names of the words independently of the color of the words. In the third part, the tasks of the first and second parts changed sequentially with a warning sound. As soon as the Stroop color-word interference test was finished, cold pressor test was applied for 3 min and right hands of the participants, up to the wrist-fold, were immersed into cold water at +4 °C. The Stroop test is known to elicit predominantly a β-adrenergic response and the cold pressor test elicits an α-adrenergic response (Pickering and Gerin 1990). Stress responses of the participants were followed via the changes in HR and BP throughout the stress protocol. HR and BP data were gathered via appropriate transducers to a data acquisition and analysis system (BIOPAC MP30, BIOPAC Systems Inc., California). Stress group underwent to the second session of taste perception test as soon as the stress protocol lasted.

Anthropometric measurements

A relationship between sensory capabilities and body weight was previously brought into attention (Griep et al. 2000; Tepper and Ullrich 2002; Simchen et al. 2006). Therefore, in order to evaluate the effect of body weight and fat distribution on taste thresholds, anthropometric measurements (body weight, body height, waist and hip circumference) were done. Body weight (kg) and height (m) were measured for the calculation of the BMI (kg m^{-2}). The values of BMI, as suggested by World Health Organization (2000), were used to group the subjects as underweight, normal, overweight or obese. Waist and hip circumferences were used to calculate WHR, which is a simple but reliable measure of intra-abdominal and total fat. The waist and hip circumference measurements were obtained in standing position. The waist circumference (cm) was measured at the site of the smallest circumference between the rib cage and the iliac crest, whereas the hip circumference (cm) was measured at the level of maximum extension of the buttocks. The WHR was calculated as waist circumference divided by hip circumference (cutoff points to define obesity was 1.0 in men and 0.85 in women as defined by World Health Organization [2000]).

State Trait Anxiety Inventory

The baseline anxiety levels of the participants were assessed using the Turkish translation of the STAI. It was translated and validated in Turkish by Oner and LeCompte (1985). This questionnaire consists of two scales: the state scale (anxiety state) and the trait scale (anxiety trait). The state scale measures the instant and temporary feelings of anxiety and trait scale measures chronic feelings of anxiety. Both scales have 20 questions with four possible responses to each question (Likert scale). In responding to the state scale, the participants rated the statement that best describes the intensity of their feelings from 1 to 4 (not at all to almost always, respectively). A high rating indicates the absence or presence of anxiety for some state anxiety items and some trait anxiety items (Spielberger 1975). The ratings of anxiety-absent items are reversed for the calculation of the score. Scores for both state and trait anxiety scales can vary from a minimum of 20 to a maximum of 80, whereas a cutoff score was 40 for state anxiety and 60 for trait anxiety in the originally defined version (Spielberger and Gorsuch 1983), whereas a score of 40 is assumed as the limit for normal in the recent studies (Livadas et al. 2011; Manocha et al. 2011).

Data analyses

Statistical analysis was performed with SPSS 15.0 for Windows (SPSS Inc., Chicago, IL). All data were given as mean ± SEM. Sample size was predetermined by power analysis ($\alpha = 0.05$ and $\beta = 0.90$). Within and between group comparisons for the taste thresholds and changes in taste threshold for control and stress groups at two trials (for group and time variables) were performed with repeated measures of analysis of variance (ANOVA). Intergroup differences based on gender or STAI scores were evaluated by

Student's *t*-test. Relation between continuous variables was determined by Spearman correlation coefficient. Variation in BP and HR throughout the experimental protocol was evaluated with repeated measures of ANOVA. Significance level was determined as $P < 0.05$.

Results

The acute stress induced by "Stroop color-word interference test" followed by "cold pressor test" in the stress group was confirmed with HR and BP measurements. HR and BP levels were significantly higher during the stress protocol ($P < 0.01$). HR, systolic and diastolic BPs of the control group did not change significantly throughout the experimental protocol (Table 3).

