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Eating After Acute Psychosocial Stress in Healthy Men and Women: Sex Differences and Endocrine Mechanisms

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Abstract

Context: Overweight and obesity have become a major health burden with a higher prevalence of obesity in women than in men. Mental stress has been discussed to play a role in this context.

Objective: We investigated endocrine mechanisms underlying eating after acute psychosocial stress and potential sex differences therein.

Methods: A total of 32 male and 31 female healthy participants underwent the Trier Social Stress Test before they tasted ice cream in a bogus taste test 15 minutes after stress. We repeatedly assessed the stress hormone cortisol and the satiety hormone cholecystokinin (CCK) in saliva as well as perceived hunger before and up to 1 hour after stress.

Results: Lower immediate total cortisol stress reactivity predicted higher hunger ($Ps \leq .004$), but was not associated with food intake ($Ps \geq .90$) or total CCK release ($Ps \geq .84$). As compared to men, women ate less after stress ($Ps < .001$) and had consistently lower levels of hunger ($Ps \leq .024$) and cortisol ($Ps \leq .008$) as well as a lower immediate total cortisol stress reactivity ($Ps = .002$). Further, they differed in the kinetics of CCK over the total experimental procedure ($Ps \leq .011$), in immediate reaction to stress ($Ps \leq .038$), and after eating ($Ps \leq .072$), with women's CCK levels continuously decreasing while men's CCK levels were reactive.

Conclusion: We found evidence for lower immediate total cortisol stress reactivity relating to higher perceived hunger, with lower cortisol levels in women. Unlike in men, CCK levels in women were not reactive to acute stress and eating and decreased continuously. Our results may suggest a higher risk for stress-induced eating in women.

Key Words: acute psychosocial stress, sex differences, cortisol, hunger, CCK

Abbreviations: ANCOVA, analysis of covariance; ANOVA, analysis of variance; AUC_G, area under the curve with respect to ground; BMI, body mass index; BTT, bogus taste test; CCK, cholecystokinin; TSST, Trier Social Stress Test.

The prevalence of overweight and obesity has substantially increased over the last decades, with a higher total prevalence of obesity in women as compared to men (1). Mental stress has repeatedly been discussed to play a role in this development (eg, (2)), but the underlying mechanisms are not clear. While most studies have focused on effects of chronic stress on risk for obesity (2–6) (but see also (7)) and eating behavior (8-11), comparably fewer studies have investigated effects of acute stress. In particular, investigation of acute stress effects on eating behavior in healthy nonobese men and women may help to understand processual aspects of underlying mechanisms.

To date, the endocrine mechanisms underlying eating behavior and related feelings of hunger in reaction to acute stress are not fully understood. At the biological level hunger and satiety are regulated by hormones of the neuroendocrine system (12, 13). In particular, appetite-stimulating hormones such as ghrelin promote food intake, while satiety-stimulating hormones such as cholecystokinin (CCK) or leptin decrease hunger, thus promoting cessation of food consumption (14, 15).

With respect to stress reactivity, ghrelin (16, 17) and leptin (18) have been shown to increase following acute mental stress induction. Similarly, CCK increases in anticipation of a sports competition (19) but its reactivity in response to acute mental stress has not yet been investigated.

In addition to hunger and satiety hormones, the stress hormone cortisol as the end product of a stress-induced activation of the hypothalamus-pituitary-adrenal axis may play a role in the regulation of eating after stress. A frequently used acute psychosocial laboratory stressor that induces strong physiological stress reactions, including those of cortisol, is the Trier Social Stress Test (TSST (20, 21)). To date, a few studies compared food intake after TSST in participants with higher vs lower cortisol responses. In normal-weight (average body mass index [BMI]<30) women, participants with higher cortisol TSST reactivity ate more after stress compared to those with lower cortisol TSST reactivity (22), with higher cortisol increases associated with higher food intake (22). In contrast, 2 other studies could not find differences (23, 24) and 2 further studies found opposite effects with higher food intake relating to lower cortisol TSST reactivity (25, 26). Results in obese participants were similarly inconclusive (23, 24). In line with the studies reporting a negative association between

cortisol stress reactivity and food intake (25, 26), acute stress is usually accompanied by reduced feelings of appetite (24, 27, 28), hunger (29), and desire to eat (30). However, studies that have investigated, notably mostly in overweight participants, the relation of hunger and appetite with stress-induced cortisol release, provide mixed findings (24, 28, 31). Finally, regarding associations between cortisol stress reactivity and hunger and satiety hormones, one study found a positive correlation between TSST-induced changes in ghrelin and cortisol in participants with and without a binge eating disorder (32). However, leptin reactivity in response to the TSST was not correlated with cortisol in postmenopausal women (18). To our best knowledge, no study to date has investigated the effects of cortisol on CCK. Taken together, studies that compare or associate cortisol stress reactivity with eating behavior, feelings of hunger, or hunger and satiety hormones have been conducted mainly in women with in part contradictory results.

Given the higher prevalence of overweight and obesity in women as compared to men, investigation of sex differences in eating behavior and feelings of hunger in reaction to acute stress may help to shed light on potential underlying mechanisms. When comparing men and women in their general eating behavior, women eat less (33, 34), choose healthier foods (35, 36), and feel more postprandial fullness than men (37). To the best of our knowledge, only 3 studies investigated sex differences in eating behavior after acute mental stress, but results differed. While one study could not observe sex differences in the amount of food consumption after a mental arithmetic task (38), men ate less after watching a stressful film compared to a control condition, whereas women tended to eat more (39). In a further study, however, men ate significantly more than women both after acute mental stress (expecting a speech task) and a control condition (40). Sex differences in eating behavior after a potent mental stressor such as the TSST, as well as in subjective ratings of hunger, or appetite respectively, after acute stress have not yet been investigated. With respect to hunger and satiety hormone levels (for review, see (41)) women have higher baseline levels of ghrelin, leptin (42, 43), and CCK (44, 45) compared to men. To the best of our knowledge, potential sex differences in the reactivity of hunger and satiety hormones to acute stress remain to be elucidated. In addition, studies investigating the effects of cortisol stress reactivity in eating after stress focused mainly on effects in women (22, 23, 25, 26, 30, 31) (but see also (24, 28)) and potential sex differences still have to be clarified.

