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Cortisol reactivity and distress-induced emotional eating

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Summary Animal studies suggest a relationship between blunted HPA-axis stress reactivity and increased stress-induced food intake in chronically stressed animals. Such a relationship can potentially explain the underlying mechanisms of emotional eating in humans. However, no studies have experimentally tested the relationship between stress-induced cortisol responses and acute food intake in high and low emotional eaters. We studied these effects in 46 female students that were preselected on the basis of extremely high (HEE) or low (LEE) scores on an emotional eating questionnaire. Using a within subject design we measured the difference of actual food intake after a control or a stress task (Trier Social Stress Test). The HEE and LEE groups did not differ in their cortisol stress reactivity but emotional eating significantly moderated the relationship between cortisol stress reactivity and the difference of food intake after stress vs control. Whereas HEE participants with a blunted cortisol stress response ate more food after distress than those with an elevated cortisol stress response, LEE participants showed no such relationship. These findings support the relevance of an animal based model on the relationship between a blunted cortisol stress response and increased stress-induced food intake for human high emotional eaters.

1. Introduction

Negative mood or distress is associated with both increased and decreased food intake (Greeno and Wing, 1994), with eating less being the typical and predominant response (Heatherton et al., 1991; Stone and Brownell, 1994; Gold and Chrousos, 2002). Distress is normally associated with activation of the hypothalamic pituitary adrenal (HPA) axis, with physiological reactions that are designed to prepare the individual for a fight or flight reaction. These adaptations include inhibition of gastric motility and the release of sugar into the bloodstream thereby suppressing hunger (Gold and Chrousos, 2002). However, so-called emotional eaters show an atypical response and eat the same amount or even more during distress (Oliver et al., 2000; Van Strien and Ouwens, 2003; Van Strien et al., 2012a). This opposite pattern may be the result of changes in the stress reactivity of the HPA-axis related to chronic stress as indicated by changes in responses of the stress hormone cortisol (e.g., Fries et al., 2005; Dallman, 2010). Revealing such a relationship in humans would be highly relevant as it may provide an explanatory mechanism

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for human emotional eating (Dallman et al., 2003a,b; Gibson, 2006; Van Strien et al., 2012a).

To our knowledge there are no previously published studies that have experimentally tested the relationship between stress-induced cortisol responses and actual acute stress-induced food intake in high and low emotional eaters. Animal studies have shown that chronically stressed rodents or rhesus monkeys who are allowed to eat calorie-dense food develop greater mesenteric fat, which in turn dampens the activity of the HPA-axis (Pecoraro et al., 2004; Arce et al., 2009; Dallman, 2010). Also in humans, it has been shown that chronically stressed people report higher scores on emotional eating, have a greater abdominal fat distribution and have dampened HPA-axis activity (Tomiyama et al., 2011). The latter authors hypothesized that highly stressed humans tend to cope with high levels of stress by engaging in stress eating, thereby developing a blunted HPA-axis responses. The evidence from this study is however largely cross-sectional and it remains to be tested whether low cortisol stress reactivity in those high emotional eaters is in turn associated with increased stress-induced food intake. Also, not all studies report reduced cortisol stress responses in emotional eating. Epel et al. (2004) and Raspopow et al. (2010) reported *increased* cortisol stress responses in emotional eaters in the context of an exam period and the Trier Social Stress Test (TSST, Kirschbaum et al., 1993), respectively. However, although blunted cortisol stress responsiveness is not necessarily a trait characteristic in emotional eaters it may still be the case that those high emotional eaters that do show blunted HPA-axis stress reactivity are the ones who show increased stress-induced food intake.

The aim of the present study was to test this animal based model of emotional eating (Dallman, 2010) in humans by assessing the relationship of the difference in food intake following a laboratory stress task or control task with cortisol reactivity in high vs low emotional eaters. Earlier, emotional eating was found to significantly moderate the participants' relation between distress and food intake, with low emotional eaters eating less after the stress than after the control task and high emotional eaters eating more (Van Strien et al., 2012a). For the present study we hypothesized that relative to low emotional eaters, high emotional eaters would show a negative association between cortisol stress reactivity and food intake after stress. Specifically: high emotional eaters with a blunted cortisol stress response were expected to eat more after the stressor than those with the typical elevated cortisol stress response.