Following acute stress exposure detection thresholds for both tastes decreased in the stress group. The glucose threshold decreased from 0.051 ± 0.007 mol L^{-1} to 0.037 ± 0.005 mol L^{-1} ($P < 0.01$) and NaCl threshold decreased from 0.016 ± 0.0015 mol L^{-1} to 0.012 ± 0.0011 mol L^{-1} ($P < 0.05$) after acute stress protocol (Figure 1). Following quite rest of the control group taste thresholds remained the same. The glucose threshold of the control group was 0.054 ± 0.009 mol L^{-1} at the first session of the taste perception test and was 0.051 ± 0.008 mol L^{-1} at the second session of the taste perception test ($P > 0.05$). The NaCl threshold was 0.017 ± 0.003 mol L^{-1} at the first session and 0.016 ± 0.002 mol L^{-1} at the second session of the taste perception test ($P > 0.05$). The thresholds for both glucose and NaCl were similar in stress and control group in the first session ($P > 0.05$) whereas both values were significantly lower in stress group compared with control group in the second session of the taste perception ($P < 0.05$).

Pre- and poststress salt thresholds were positively correlated with trait anxiety scores of the subjects (prestress: $r = 0.317$, $P = 0.022$; poststress: $r = 0.682$, $P = 0.0001$) (Figure 2A and

Table 3 Heart rate, systolic and diastolic BP values of the stress and control groups before, during and after acute stress

Test conditions	HR (beat per min)	SBP (mmHg)	DBP (mmHg)
Stress group (*n* = 35)			
Prestress test	82 ± 15	105 ± 12	70 ± 9
End of the first part	92 ± 15*	113 ± 12*	74 ± 7*
End of the second part	97 ± 15*	115 ± 11*	76 ± 7*
End of the third part	104 ± 14*	115 ± 10*	76 ± 7*
End of the cold pressor test	110 ± 12*	116 ± 13*	78 ± 8*
Control group (*n* = 16)			
After first session	73 ± 2	115 ± 4	71 ± 3
Before second session	74 ± 1	108 ± 5	71 ± 2

Values are given as mean ± SEM. All values of the stress group were compared with the prestress values of the same group (*$P < 0.01$). HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure.
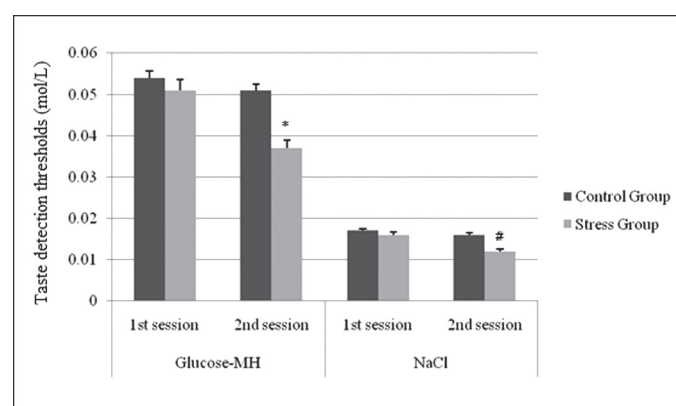
Figure 1 Taste detection thresholds of the control and the stress groups. First session is before the stress protocol and second session is after the stress protocol for the stress group, values are mean ± SEM, *$P < 0.01$ stress versus control group and first versus second session in stress group, #$P < 0.05$ stress versus control group and first versus second session in stress group.

Figure 2 Correlation of trait anxiety scores with salt detection thresholds measured **(A)** before and **(B)** after stress procedure.

Figure 3 Correlation of WHR with salt detection thresholds measured before stress procedure.

2B). Trait anxiety scores of the stress group were also negatively correlated with the decrease in salt thresholds after acute stress exposure ($r = –0.345$, $P = 0.004$). There was no correlation between pre- and poststress glucose threshold and state or trait anxiety scores. On the other hand, the state anxiety levels of the stress group were positively correlated with the decrease in glucose thresholds ($r = 0.548$, $P = 0.001$).

Our results pointed out a correlation between intra-abdominal and total body fat and salty taste sensitivity, represented by lower threshold. This relationship was demonstrated for prestress salt thresholds. Although there was no correlation for BMI, WHR was negatively correlated with prestress salt thresholds of the subjects ($r = –0.532$, $P = 0.008$) (Figure 3). The decrease in sweet and salty taste thresholds after stress protocol in the stress group was not correlated with BMI or WHR. These correlations were comparable in both genders.