Here, we set out to investigate endocrine mechanisms underlying hunger and eating after acute psychosocial stress in healthy men and women. As endocrine measures, we repeatedly assessed cortisol and CCK from saliva measured before and up to 60 minutes after stress induction. First, we investigated whether cortisol release in response to acute stress relates to food intake in a subsequent bogus taste test (BTT) starting at immediate cortisol stress reactivity 15 minutes after the TSST (eg, (20, 46)), as well as to the reactivity of CCK and sensation of hunger. To avoid sex differences in the salivary cortisol stress reactivity, we invited female participants without oral contraceptive use in the luteal phase of their menstrual cycle, as comparable cortisol reactivity in response to the TSST for men and women has been found only under these conditions (47-49). In line with the previously described reasoning, we hypothesized lower immediate (ie, up to 15 minutes after stress cessation) total cortisol stress reactivity to predict higher food intake (25, 26), higher hunger in reaction to stress, and correspondingly lower levels of CCK. Second, we tested for potential sex differences in CCK and hunger in immediate reaction to stress induction and quantity of food intake in the subsequent BTT. Here, we expected men to eat more than women in the taste test after stress as we considered the general sex differences in the amount of food intake (33) to outweigh potential additional differential stress effects on food intake (39). With respect to feelings of hunger, we hypothesized men to repeatedly report higher levels hunger throughout the experiment. Moreover, given the baseline differences in satiety hormones (42-45) and the food intake differences after stress between men and women (39), we expected sex differences in CCK in the immediate reaction to stress and after food intake.

Materials and Methods

Study Participants

We recruited apparently healthy male and female volunteers aged between 18 and 48 years by advertisement and mailing lists. Interested individuals were screened by an online questionnaire asking for the following exclusion criteria: excessive alcohol or drug use, smoking more than 5 cigarettes per day, acute as well as chronic physical or mental illness, medication, use of oral contraceptives, and food intolerances. To compare immediate cortisol stress reactivity between men and women, eligible female participants were invited during the luteal phase of their menstrual cycle based on information regarding the date of the first day of their last menstruation and the typical length of their menstrual cycle (47-49). All participants provided written informed consent and were compensated with 20€ or 3 subject hours. The study was approved by the ethics committee of the University of Konstanz.

Design and Procedure

In anticipation of the experimental session, participants were asked to refrain from coffee on the study day. In addition, they had to abstain from meals and beverages other than water and not to brush their teeth 2 hours prior to their appointment. Furthermore, they had to abstain from excessive exercise within 48 hours before study participation. To control for diurnal variations in cortisol secretion (50), experimental sessions started between 2 PM and 4 PM. On arrival, participants were welcomed and seated in a quiet room before they were provided with complete written and oral descriptions of the study. The study participants were informed that they would be engaged in a challenging task and a taste test during the course of the experimental procedure. Participants were administered 200 mL of grape juice 40 minutes prior to the start of the TSST (discussed later) (51) before they completed questionnaires. Participants' height and weight (Sanitas SBG39) were measured before they were exposed to the TSST in a separate room. Fifteen minutes after the stress cessation actual food intake was assessed during a 15-minute BTT (52) (discussed later). Saliva samples for cortisol and CCK assessment were taken immediately before (−1 minute (S1)) and 1 minute (S2), and 10 (S3), 20 (S4), 30 (S5), 45 (S6), and 60 minutes (S7) after stress cessation. While samples S1 to S3 allow for testing of immediate (total) cortisol stress reactivity alone, the subsequent samples assess immediate reactivity to food take during the BTT with assessment before (S3), during

(S4), and after food intake (S5-S7). In addition, the sensation of hunger (using a visual analog scale) was recorded at each sampling time point before and after stress.

Induction of Acute Psychosocial Stress

The TSST is a well-standardized procedure to reliably induce psychosocial stress and resulting neuroendocrine responses (20, 21). The procedure comprises a preparation period after a short introduction by the experimenter (5 minutes), a simulated job interview (5 minutes), and an arithmetic task (5 minutes). The test took place in a separate room in front of an unknown panel of 2 evaluators and a conspicuous video camera and microphone. The panel members, dressed in white laboratory coats, were introduced as experts in evaluation of nonverbal behavior.

Bogus Taste Test

BTTs are used to assess actual food intake and simultaneously omit potential biases of self-reports and retrospective memories of eating behavior (52). Fifteen minutes after stress cessation, 3 different flavors of ice cream (each approximately 72 g and 225.3 kcal) were served to the participants, who were asked to evaluate the taste, texture, and their preference of the flavors. In detail, the taste test included 20 fake questions per ice cream flavor (eg, "How much do you like this ice cream?" or "How likely is it that you would purchase this ice cream?") to be answered on a 4-point scale (52). Participants were told to taste and eat as much as they liked in the following 15 minutes. As outcome, ice cream bowls were weighed before and after the taste test to calculate the quantity of ice cream (in grams) that had been consumed.