2. Method

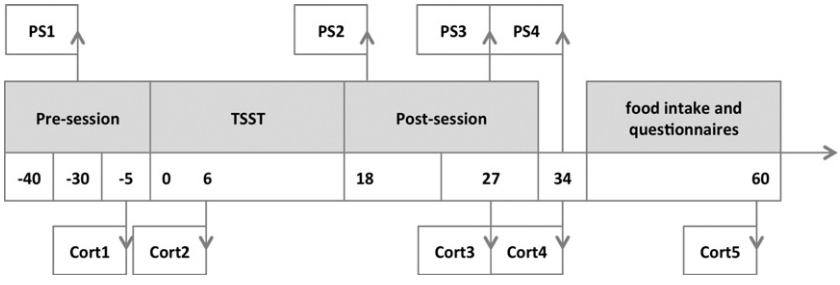
2.1. Sample

Participants were recruited from a pool of female students taking introductory psychology or pedagogy courses who had completed the emotional eating scale of the Dutch Eating Behaviour Questionnaire (DEBQ; Van Strien, 2010) and a questionnaire on exclusion criteria. Exclusion criteria included taking medications that could influence the cortisol response. Because most Dutch female students use hormonal contraceptives, it was not possible to exclude participants using hormonal contraceptives, so the possible confounding effect of use of hormonal contraceptives was controlled for. Participants with DEBQ scores below 1.82 or above 3.25 (corresponding to the 20th and 80th percentiles of the Dutch norm group of females) were invited to participate in a study on 'health and physiology'. A total of 47 female students agreed to participate. However, cortisol values of one high emotional eater turned out to be invalid so our final sample had 46 participants, 23 low and 23 high emotional eaters (LEE and HEE, respectively). The mean age of the sample was 19.68 years (SD = 1.86) and the mean body mass index (BMI = weight (kg)/height (m^2) was 21.27 (SD = 2.66). The study protocol was approved by the ethical board of the Faculty of Social Sciences of the Radboud University Nijmegen (ECG 29042010).

2.2. Procedures

Two laboratory sessions were completed on consecutive weekdays. Participants were instructed to wake up at least two and a half hours before the experiment and to refrain from intake of alcohol or drugs. For 1 h prior to the experiment they were not allowed to smoke, to engage in physical exercise (including cycling), to eat or drink (only water was allowed), or to brush their teeth. Experimental sessions were scheduled between 1100 h and 1500 h to minimize the effects of diurnal rhythms on HPA-axis reactivity. This testing time was chosen in order to follow the procedure by Appelhans et al. (2010) who also gave their participants access to food immediately after the task. Each participant was tested at the same time on both days. Upon arrival on the first day participants were asked to fill out an informed consent form.

On the first test day, participants were subjected to the control condition, which followed a similar protocol to that of the stress condition (see below, see also Flow chart 1).



Flow chart 1 Schematic depiction of the stress procedure. Time: start TSST = 0; TSST is Trief Social Stress Test; PS1—PS4; consecutive assessments of perceived stress and hunger; Cort1—Cort5: consecutive assessments of salivary cortisol.

However, in the control condition instead of performing stressful tasks in front of a jury, the participants had to rate six different fabrics (wool, fur, felt, silk, linen, and cotton), on various attributes (e.g., softness, pleasantness, warmth). This task was completed in the same room and for the same amount of time as the stress tasks, with the difference that the participants were left alone in the experimental room during the task. After 15 min the experimenter took them to a separate room to fill out several questionnaires at a table which also held a glass of water and four bowls filled with, respectively, white grapes, pieces of carrot, M&Ms and pieces of butter—cake. The experimenter left the room, saying "Please help yourself to the water and the food. You have earned it.' After 20 min the experimenter returned to take the participants to another room to perform a 15-min computer task (not relevant to the present study).

