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The relationship between cortisol responses to laboratory stress and cortisol profiles in daily life

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ABSTRACT

Relationships between cortisol responses to laboratory stress and cortisol output over the day have not been studied extensively. We tested associations between cortisol responses to a set of laboratory challenges (colour/word interference and mirror tracing) and three aspects of cortisol output over the day, namely total area under the curve (AUC_{day}), the cortisol awakening response (CAR) and the slope of cortisol decline over the day. Participants were 466 men and women aged 54–76 years. We found that cortisol responses to laboratory stress were positively associated with cortisol AUC_{day} independently of sex, age, socioeconomic status, smoking, body mass index, and time of laboratory testing ($B = 0.212$, 95% C.I. 0.143–0.282, $p < 0.001$). No associations between laboratory responses and the CAR or cortisol slope were observed. The laboratory–field association was not moderated by demographic or psychosocial factors. The study provides evidence for the ecological validity of acute laboratory stress testing.

1. Introduction

The investigation of cortisol responses to acute mental stress in the laboratory is an important technique in psychoneuroendocrinology (Dickerson & Kemeny, 2004; Kudielka & Wust, 2010). Procedures such as the Trier Social Stress Test (TSST, Kirschbaum, Pirke, & Hellhammer, 1993) and other behavioural challenges have been used to study the impact of stress on hypothalamic-pituitary-adrenocortical (HPA) axis function in relation to demographic factors, background stress, psychological characteristics, cognitive function, early life experience, and physical and mental health conditions (Burke, Davis, Otte, & Mohr, 2005; Chida & Hamer, 2008; Kajantie & Raikkonen, 2010). This work primarily involves examining individual or group differences in the magnitude or duration of cortisol responses and other markers of HPA function. Laboratory mental stress testing has several methodological strengths, including the precise delineation of the profile of responses to standardised stimuli under controlled conditions in which the confounding effects of concurrent activities and exposures are eliminated (Steptoe, 2007).

Studying cortisol responses to laboratory stress suffers from the same limitations as those of psychophysiological mental stress testing more generally: namely, it involves assessing acute responses to arbitrary short-term behavioural stimuli under artificial conditions that are seldom encountered in everyday life. Since most research is cross-sectional, it is not clear whether variations in response to stress are relevant to the development of physical or mental health problems or are consequences of these conditions. The notion underlying the strategy of studying biological responses to laboratory stress is that individual differences in response magnitude or duration reflect the way people react biologically in everyday life. A person who is highly reactive in the laboratory will experience repeated episodes of heightened biological activity in their lives. These effects will lead to sustained differences in biological activity in everyday life, and will over months and years subsequently impact on health risk (Steptoe, 2007). The validity of mental stress testing is therefore typically assessed in two ways. The first is to evaluate whether variations in biological responses to laboratory stress predict future health outcomes or the development of clinical conditions. This issue has been examined extensively in relation to cardiovascular stress responses (Chida & Steptoe, 2010), but evidence related to cortisol stress responses is limited. Our group has shown that individuals with larger cortisol responses to laboratory stress are at increased risk of developing hypertension (Hamer & Steptoe, 2012), and of showing accelerated progression of subclinical coronary atherosclerosis as indexed by coronary artery calcification (Hamer, Endrighi, Venuraju, Lahiri, & Steptoe, 2012). Another study showed no association between cortisol responses and changes in self-reported body mass 4–7 years later (Phillips, Roseboom, Carroll, & de Rooij, 2012). No studies relating variations in cortisol responses to laboratory stress prospectively with depression, atopic conditions, or other health outcomes have yet been

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reported, so the relevance of individual differences in responsivity to future health risk remains untested in these conditions.

The second approach to evaluating the validity of mental stress testing is to test relationships with function in everyday life. The 'laboratory–field' problem has been studied intensively with respect to blood pressure and heart rate responses, comparing individual differences in acute responses to laboratory stress with values recorded over the day using ambulatory monitoring (Kamarck, Schwartz, Janicki, Shiffman, & Raynor, 2003; Turner et al., 1994). The consensus is that individual differences in levels of blood pressure and heart rate correlate well across the two situations (so people with higher blood pressure during stress tasks in the laboratory display higher ambulatory blood pressure over the day), but that the relationship between responses to acute laboratory stress (measured as change from baseline) and values recorded in daily life are less robust. A number of explanations have been put forward, including the possibility that the strength of associations depends on the type of laboratory task, the level of stress experienced in everyday life, and the reliability of the estimates of acute stress responses (Manuck, 1994; Steptoe, Cropley, & Joekes, 2000).