Repeated Assessment of Hunger

To assess sensations of hunger before and after stress as well as before and after the BTT, that is, food intake, participants evaluated their current hunger ("Do you feel hungry right now?") and on a visual analog scale ranging from 0 ("Not hungry at all") to 10 ("Very hungry") with each Salivette at each sampling time point (ie, −1, 1, 10, 20, 30, 45, and 60 minutes (S1-S7)).

Biochemical Analyses

Saliva samples for cortisol and CCK determination were collected using Salivettes (Sarstedt), centrifuged and aliquoted into Eppendorf tubes, and stored at −20 °C until biochemical analyses. To prepare biochemical analyses, saliva samples were thawed and centrifuged at 2500*g* for 10 minutes. Biochemical analyses of cortisol were performed using a competitive enzyme-linked immunosorbent assay (ELISA, IBL International GmbH) (Tecan (IBL) catalog No. RE52611, RRID:AB_3064818). Intra-assay and inter-assay coefficients of variation were less than or equal to 13.2%. CCK was determined using a competitive inhibition enzyme immunoassay for the in vitro quantitative measurement of CCK (ELISA, Biomatik Corporation) (Biomatik catalog No. EKL54157, RRID:AB_3064819). Intra-assay and inter-assay coefficients of variation were less than or equal to 12%. Due to an insufficient amount of saliva, CCK could not be determined sufficiently (missing baseline and at least 2 further missing time points) analyzed in 4 male participants. These 4 men were excluded from analyses including CCK measures.

Statistical Analyses

Statistical analyses were performed using SPSS (version 26.0) statistical software packages for MacIntosh (IBM SPSS Statistics). All tests were 2-tailed with level of statistical significance set at P less than .05. No outliers were excluded. We determined f from partial η^2 (η_p^2) values using G*Power3.1. Effect size parameters f and R^2 changes are reported where appropriate (effect size conventions; small: $f = .10$, $\Delta R^2 = 0$ .02; medium: $f = .25$, $\Delta R^2 = 0$ .13; large: $f = .40$, $\Delta R^2 = 0.26$) (53). For all participants, we calculated BMI by the formula BMI = kg/m^2.

To compute sex differences in participant characteristics as well as baseline assessments of cortisol and eating-related parameters (ie, CCK, hunger, and quantity of food intake) we used a univariate analysis of variance (ANOVA). To confirm that the TSST significantly induced immediate cortisol stress reactivity, we calculated a repeated-measures analysis of covariance (ANCOVA) with repeated assessment of cortisol (S1-S7) as a manipulation check.

To analyze our first study question, that is, potential stress-induced cortisol effects on eating-related measures, we aggregated the immediate total cortisol stress reactivity in response to the TSST as area under the curve with respect to ground (AUC_G) (54) from baseline to 10 minutes after the TSST and thus before beginning the BTT ($AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$). We first calculated a linear regression analysis with cortisol $AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$ as a predictor and food intake as the dependent variable. Second, we tested for potential effects of immediate total cortisol stress reactivity on hunger and CCK levels over the course of the experimental procedure. For this purpose, we aggregated the repeated measures of hunger and CCK respectively as AUC_Gs from baseline to 60 minutes after the TSST (hunger $AUC_{G\text{-}S1\text{-}S7}$; CCK $AUC_{G\text{-}S1\text{-}S7}$). We calculated regression analyses with cortisol $AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$ as the independent variable and aggregated hunger ($AUC_{G\text{-}S1\text{-}S7}$) or CCK levels ($AUC_{G\text{-}S1\text{-}S7}$) as the respective dependent variable. Post hoc testing of significant regression analyses comprised separate regression analyses for the different phases of the experimental procedure, that is, stress peak reactivity alone (Peak-S1-S3), immediate reactivity to food intake after stress during the BTT (BTT-S3-S5), and the final phase after completion of food intake (After-BTT-S5-S7). We therefore computed AUC_Gs of significant repeatedly assessed dependent variables for the 3 phases of the experimental procedure ($AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$, $AUC_{G\text{-}BTT\text{-}S3\text{-}S5}$, $AUC_{G\text{-}After\text{-}BTT\text{-}S5\text{-}S7}$). To determine possible interactions with immediate total cortisol stress reactivity, complementary post hoc testing comprised repeated-measures ANCOVAs with significant dependent variables for the 3 phases of the experimental procedure (ie, S1-S3, S3-S5, and S5-S7) and immediate total cortisol stress reactivity (cortisol $AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$) as the linear independent variable.

Our second study aim was to test for potential sex differences in CCK and hunger in reaction to stress induction and quantity of food intake in the subsequent BTT. We calculated a repeated-measures AN(C)OVA with repeated assessment of cortisol (S1-S7) as the dependent and sex as the independent variable to test whether cortisol stress reactivity would be comparable between men and women in the luteal phase of the menstrual cycle.

To reveal potential sex differences in the endocrine mechanisms of hunger and eating after acute psychosocial stress, we first calculated an univariate AN(C)OVA with quantity of food intake as the dependent variable and sex as the independent variable. Second, we calculated repeated-measures AN(C)

OVAs with CCK and hunger (S1-S7) as dependent variables. Post hoc testing of significant repeated-measures AN(C)OVAs comprised separate analyses of CCK and hunger in the different phases of the experimental procedure.

We conducted all analyses of study question 1 with the control for potential confounding effects of sex alone in addition to age and BMI. Sex differences in study question 2 were analyzed without and with controlling for age and BMI. We aimed at recruiting women in the luteal phase of their menstrual cycle. However, post hoc verification of cycle phase revealed that on the experimental day 3 women were in the follicular phase. Therefore, we additionally controlled for cycle phase in all cortisol analyses. We applied the Huynh-Feldt correction for repeated measures.