On the second test day the subjects were subjected to a modified version of the Trier Social Stress Task (Kirschbaum et al., 1993). This distress manipulation involves a social evaluative threat, known to elicit significant cortisol responses in the majority of participants (Dickerson and Kemeny, 2004). Briefly, the task consisted of preparing (5 min) and delivering (5 min) a speech, followed by a serial subtraction task (4 min). The speech and subtraction task were presented in front of a two-person jury who sat behind a table and wore white doctors' coats. Participants were told the task was being videotaped and that they would be judged on the quality of their performance. The participant had to stand in stockinged feet on a Wii© balance board that measured kinesthetic activity (for a research question not relevant to the present study). After the stress task, when the jury had left the room, the participant was asked to fill out a set of questionnaires in that room while waiting for the jury's judgement of the performance — in this manner the stressfulness of the public speaking task was extended by a prolonged period of waiting for the results. After 4 min the experimenter returned to say: '*There has been a problem with the videotape, and it may be necessary to redo the task*' (negative feedback), and left again. After another 5 min the experimenter returned again, this time with a negative feedback on the performance. After a further 5 min the experimenter returned and said that the problem had been solved, and that the jury's judgment was positive. The prolongation of the stress period was done in order to be able to sample post-stress saliva before the participant was offered food. The participant was then taken to a separate room to fill out a further set of questionnaires. Once again the table held a glass of water and bowls with food, and participants were invited to help themselves to the water and the food in the same words as the previous day. After 20 min the experimenter returned to measure the weight and height of the participants in light clothing and stockinged feet. Finally the participant was debriefed, thanked and paid with course credits.

Saliva samples were collected by passive drooling at five identical time points on each study day (see Flow chart 1 for times as related to the protocol). Each sample contained at least 1.5 ml of saliva. At the end of the session the samples were frozen at −20 °C. It should be noted that the experimenter was kept blind to the emotional eating status of the participants and that all participants were unaware that their food intake was being measured. Furthermore, we deliberately chose to always start with the control condition and not to counterbalance the order of the two conditions because we considered that presenting the stress condition first could have unwanted effects. Namely, there could be carry-over effects from the stress to the control condition due to anticipation stress, and also, participants could more often decide to end participation after having gone through the stress session.

2.3. Measures

Eating behaviour was assessed with the Dutch Eating Behaviour Questionnaire (DEBQ; Van Strien, 2010). The DEBQ has 33 items, 13 on emotional eating (e.g., "Do you have a desire to eat when you are irritated?"), 10 on external eating (e.g., "If food smells and looks good, do you eat more than usual?") and 10 on restrained eating (e.g., "Do you try to eat less at mealtimes than you would like to eat"?). All items have to be rated on a 5-point scale with response categories that range from 1 'never' to 5 'very often.' For each of the three scales scores were obtained by dividing the sum of the scores on the individual items by the total number of items on that scale. The three scales have good internal reliability and good construct and predictive validity (Van Strien and van de Laar, 2008; Van Strien et al., 2012a,b). In the present sample, Cronbach's alphas for the scale on emotional eating, external eating and restrained eating were $\alpha = 0.97$, $\alpha = 0.96$ and $\alpha = 0.88$, respectively.

2.3.1. Mood and hunger

On both days, ratings of mood and hunger were measured upon arrival and at three more time points: immediately after the task, after the negative feedback, and after the final positive feedback and during the food intake (see Flow chart 1 for the time scheme). The Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) was used to measure, on a 5-point scale ('not at all' to 'extremely'), the degree to which participants experienced 10 positive and 10 negative affects. The descriptor 'hungry' was inserted among the PANAS items so that hunger could be evaluated without alerting the participants to the true nature of the study.

2.3.2. Food intake

Before and after participants ate, the bowls with grapes, carrots, M&Ms, and butter cakes were weighed with a professional balance (model 200, KERN®). We then translated weight into calories for each food type, and summed the caloric intake over the food.

2.3.3. Cortisol

Saliva samples for cortisol determination were collected at 5 time points during the protocol (see Flow chart 1). The cortisol samples were analysed at the Psychobiology Laboratory of the University of Trier, Germany, using a competitive solid phase time-resolved fluorescence immunoassay with fluorometric end point detection (DELFIA). The inter-assay coefficients of variation ranged between 7.1% and 9.0%; the sensitivity of the assay was 0.173 nmol/l.