Discussion

The novel finding of this study is decreased taste thresholds for both glucose and NaCl in response to acute stress in individiuals with normal anxiety scores. Although the basic neurochemistry of the stress response is now well understood, much remains to be discovered about how the components of this system interact throug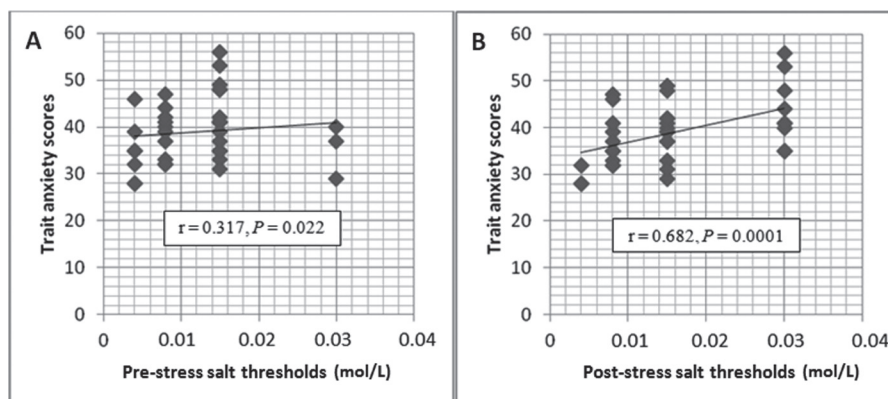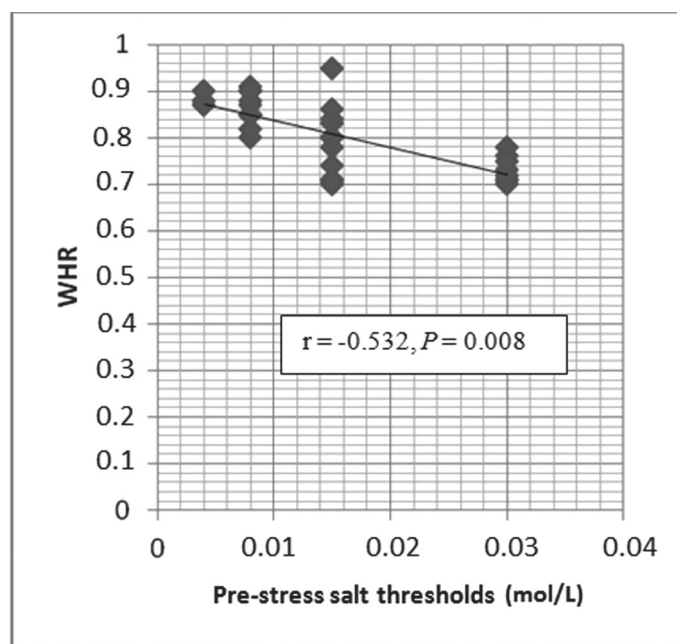hout the body, particularly with the gustatory system. Taste is a complex sensory information and our understanding about taste sensitivity regulation is limited. Recent advances have elucidated a number of neurotransmitter and neuropeptide signaling agents in the taste bud that may regulate taste sensitivity (Herness et al. 2002a, 2002b; Ogura 2002; Kaya et al. 2004; Zhao et al.

2005; Zhang et al. 2010). A large body of evidence suggests that noradrenaline, the main sympathetic neurotransmitter, is one of these agents. The effect of noradrenaline, on taste sensation was demonstrated almost 30 years ago (Morimoto and Sato 1982). It was shown that responses in gustatory afferent nerves were enhanced by exogenous noradrenaline. In a recent study about the neurotransmitter modulations on different taste thresholds, it was demonstrated that taste receptor cells respond to noradrenaline (Heath et al. 2006). This study showed that noradrenaline reuptake inhibitors alter human taste thresholds for sour and bitter, but not for sweet or salt, via a peripheral mechanism. Albeit our results seem contrary to the findings of Heath et al. (2006), the participants of the above-mentioned study were not in a stressed state. Stress reaction is a complex issue with the release of stress-related hormones like adrenaline, noradrenaline and cortisol. These hormones together may have complex and confounding effects on taste perception. An *in vitro* study showed that taste receptors can selectively take up noradrenaline synthesized in catecholaminergic nerve fibers, repackage and release it on depolarization (Dvoryanchikov et al. 2007). It was also demonstrated that exogenous noradrenaline enhances a calcium-activated chloride current via β-adrenergic receptors in taste receptor cells (Herness and Sun 1999; Herness et al. 2002a) and taste cells respond to both α- and β-adrenoceptor agonists (Herness et al. 2002b). Collectively, these data imply that adrenergic signaling, thereby SNS, can play modulatory roles in processing of gustatory information. Our results too contribute to the role of SNS and peripheral regulation of taste sensitivity.

