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Effects of orthostasis on endocrine responses to psychosocial stress

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ABSTRACT

Standardized psychological procedures have been designed to induce physiological stress responses. However, the impact of standing (orthostasis) on the physiological reaction after psychological stress remains unclear. The purpose of the current analysis was to examine and quantify the relative contribution of orthostasis to the physiological stress response by comparing a "standing with stress" to a "standing without stress" condition. We investigated the effect of standing with and without stress on responses of the sympathetic–adrenomedullary (SAM) system and the hypothalamic–pituitary–adrenal (HPA) axis using a standardized psychosocial stress protocol (Trier Social Stress Test) and a non-stress condition in a repeated measures design. Subjects ($N = 30$) were exposed to both conditions in randomized order and had to maintain a standing, upright position for 10 minutes. In the "standing with stress" condition, significant increases in repeatedly assessed plasma norepinephrine (NE) and epinephrine (EP), as well as in saliva cortisol were found, while in the "standing without stress" condition, no significant changes in plasma epinephrine and saliva cortisol were observed. Calculations of the relative contribution of orthostasis to physiological stress responses revealed that 25.61% of the NE increase, 82.94% of the EP increase, and 68.91% of the cortisol increase, could be attributed to psychosocial stress adjusted for the effects of orthostasis and basal endocrine output. Although these results are indicative for a marked endocrine reaction that is caused by psychosocial stress alone, our findings show that the contribution of orthostasis must be taken into account when interpreting endocrine data collected in a psychosocial stress test.

1. Introduction

The role of stress in everyday life and the detrimental consequences it might exert on our body have been widely examined in the past decades (McEwen, 1998). Assessment of hormonal changes in the two main components of the physiological stress system, i.e. the sympathetic-adrenomedullary (SAM) system and the hypothalamic–pituitary–adrenal (HPA) axis, is essential in psychobiological research to examine the effects of stress and a possible relation to future disease risk (Chida and Steptoe, 2010). Reactivity to psychological stressors in the laboratory has been extensively studied. A plethora of psychological stress paradigms has been developed, albeit with mixed results with regard to endocrine reactivity (Biondi and Picardi, 1999). On the other hand, physiological maneuvers and techniques have been used to evaluate the function of the SAM system (e.g., Valsalva maneuver, cold pressor test, static and dynamic exercise, upright posture (Oribe, 1999)) and the HPA axis (e.g., pharmacological stimulation tests (Heim, Ehlert, and Hellhammer, 2000)).

A frequently cited meta-analysis identified two characteristics of acute psychological stressors and tests that reliably induce changes in HPA axis, i.e. uncontrollability and social-evaluative threat (Dickerson and Kemeny, 2004a). A similar analysis for SAM system changes due to psychological stress is not available. However, research suggests that the SAM reacts more broadly and more sensitively to different challenges, such as physical activity or emotional arousal, than does the HPA axis. As can be seen from the above mentioned stressors used to stimulate the SAM system, it is of pivotal importance to have information about how much of the variance is contributed by psychological characteristics (e.g., uncontrollability) and how much of the variance of the effect can be attributed to physical characteristics of the stressor (e.g., standing). In the laboratory, exposure to psychological stressors is usually studied with subjects in a sitting position. However, the simulation of real-life situations, especially when examining the effects of psychosocial stress on human subjects, demands a more realistic setting. Thus, some stress tests have been conducted with the subjects being examined in an upright, standing position (e.g. Kirschbaum, Pirke, and Hellhammer, 1993). Furthermore, subjects are also often required to walk between different rooms in a laboratory (i.e. resting and testing room). Besides these well-established stress tests (Dickerson and Kemeny, 2004a; Gaab et al., 2003; Roy, 2004; Singh, Petrides, Gold, Chrousos, and Deuster, 1999), effects of orthostasis on parameters of the SAM system and the HPA axis have been observed (Bie, Secher,

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Astrup, and Warberg, 1986; Matzen, Secher, Knigge, Bach, and Warberg, 1992; Vlcek, Radikova, Penesova, Kvetnansky, and Imrich, 2008). Assuming the upright posture leads to sharp rises in catecholamine concentrations, especially in norepinephrine (NE). In healthy subjects, changing posture from lying to sitting results in higher concentrations of NE, and in epinephrine (EP) (Cameron et al., 1987), and lying vs. walking increases NE, but not EP concentrations (Robertson et al., 1979). Changing from lying to standing results in higher NE levels (Christensen and Brandsborg, 1976; Cryer, Santiago, and Shah, 1974; Lechin et al., 1995a; Lechin et al., 1995b; Paramore, Fanelli, Shah, and Cryer, 1998; Schofl, Becker, Prank, von zur Muhlen, and Brabant, 1997; Vlcek et al., 2008), whereas in EP, the results are inconclusive: some studies found higher concentrations (Paramore et al., 1998; Schofl et al., 1997; Vlcek et al., 2008), others found no changes (Christensen and Brandsborg, 1976; Cryer et al., 1974; Lechin et al., 1995a; Lechin et al., 1995b). Finally, changing from sitting to standing leads to increased NE and EP (Tulen, Boomsma, and Man in 't Veld, 1999). Orthostatic challenge (via tilt) also results in a rise of other physiological parameters, such as heart period and blood pressure (Hatch, Klatt, Porges, Schroeder-Jasheway, and Supik, 1986). With regard to HPA axis activation, increases in cortisol when changing from a sitting to a standing position have also been found. Plasma cortisol was shown to rise in response to 40 minutes of standing (Abalan et al., 1992). The same was true for salivary cortisol in a balanced cross-over design examining the effect of 20 minutes of sitting, lying, or being in the upright posture with increasing values only in the upright condition (Hennig et al., 2000). However, there are also studies lacking any evidence of postural shift affecting salivary cortisol (Hucklebridge, Mellins, Evans, and Clow, 2002).

