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The final definitive version in Psychoneuroendocrinology is available online at:

https://dx.doi.org/10.1016/j.psyneuen.2018.12.220

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Accepted Manuscript

Title: Repressors exhibit lower cortisol reactivity to group psychosocial stress

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PII: S0306-4530(18)30737-6
DOI: https://doi.org/10.1016/j.psyneuen.2018.12.220
Reference: PNEC 4183

To appear in:

Received date: 19 July 2018
Revised date: 10 October 2018
Accepted date: 14 December 2018

Please cite this article as: Oskis A, Smyth N, Flynn M, Clow A, Repressors exhibit lower cortisol reactivity to group psychosocial stress, *Psychoneuroendocrinology* (2018), https://doi.org/10.1016/j.psyneuen.2018.12.220

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Repressors exhibit lower cortisol reactivity to group psychosocial stress

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Keywords: repress; anxiety; TSST-G; HPA axis; cortisol reactivity; non-responders

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Highlights

- Repressors have lower cortisol reactivity to group psychosocial stress
- Repressors self-report less subjective stress compared to other coping styles
- Repressor psychophysiological dissociation differs for HPA axis and ANS reactivity
- Blunted cortisol reactivity may be an adverse health trajectory for repressors

Abstract

Repressors are well-known to monitor potential psychosocial threats to their self-esteem and self-concept. In research, repressors are traditionally categorised as those scoring low on trait anxiety and high on defensiveness (as measured by social desirability scales). Examining repressors' cortisol reactivity to a group socio-evaluative laboratory stressor could be an important way to extend work on the classic 'repressor dissociation', which proposes that this group experience higher levels of physiological stress, but lower levels of subjective affect during stressful situations. Research however has focused mainly on repressors' higher, more risk-prone levels of autonomic, rather than hypothalamic-pituitary-adrenal (HPA), reactivity to stressful stimuli. We assessed cortisol reactivity using a group-based acute psychosocial stressor, the Trier Social Stress Test for groups (TSST-G), which required participants to individually perform public speaking and mental arithmetic tasks in front of up to six other group members, as well as an evaluative panel of judges. Seventy-seven healthy young females (mean age ± SD: 20.2 ± 3.2 years) took part, of which 64 met the conventional criterion for a response to the TSST-G (<15.5% increase from baseline sample). The Stress-Arousal Checklist was completed pre- and post-TSST-G. Participants also completed the Perceived Stress Scale, the Trait Anxiety Inventory and the Marlow-Crowne Social Desirability Scale. The latter two measures were used to provide a categorisation of repressive coping style. Participants identified as repressive copers exhibited significantly lower cortisol reactivity during the TSST-G. Repressors also self-reported less subjective stress. These findings provide some evidence against the notion of

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the repressor dissociation and are discussed in terms of how cortisol hyporeactivity may be a pathway through which repressive coping adversely affects health.

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1. Introduction

The study of repression is a central concept in research on stress and coping, and now lies at the intersection of several important areas of psychological science, including the cognitive, clinical, biological and health fields. The concept however has its origins in psychoanalytic traditions. Repression was first conceptualised by Freud (1909) as part of his formulation regarding the origin of neurosis. Freud proposed that repression, in other words, pushing out of consciousness thoughts and feelings that do not fit in with one's view of the self, was the primary defence mechanism in neurosis. Whilst his idea became a cornerstone of psychoanalytic theory, repression has been somewhat difficult to operationalise as a construct for empirical research purposes, given the role of the unconscious in characterising this defence. Although this initially lead to a paucity of systematic research into the causes and consequences of repression, one bridge from this original psychoanalytic tenet to empirical investigation has been via work on defensiveness, also known as the repressive coping style. Whilst repressive coping has been related to several relevant constructs, including higher levels of trait emotional intelligence, self-estimated intelligence, functional impulsivity and stoicism (Furnham et al., 2010), two constructs remain central to its classification: low trait anxiety and high social desirability (generally interpreted to indicate defensiveness) (Weinberger et al., 1979). Operationalising the style in this way unlocked a wave of investigation, and over 30 years of research since has shown that the repressive coping style characterises between 10-50% in different populations (Myers, 2010).

Repressors have been described as preoccupied with mastering negative emotion and rigorously controlling their behaviour, and are viewed as valuing a rational, non-emotional approach to life (Weinberger et al., 1979). Early work typically described repressors as

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reporting low levels of distress and anxiety but exhibiting high levels of autonomic nervous system (ANS) stress reactivity (e.g. Asendorpf and Scherer, 1983; Derakshan and Eysenck, 1997; Weinberger et al., 1979). Significantly, repressive coping has been linked with poor health outcomes, particularly in cardiac patients (Frasure-Smith et al., 2002). For example, in one study, after controlling for various psychological and physiological variables, repressive coping was shown to be an independent predictor associated with a two-fold increased risk of death, myocardial infarction and other cardiac events (Denollet et al., 2008). Together these studies offer compelling evidence that the repressive coping style is associated with negative health outcomes. This is consistent with the observation that exaggerated cardiovascular responses to stress are predictive of future cardiovascular disease risk (see Lovallo, 2005).