A recently discovered signaling system modulating emotional responses to environmental impacts is constituted by the endocannabinoid system. Stress has a major effect on the endocannabinoids, and once the endocannabinoid system is activated, it executes anxiolytic and stress-relieving functions. Besides their other physiological functions, endogenous cannabinoids affect appetite and stimulate food intake (Jamshidi and Taylor 2001; Kirkham et al. 2002; Cota et al. 2003). Endocannabinoids not only stimulate food intake via central systems but also may increase palatability of foods by enhancing peripheral taste responses. This assertion was supported with a recent study showing that endocannabinoids enhanced sweet taste responses in the peripheral taste system of mice (Yoshida et al. 2010). The major limitation of this study is lacking of measurements regarding the activation of the endocannabinoid system, restricts our comments about this system's activation and its impact on central or peripheral processing of taste perception during stress and it needs further studies.

Everybody experiences anxiety when faced with a stressful situation. Stress and anxiety do not define the same condition, but they tend to reinforce and perpetuate each other. Stress is a nonspecific biological response to a stressor (environmental, physiological or psychological) an organism

encounters and anxiety is a cognitive response with feelings of worry, nervousness and apprehension (Panksepp 2004). So to speak, stress level of a person is strongly correlated with the anxiety level. In this study, we also aimed to determine whether there was a relationship between baseline anxiety level of the participants and both basal and poststress taste thresholds. We demonstrated that prestress salt thresholds of the participants were positively correlated with their trait anxiety scores. In other words, with an increase in baseline anxiety level, baseline salty taste thresholds were shifted to a higher concentration. Similarly, Heath et al. (2006) reported a significant positive correlation between trait anxiety scores and salt taste thresholds, in support of our findings. These data suggest that healthy anxious individuals may have higher salty taste thresholds compared with less anxious individuals, which in long term may lead to high salt intake and related diseases like hypertension. We also demonstrated that trait anxiety scores of the participants were negatively correlated with the decrease in salt thresholds, that is subjects with increased general anxiety level have responded less to the acute stress conditions in the context of taste threshold changes. This fact can be explained by the altered salty taste sensitivity under the effect of chronic sympathoadrenal activity and their salty taste sensitivity has already altered. It is possible that gustatory system generates an adaptive response for salt sensitivity during prolonged anxiety conditions.

Although Al'Absi et al. found attenuated sweet taste perception especially in participants with high trait negative affect, our results positively correlated the state anxiety (the instant and temporary feelings of anxiety) levels of the participants with the decrease in glucose thresholds. To participate in an experiment is perceived as stress for some individuals and they get excited before the experimental protocol, resulting in higher state anxiety levels. Therefore, the higher state anxiety levels and anxiety-related physiological changes probably potentiate the effect of acute stress protocol and may explain our results. Although our results seem to be conflicting with the above-mentioned study, as it was pointed out by the authors single concentration was tested and they correlated taste sensation with negative and positive affect after stress. Albeit the data of two studies are not fully comparable, both results will have implications in understanding the role of stress in regulation of taste sensation and appetite.