All data were tested for normal distribution and homogeneity of variance using Kolmogorov-Smirnov and Levene's tests prior to statistical analyses. All measures showing a skewed distribution (age, BMI, repeated cortisol, CCK, and hunger measures as well as cortisol $AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$, cortisol $AUC_{G\text{-}BTT\text{-}S3\text{-}S5}$, cortisol $AUC_{G\text{-}After\text{-}BTT\text{-}S5\text{-}S7}$, and CCK $AUC_{G\text{-}S1\text{-}S7}$) were log-transformed. While log-transformed data were used for modeling and testing, we depict untransformed data in Table 1 and in Figs. 1 to 5.

Results

Participant Characteristics

Table 1 provides the demographic characteristics and eating-related measures of the 32 male and 31 female participants. Men and women did not differ in age and BMI ($Ps \geq .22$), although men showed a higher food intake ($P < .001$) after stress as well as higher cortisol (with and without control for menstrual cycle phase: $P = .002$) and hunger baseline levels ($P = .051$). Women had higher levels of baseline CCK toward a trend level of significance ($P = .093$).

Cortisol Stress Reactivity as a Predictor of Eating-related Measures

Cortisol

Across all participants, the TSST induced significant increases in cortisol (main effect of time: S1-S7: $F(2.40,146.46) = 4.50$; $P = .008$; $\eta_p^2 = .07$, $f = .27$; with all covariates: $F(2.63,152.46) = 3.30$; $P = .027$; $\eta_p^2 = .05$, $f = .23$), with highest levels observed 10 minutes after stress cessation (see Fig. 1).

Eating-related Parameters

Immediate total cortisol stress reactivity in terms of AUC_G from baseline to peak ($AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$) did not predict the amount of food intake (in grams) and thus eating after stress ($Ps \geq .90$) nor was it associated with CCK $AUC_{G\text{-}S1\text{-}S7}$ ($Ps \geq .84$). However, lower cortisol $AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$ predicted higher hunger over the total experimental procedure (hunger $AUC_{G\text{-}S1\text{-}S7}$: $\beta = -.38$; $P = .004$; $\Delta R^2 = 0.20$; with all covariates: $\beta = -.39$; $P = .003$; $\Delta R^2 = 0.24$; see Fig. 2). Post hoc analyses revealed that lower cortisol $AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$ predicted higher hunger in particular during peak reactivity from S1 to S3 (hunger $AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$: $\beta = -.35$; $P = .007$; $\Delta R^2 = 0.19$; with all covariates: $\beta = -.36$; $P = .007$; $\Delta R^2 = 0.21$) as well as in immediate reaction to food intake from S3 to S5 (hunger $AUC_{G\text{-}BTT\text{-}S3\text{-}S5}$: $\beta = -.46$; $P < .001$; $\Delta R^2 = 0.27$; with all covariates: $\beta = -.48$; $P < .001$; $\Delta R^2 = 0.31$). After food intake (S5-S7), associations between cortisol and hunger disappeared ($Ps \geq .075$). There was a significant interaction effect of immediate total cortisol stress reactivity with hunger in reaction to food intake (S3-S5: $F(1.69,101.32) = 3.86$; $P = .031$; $\eta_p^2 = .06$; $f = .25$; with all covariates: $F(1.78,101.59) = 3.67$; $P = .034$; $\eta_p^2 = .06$; $f = .25$; see Fig. 3). There were no further interactions with hunger, neither in reaction to stress (S1-S3: $Ps \geq .25$) nor after food intake (S5-S7: $Ps \geq .47$).

Sex Differences in Cholecystokinin, Hunger, and Food Intake After Stress

Cortisol

As intended by our recruiting procedure, men and women did not differ in their cortisol reactivity in response to the TSST (interaction sex-by-time, $Ps \geq .10$; see Fig. 1). However, there was a main effect of sex with overall higher cortisol secretion in men (S1-S7: without covariates: $F(1,61) = 8.56$; $P = .005$; $\eta_p^2 = .12$; $f = .37$; with all covariates: $F(1,58) = 7.59$; $P = .008$; $\eta_p^2 = .12$; $f = .37$). As compared to men, women showed lower immediate total cortisol stress reactivity ($AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$: without covariates: $F(1,61) = 10.88$; $P = .002$; $\eta_p^2 = .15$; $f = .42$; with all covariates: $F(1,58) = 10.36$; $P = .002$; $\eta_p^2 = .15$; $f = .42$) and lower total cortisol reactivity to eating ($AUC_{G\text{-}BTT\text{-}S3\text{-}S5}$: without covariates: $F(1,61) = 6.46$; $P = .014$; $\eta_p^2 = .10$; $f = .33$; with all covariates: $F(1,58) = 5.18$; $P = .027$; $\eta_p^2 = .08$; $f = .29$) but they did not differ in their total cortisol after food intake ($AUC_{G\text{-}After\text{-}BTT\text{-}S5\text{-}S7}$: $Ps \geq .078$; see Table 1).