The mismatch in self-reported and physiological stress responding observed in repressive coping, termed the 'repressive dissociation' (Bonanno et al., 1995), is thought to interfere with effective coping and, paradoxically, promote the observed exaggerated physiological responses to stress (Myers, 2010). This proposal is rooted in General Systems Theory (Schwartz, 1990), and provides a conceptual link as to how the repressive response pattern leads to dysregulation of both ANS and immune dysregulation, which in turn may increase the risk of physiological disease. Repressive dissociation, which manifests in higher, and thus more risk-prone, levels of somatic stress, seems only apparent in those classified as repressors; the three other groups that can be operationalised from patterns of trait anxiety and defensiveness (high anxious, low anxious and defensive high anxious) typically show different patterns of responses (Asendorpf and Scherer, 1983; Derakshan and Eysenck, 1997; Lambie and Baker, 2003) Although the repressor dissociation is a widely replicated finding, most studies adopting the Weinberger approach (i.e. low self-report trait anxiety and high defensiveness) have focused on ANS reactivity to stressful stimuli, as indexed by either cardiovascular activity or electrodermal activity (EDA). There is limited research on

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hypothalamic-pituitary-adrenal (HPA) axis responses to stress in repressors. But available evidence is consistent with the hypothesis that the HPA axis is dysregulated. For example, when compared with high trait-anxious people (those who exhibit high levels of self-report trait anxiety, but low levels of defensiveness), repressors were found to have aberrant patterns of basal cortisol (Brown et al., 1996). Also relevant is that that high social desirability and low trait anxiety have each been previously investigated in relation to cortisol dysregulation (see Habersaat et al., 2017; Lam et al., 2009; Mikolajczak, et al., 2007).

Very little research has been undertaken on the potential role of HPA axis dysfunction as a pathway to ill health in repression, which is important given that repressors comprise up to 50% of chronic illness groups (Myers, 2010). It is clear that flat *diurnal* patterns of cortisol secretion are linked with a wide range of negative health outcomes including cardiovascular disease and cancer (Adam et al., 2017). Consistent with this notion, repressors with metastatic breast cancer show a flatter cortisol diurnal slope than truly low-anxious participants (Giese-Davis et al., 2004). Yet, there is limited research regarding HPA axis reactivity in repressors, which is interesting given that physiological reactivity is central to the concept of the repressor dissociation (Myers, 2010). A more recent study has demonstrated blunted (not exaggerated) cardiovascular and cortisol responses to stress in healthy young participants categorised as having low disgust sensitivity, which is related to a defensive coping style (Rohrmann et al., 2008). These data provide a rationale for further investigation of HPA axis function in repression and are interesting, as it is now becoming more apparent that both exaggerated and blunted physiological responding to stress is predictive as a poor prognostic indicator (Carroll et al., 2017).

Some studies however have not been able to replicate the finding of dysregulated physiological measures of distress in repressors. For example, Jørgensen and Zachariae (2006) found no difference between repressors and true low-anxious participants during or

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after stress tasks, on EDA or any heart rate variability-related parameter. These inconsistent findings have been thought to reflect differences in the experimental challenges used, and ANS reactivity (as measured by EDA) has been suggested to only be potentially elicitable in emotionally threatening tasks and/or specific social contexts (Barger et al., 1997). This has influenced the choice of task used to investigate the psychophysiological consequences of repressive coping in previous research. One study protocol required participants to purposely draw attention to negative aspects of themselves (Jørgensen and Zachariae, 2006). This was thought more likely to induce a repressive coping response, given that at the heart of this coping style is the maintenance of a positive self-evaluation, which is central to protecting one's self-image *and* social image (Myers, 2010).

The Trier Social Stress Test is a well-established and highly reliable acute stress laboratory protocol. Versions for individuals or groups exist (the TSST and TSST-G, respectively, (Kirschbaum et al., 1993; von Dawans et al., 2011), but common to both is the inclusion of elements of social-evaluative threat and uncontrollability. It is these features that make the TSST a standardised and reliable method of inducing HPA axis responses, as indexed by large cortisol changes. The TSST-G is starting to be used more extensively (Guez et al., 2016; Smyth et al., 2015), and in line with the current study's focus on repressive coping, the group element of the TSST provides an important opportunity to reinforce the impact of social dynamics on stress reactivity. The group version may also be a particularly apt choice of paradigm given previous findings showing that a threatening social evaluation in the presence of an audience interferes with a repressor's ability to repress negative self-evaluations (Baumeister and Cairns, 1992).

The rationale for the current study was to examine stress-induced cortisol reactivity and recovery to psychosocial stress in relation to defensiveness and anxiety to allow comparison between repressor, defensive high anxious, low and high anxious groups. We also measured subjective (self-report) stress and arousal and considered the issue of

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responding/non-responding (Bellingrath and Kudielka, 2008; Miller et al., 2013). To avoid intrusive effects of age and gender (see Smyth et al., 2013) we examined an all-female, healthy young population. We hypothesised evidence of HPA axis dysregulation in the repressor group, particularly in terms of cortisol reactivity.

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2. Methods

2.1 Participants

Eighty-one females were recruited from the academic community for this research, and an all-female sample was used to control for sex differences. Course credits were given for participation. The data presented here are based on 77 individuals; cortisol data were missing for one person due to insufficient volume for assay purposes, two participants did not complete the questionnaires in their entirety and another participant was removed from the data set due to their cortisol data being greater than five standard deviations above the mean for each sample (their data remained as outliers even following square root transformation). The group of 77 that remained ranged from 18 to 33 (mean ± SD: 20.2 ± 3.2) years.