Although, it is not the primary aim of this study, interaction of adipokines, directly related to body fat amount, with taste perception should be taken into consideration (Shigemura et al. 2004; Niki et al. 2010). A relationship between body weight and sensory capabilities was previously demonstrated. Past research has mainly focused on sensitivity for sweet taste; therefore, little information is available about the association between body weight and the perception of sour, bitter and salty taste. Simchen et al. (2006) reported that in younger adults overweight was associated with lower sour and bitter taste perception. Studies focusing on the relationship between taste sensitivity to the bitter taste of 6-*n*-propythiouracil (PROP) revealed a negative association between PROP sensitivity and BMI (Tepper 1999; Tepper and Ullrich 2002; Padiglia et al. 2010). In contrast, a positive association between BMI and preference for flavor-amplified yoghurt was observed in another study (Griep et al. 2000). Even though we found no correlation between BMI and taste thresholds, the WHR was negatively correlated with prestress salt thresholds of the participants. BMI is widely used for classification of overweight and obesity, but it does not account for the wide variation of the body fat distribution and abdominal fat mass (World Health Organization, 2000). WHR, on the other hand, has been shown to be the best simple measure of both intra-abdominal and total fat (Lemieux et al. 1996; Han et al. 1997). Several studies have reported a stronger positive association between obesity-related morbidity (Ho et al. 2001; Visscher et al. 2002) and abdominal adiposity (measured by WHR) than with overall adiposity (as measured by BMI). To the best of our knowledge, this study is the first to demonstrate a relationship between WHR and salty taste sensitivity. Similar to our results, Pasquet et al. (2007) demonstrated a relationship between body weight and taste sensitivity for salt. They found an increased sensitivity to sodium chloride in massively obese adolescents. The decreased salty taste thresholds that we observed with subjects who have higher WHR may be associated with obesity-related metabolic and/or hormonal changes and needs further investigation.

In conclusion, our findings show that sweet and salt taste thresholds are modulated during stressful conditions. Our data also demonstrated a relationship between taste sensitivity and baseline anxiety level of healthy individuals. These modulations may be important to understand the appetite alterations in individuals under stressful conditions, which deserve further studies.

Acknowledgements

References

Adam TC, Epel ES. 2007. Stress, eating and the reward system. Physiol Behav. 91:449–458.

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.

Amsterdam JD, Settle RG, Doty RL, Abelman E, Winokur A. 1987. Taste and smell perception in depression. Biol Psychiatry. 22:1481–1485.

Anderson GH. 1995. Sugars, sweetness, and food intake. Am J Clin Nutr. 62:195S–201S, discussion 201S–202S.

Axelrod J, Reisine TD. 1984. Stress hormones: their interaction and regulation. Science. 224:452–459.

Bartoshuk LM, Duffy VB, Hayes JE, Moskowitz HR, Snyder DJ. 2006. Psychophysics of sweet and fat perception in obesity: problems, solutions and new perspectives. Philos Trans R Soc Lond, B, Biol Sci. 361:1137–1148.

Charmandari E, Tsigos C, Chrousos G. 2005. Endocrinology of the stress response. Annu Rev Physiol. 67:259–284.

Cornsweet TN. 1962. The staircrase-method in psychophysics. Am J Psychol. 75:485–491.

Cota D, Marsicano G, Tschop M, Grubler Y, Flachskamm C, Schubert M, Pagotto U. 2003. The endogenous cannabinoid system affects energy balance via central or exigenic drive and peripheral lipogenesis. J Clin Invest. 112:423–431.

DeMet E, Stein MK, Tran C, Chicz-DeMet A, Sangdahl C, Nelson J. 1989. Caffeine taste test for panic disorder: adenosine receptor supersensitivity. Psychiatry Res. 30:231–242.

Dess NK, Edelheit D. 1998. The bitter with the sweet: the taste/stress/temperament nexus. Biol Psychol. 48:103–119.

Dvoryanchikov G, Tomchik SM, Chaudhari N. 2007. Biogenic amine synthesis and uptake in rodent taste buds. J Comp Neurol. 505:302–313.

Frijters JE, Rasmussen-Conrad EL. 1982. Sensory discrimination, intensity perception, and affective judgment of sucrose-sweetness in the overweight. J Gen Psychol. 107(2d Half):233–247.

García-Prieto MD, Tébar FJ, Nicolás F, Larqué E, Zamora S, Garaulet M. 2007. Cortisol secretary pattern and glucocorticoid feedback sensitivity in women from a Mediterranean area: relationship with anthropometric characteristics, dietary intake and plasma fatty acid profile. Clin Endocrinol (Oxf). 66:185–191.

Gluck ME, Geliebter A, Hung J, Yahav E. 2004. Cortisol, hunger, and desire to binge eat following a cold stress test in obese women with binge eating disorder. Psychosom Med. 66:876–881.

Griep MI, Mets TF, Massart DL. 2000. Effects of flavour amplification of Quorn and yoghurt on food preference and consumption in relation to age, BMI and odour perception. Br J Nutr. 83:105–113.

Grinker JA, Gropman-Rubin J, Bose K. 1986. Sweet preference and body fatness: neonatal data. Nutr Behav. 3:197–209.