Before carrying out the statistical analyses, cortisol data were scrutinized for outliers, defined as 3 SD above or below the mean for each assessment. Out of 460 cortisol assessments, 5 outliers above the mean and 0 below the mean were

determined. These five values were winsorized by replacing the outlying values with the value of 3 SD above the mean.

Cortisol reactivity was calculated by computing the difference between the area under the curve with respect to increase (AUCi) in the stress condition and the AUCi in the control condition (deltaAUC) using the formula for AUCi by Pruessner et al. (2003). In the analyses with cortisol as the dependent variable, the salivary cortisol data were log-transformed to achieve normality of the data.

2.3.4. Confounders

Smoking, use of alcohol and use of oral contraceptives were measured with yes/no questions: 'Do you smoke/use alcohol/use oral contraceptives?' Weekly physical activity was measured with the question: how many days in a normal week do you engage in moderate physical activity for at least 60 min during which your breath goes somewhat quicker than normal (for example doing cycling or walking). Response categories were not a single day (1) to 7 days a week (8), but they were recoded into a dummy variable: $\leq$1 day (=0, n = 28) and $>$1 day (=1, n = 19). For people with ages below 20 years the Dutch Norm for Healthy Physical Activity requires a person to be active for at least 60 min on at least five days a week in summer and winter (Kemper et al., 2000).

2.4. Analytic plan

All analyses were carried out using SPSS version 15.0 (SPSS Inc., Chicago). With repeated measures GLM we conducted various manipulation checks by assessing the effect of time on the various cortisol values in both the control and the stress condition, in addition to the main effect of condition (control vs stress) on the cortisol response over time. The possible confounding effect of use of hormonal contraceptives, smoking, use of alcoholic drinks, and physical activity was controlled for. In the same manner we also assessed the main effect of condition (control vs stress) on the mood response over time. Subsequently, we assessed main effect of emotional eating on the cortisol response over time in both the control and the stress condition, in addition to the emotional eating $\times$ condition interaction effect on cortisol response over time, controlling for the possible confounders. Greenhous-Geisser corrections were applied where appropriate.

Next, we computed the difference between food intake (in kcal) in the stress condition and the control condition, a positive value meaning a higher intake in the stress condition (henceforth: delta kcal). With hierarchical regression analyses we tested the interaction of emotional eating with cortisol reactivity (see cortisol data preparation), on delta kcal. The possible confounding effect of use of hormonal contraceptives, smoking, use of alcoholic drinks, and physical activity was controlled for. In these analyses we also controlled for pre-task hunger (the mean of pre-task hunger on the control and on the stress day.

Because of the high interrelations between emotional eating, external eating and dietary restraint ($.45 < r$'s $< .54$, $p < .001$), we corrected in additional analyses for external eating and dietary restraint. To avoid multicollinearity in the regression analyses, all variables were centred before computing interaction terms (Aiken and West, 1991).

3. Results

3.1. Preliminary analyses

Of the total of 46 participants, 35 reported using hormonal contraceptives, 6 reported smoking, and 40 reported taking alcoholic drinks. On both testing occasions all participants reported waking up at least 2.5 h before arrival at the lab. HEE had significantly higher BMI's than LEE (mean (SD) = 22.10 (3.2) vs 20.25 (1.4); p = .02) and also their scores on external eating were significantly higher (mean (SD) = 3.76 (.47) vs 2.87 (.67); $p < .001$). There were no significant differences between the two groups with respect to dietary restraint (p = .13), use of hormonal contraceptives (p = .74), and weekly physical activity (p = .14). There was, however, a borderline significant difference with regard to drinking (p = .08) and smoking (p = .08), with HEE reporting drinking and smoking more often.

3.2. Manipulation check

3.2.1. Cortisol

Fig. 1 shows the mean uncontrolled cortisol values in the control and stress condition at the five time points.