Exclusion criteria reflected certain variables known to affect cortisol reactivity, including current medication, illness and history of psychiatric illness. Menstrual cycle and body mass index (BMI) have both been shown to affect cortisol reactivity (see Smyth et al., 2013), thus height, weight and the number of days since last period were all recorded, as was the use of oral contraceptives (two participants) and smoking status, for examination in relation to cortisol indices.

2.2 Measures

Two self-report measures widely used in the field (see Walsh et al., 2015; Weinberger et al., 1979) were used to categorise repressive coping style:

State-Trait Anxiety Inventory (STAI-T) (Spielberger et al., 1983): The STAI-T contains 20 items, each rated using a four-point likert scale, which collectively assess trait levels of

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anxiety. Total scores range from 20-80, with higher scores indicating higher trait anxiety. In the present study, Cronbach's alpha was .86.

Marlowe-Crowne Social Desirability Scale (MC-SDS) (Crowne and Marlowe, 1960): The MC-SDS is a 33-item scale which uses forced-choice, true-false items concerning everyday behaviours to assess whether respondents are concerned with social approval. Total scores range from 0-33. Greater scores indicate a tendency to respond in a socially desirable way, a stronger inclination to emotional inhibition and greater self-concealment (in other words, defensiveness). Cronbach's alpha in the current study was .74

Participants also completed the:

Perceived Stress Scale (PSS) (Cohen et al., 1983): The PSS includes 10 items in the format of direct queries about how unpredictable, uncontrollable and overloaded respondents perceive their lives to be, as well as their feelings and thoughts about perceived stress. Each item is rated on a four-point scale. A higher total score indicates greater perceived stress, with scores ranging from 0-40. In this study, Cronbach's alpha was .86.

Stress Arousal Checklist (SACL) (Mackay et al., 1978): The SACL uses an adjective checklist to measure situational stress and arousal. The arousal score is derived from 11 adjectives and the stress score from 19 adjectives. A four-point response system is used to rate each 'positive' or 'negative' adjective, as related to the specific construct. Higher scores indicate greater stress and arousal (i.e. for the stress measure this indicates that a person is more worried, apprehensive, uneasy and distressed, less peaceful, contented, comfortable and relaxed, and for arousal, more alert, active, energetic, less drowsy, tired and sluggish) respectively. In this study, Cronbach's alphas were good for each of the positive and negative stress and arousal dimensions, for both the pre- and post-TSST-G measures (alphas ranged from .73 to .83).

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2.3 Procedure

Ethical approval was provided by the University of Westminster Ethics committee. After recruitment and an initial briefing and test session, volunteers took part in the TSST-G during afternoon sessions between 13:00 and 15:00 hr. This timing was in line with best practice guidelines to control for basal changes in morning cortisol secretion (Kirschbaum et al., 1993; Oskis et al., 2009; Smyth et al., 2013). For 30 minutes prior to the session, participants were asked to refrain from food, caffeine, alcohol, exercise and smoking.

Three phases comprise the TSST-G (von Dawans et al., 2011): the group preparatory period (30 min); the group stress task period (22 min); and a group resting and debriefing period (40 min). During the preparatory period, groups of up to six participants met in Room 1 where they were informally seated around a single table and introduced to the experimenter. At this point in the session participants were free to speak to each other.

Participants provided informed written consent and then individually and silently completed demographic questions, wrote down the date of their last menstruation and filled in the STAI-T, MC-SDS and PSS. Each participant was given a large sticker with a number between one and six and they were told that they would be identified with this number during the task period. Participants were also informed that the numbers would be called in a random order. They were then given a demonstration of the saliva-sampling method. The last part of the preparatory period involved telling participants that they would now be given 10 minutes of quiet time to prepare notes for a mock job interview, which was to include preparing a free two-minute speech to introduce themselves to a committee for an application process for a job of their choosing. Participants were instructed to try to convince the committee that they were the most suitable candidate for the position. The baseline saliva sample was collected immediately at the end of this. Participants subsequently filled in the SACL.

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Participants were then taken from Room 1 to Room 2 for the start of the second phase. They were told however, that they had to leave all their notes behind. In Room 2, a committee, formed of one man and one woman, was already sat behind a table wearing laboratory coats. Participants were instructed to stand in a straight line in front of the committee. Two cameras visibly pointed at the participants. A committee member called the number of each participant in turn, in a random order, to make their two-minute speech. Committee members used standard responses (e.g. “You still have time, please continue”) if participants finished their speech before the two minutes. At this point a saliva sample was taken. After each participants had given their speech, the committee members instructed the participant to serially subtract the number 17 from a given number (e.g. 7848) as fast and accurately as possible for 80 seconds. The same participant order as for the speech was maintained for the arithmetic task. To control for learning effects, each participant received an individual starting number. If participants failed during this task they were again met with a standard response (e.g. “You made a mistake please start again from the number”) (von Dawans et al., 2011). Another saliva sample was collected at this time. Following completion of the TSST-G, participants were straightaway returned to Room 1, where they collected saliva samples every 10 minutes up to 40 minutes following the test period. After this, they completed the SACL again. Participants then received a debrief, and this completed the process.

