



Are salivary gonadal steroid concentrations influenced by acute psychosocial stress? A study using the Trier Social Stress Test (TSST)

Daniela Schoofs*, Oliver T. Wolf

Department of Cognitive Psychology, Ruhr-University Bochum, Germany

ARTICLE INFO

Article history:

Received 8 July 2010

Received in revised form 11 January 2011

Accepted 12 January 2011

Available online 21 January 2011

Keyword:

Acute stress

HPA-axis

HPG-axis

Cortisol

sAA

Progesterone

Testosterone

Estradiol

ABSTRACT

It is well documented that acute stress activates the sympathetic nervous system (SNS) and the Hypothalamus–Pituitary–Adrenal (HPA) axis. Results regarding the hypothalamus pituitary gonadal (HPG) axis, in contrast, are less consistent. Stress-associated increases as well as decreases have been reported for testosterone and estradiol. In the present study, healthy young male ($n = 39$) and female participants ($n = 44$, all tested in the luteal phase) were randomly assigned to a well-evaluated psychosocial stress protocol (“Trier Social Stress Test”, TSST) or to a non-stressful control condition (“Placebo-TSST”). Salivary concentrations of cortisol, alpha-amylase, testosterone, progesterone, and estradiol were measured immediately before and twice (10 and 25 min) after the treatment. As was to be expected, cortisol- and sAA-concentrations increased in response to the stressor. Stressed men showed a more pronounced increase of cortisol than stressed women. In contrast, acute stress did not affect testosterone-, progesterone-, and estradiol-concentrations. The results of the present study suggest that an acute psychosocial laboratory stressor has no strong rapid effects on salivary gonadal steroids. In line with several previous studies the findings might suggest that stress-induced changes in gonadal steroids occur in response to physical stressors, to competitive stressors or to more severe stressors only.

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1. Introduction

The term stress is used to describe experiences that put a high demand on emotional and physiological processes (McEwen, 2007). Physiological processes include secretion of glucocorticoids (GCs, in humans primarily cortisol) and catecholamines (adrenaline and noradrenaline) to facilitate adaption. The release of these stress messengers is promoted by an activation of the sympathetic nervous system (SNS) and the Hypothalamus–Pituitary–Adrenal (HPA) axis. The response of the HPA-axis is initiated by the release of corticotropin-releasing hormone (CRH) from the paraventricular nucleus (PVN) in the hypothalamus. Higher CRH-concentrations stimulate secretion of the pituitary adrenocorticotropic hormone (ACTH), which in turn activates the release of cortisol by the adrenal glands (Charmandari et al., 2005). The described processes launch an immediate enhancement (among other effects) of oxygen and glucose availability (de Kloet et al., 2005; Sapolsky et al., 2000), both of which provide energy for adaptive mechanisms. However, considering the limited energy resources of an organism, it would make sense to reduce energetically expensive processes that are not directly related to the adaptive response (e.g. digestion, growth, reproductive behaviour; Sapolsky et al., 2000). Indeed, based on his observations,

Selye had assumed that stress disrupts reproductive behaviour in animals as early as 1939 (Selye, 1939). It seems reasonable that these effects are modulated by interactions between the HPA-axis and the Hypothalamus–Pituitary–Gonadal (HPG) axis. The HPG-axis orchestrates the release of sex steroids, including testosterone (T), estradiol (E2), and progesterone (PROG), from the gonads and the adrenals (Rivier and Rivest, 1991; Williamson et al., 2005).

The assumption of a close interaction between both axes is supported by studies reporting evidence that exogenous GCs suppress the release of gonadotropins (LH and FSH) from the pituitary in different animal species (Breen and Karsch, 2006). Further studies employing acute or chronic stress protocols (e.g. immobilization, foot shock, sleep deprivation etc.) found stress-induced changes in T-, E2-, and PROG-concentrations in animals (Andersen et al., 2004; Chichinadze and Chichinadze, 2008; Shors et al., 1999). In regard to male rodents, most studies reported significant decreases in T and E2, while corticosterone (as a main GC in rodents) and PROG usually increased after stress-induction (Andersen et al., 2004; Dong et al., 2004; Orr et al., 1994). In contrast, one study observed higher E2 levels in female rodents after exposure to an acute stressor, although the magnitude of the effect was additionally modulated by the specific cycle stage (Shors et al., 1999).