Table 1. Participant characteristics and baseline assessment

	$N = 63$	Men ($n = 32$)	Women ($n = 31$)	P
Age, y	23.22 ± 0.72 (18-48)	22.97 ± 0.97 (18-48)	23.48 ± 1.09 (18-46)	.74
BMI	22.79 ± 0.41 (17.89-34.58)	23.23 ± 0.54 (17.89-30.12)	22.33 ± 0.63 (17.94-34.58)	.22
Food intake, g	121.98 ± 7.31 (6.42-212.21)	147.38 ± 9.83 (56.36-212.21)	95.75 ± 8.75 (6.42-185.71)	<.001
Cortisol, nmol/L (S1)	3.96 ± 0.33 (0.63-10.71)	4.76 ± 0.45 (1.45-10.60)	3.13 ± 0.43 (0.63-10.71)	.002
$AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$	160.86 ± 11.15 (26.36-435.94)	191.17 ± 14.80 (56.82-356.11)	129.57 ± 14.99 (26.36-435.94)	.002
$AUC_{G\text{-}BTT\text{-}S3\text{-}S5}$	174.69 ± 14.63 (12.41-660.47)	201.64 ± 18.47 (49.68-476.93)	146.87 ± 22.02 (12.41-660.47)	.014
$AUC_{G\text{-}After\text{-}BTT\text{-}S5\text{-}S7}$	160.60 ± 14.17 (16.56-663.44)	176.99 ± 18.94 (39.02-583.33)	143.68 ± 21.02 (16.56-663.44)	.078
CCK, pg/mL (S1)	372.22 ± 54.79 (7.75-2071.94)	297.83 ± 63.00 (7.75-1405.91) ($n = 28$)	439.42 ± 86.62 (15.58-2071.94)	.093
Hunger (S1)	4.02 ± 0.36 (0.00-9.79)	4.70 ± 0.51 (0.00-9.79)	3.33 ± 0.49 (0.00-9.38)	.051

Abbreviations: AUC_G, area under the curve with respect to ground; BMI, body mass index; BTT, bogus taste test; CCK, cholecystokinin.

Eating-related Parameters

Men ate significantly more ice cream after stress than women (without covariates: $F(1,61) = 15.33$; $P < .001$; $\eta_p^2 = .20$; $f = .50$; with covariates: $F(1,59) = 12.48$; $P < .001$; $\eta_p^2 = .18$; $f = .47$). Moreover, men and women significantly differed in the kinetics of CCK secretion (interaction sex-by-time, S1-S7: without covariates: $F(4.01,228.57) = 3.83$; $P = .005$; $\eta_p^2 = .06$; $f = .25$; with covariates: $F(4.09,224.94) = 3.29$; $P = .011$; $\eta_p^2 = .06$; $f = .25$; see Fig. 4; main effect of sex, $Ps \geq .30$). While women had borderline significantly higher CCK levels at baseline (S1: without covariates: $F(1,57) = 2.93$; $P = .093$; with covariates: $F(1,55) = 3.36$; $P = .072$; see Table 1), post hoc analyses revealed that women's CCK levels continuously decreased after stress while men's CCK levels showed a stress reaction with levels decreasing immediately after stress and recovering until 10 minutes after stress cessation (interaction sex-by-time, S1-S3: without covariates: $F(1.94,110.56) = 3.65$; $P = .030$; $\eta_p^2 = .06$; $f = .25$; with covariates: $F(2.0,110.0) = 3.36$; $P = .038$; $\eta_p^2 = .06$; $f = .25$; main effect of sex, $Ps \geq .08$). While men and women did not differ in their CCK reactivity in immediate reaction to eating (interaction sex-by-time, S3-S5: $Ps \geq .64$; main effect of sex, $Ps \geq .25$). While CCK further decreased after eating in women, it increased in men at a trend level toward significance (interaction sex-by-time, S5-S7: without covariates: $F(1.77,101.05) = 3.15$; $P = .053$; with covariates: $F(1.85,101.93) = 2.76$; $P = .072$; main effect of sex, $Ps \geq .60$).

In terms of hunger, women indicated lower overall levels of hunger (main effect of sex, S1-S7: without covariates: $F(1,61) = 6.00$; $P = .017$; $\eta_p^2 = .09$; $f = .31$; with covariates: $F(1,59) = 5.38$; $P = .024$; $\eta_p^2 = .08$; $f = .29$), but men and

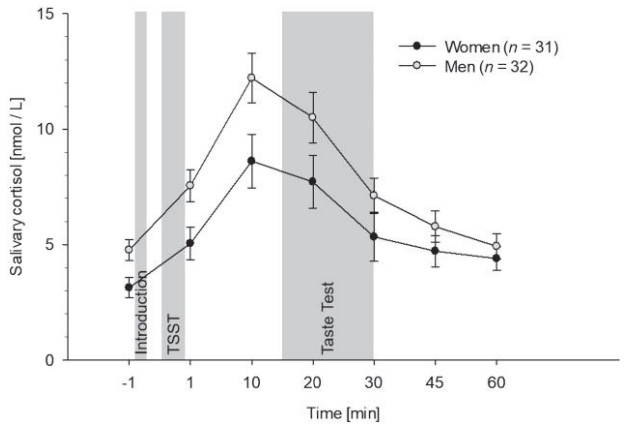

Figure 1. Cortisol reactivity of men and women over the total experimental procedure: stress peak reactivity (S1-S3), immediate reactivity to food intake after stress during the bogus taste test (S3-S5), and after the bogus taste test (S5-S7) (mean ± *SEM*).