For both the control and the stress condition there was a significant effect of time on the transformed cortisol values (controlling for the possible confounders) (control condition: $F(1.955, 80.173) = 6.806$, $p < .01$, partial eta squared = .14; stress condition: $F(1.618, 66.356) = 9.810$, $p < .001$, partial eta squared = .19), with the linear model reaching the highest significance in the control condition ($F(1, 41) = 9.651$, $p < .01$, partial eta squared = .19) and the cubic model reaching the highest significance in the stress condition ($F(1,41) = 27.315$, $p < .001$ partial eta squared = .40). There also was a significant overall moderator effect of the stress

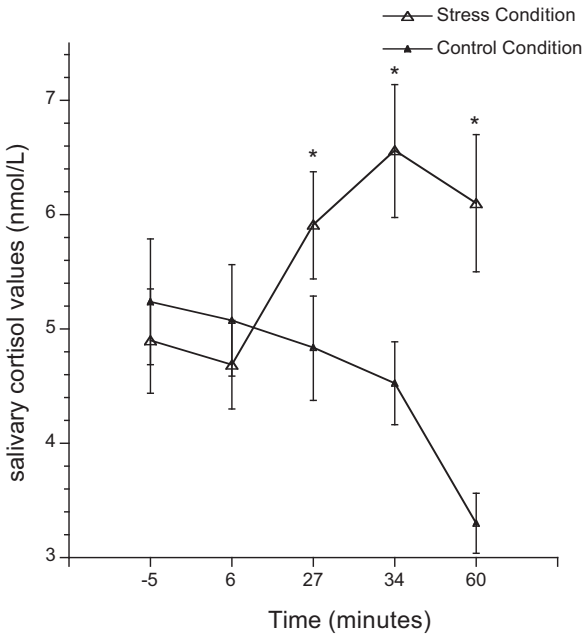


Figure 1 The moderator effect of the stress condition on raw cortisol values over time.

condition on cortisol values over time ($F(5, 37) = 5{,}835$, $p < .001$, partial eta squared = .44) with significant effects for time 4 ($F(1, 41) = 5{,}865$, $p \leq .05$, partial eta squared = .13) and time 5 ($F(1, 41) = 16{,}219$, $p < .001$; partial eta squared = .28. (With transformed cortisol values the significant effect for time 3 (Fig. 1) was no longer present.)

3.2.2. Negative mood

The scales for negative mood at the time points 1−4 showed massive skewness and kurtosis. Only by computing the difference between negative mood in the stress condition and the control condition (henceforth: delta negative mood) the negative mood values could be normalized. Positive values of delta negative mood meant more negative mood in the stress condition. The stress manipulation significantly moderated delta negative mood over time ($F(2.150, 92.429) = 28.982$, $p < .001$, partial eta squared = .401) with the Quadratic model reaching the highest significance ($F(1, 43) = 42{,}419$, $p < .001$, partial eta squared 0.50). The mean (SD) of the delta negative mood values at times 1 through 4 were, respectively:T1: −.14 (.32); T2: .46 (.55); T3: .42 (.62) and T4: .11 (.33). Significant mean differences (Bonferroni corrected) in negative mood were obtained between time points 1 and 2 ($p < .001$), 1 and 3 ($p < .001$), 1 and 4 ($p < .01$), 2 and 4 ($p < .001$) and 3 and 4 ($p < .010$).

3.3. Effect of emotional eating on cortisol values over time

For both the control and the stress condition there were no significant effects of emotional eating on cortisol values over time (controlling for the possible confounders) (p's $> .10$). There also was no emotional eating × condition interaction effect on cortisol response over time, controlling for the possible confounders ($p > .10$). Though differences were not significant, HEE showed lower cortisol values in the stress condition than LEE (see Fig. 2).

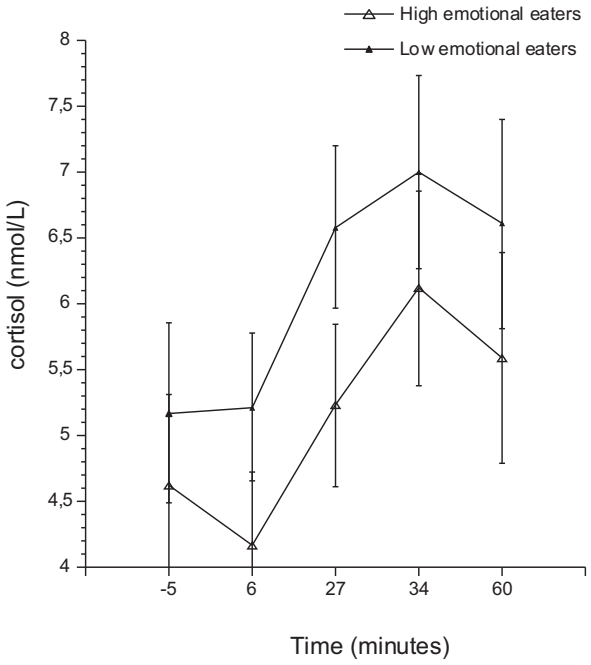


Figure 2 Mean cortisol values of high and low emotional eaters in the stress condition (controlling for the possible confounders).