Han TS, McNeill G, Seidell JC, Lean ME. 1997. Predicting intra-abdominal fatness from anthropometric measures: the influence of stature. Int J Obes Relat Metab Disord. 21:587–593.

Heath TP, Melichar JK, Nutt DJ, Donaldson LF. 2006. Human taste thresholds are modulated by serotonin and noradrenaline. J Neurosci. 26:12664–12671.

Herness MS, Sun XD. 1999. Characterization of chloride currents and their noradrenergic modulation in rat taste receptor cells. J Neurophysiol. 82:260–271.

Herness S, Zhao FL, Kaya N, Lu SG, Shen T, Sun XD. 2002a. Adrenergic signalling between rat taste receptor cells. J Physiol. 543:601–614.

Herness S, Zhao FL, Lu SG, Kaya N, Shen T. 2002b. Expression and physiological actions of cholecystokinin in rat taste receptor cells. J Neurosci. 22:10018–10029.

Ho SC, Chen YM, Woo JL, Leung SS, Lam TH, Janus ED. 2001. Association between simple anthropometric indices and cardiovascular risk factors. Int J Obes Relat Metab Disord. 25:1689–1697.

Jamshidi N, Taylor DA. 2001. Anandamide administration into the ventromedial hypothalamus stimulates appetite in rats. Br J Pharmacol. 134:1151–1154.

Kaya N, Shen T, Lu SG, Zhao FL, Herness S. 2004. A paracrine signaling role for serotonin in rat taste buds: expression and localization of serotonin receptor subtypes. Am J Physiol Regul Integr Comp Physiol. 286:R649–R658.

Kirkham TC, Williams CM, Fezza F, Di Marzo V. 2002. Endocannabinoid levels in rat limbic forebrain and hypothalamus in relation to fasting, feeding and satiation: stimulation of eating by 2-arachidonoyl glycerol. Br J Pharmacol. 136:550–557.

Lemieux S, Prud'homme D, Bouchard C, Tremblay A, Després JP. 1996. A single threshold value of waist girth identifies normal-weight and overweight subjects with excess visceral adipose tissue. Am J Clin Nutr. 64:685–693.

Livadas S, Chaskou S, Kandaraki AA, Skourletos G, Economou F, Christou M, Boutzios G, Karachalios A, Zerva A, Xyrafis X, et al. 2011. Anxiety is associated with hormonal and metabolic profile in women with polycystic ovarian syndrome. Clin Endocrinol. 75:698–703.

Malcolm R, O'Neil PM, Hirsch AA, Currey HS, Moskowitz G. 1980. Taste hedonics and thresholds in obesity. Int J Obes. 4:203–212.

Manocha R, Black D, Sarris J, Stough C. 2011. A randomized, controlled trial of meditation for work stress, anxiety and depressed mood in full-time workers. Evid Based Complement Alternat Med. doi: 10.1155/2011/960583.

Miller SM, Naylor GJ. 1989. Unpleasant taste—a neglected symptom in depression. J Affect Disord. 17:291–293.

Morimoto K, Sato M. 1982. Role of monoamines in afferent synaptic transmission in frog taste organ. Jpn J Physiol. 32:855–871.

Nakagawa M, Mizuma K, Inui T. 1996. Changes in taste perception following mental or physical stress. Chem Senses. 21:195–200.

Newman E, O'Connor DB, Conner M. 2007. Daily hassles and eating behaviour: the role of cortisol reactivity status. Psychoneuroendocrinology. 32:125–132.

Niki M, Jyotaki M, Yoshida R, Ninomiya Y. 2010. Reciprocal modulation of sweet taste by leptin and endocannabinoids. Results Probl Cell Differ. 52:101–114.

Ogura T. 2002. Acetylcholine increases intracellular Ca^{2+} in taste cells via activation of muscarinic receptors. J Neurophysiol. 87:2643–2649.

Oliver G, Wardle J, Gibson EL. 2000. Stress and food choice: a laboratory study. Psychosom Med. 62:853–865.

Oner N, LeCompte A. 1985. Manual of State and Trait Anxiety Inventory. Istanbul: Bogazici University Press. p. 10–16.