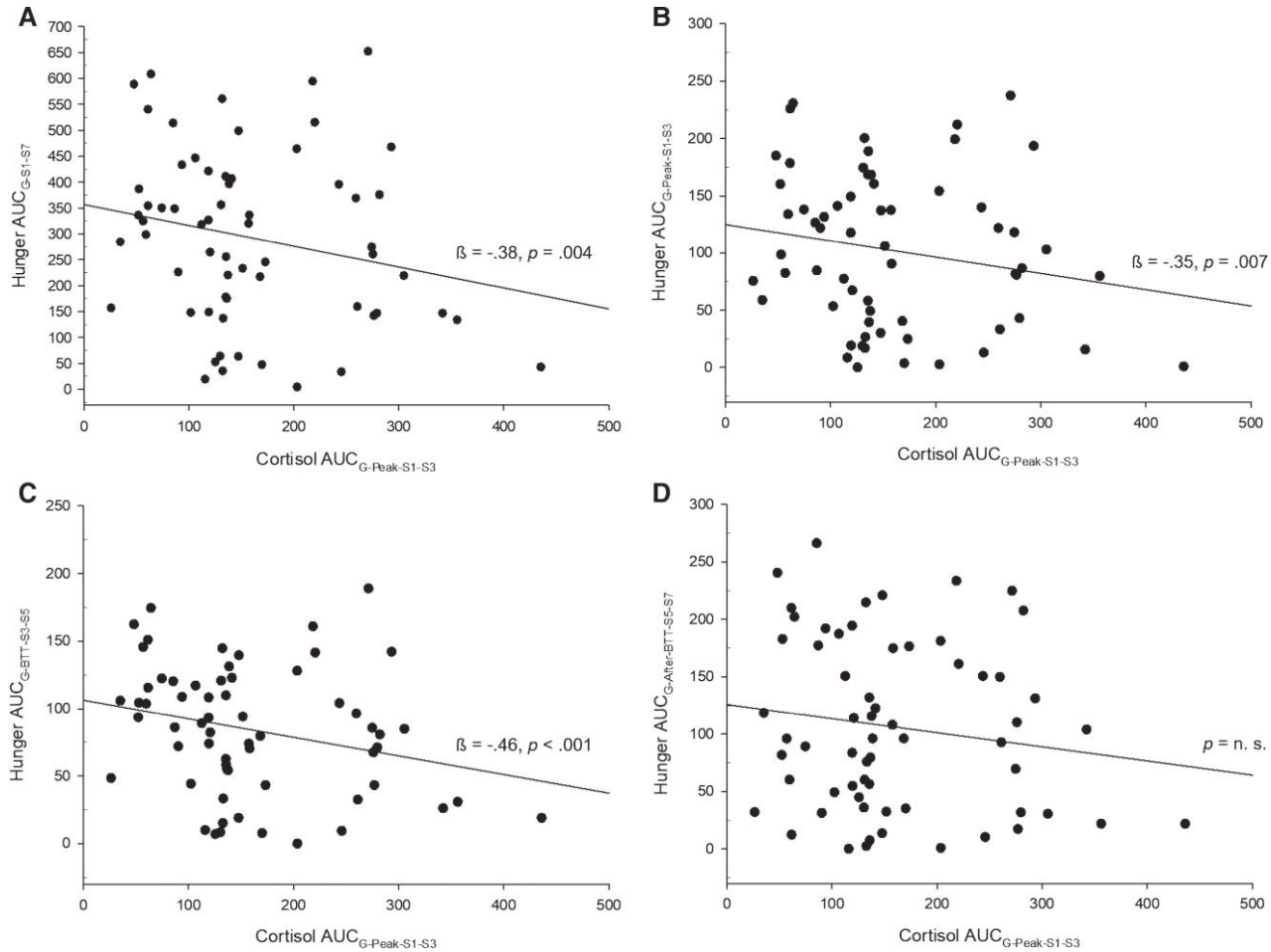

Figure 2. The effects of the immediate total cortisol stress reactivity ($AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$) on hunger over A, the total experimental procedure (Hunger $AUC_{G\text{-}S1\text{-}S7}$); B, during stress peak reactivity (Hunger $AUC_{G\text{-}Peak\text{-}S1\text{-}S3}$); C, in immediate reaction to food intake after stress during the bogus taste test (Hunger $AUC_{G\text{-}BTT\text{-}S3\text{-}S5}$); and D, after the bogus taste test (Hunger $AUC_{G\text{-}After\text{-}BTT\text{-}S5\text{-}S7}$).

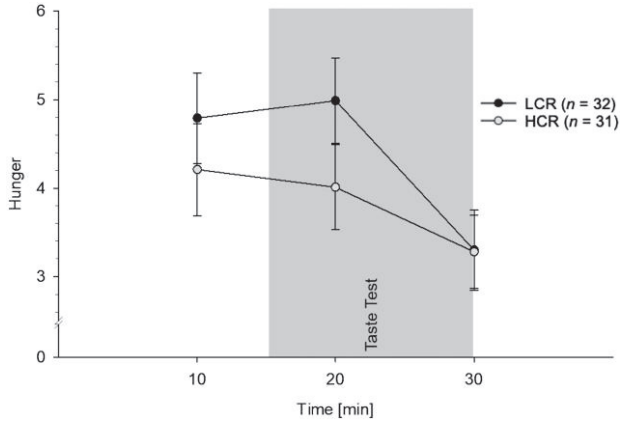

Figure 3. Interaction effect of the immediate total cortisol stress reactivity and hunger in reaction to food intake. The figure depicts hunger levels from S3 to S5 for individuals with immediate total cortisol stress reactivity higher (HCR) and lower (LCR) than median (mean ± *SEM*).

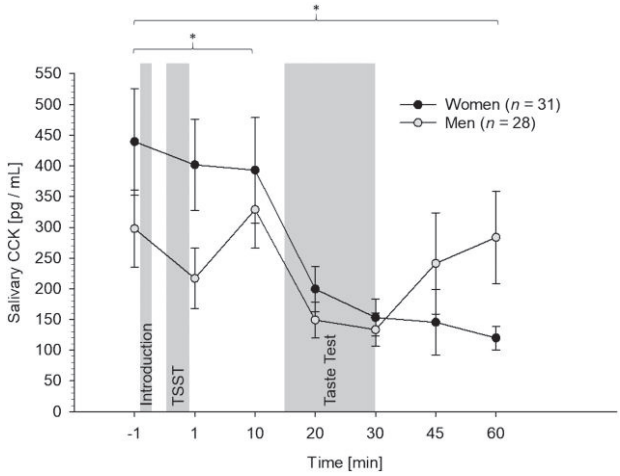

Figure 4. Cholecystokinin (CCK) secretion of men and women over the total experimental procedure: stress peak reactivity (S1-S3), immediate reactivity to food intake after stress during the bogus taste test (S3-S5), and after the bogus taste test (S5-S7) (mean ± *SEM*). Asterisks indicate significant sex differences in the kinetics of CCK secretion (*P less than .05).

women did not significantly differ in their reactivity over the course of the experimental procedure (interaction sex-by-time, S1-S7: $Ps = .72$; see Fig. 5).