3.4. Cortisol reactivity, emotional eating and food intake

The mean (SD) of cortisol reactivity (AUCi) in the stress and control condition was 3.13 (9.13) and −2.02 (6.90), respectively. The mean (SD) of deltaAUCi, the cortisol reactivity of the stress minus the control condition, was 5.15 (12.45) with a range of −24.84 to 44.98. A total of 34.8% of the participants ($n = 16$) had values below zero. The mean food intake (kcal) in the control condition did not significantly differ from the mean food intake in the stress condition (mean (SD) = 169.57 (130.04) vs 157.76 (181.86), $p = .80$). This also held for the types of food consumed. The quantities in grams for the control vs stress conditions were the following: carrots, mean (SD) = 7.43 (14.67) vs 5.66 (12.70); grapes, mean (SD) = 39.64 (42.90) vs 37.67 (41.56); butter cake, mean (SD) = 19.35 (18.69) vs 17.19 (21.95); M&Ms, mean (SD) = 10.87 (15.04) vs 14.57 (24.63), all p's $> .10$.

Table 1 shows the partial correlations (controlling for the confounders) of AUCi stress, AUCi control and overall cortisol reactivity (deltaAUCi) with food intake in the control condition, food intake in the stress condition, emotional eating, external eating and dietary restraint. Please note the significant partial correlations of AUCi control, AUCi stress and overall cortisol reactivity (deltaAUCi) with food intake in the stress condition ($r = .37$, $p = .01$; $r = -.32$, $p = 045$; $r = -.48$, $p = .002$, respectively).

With hierarchical regression analyses we tested the interaction of emotional eating with cortisol reactivity (deltaAUCi) on delta kcal (the difference between food intake in the stress and the control condition). In addition to the possible cortisol confounders, we also controlled in these analyses for pre-task hunger.

There was a significant effect for cortisol reactivity (deltaAUCi) on delta kcal (unstandardised coefficient (B) = −8.466, $p < .001$). There was a borderline significant effect for emotional eating on delta kcal ($B = 82.638$, $p = .072$). There also was a significant moderator effect of

Table 1 Partial correlations of AUCi control, AUCi stress and cortisol reactivity (deltaAUCi) with food intake and the eating styles (controlling for the confounders).

	Food intake control	Food intake stress	Emotional eating	External eating	Restrained eating
AUCi control	−0.22	0.37*	0.23	0.23	−0.12
AUCi stress	−0.21	−0.32*	−0.12	−0.12	−0.21
deltaAUCi	0.005	−0.48*	−0.24	−0.23	−0.06

* $p < .05$.

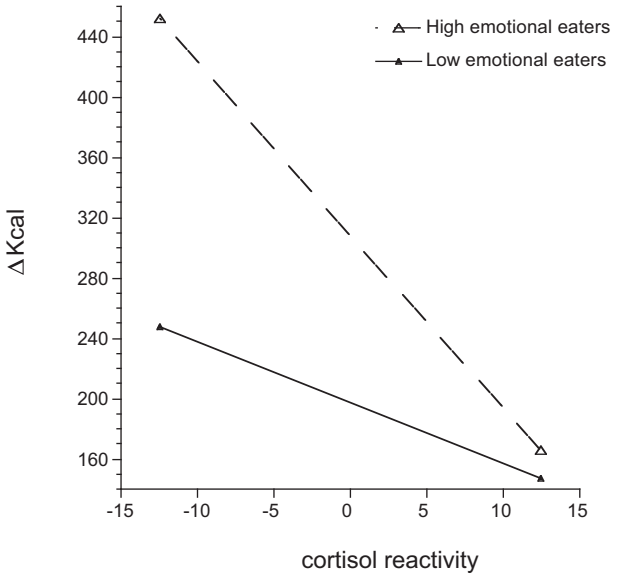


Figure 3 Regression lines depicting levels of cortisol reactivity for the high and low emotional eaters in relation to delta kcal (food intake (kcal) in the stress condition minus food intake in the control condition) showing that high emotional eaters with blunted cortisol responses have relatively increased food intake after stress.