2.3.1 Saliva sample collection

Salivettes (saliva sampling devices, Sarstedt Ltd., Leicester, England) were used to collect saliva. Cortisol was extracted and measured from the cotton swab of the salivette device. The protocol included seven sample collections; at baseline (immediately before the TSST-G: sample 1, at 0 min, sample A on Figure 1), immediately after the public speaking task (sample 2, at 12 min, sample B on Figure 1), after the mental arithmetic task (sample 3, at

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22 min, sample C on Figure 1), and every 10 min up to 60 min (sample 4, at 32 min sample 5, at 42 min, sample 6, at 52 min, and sample 7, at 62 min). This sampling profile therefore included the rise in cortisol, the cortisol peak, and the decline of cortisol.

Saliva samples were frozen at -20°C until assayed at the University of Westminster. Samples were thawed and centrifuged at a relative centrifugal force (RCF) of 1500 X g, at 3,500 revolutions per minute using a centrifuge, for 10 minutes. Cortisol concentrations were determined by enzyme linked immuno-sorbent assay developed by Salimetrics LLC (USA). The standard range in the assay was 0.33–82.77 nmol/l. Intra and inter-assay variations were both below 10%.

2.4 Treatment of data and statistical analysis

Cortisol data were moderately skewed so a square root transformation was applied to normalised distributions. Cortisol concentrations shown in figures however, represent original units. A repeated measures analysis of variance (ANOVA) was used to examine differences in cortisol over sampling time. The pattern of cortisol secretion was assessed using within-subjects contrasts. Where sphericity assumptions were violated, Greenhouse-Geisser corrections were applied.

In line with the approach proposed by Miller et al., (2013), each participant's cortisol reactivity was computed as their peak sample minus baseline (sample 1). Cortisol recovery was computed as each participant's peak sample minus the last sample (sample 7). Pearson's correlation coefficients were used to examine relationships between cortisol composites, the STAI-T, MC-SDS, PSS, SACL and demographics variables. To examine differences in repressive coping, participants were grouped in the same way as Walsh et al. (2015), based on the widely-used practice of using median split scores on the STAI-T and the MC-SDS to identify high and low cut-offs for grouping purposes. In our study, high

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anxiety was defined as a STAI-T score of 46 or more and low anxiety as a score of 45 or less; high defensiveness was defined as a MC-SDS score of 19 or more and low defensiveness as a score of 18 or less. Four groups of participants, representing the four combinations of low and high trait anxiety and defensiveness, were identified. In the whole sample, those with low levels of both characteristics formed the 'low anxious' group (n = 17), and those with high levels of both were the 'defensive high anxious group' (n = 8). High anxiety coupled with low defensiveness comprised the 'high anxious' group (n = 27). Participants with low anxiety and high defensiveness were categorised as 'repressors' (n = 25).

One-way between-subjects ANCOVA/ANOVAs explored group differences in baseline cortisol concentrations, reactivity and recovery composite measures (covarying for age), and also PSS scores. Bonferroni post-hoc tests were applied as necessary. Mixed ANOVAs, with repressive coping group as the between-subjects factor and time as the within-subjects factor were used to assess changes in SACL stress and arousal levels over the course of the experiment. Chi-square was used to examine the association between repressive coping group and whether a response to the TSST-G occurred.

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3. Results

The TSST-G induced an overall cortisol response in this sample ($F_{(2.6, 189.9)} = 7.061$, $p < .001$, partial $\eta^2 = .088$), illustrated in Figure 1. Within-subjects contrasts revealed a significant quadratic effect ($F_{(1, 73)} = 23.157$, $p < .001$, partial $\eta^2 = .241$), where, on average, cortisol increased from baseline, peaked at the fourth sampling point (10 min after the completion of the TSST-G) and subsequently declined.

Insert Figure 1 about here

The sample was subsequently categorised based on whether they showed a percentage baseline-to-peak cortisol increase of 15.5% in response to the TSST-G (see Miller et al., 2013). In line with this criterion, there were 64 responders in the sample. All analyses reported here were carried out on this group of responders. There were no significant differences in any of the variables measured in this study (demographic or psychosocial) between the responders and non-responders (F values ranged from 0.043 - 2.713, all p values > .05).

Relationships between cortisol data, demographic data and psychosocial measures were examined using focused composite cortisol indices of reactivity (as recommended by Miller et al., (2018); individual peak sample minus baseline) and recovery (individual peak sample minus sample 7). Descriptive statistic and intercorrelations for all variables are presented in Table 1.

Enter Table 1 about here

There was a significant positive relationship between cortisol reactivity and trait anxiety. Trait anxiety was also significantly correlated with defensiveness, albeit negatively, indicating that

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those individuals with higher trait anxiety were less concerned with social approval (in other words, less defensive). Defensiveness was also negatively associated with pre-TSST-G levels of stress. Trait anxiety was positively correlated with pre-TSST-G stress and total perceived stress scores. Perceived stress was negatively correlated with pre-TSST-G arousal levels, but positively with post-TSST-G stress levels. Pre-arousal significantly correlated with post-arousal. Age was positively associated with both cortisol reactivity and recovery. Other descriptive variables of interest with regards to cortisol activity (BMI, menstrual cycle phase and smoking status) were not significantly related to any cortisol index. Excluding the two participants who were taking oral contraceptives did not affect any of the results reported here.