Discussion

Here, we set out to investigate endocrine mechanisms underlying hunger and eating after acute psychosocial stress in healthy men and women. We repeatedly assessed the stress hormone cortisol and the satiety hormone CCK from saliva measured before and up to 60 minutes after stress induction in terms of the TSST. Our first study aim was to investigate whether the immediate total cortisol stress reactivity relates to food intake after stress, as well as to the reactivity of CCK and sensation of hunger in immediate reaction to stress induction, in reaction to the BTT, and after the taste test.

Our results showed for the first time that lower immediate total cortisol stress reactivity in response to the TSST significantly related to a higher perceived hunger over the entire course of the experimental procedure, and in particular, in immediate response to the TSST, in response to the taste test and thus eating, and toward a trend level of significance after food intake. Our results point to potential endocrine underpinnings of previous study findings of reduced feelings of appetite (24, 27, 28), hunger (29), and desire to eat (30) after acute stress as compared to nonstress. A reduced feeling of hunger and food intake after acute stress has been interpreted as an appropriate and adaptive reaction, allowing the body to prepare for a fight-or-flight response (eg, (55, 56)), which according to our findings may be regulated by the amount of the cortisol stress response. Moreover, our results on a negative association between cortisol and hunger are in line with evidence from patients with seasonal depression who have both attenuated circadian cortisol secretion (57) on the one hand and increased appetite and weight gain on the other hand (58). The effects of the immediate total cortisol stress reactivity on hunger might not, however, be independent of the type of stressor, as a previous study using the Cold Pressor Test and thus a physical stressor to induce stress found a positive association between cortisol stress reactivity and hunger in overweight women (31).

In contrast to our hypotheses and to the previously described results regarding hunger, immediate total cortisol

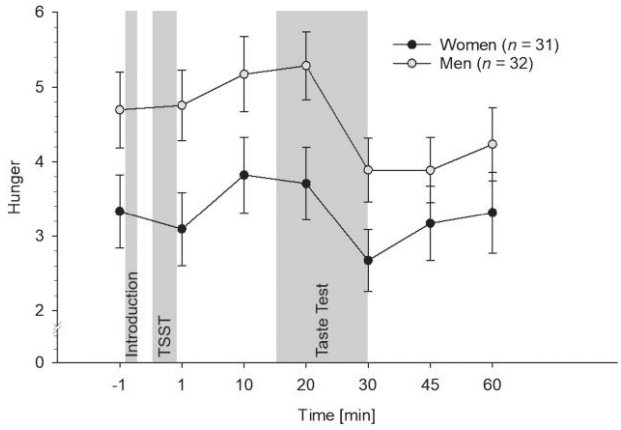

Figure 5. Hunger of men and women over the total experimental procedure: stress peak reactivity (S1-S3), immediate reactivity to food intake after stress during the bogus taste test (S3-S5), and after the bogus taste test (S5-S7) (mean ± *SEM*).

stress reactivity was not related to the amount of food intake after stress. Most previous studies were able to demonstrate a relationship between the stress-induced cortisol response and the amount of food intake in (on average) normal-weight women (22, 25, 26) and obese participants (23, 24), although the direction of the associations varied considerably. In line with our results, a previous study in lean participants could not find differences concerning eating behavior after acute stress in cortisol high reactors as compared to cortisol low reactors (23). In that study, participants were encouraged to eat by reminding them that they had not eaten since morning and by telling them that uneaten food would be discarded (23). It is possible that such statements put the participants under pressure, resulting in biased food intake notably in lean participants. Although our BTT was successfully used in an earlier study in the context of stress and eating (52), our participants may not have felt comfortable eating as much or as little as they wanted in the time-limited taste test, possibly leading to a bias in our results.

In line with a previous TSST study (18), in which reactivity of the satiety hormone leptin was unrelated to cortisol reactivity, we could not find an association between immediate total cortisol stress reactivity and CCK release, indicating that cortisol release during acute stress might not relate to satiety hormones in general. We interpret these findings in that the negative effects of the immediate total cortisol stress reactivity on hunger after acute stress seem to be mediated differently, most likely on a more central level. More precisely, food intake and energy homeostasis are regulated centrally, mainly in the arcuate nucleus of the hypothalamus (59-61). The arcuate nucleus comprises among others the orexigenic peptides neuropeptide Y and agouti-related peptide, which both stimulate food intake (60, 61). Evidence from a rodent study suggests that stress-induced hypothalamus-pituitary-adrenal axis activation may reduce the number and expression of agouti-related peptide-producing cells and thus act as an anorexigenic substance (13, 62).