A few studies have assessed the relationship between cortisol responses to laboratory stress with the cortisol awakening response (CAR) in daily life, with largely negative results. For example, Schmidt-Reinwald et al. (1999) found no association between cortisol responses to the TSST and the CAR in a study of 22 young adults, a result that was replicated in a study of 21 student teachers (Wolfram, Bellingrath, & Kudielka, 2011). But a negative correlation was observed by Quirin, Pruessner, and Kuhl (2008), with smaller CARs in individuals who were more stress responsive in the laboratory. Other groups have reported between-group differences in cortisol responses to laboratory stress but not the CAR, or vice versa, further implying that the two phenomena are not closely related (Buske-Kirschbaum, Ebrecht, & Hellhammer, 2010; Petrowski, Herold, Joraschky, Wittchen, & Kirschbaum, 2010). To the best of our knowledge, the only study to investigate associations between cortisol responses to laboratory stress and cortisol over the day was an investigation of 87 employed men (van Eck, Nicolson, Berkhof, & Sulon, 1996); this showed a positive relationship between pre-stress baseline cortisol in the laboratory and measures taken at a similar time in daily life, but no correlation between laboratory stress responses and assessments at other times. The first aim of the present study was therefore to test in a large sample of older men and women the association between cortisol responses to laboratory stress and salivary cortisol over the day. We explored three aspects of cortisol dynamics: the CAR, cortisol output over the whole day computed as area under the curve (AUC_{day}), and the cortisol slope over the day, and tested whether associations with cortisol responses to laboratory stress were independent of baseline (pre-stress) cortisol levels.

The second aim of the study was to discover whether associations between cortisol responses to laboratory stress and cortisol in daily life were moderated by other factors. If the relationship between the magnitude of responses to acute stress and values recorded in everyday life varies with demographic or other factors, then comparisons involving different groups may be compromised. There is evidence that cortisol responses to laboratory stress vary with sex, age, depression and ongoing stress (Burke et al., 2005; Chida & Hamer, 2008; Kajantie & Phillips, 2006), while cortisol output over the day varies with socioeconomic status (SES) and affect in daily life (Adam, Hawkley, Kudielka, & Cacioppo, 2006; Kumari et al., 2010). It is conceivable therefore that the correlation between measures in the laboratory and over the day differs with the levels of these factors. Such associations would limit the generalizability of cortisol responses to laboratory stress. We therefore carried out moderator analyses of the laboratory–field relationship, testing differences in relation to sex, age, SES (defined by grade of employment), chronic stress (operationalised as financial strain, lack of social cohesion, social isolation and loneliness), subjective stress response to the task, depressive symptoms, and affect balance over the day evaluated using ecological momentary assessments (EMA).

2. Methods

2.1. Participants

We analysed data from the Heart Scan Study, which involved a sample of healthy older adults ($n = 543$) drawn from the Whitehall II cohort in 2006–2008 to investigate the association between physiological reactivity to behavioural stressors and subclinical coronary artery calcification (Hamer, O'Donnell, Lahiri, & Steptoe, 2010; Kidd, Hamer, & Steptoe, 2011). The Whitehall II study is a well-established epidemiological study of socioeconomic, psychosocial and biological risk factors for coronary heart disease and other disorders of ageing, involving men and women in the British civil service (Marmot & Brunner, 2005). Criteria for inclusion in the Heart Scan Study were white European origin, no history or objective signs of coronary heart disease (CHD), hypertension, or inflammatory disease, no history of mental illness, or any medication that might affect cortisol levels, including hormone treatment. All female participants reported postmenopausal status. All participants who had complete cortisol data from both the laboratory stress session and samples over the day were included in the analyses. Complete cortisol over the day was missing from 69 cases, and a further eight were eliminated because cortisol assays from the laboratory stress session were unsatisfactory. The final sample therefore consisted of 466 individuals, of whom 47% were women, and 53% men. There was no difference in demographic characteristics between those who did and did not provide complete cortisol data. Ethical approval for the study was given by the Research Ethics Committee for University College London/UCL Hospitals.

2.2. Laboratory stress procedures

Laboratory stress testing took place either in the morning (starting at 9:15 am) or afternoon (starting at 1:30 pm). Multiple physiological markers were measured during the acute stress testing in the laboratory; however, for the purposes of this paper we report the cortisol response only. After anthropometric measurements, a cannula was inserted for drawing blood. Blood pressure and heart rate were recorded continuously using a Finometer, a device that monitors blood pressure from the finger using the vascular unloading method (Guelen et al., 2008). The acute stress protocol was as follows: after a 30-min rest period following cannulation, a 5 min period of baseline blood pressure was carried out, together with a resting saliva sample, and blood sample. Two behavioural tasks were administered in a random order. The first task was a mirror tracing task, and the second was a computerised colour/word interference task (Kidd et al., 2011; Steptoe et al., 2002). Each task lasted 5 min, and task order was randomised across participants. Saliva samples were collected immediately after the tasks were completed, and then at 20, 45, and 75 min after the tasks for the assessment of salivary cortisol. Monitoring of cardiovascular activity continued throughout the study, with further blood draws at 45 min and 75 min post task. Participants were asked to rate their level of stress at baseline, immediately after each task, and during recovery on a seven point Likert scale ranging from 1 (no stress) to 7 (feeling very stressed). Scores from both stress tasks were aggregated to produce one average stress task score.