Daily hassles and eating behaviour: The role of cortisol reactivity status

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Summary
Previous research has shown high cortisol reactors to consume a greater amount of snack foods than low reactors following a laboratory stressor. The current study tested whether high cortisol reactors also consume more snacks than low reactors in response to field stressors. Fifty pre-menopausal women completed a laboratory stressor, provided saliva samples to assess cortisol reactor status and then completed daily hassles and snack intake diaries over the next fourteen days. Hierarchical multivariate linear modelling showed a significant association between daily hassles and snack intake within the overall sample, where an increased number of hassles was associated with increased snack intake. This significant positive association between number of hassles and snack intake was only observed within the high cortisol reactors and not within the low cortisol reactors. These findings suggest that high cortisol reactivity to stress promotes food intake. Furthermore, the eating style variables of restraint, emotional eating, external eating and disinhibition were more strongly associated with snack intake in high reactors than in low reactors. This suggests that cortisol reactivity may in part account for the moderating role of eating style on stress-induced eating. The results are discussed within the context of future health risk.

1. Introduction
It is becoming more apparent that stress and negative affect not only have direct effects on health but also indirect effects through behavioural changes, including changes in the type and amount of food consumed (e.g., Macht and Simons, 2000; O’Connor et al., 2000; O’Connor and O’Connor, 2004). Laboratory and self-report studies demonstrate that individuals respond differently in their eating response to stress with gender, bodyweight and the eating style variables of restraint, emotional eating, external eating and disinhibition acting as significant moderators of the stress–eating relationship (McKenna, 1972; Herman and Polivy, 1975; Grunberg and Straub, 1992; Greeno and Wing, 1994; Conner et al., 1999; Oliver et al., 2000; Van Strien et al., 2000; O’Connor et al., 2005). Although, while research has
identified a number of important moderators of stress-induced eating, relatively little is known about the underlying mechanisms.

One possible mechanism for stress-induced eating concerns the activity of the hypothalamic–pituitary–adrenal axis during stress, particularly the release of glucocorticoids from the adrenal cortex. Sapolsky (1998) proposed that corticotropin releasing hormone (CRH) and glucocorticoids (GC) have opposing effects on appetite, such that food intake is inhibited by CRH and promoted by GC production. Direct manipulations of GC levels support their association with appetite and food intake. Adrenalectomised rats unable to secrete GC have been shown to consume smaller amounts of carbohydrate relative to other macronutrients (Laugero, 2001), and GC appears to protect against the hypophagic effects of leptin (Zakrzewska et al., 1997). In humans, the administration of glucocorticoids in humans has been shown to increase energy consumption, especially carbohydrates and proteins (Tataranni et al., 1996).

Furthermore, the release of GC during stress has been associated with increased snack intake. In a laboratory investigation of snack intake in women after stress exposure, Epel et al. (2001) reported that during stress recovery high cortisol reactors consumed more than low reactors, especially of high fat, sweet foods. Therefore individual differences in the stress–eating response could be dependant on GC activity to stress, such that high cortisol reactors consume a greater amount when stressed than do low reactors. As yet, this cortisol reactivity theory of stress-induced eating has only been tested in the laboratory and not in the field. To test whether the effect is limited to the laboratory it is essential to replicate and extend Epel et al.’s (2001) findings in a more natural setting.

Field studies of stress-induced eating usually require individuals to complete diary records of workload, hassles and food intake (e.g., Steptoe et al., 1998; Conner et al., 1999), allowing the researcher to measure natural eating behaviour in response to real-life stressors. Previously, diary studies of stress-induced eating have compared overall snack intake across high and low stress weeks (e.g., Steptoe et al., 1998). However, by averaging intake across days and weeks subtle daily variations in stress and intake may be lost. Multivariate linear modelling enables researchers to test between-person associations and within-person daily variations in measures (Affleck et al., 1999), and would therefore facilitate an investigation of the relationship between daily stress and snack intake. Despite this advantage, only one previous study has employed this method of analysis to examine fluctuations in intake with daily stress (O’Connor et al., 2005).

The present study aimed to test whether the relationship between hassles and snacks outside the laboratory differs between high and low cortisol reactors as an extension of Epel et al.’s (2001) study and a test of whether GC release could account for variations in stress-induced eating. The study also aimed to test whether the relationship between eating style and snacking differed according to cortisol reactivity status. Following the procedure of Epel et al. (2001), pre-menopausal women were exposed to laboratory stressors to establish cortisol reactivity status and required to report daily hassles and snack intake in diaries over 2 weeks. Because of reported gender differences in cortisol reactivity to stress (e.g., Kudielka and Kirschbaum, 2005) and a greater prevalence of stress-induced food intake in females (e.g., Grunberg and Straub, 1992), only females were included in the current study. It was predicted that high cortisol reactors would show a stronger positive association between daily hassles and snack intake than low reactors.

2. Methods

2.1. Participants

Fifty-five pre-menopausal women completed the laboratory tasks. Of these, four did not return both diaries and one woman did not produce sufficient saliva for analysis. Therefore complete data were obtained and is reported from 50 women. The participants had a mean age of 33.96 years (SD = 6.18) and mean body mass index (BMI) of 23.34 (SD = 3.62). Participants were recruited using a County Council Bulletin in an advert to take part in a ‘Women and Eating Study’. Exclusion criteria were based on factors previously shown to affect baseline or reactive cortisol levels. Participants were not included in the study if they had been diagnosed with a neuroendocrine or metabolic disorder, had a history of eating disorders or depression (Ellenbogen et al., 2002), were postmenopausal or taking oral contraceptives (Kirschbaum and Hellhammer, 1989; Pruessner et al., 1997). Menstrual cycle phase was left random, although cortisol levels are reportedly higher around the luteal phase (Kirschbaum et al., 1999). Participants were paid 20 pounds (approximately $36) for completing all parts of the study.

2.2. Procedure

Participants’ cortisol reactivity was individually tested in the laboratory in the afternoon, to control for the waking response (Pruessner et al., 1997) and because cortisol stress reactivity is greater in the afternoon (Kirschbaum and Hellhammer, 1989). Moreover, time of day has also been found to influence levels of anxiety during stressful encounters (Willis, O’Connor and Smith, 2005). They were asked not to smoke during the hour before testing due to the associated rise in cortisol (Morgan et al., 2004), nor to exercise or drink alcohol on the test day. On arrival at the laboratory, the participant was given an information sheet about the study before providing written consent. She was measured and weighed, and asked to provide a first saliva sample (baseline measure one). The participant then relaxed for fifteen minutes with a selection of magazines while listening to a ‘Classical Chillout’ compact disc (Circa Records Ltd, 2001), before providing a second baseline saliva sample. The 15-min stress induction procedure was then conducted. The stress manipulation was based on the Trier Social Stress Test (Kirschbaum et al., 1993). For the first five minutes, each participant was asked to prepare a 5-min presentation of their opinion on a controversial topic from a list (topics included abortion, sexual equality and cannabis legalisation), for later assessment by psychologists who were experts in body language. The participant then performed the presentation for five minutes in front of the
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