In addition, it should be noted that the release of sex steroid hormones in male as well as in female laboratory animals seems to depend on the distinct type of the stressful experience and the associated corticosterone response to the specific stressor since some types of stressors did induce lower or none stress-dependent changes of those hormones (Andersen et al., 2004; Shors et al., 1999). One

* Corresponding author at: Department of Cognitive Psychology, Ruhr-University Bochum, Universitätsstr. 150, D-44780 Bochum, Germany. Tel.: +49 234 32 29233; fax: +49 234 32 14308.

E-mail address: Daniela.schoofs@rub.de (D. Schoofs).

study systematically investigating the influence of stress on gonadal steroids submitted rats to five different chronic stress groups for four days (Andersen et al., 2004). The stressors were applied either twice a day for periods of 1 h (restraint stress, footshock, cold, and forced swimming) or for 96 h (paradoxical sleep deprivation; PSD). While PSD and footshock resulted in significantly lower T- and E2-concentrations and higher PROG-levels, cold and restraint stress induced solely lower T- and lower E2-levels, respectively. However, it should be noted that significant corticosterone changes were observed only in those groups exposed to PSD and footshock. In line with the previously mentioned results Shors et al. (1999) observed no changes of estradiol after an acute swim stressor in female rats but in contrast to prior observation made in male rats found elevated E2 levels in females after tailshock. The results did not depend on corticosterone changes since both stressors induced significant corticosterone elevations.

Possible mechanisms explaining the described influences of stress and/or GC on the HPG-axis are still under discussion, but existing results suggest that stress might affect the HPG axis on three levels (Charmandari et al., 2005; Rivier and Rivest, 1991). On the first level, stress might inhibit secretion of the gonadotropin releasing hormone (GnRH) by the hypothalamus, while on the second level, it could interfere with the GnRH-induced release of the luteinizing hormone (LH) and the follicle-stimulating hormone (FSH) by the pituitary. Finally, stress might alter responsiveness of the gonads for gonadotropins.

Only few studies have investigated the effects of acute stress on sex steroid-concentrations in humans (Gerra et al., 2000; Heinz et al., 2003) – the influence of gender on the magnitude of the response of the HPA-axis on the other hand has attracted much more attention. Regarding this issue, the majority of studies employing standardized acute laboratory stressors (for example the Trier Social Stress Test (TSST); Kirschbaum et al., 1993) have shown significantly larger stress-induced salivary cortisol-concentrations in male compared to female participants (Kajantie and Phillips, 2006; Kudielka et al., 2009). In addition, the response of the HPA-axis in women seems to depend on the distinct menstrual cycle stage, with women in the luteal phase displaying similar stress-induced cortisol-levels as men and higher concentrations compared to women in the follicular phase and to those taking oral contraceptives (Kajantie and Phillips, 2006; Kudielka and Kirschbaum, 2005). Some authors have suggested that sex differences in the HPA-axis-response might be generated by protective effects of circulating estrogens (Bowman et al., 2001; Charney, 2004; Luine, 2002). However, dimorphisms in brain function, differences in corticosteroid-binding globulin-levels, and gonadal and adrenal interactions at the genomic and cellular levels are also discussed as possible mechanisms (Chichinadze and Chichinadze, 2008; Handa et al., 1994; Viau, 2002).