emotional eating on the relation of cortisol reactivity with delta kcal ($B = -.8.094$, $p = .013$; R^2 change = .093), which remained significant when we additionally controlled for dietary restraint and external eating ($B = -7.467$; $p = .028$; R^2 change = .075). (In the full model the main effect for cortisol reactivity on delta kcal remained significant ($B = -8.273$, $p = .001$) and the main effect for emotional eating on delta kcal became significant ($B = 125.444$, $p = .045$.) The R^2 associated with the final model was .50.

In a post hoc hierarchical regression analysis on the full model the nature of this interaction effect was determined (see Holmbeck, 2002). The regression for high emotional eating indicated a negative association of cortisol reactivity on delta kcal ($B = -11.525$; $p < .001$). In contrast, results of the regression for low emotional eating indicated no significant association of cortisol reactivity on delta kcal ($B = -0.308$; $p = .139$). Regression lines depicting levels of cortisol reactivity for the high and low emotional eaters in relation to delta kcal are plotted in Fig. 3.

For illustrative purposes, Table 2 shows the food intake data (kcal) in the stress vs control condition for HEE and LEE with a blunted vs typical cortisol stress response (controlling for the confounders). The classification of participants with a blunted vs typical cortisol stress response was obtained by artificially dichotomizing cortisol stress reactivity by means of a median split (note, however, that this procedure is notoriously vulnerable to misclassification of research participants and spurious effects; Maxwell and Delaney (1993).

4. Discussion

In the present study on female students with extreme scores on emotional eating, we tested the hypothesis that high emotional eaters with a blunted cortisol stress response would eat more after the stressor than those with the typical elevated cortisol stress response. This hypothesis was indeed supported: in HEE there was a negative association of cortisol reactivity with delta kcal (food intake in the stress condition minus food intake in the control condition). In contrast, in LEE cortisol reactivity was not significantly associated with delta kcal.

The finding that high emotional eating in combination with *low* cortisol reactivity was associated with higher stress-induced food intake, seems contradictory to outcomes of a study by Newman et al. (2007), where emotional eating in combination with high cortisol stress reactivity in women was associated with higher snack intake. However, in that study snack intake was measured by means of a fourteen-day snack intake diary as opposed to the objective post-stress measures of the present study. Different mechanisms may be at play if the period between the stressor and the food intake is prolonged. A further difference is that we studied these effects in female students with extremely high or low scores on emotional eating. These methodological differences may explain the discrepancy between the present findings and those of Newman et al. (2007). Our present findings are, however, in line with those of Tomiyama et al. (2011), who found that high stress women (caregivers of chronically ill children) reported more emotional eating and showed a blunted cortisol stress response in comparison with low stress women (caregivers of healthy children).

Also, the present trend ($p < .10$) suggesting that food intake after distress (regardless of emotional eating status) was negatively related to cortisol reactivity seems at odds with two studies finding positive associations between cortisol reactivity and food intake (Epel et al., 2001; Newman et al., 2007). As in the present study, Epel et al. (2001) assessed actual post-stressor food intake, but they conducted their study between 1600 h and 1730 h. Our study was

Table 2 Mean (*M*) and standard error (SE) of the total food consumption (kcal) in the control and stress conditions split by emotional eating (low vs high) and cortisol stress reactivity (low-blunted vs high-typical) controlling for hunger, smoking, drinking, physical activity and use of oral contraceptives.

Emotional eating	Cortisol stress reactivity	Control-day		Stress-day	
		M	SE	*M*	SE
Low	Low ($n = 10$)	226.44	44.51	177.34	55.74
	High ($n = 13$)	144.80	38.13	88.45	47.75
High	Low ($n = 13$)	164.61	38.13	310.39	48.10
	High ($n = 10$)	151.36	44.23	112.65	55.39

conducted at midday, and it is possible that associations between cortisol reactivity and food intake are affected by time of day. The present findings, however, support those of Appelhans et al. (2010) and Raspopow et al. (2010) where food intake was also negatively associated to cortisol reactivity to the TSST.