3.1 Repressive coping and cortisol secretion in the TSST-G

The following analyses were run for the n = 64 group of responders (16 low anxious, 23 high anxious, 8 defensive high anxious and 17 repressors). There was no significant association between repressive coping group and if a response to the TSST-G occurred (χ^2 = 7.245, p = .064). There were no significant differences in baseline sample concentrations between groups ($F_{(3,60)}$ = 1.001, p = .399, partial η^2 =.048). Differences between repressive coping groups cortisol reactivity in the TSST-G cortisol secretion were explored, and because of the significant relationship with age, this was included as a covariate. There was a significant main effect of group on cortisol reactivity ($F_{(3,59)}$ = 4.486, p = .007, partial η^2 =.186). Bonferroni post-hoc tests revealed that both repressor and low anxious groups had significantly lower reactivity during the TSST-G compared to those who were defensive high anxious (p = .004 for repressors, and p = .030 for low anxious, respectively. See Figure 2).

Since our interest was in repressors specifically, to see if the repressor group difference held exclusively, we followed the common approach within the repression literature of collapsing

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the defensive high anxious, high anxious and low anxious groups into a 'non-repressor' group (see Myers, 2010) and re-ran the above analyses. Once again, using the cortisol reactivity composite measure revealed a significant main effect of group, with repressors exhibiting lower reactivity ($F_{(1,61)} = 4.178$, $p = .045$, partial $\eta^2 = .064$).

Insert Figure 2 about here

In terms of the cortisol recovery, there was no significant effect of group when looking at the cortisol recovery composite ($F_{(3,59)} = 0.328$, $p = .805$, partial $\eta^2 = .016$). Examining cortisol recovery in terms of repressor vs. non-repressor also revealed a non-significant effect ($F_{(1, 61)} = 0.275$, $p = .602$, partial $\eta^2 = .004$)).

3.2 Repressive coping and stress and arousal

Repressive coping group had a significant effect on the PSS ($F_{(3,60)} = 5.245$, $p = .003$, partial $\eta^2 = .208$), where repressors self-reported less total perceived stress than high anxious ($p = .030$) participants. (See Figure 2).

For the SACL, there was a significant effect of repressive coping group on pre- to post-test stress ($F_{(3,60)} = 2.999$, $p = .038$, partial $\eta^2 = .130$), where repressors had lower stress compared to the high anxious group ($p = .031$). There was no significant difference in arousal levels ($F_{(3, 60)} = 0.270$ $p = .847$, partial $\eta^2 = .013$). Full descriptive statistics for each group can be seen in Table 2.

Insert Table 2 about here

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Insert Figure 3 about here

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4. Discussion

The present study found a significant effect of repressive coping group on cortisol reactivity, where those participants identified as repressive copers exhibited significantly lower reactivity in response to the TSST-G, particularly compared to the defensive high anxious group. Repressors also self-reported less stress, especially in relation to high anxious participants.

This is the first time that a standardised group socio-evaluative laboratory stressor has been used to investigate differences in repressive coping. The use of the TSST-G was particularly fitting for this purpose, given its psychosocial approach and its purposeful threat to the social self. We found that during the TSST-G those with a repressive coping style exhibited lower cortisol reactivity compared to the defensive high anxious group. Our findings concur with the cognitive approach to trait anxiety, specifically the four-factor theory of anxiety, which considers that repressors have biases processing the four sources of information necessary for the experience of anxiety: behaviour, cognitions, external stimuli and internal physiological stimuli (Eysenck, 2000). Whereas repressors minimise the threateningness of these four sources of information, individuals experiencing higher anxiety exaggerate this threat. This may provide some explanation for our cortisol findings, as well as for our data showing that repressors also had lower levels of self-reported stress compared to the high anxious group.

Our findings also concur with work investigating coping styles and attentional bias towards affect-laden information, particularly that involving threat. Previous work suggests that the higher the individual's level of anxiety, the greater the allocation of attention to threatening rather than non-threatening information (Eysenck, 2000). The difference between participants with higher trait anxiety and repressors is that in threatening situations, repressors' lower level of anxiety would not cause them to seek out knowledge about the

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stressor in a sensitising or monitoring fashion. Instead, repressors automatically direct their attention away from threat-related stimuli. This is supported by experimental evidence showing that repressors have a greater ability to shift their attention away from socially-threatening words and from socially threatening cues, as well as self-related threatening stimuli, such as negative feedback (Mohiyeddini, 2017).