To examine effects of HPA-axis activation on sex steroids in humans many researchers have studied competitive situations such as sport tournaments (Bateup et al., 2001; Kivlighan et al., 2005; Suay et al., 1999) or cognitive competitions (e.g. Japanese chess or computer games; Gladue, 1989; Hasegawa et al., 2008; Mazur, 1997). In men a variety of sports competitions (e.g. rowing, judo; Kivlighan et al., 2005; Suay et al., 1999) as well as cognitive competitive situations, which lack the physical component, seems to increase anticipatory (Mazur, 1997) or post-competition cortisol and T concentrations (Gladue, 1989; Hasegawa et al., 2008). The results are less homogeneous for women. Studies employing sport tournaments observed enhanced post-competition cortisol- and T-levels (Bateup et al., 2001), others merely found significant cortisol increases and unchanged T concentrations (Kivlighan et al., 2005), while one study using a cognitive competition reported no changes at all (Mazur, 1997). In reality, the issue is even more complicated, since the magnitude of hormonal changes seems to depend on further psychological variables such as the individual experience in the specific competitive situation, winning or losing the competition, and the strength of team bonding (Gladue, 1989; Kivlighan et al., 2005).

In comparison to the number of studies examining sex differences in the endocrine stress-response and studies investigating the impact of competitive situations on changes in HPA- and HPG-activity, it is striking that only very few studies have employed standardized laboratory stressors to address the question of how stress affects sex steroid concentrations in humans.

Existing results for male participants are rather heterogeneous. It was found that metabolic stress (glucose deprivation, Elman and Breier, 1997) and anticipatory stress before a one day clinical research protocol (Schulz et al., 1996) significantly increased cortisol- and decreased T-levels while a public oral presentation on a scientific conference (Heinz et al., 2003) and a combined laboratory stressor (mental arithmetics, Stroop-task, and public speaking; Gerra et al., 2000) didn't induce T-changes in healthy participants. However, it should be noted that the latter study tested peripubertal male participants, whose endocrine state and response pattern probably differ considerably from male adults. Regarding the influence of stress on PROG it was reported that, consistent with results from animal studies, metabolic stress induced a rise of PROG-concentrations (Elman and Breier, 1997). In contrast to PROG, E2-levels were not influenced by a stressful public speaking situation (Heinz et al., 2003). Only one study regarding female participants was found in the literature. However, in this study, no stress induction was employed, but hydrocortisone was administered for several days to healthy young women (Saketos et al., 1993). This treatment led to decreased PROG-levels, while having no effect on E2 concentrations.

In summary, animal and human studies on male and female individuals suggest that stress and/or enhanced GC concentrations can influence the HPG-axis. However, results in animals are relatively homogeneous. In rodents most studies found stress induced decreases in T and E2 and an enhancement of corticosterone and PROG (Andersen et al., 2004; Dong et al., 2004; Orr et al., 1994). In human studies the picture is less consistent and results seem to be additionally modulated by various psychological variables (Gladue, 1989; Kivlighan et al., 2005). One obvious explanation might be the lack of studies using well standardized stressors, which reliably induce a robust endocrine stress response. One laboratory stressor which meets this criterion is the TSST, a well-evaluated psychosocial stressor which reliably induces significant activation of the HPA-axis and the SNS (Dickerson and Kemeny, 2004; Kirschbaum et al., 1993). Thus, we were interested in examining the effects of the TSST (as one of the most employed psychosocial laboratory stressors in humans) on physiological stress markers and sex steroids in young male and female adult humans. The TSST can be characterized as a paradigm which combines motivated performance with uncontrollability and social evaluative threat (Dickerson and Kemeny, 2004). It induces feelings of anxiety and shame (Dickerson et al., 2008) and can be described as a situation of experimentally induced failure. Further on, a well matched control situation for the TSST exists: the Placebo-TSST (Het et al., 2009), which does not induce a cortisol response. Based on this conceptualisation and on results from animal studies we expect decreased T-levels in response to the TSST, while PROG is expected to increase (Andersen et al., 2004; Elman and Breier, 1997; Schulz et al., 1996). Regarding E2, various studies have yielded no consistent results (Andersen et al., 2004; Saketos et al., 1993; Shors et al., 1999), therefore it is hardly possible to predict the direction of a potential stress effect.

2. Methods

2.1. Participants

Eighty-three healthy young male ($n = 39$; average age \pm SD = 24.85 ± 4.06) and female ($n = 44$; average age \pm SD = 24.73 ± 3.90) participants participated in the experiment. All participants took part in one of two studies investigating the effects of stress on memory performance in a working memory (Schoofs et al., 2008b) or a declarative memory

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