Our results may be related to the food being offered immediately after the stressor (1 min after finalization and 34 after initiation of the stressor), when the activity of the HPA axis was still high. Hypothalamic corticotropin-releasing hormone (CRH) is secreted early in the HPA axis cascade, resulting in later secretion of adrenal cortisol. CRH has a potent anorectic effect (Mastorakos and Zapanti, 2004). Cortisol has a suppressing effect on the production of CRH, but this effect would only begin to work 35—45 min after stressor onset (Dallman, 2003). Hence, the CRH secretion had probably still not been inhibited when the post-stressor food was offered, and therefore, the anorectic effects of CRH were not yet suppressed. In other words, while HEE with low cortisol reactivity might immediately engage in post-stressor food intake, HEE with high cortisol reactivity might do this only later, when the anorectic effects of CRH have been suppressed. More research into the links between HPA axis physiology and the timing of post-stressor food intake clearly needs to be done to elucidate these issues further.

The mechanism proposed above constitutes a proximal explanation of the negative links between cortisol reactivity and food intake. However, the patterns found can also be explained as the result of long-lasting physiological alterations. Namely, the present findings with HEE could be in support of Dallman's (2010) chronic stress response network model. According to this model, highly stressed people may have coped with high levels of stress by engaging in stress eating, thereby developing blunted HPA axis responses as a reflection of an adaptive downregulation secondary to emotional overeating. Alternatively, a blunted cortisol response may also have been primary, instead of secondary, to emotional eating. A blunted cortisol response has been related to adverse early life experiences (Elzinga et al., 2008). In this line of thought emotional eating would be the consequence of a lowered HPA axis functioning, which would explain why emotional eaters are more receptive to the reinforcing value of food and use food as 'self-medication' to blunt effects of negative emotions.

4.1. Strengths and limitations

A strength of the present study is the use of a within subject design where participants acted as their own control group. A further strength is that the use of groups with extreme scores on emotional eating is associated with higher efficiency of detecting interaction effects (McClelland and Judd, 1993). Additionally, the use of groups with extreme scores provides interesting insights into the physiology of women with (pre-) clinical levels of emotional eating. A further strength is that in all the analyses we controlled for the other two eating styles, so the present moderator effect of emotional eating on the relation between cortisol reactivity and food intake appears to be a robust, independent effect.

With respect to limitations, the relatively small sample size may have reduced the power to reveal small (interaction) effects. A further consideration is that a large number of our participants were tested at the end of the morning, and we cannot rule out the possibility that the stress induced cortisol response is somewhat blurred due to the diurnal decline in the morning. Additionally, emotional eating has been closely associated with binge eating, depressive feelings, poor impulse regulation and alexithymia (Van Strien et al., 2005, 2010; Ouwens et al., 2009). Therefore, it is highly probable that our subjects with high emotional eating had other psychosocial symptomatology such as depressive symptoms. This is a limitation of this study that should be addressed in future studies with larger numbers of participants. Another limitation is that the present experiment was conducted on predominantly normal weight female emotional eaters, so the study needs replication with high and low emotional eaters with overweight. Because we used females only, it remains to be seen whether the same results would hold for men. A further limitation is that we cannot rule out the possibility that social desirability or acquiescence may have affected scores on emotional, external and restrained eating. Finally, the present findings would need replication outside the laboratory, for example by using field stressors and assessing post-stressor food intake by means of snack intake diaries in the hours following the stressor.

5. Conclusion

Whereas HEE participants with a blunted cortisol stress response ate more food after distress than those with an elevated cortisol stress response, LEE participants showed no such relationship. These findings would suggest support for the relevance of an animal-based model on the relationship between blunted cortisol stress responses and increased stress-induced food intake for human high emotional eaters.

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Conflict of interest

Tatjana van Strien has a copyright and royalty interest in the Dutch Eating Behaviour Questionnaire (DEBQ) and manual.

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