



Cortisol reactivity and distress-induced emotional eating

Tatjana van Strien^{a,*}, Karin Roelofs^b, Carolina de Weerth^b

^a Behavioural Science Institute and Institute for Gender Studies, Radboud University Nijmegen, Nijmegen, The Netherlands

^b Behavioural Science Institute, Radboud University Nijmegen, Nijmegen, The Netherlands

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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.

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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

* Corresponding author. Tel.: +31 243615596.
E-mail address: T.vanStrien@psych.ru.nl (T. van Strien).

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 (Tomiya 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 Rasopow 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

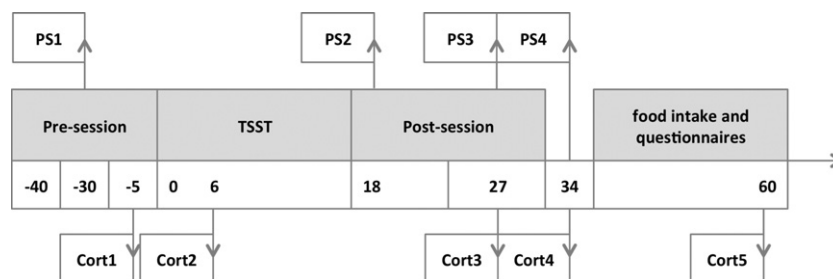
2.1. Sample

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 (m²) 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 Trier Social Stress Test; PS1–PS4; consecutive assessments of perceived stress and hunger; Cort1–Cort5: consecutive assessments of salivary cortisol.

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