These findings of postural changes stimulating the SAM system and the HPA axis make it essential to consider orthostasis-induced changes in the respective systems when examining psychosocial stress effects. Studies investigating healthy subjects in either sitting or standing positions or those which require walking from one room to the next, thus risk a possible confounding effect by orthostasis. It might very well be that the observed results are just due to standing or walking rather than due to the psychosocial or mental stress elicited by the task. Addressing the possible confounding effect of orthostasis, Tulen et al. (1999) found no differences in plasma catecholamines between standing and sitting for subjects doing the word–color–Stroop test. There is also evidence that during another well-examined standardized psychosocial stress test, the Trier Social Stress Test (Kirschbaum et al., 1993), postural changes have an impact on autonomic measures. Research indicates that asking the participants to assume an upright position for 10 minutes as a control condition results in heart rate and heart rate variability changes comparable to the TSST condition (Rohleder, Wolf, Maldonado, and Kirschbaum, 2006). In this study, salivary cortisol was unaffected by postural changes during the control condition. Replicating and extending this finding, our group was able to show an effect of standing during such a control condition on NE while EP seemed to be unaffected by postural changes (Nater et al., 2006). These findings were confirmed by a third TSST study that showed increases in heart rate, blood pressure, NE, aldosterone and renin activity to postural changes, while ACTH, cortisol and EP remained unaffected (Mlynarik, Makatsori, Dicko, Hinghofer-Szalkay, and Jezova, 2007). Furthermore, changes of serum cholesterol levels were shown in response to mental stress and standing, but controlling for body shift-induced changes in hemoconcentration diminished the effect of both tasks (Muldoon et al., 1992). The combined effect of postural and mental stress on hemoconcentration was examined in a well-designed study on the effects of head-up-tilt and a mental stressor both alone and in combination on rheological and cardiovascular measures. Changes in blood pressure, total peripheral resistance, plasma volume and hematocrit were highest in the combined condition while cardiac responses did not differ from those to postural stress alone (Veldhuijzen Van Zanten, Thrall, Wasche, Carroll, and Ring, 2005).

In this present work, we will extend previous work by calculating the amount by which psychosocial stress will change endocrine (i.e. catecholamines and cortisol) activity in comparison with changes in body posture. The findings from this study will have implications for the assessment of the relative contribution of orthostasis on psychosocial stress responses and might guide future research in choosing a study design that may take into account orthostasis as a potentially confounding factor. We studied the influence of standing on both catecholamines and cortisol in a balanced exposition to a stress and a no-stress condition in a repeated measures design. This procedure allows us to separate the physiological component and the psychological component in a psychobiological stress paradigm.

It is hypothesized that EP and cortisol would not significantly change in response to the "standing without stress" compared to the "standing with stress" condition, while NE is expected to increase in both conditions. It is further expected that a considerable percentage of the change of endocrine markers is just due to postural shift.

2. Methods and materials

2.1. Participants

Subjects were recruited through advertisement at the University of Zurich and the Swiss Federal Institute of Technology, Zurich to participate in a larger project on the effects of psychosocial stress on various stress markers. Stress effects on salivary cortisol and α-amylase, plasma catecholamines, and heart rate variability have been previously reported (Nater et al., 2006). Thirty healthy male subjects participated in the study twice, during a "standing with stress" and during a "standing without stress" condition (for subject characteristics see Table 1). All subjects were medication-free and were non-smokers. Subjects with any acute or chronic somatic or psychiatric disorder (as evidenced by self-report) were excluded from the study. Participants were told not to perform any strenuous physical activity 48 hours prior to the experiment and to cease all sporting activities during the time of the study. Intake of ethanol and caffeine was forbidden 18 hours prior to the experiment. At least 60 minutes before the study, subjects were not allowed to eat or brush their teeth so as to avoid gingival bleeding. After the subjects were provided with complete written and oral descriptions of the study, written informed consent was obtained. The subjects were remunerated for participation in the study with 80 Swiss Francs. This study adhered to human experimentation guidelines of the Helsinki Declaration. The study protocol was formally approved by the ethics committee of the University of Zurich and the ethics committee of the Canton of Zurich.

2.2. Procedures

All subjects were exposed to a standardized control condition ("standing without stress") and the Trier Social Stress Test (TSST) ("standing with stress") with a gap of 2 weeks (±3 days) in between.

Table 1
Subject characteristics and AUC_G values.

$N = 30$	"Standing with stress"	"Standing w/o stress"
Age in years	24.8 ± 2.4	
Height in cm	182.3 ± 7.7	
Weight in kg	74.8 ± 9.2	
BMI in kg/m^2	22.5 ± 2.0	
AUC_G cortisol	518.04 ± 261.68	306.70 ± 246.86
AUC_G epinephrine	1 565.17 ± 902.83	855.58 ± 542.04
AUC_G norepinephrine	12 706.58 ± 4 963.14	10 116.25 ± 2 840.25

Abbreviations: BMI = body mass index; AUC_G = area under the curve with respect to ground (calculated using the trapezoid formula, see Pruessner et al. (2003)).