Padiglia A, Zonza A, Atzori E, Chillotti C, Calò C, Tepper BJ, Barbarossa IT. 2010. Sensitivity to 6-n-propylthiouracil is associated with gustin (carbonic anhydrase VI) gene polymorphism, salivary zinc, and body mass index in humans. Am J Clin Nutr. 92:539–545.

Panksepp J. 2004. Textbook of biological psychiatry. Hoboken, NJ: Wiley-Liss.

Pasquet P, Frelut ML, Simmen B, Hladik CM, Monneuse MO. 2007. Taste perception in massively obese and in non-obese adolescents. Int J Pediatr Obes. 2:242–248.

Pehlivanoglu B, Bayrak S, Gurel EI, Balkanci ZD. 2012. Effect of gender and menstrual cycle on immune system response to acute mental stress: apoptosis as a mediator. Neuroimmunomodulation. 19:25–32.

Pehlivanoglu B, Durmazlar N, Balkanci ZD. 2005. Computer adapted Stroop colour-word conflict test as a laboratory stress model. Erciyes Med J. 58:58–63.

Pickering TG, Gerin W. 1990. Cardiovascular reactivity in the laboratory and the role of behavioral factors in hypertension: a critical review. Ann Behav Med. 12:3–16.

Sartor F, Donaldson LF, Markland DA, Loveday H, Jackson MJ, Kubis HP. 2011. Taste perception and implicit attitude toward sweet related to body mass index and soft drink supplementation. Appetite. 57:237–246.

Shigemura N, Ohta R, Kusakabe Y, Miura H, Hino A, Koyano K, Nakashima K, Ninomiya Y. 2004. Leptin modulates behavioral responses to sweet substances by influencing peripheral taste structures. Endocrinology. 145:839–847.

Simchen U, Koebnick C, Hoyer S, Issanchou S, Zunft HJ. 2006. Odour and taste sensitivity is associated with body weight and extent of misreporting of body weight. Eur J Clin Nutr. 60:698–705.

Spielberger CD, Gorsuch RL. 1983. Manual for the State-Trait Anxiety Inventory (Form Y): ("self-evaluation questionnaire") ([Rev.]. ed.). Palo Alto, CA: Consulting Psychologists Press, Inc.

Spielberger CD. 1975. The measurement of state and trait anxiety: conceptual and methodological issues. New York: Raven Press.

Steiner JE, Rosenthal-Zifroni A, Edelstein EL. 1969. Taste perception in depressive illness. Isr Ann Psychiatr Relat Discip. 7:223–232.

Tepper BJ, Ullrich NV. 2002. Influence of genetic taste sensitivity to 6-n-propylthiouracil (PROP), dietary restraint and disinhibition on body mass index in middle-aged women. Physiol Behav. 75:305–312.

Tepper BJ. 1999. Does genetic taste sensitivity to PROP influence food preferences and body weight? Appetite. 32:422.

Visscher TL, Kromhout D, Seidell JC. 2002. Long-term and recent time trends in the prevalence of obesity among Dutch men and women. Int J Obes Relat Metab Disord. 26:1218–1224.

Wardle J, Steptoe A, Oliver G, Lipsey Z. 2000. Stress, dietary restraint and food intake. J Psychosom Res. 48:195–202.

World Health Organization. 2000. Obesity: preventing and managing the global epidemic: report of a WHO consultation. Geneva: World Health Organization.

Woschnagg H, Stollberger C, Finsterer J. 2002. Loss of taste is loss of weight. Lancet. 359:891.

Yoshida R, Ohkuri T, Jyotaki M, Yasuo T, Horio N, Yasumatsu K, Ninomiya Y. 2010. Endocannabinoids selectively enhance sweet taste. Proc Natl Acad Sci USA. 107:935–939.

Zellner DA, Loaiza S, Gonzalez Z, Pita J, Morales J, Pecora D, Wolf A. 2006. Food selection changes under stress. Physiol Behav. 87:789–793.

Zhang Y, Kolli T, Hivley R, Jaber L, Zhao FI, Yan J, Herness S. 2010. Characterization of the expression pattern of adrenergic receptors in rat taste buds. Neurosci. 169:1421–1423.

Zhao FL, Shen T, Kaya N, Lu SG, Cao Y, Herness S. 2005. Expression, physiological action, and coexpression patterns of neuropeptide Y in rat taste-bud cells. Proc Natl Acad Sci USA. 102:11100–11105.