Our second study aim was to test for potential sex differences in CCK and hunger in reaction to our experimental procedure and quantity of food intake after acute stress. In line with our hypotheses and with previous studies pointing to lower food intake in women in general (33, 34, 40) and after acute stress (40), women ate significantly less ice cream during the taste test and reported a lower perception of hunger over the course of the experimental procedure than men. Regarding men's higher body weights and consequently greater daily energy requirements (40), these results have not been surprising. Notably, there were no reactivity differences in hunger in reaction to the experimental procedure between men and women. Our recruiting procedure with women recruited in the luteal phase of the menstrual cycle (but with 3 exceptions) was intended to prevent sex differences in the kinetics of the cortisol stress reactivity. Indeed, despite a main effect of sex with women showing overall lower cortisol levels and thus a lower immediate total cortisol stress reactivity, the kinetics of the reactivity of cortisol in response to stress, that is, the interaction sex-by-time, did not differ between men and women. Given this comparable cortisol stress reactivity and given that a previous study reported higher psychological reactivity to the TSST in women compared with men (63), we do not assume that the TSST was less stressful for women than for men. Notably, women in general (except for women without hormonal contraceptives in the luteal phase of the menstrual cycle) show lower cortisol responses to acute stress than men (47-49). This generally lower cortisol reactivity in women together with our results of lower immediate total cortisol stress reactivity (even in the luteal phase) and the observed negative association between immediate total cortisol stress reactivity and perceived hunger, may suggest that women have a lower stress-induced hunger inhibition than men. Future studies are needed to test if the lower total cortisol reactivity to stress in women induces a lower hunger inhibition compared to nonstress that in turn puts women at higher risk for stress-related eating.

With respect to CCK, men and women differed in the kinetics of CCK over the course of the experiment. This difference was evident at baseline, with women having slightly higher CCK levels than men (of borderline significance). This finding is in line with previous studies pointing to higher gastrointestinal hormone levels (42, 43), including CCK (44, 45), in women. The difference in CCK between men and women became more evident during the immediate response to stress, with women's CCK levels constantly decreasing while men's CCK levels showed a stress reaction with levels decreasing immediately after stress and recovering until 10 minutes after stress cessation. In addition, we found that CCK further decreased in women after eating while it tended to increase in men. This finding differs from a previous study in which women had a greater prandial CCK secretion than men (44). Notably, in that study there was no stress induction before eating, which may point to a potential effect of previous acute psychosocial stress induction. Taken together, our results with consistent decreases and lack of CCK reactivity, both in reaction to acute stress and to eating after stress in women, suggest that the CCK secretion seems to be less reactive in women than in men. Accordingly, women could be at higher risk for stress-induced eating, as the observed missing CCK increases, in particular in response to eating (60), could possibly lead to a lower satiation (60) and thus prevent the termination of food intake (14). Notably, additional controlling for the menstrual cycle phase regarding analyses of sex differences in the amount of food intake, hunger, and CCK did not substantially change results except that the main effect of sex in hunger (S1-S7: with covariates age and BMI: $F(1,59)= 5.38$; $P = .024$; $\eta_p^2 = .08$; $f = .29$; with covariates age, BMI, and menstrual cycle phase: $F(1,58) = 3.01$; $P = .088$; $\eta_p^2 = .05$; $f = .23$) became borderline significant.

Taken together, despite healthier general eating habits compared to men (ie, by eating less (33, 34) and choosing healthier foods (35, 36)), our findings on immediate total cortisol stress reactivity and related hunger combined with the satiety hormone CCK suggest that women might be at higher risk for stress-related eating after acute stress. Indeed, studies investigating the effects of chronic stress on eating behavior confirm that women are at higher risk for increased stress-related food consumption (4, 64-66) and weight gain (67). In this context, uncontrolled and emotional eating in women but not in men has been proposed to play a mediating role (64). However, whether our findings point to potential endocrine mechanisms underlying higher stress-related obesity risk in women remains to be elucidated. With respect to clinical implications, light therapy has been shown to elevate low cortisol levels in the morning (68) and to reduce body fat and appetite in overweight women (69). Future studies are needed to investigate the effects of light therapy in the context of stress-related eating.

Strengths of our study include the fact that our study sample comprised men and women, allowing us to test for sex differences in eating after acute stress. Further, we invited female participants in the luteal phase of their menstrual cycle to allow for comparable cortisol stress reactivity between men and women (47-49).

A limitation of our study is the use of a cover story (ie, the BTT) to encourage the participants to eat after stress induction, which may have led to a bias in our results. Our study design without a nonstress control condition was not intended and consequently did not allow us to investigate direct stress effects on the amount food intake as well as hunger or CCK kinetics compared to nonstress. Therefore, investigation of direct stress effects in these measures warrants further research. Also, whether our findings are generalizable to populations other than mostly normal-weight healthy young men and women such as obese individuals remains unclear. Indeed, appetite and satiety hormones are suggested to be differentially regulated in obese individuals (59) and potential

stress effects need to be elucidated in this context. In addition, future studies are also needed to examine psychological variables that may affect the effects of stress on eating behavior such as restrained eating (8, 66, 70, 71), emotional eating (8, 29, 40, 71-73), or stress eating (52, 74, 75). Finally, in our study, participants were required to start eating 15 minutes after stress cessation to elucidate eating during cortisol peak stress reactivity. Testing of later effects of stress on eating after cessation of the cortisol response remains to be elucidated.

Taken together, we found evidence for a lower immediate total cortisol stress reactivity relating to higher perceived hunger but not to food intake or the satiety hormone CCK. Further, CCK levels of women continuously decreased while those of men were more reactive to acute stress and eating after stress. Given the observed lower cortisol levels in women (main effect of sex), the negative association between immediate total cortisol output in reaction to stress and hunger together with the observed sex differences in the satiety hormone CCK (with constantly decreasing levels of the satiety hormone CCK in women) may point to a potential mechanism underlying the higher risk for stress-induced eating in women. Future studies are needed to further elucidate and to better understand the endocrine underpinnings of stress-related obesity risk, especially in women.

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Disclosures

The authors have nothing to disclose. There are no conflicts of interest.

Data Availability

Data available on reasonable request from the authors.

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