The converging evidence regarding repressors' treatment of socially- and personally-threatening information appears to extend across multiple cognitive domains. Repressors have been shown to have biases involving attentional, interpretative and memory mechanisms. For example, repressors minimise the reporting of personally-aversive events, giving summarised accounts rather than specific contextual detail, and they experience an absence of cognitive activity when recalling personally-threatening information (Saunders et al., 2014). In particular, memory deficits for negatively-valanced autobiographical memories from both childhood and adulthood have been found in repressors (Raes et al., 2006). Memory deficits have previously been linked to blunted cortisol reactivity, with evidence that lower TSST reactivity is associated with lower memory function in low-income children (Raffington et al., 2018). Repressors especially minimise the reporting of affective memories involving fearful and self-conscious experiences (Myers, 2010). Our study has shown that in a paradigm such as the TSST-G, where the stress is more personally-relevant, repressors minimise not only cognitively/affectively, but also biologically. We found that the repressor group exhibited a uniform 'low stress' response across self-report and cortisol reactivity. This supports the recent idea that blunted cortisol responses reflect failure to respond to active challenges that require optimal motivational and emotional processing systems (Ginty, 2013). Our repressor findings fit this proposed 'biological disengagement' (Ginty, 2013), and furthermore suggest a type of psychological or emotional disengagement too.

Interestingly, the less reactive profile of repressors presents an opposing picture to cortisol reactivity in those with the opposite emotion regulation strategy, suppressors. Suppression,

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unlike repression, involves regulating emotion by inhibiting ongoing emotional expression, and suppressors have been found to exhibit exaggerated cortisol responses to the TSST (Lam et al., 2009). Taken together, our study provides further support for the idea that the two response-focused emotion regulation strategies of repression and suppression are physiologically, as well as conceptually different.

Our finding however, that repressors had lower cortisol reactivity, is contrary to what might be expected when considering the proposed 'repressor dissociation' (Weinberger et al., 1979). In support of this idea, we did find that repressors reported lower levels of subjective stress. However, this lower self-report affect was not accompanied by higher reactivity, unlike previous studies, where this repressive dissociation has been demonstrated (e.g. Derakshan and Eysenck, 1997; Lambie and Baker, 2003). Not all studies however have been able to replicate the repressive dissociation, and similar to our study, some have not found differences in physiological reactivity between repressors and low anxious participants (see Barger et al., 2000; Jørgensen & Zachariae, 2006). It has been suggested that it is the autonomic measure of EDA that consistently identifies repressors as most reactive (Barger et al., 1997; Weinberger et al., 1979), and that this increased reactivity is shown in repressors regardless of the other coping groups for comparison (Barger et al., 2000). Whilst differences in operationalisation of repressive coping and choice of physiological measures might account for the discrepancies across studies, it may be that repressor differences in HPA axis activation do not necessarily parallel the differences in their autonomic activity. One interesting suggestion is that autonomic blunting does eventually follow limbic/HPA blunting, and hypothalamic dysfunction is the trigger for primary motivational and emotional dysregulation that subsequently develops into poor peripheral homeostasis (Cărnuţă et al., 2015). Thus, if HPA axis dysregulation represents a primary level of physiological dysfunction in repressors, it may be that cortisol offers a clearer window into changes specifically associated with the stress response than ANS measures do. Measures such as EDA are also subject to the issue of non-responding (Farrow et al., 2013), and it is

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noteworthy that the issue of ANS non-responding has not been considered in previous work on repression. More recently, the idea that elevated ANS responses represent 'stress' has been questioned, with the suggestion that different ANS measures, such as EDA and heart rate, link differentially to arousal and valence aspects of affective experience (Farrow et al., 2013). Ultimately, as we did not find discrepant responding in repressors, this provides some evidence against the notion of the repressor dissociation, as it has been traditionally conceptualised.

In the present study, repressors, compared to the entire group of non-repressors did present as having less cortisol reactivity. In previous work on chronically stressed participants, this qualifies as 'blunted' cortisol reactivity (see Miller et al., 2013). Several adverse long-term health outcomes found in repressors have also been associated with blunted cortisol stress reactivity, for example impaired memory functioning, poor lung function and cancer (Giese-Davis et al., 2006 Myers; 2010; Raffington et al., 2018). However, we are mindful of labelling the repressor group's pattern as such at this stage, and more data are needed to ascertain whether repressors are truly blunted, both from investigations of reactive and basal cortisol secretion. The term blunted has most often been associated with clinical/pathological populations, and one hypothesis is that blunted stress reactivity may reflect an adapted stress response due to down-regulation of receptors following chronic stress exposure (Miller et al., 2013). In the case of repressors, their constant (albeit unconscious) monitoring of their self-image could be construed as a chronic stressor. We therefore propose that our finding of similarly low cortisol reactivity in both the repressor and low anxious groups could be a result of different physiological trajectories, where the repressor group's stress reactivity has become blunted due to repeated stimulation of their defensiveness. This same quality may also leave repressors susceptible to stress-related diseases; if repressors allocate greater resource to monitoring whether or not their self-concept is threatened, this would serve to reduce their capacity to cope adaptively with external demands. These physiological suggestions sit well with what is known about repressors, in that they are genuine when they

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report experiencing low anxiety because of their capacity to self-deceive, which makes them qualitatively different from those who are truly low anxious individuals. Although further work is needed to disentangle the role of cortisol secretion in the repression-adverse health relationship, the importance of considering anxiety in the context of high or low defensiveness is clear, given that the repressive coping style, and *not* the low anxious style, has been associated with poorer treatment response and health outcomes (Lewis et al., 2012).

One notable feature of the present study was our consideration of responding to the TSST-G, which is an issue that is increasingly being highlighted in research on HPA axis reactivity (Miller et al., 2013). Non-responding is defined differently in the literature, but most recently it has been defined as a 15.5% increase from baseline to peak (see Miller et al., 2013). When investigating associations between cortisol stress reactivity it is crucial to account for non-responding as it is different from a blunted/lower cortisol response (Bellingrath and Kudielka, 2008). Failing to account for this could result in lower overall mean cortisol stress reactivity, which might then be construed as significantly blunted responding. We are confident that our findings tell a story about repressor, rather than non-responder, differences in cortisol reactivity, and this is one of the strengths of our study. Another strength is the present general good health of our sample; it is interesting that the less reactive cortisol profile of repressors mirrors that of individuals with adverse health status. As such, our data may provide a prognostic view into the future health of our repressor group. The cross-sectional design of the present study however, is a limitation, and does not permit us to draw any firm conclusions about health outcomes.

To obtain a better picture of how stress reactivity affects the health of repressors, future work should aim to measure cardiovascular responses, using heart rate or another measure of the sympathetic nervous system activity, and HPA axis responses in parallel. Also, in terms of expanding measurement, to provide a more complete account of repressors' pattern of

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response it may be useful to include a short measure of self-report stress at an appropriate point during the TSST-G. We also acknowledge the very small number of defensive high anxious participants in the present study (but this reflects the low prevalence of this category in general society, see this point considered in Lewis et al., 2012). Replication with greater numbers of defensive high anxious participants is required, particularly as there is a trend in the repressor literature for studies choosing not to include this group at all (Myers, 2010). Arguably, it is important for studies to include this group, even in the case of ours with low numbers, to be confident that repressor differences are due to the unique combination of low anxiety and high defensiveness. Although the generalisability of our results regarding the defensive high anxious group are limited, our findings suggest a hyper-reactive response to the TSST-G compared to the other groups. Our results are also restricted to healthy young females. Given that sex differences have previously been found in work on both repressive coping (Diehl et al., 1996) and cortisol reactivity (Smyth et al., 2013), it may be interesting to carry out a similar study with healthy young males.

4.1 Conclusions

In conclusion, the present study found that those classified as repressors exhibit a lower cortisol response to a group stressor involving negative social-evaluative threat and uncontrollability. Our findings suggest that lesser cortisol reactivity in those with a repressive coping style may be a means of maintaining both a positive self-evaluation and social-self and may be part of an overall strategy to inhibit negative affect. This finding of a distinct cortisol reactivity profile could have physiological relevance to the repression-adverse health link.

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Conflicts of interest

The authors report no conflicts of interest.

Contribution of the authors

The work presented in this manuscript was undertaken collaboratively by all authors. All authors contributed to the research process and have approved the final manuscript.

CRediT author statement

Andrea Oskis: Conceptualization, Methodology, Data Curation. Writing- Original draft preparation. **Nina Smyth:** Conceptualization, Methodology, Laboratory Analyses; Data Curation. Writing- Original draft preparation. **Maria Flynn**: Methodology, Data Curation. Writing- Original draft preparation. **Angela Clow**: Conceptualization, Methodology, Data Curation. Writing- Original draft preparation, Team Supervision.

Acknowledgements

We would like to acknowledge the financial support provided by the Bial Foundation. Without this support the work would not have been possible.

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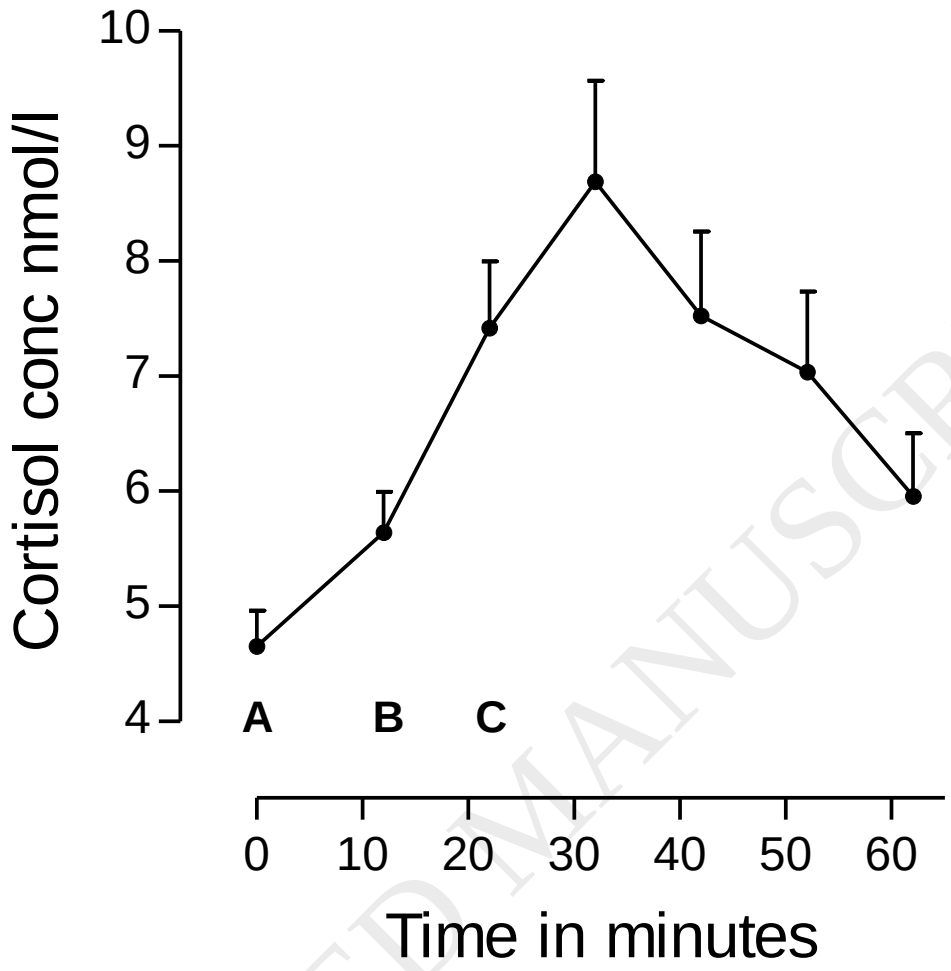

Figure 1 Figure 1 Mean (±S.E.M.) salivary free cortisol concentrations (nmol/l) for cortisol responders (*n* = 64) over the course of the TSST-G. A: immediately prior to the task; B: after the public speaking task element; C: after the mental arithmetic element i.e. the end of the task.

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Figure 2 Repressive coping group differences in cortisol reactivity in the TSST-G (*n* = 64). Repressor and low anxious groups had significantly lower reactivity during the TSST-G compared to those who were defensively high anxious (*p* = .004 for repressors, and *p* = .030 for low anxious, respectively)

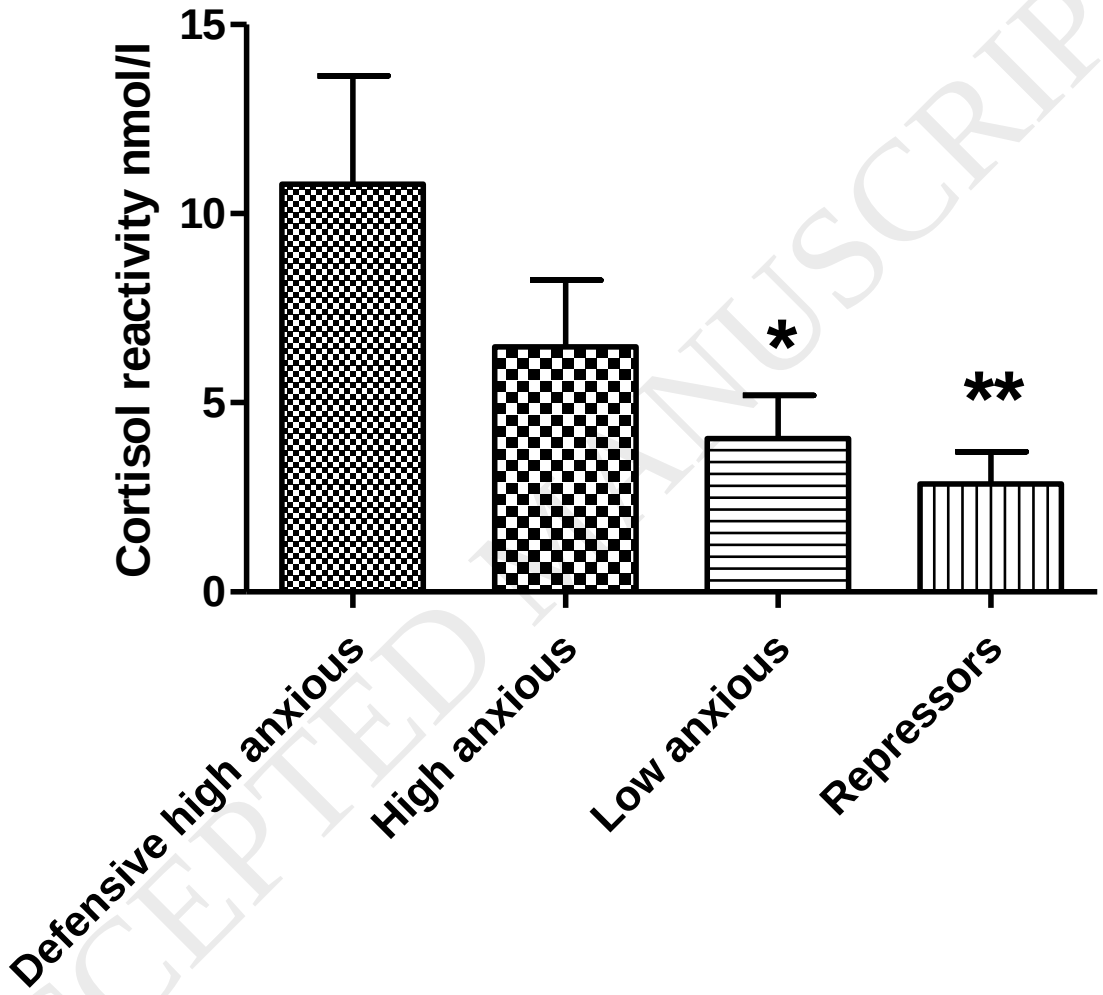

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Figure 3 Group differences in the perceived stress scores (PSS) of cortisol responders (*n* = 64). There was a significant difference between the high anxious and repressive coping groups

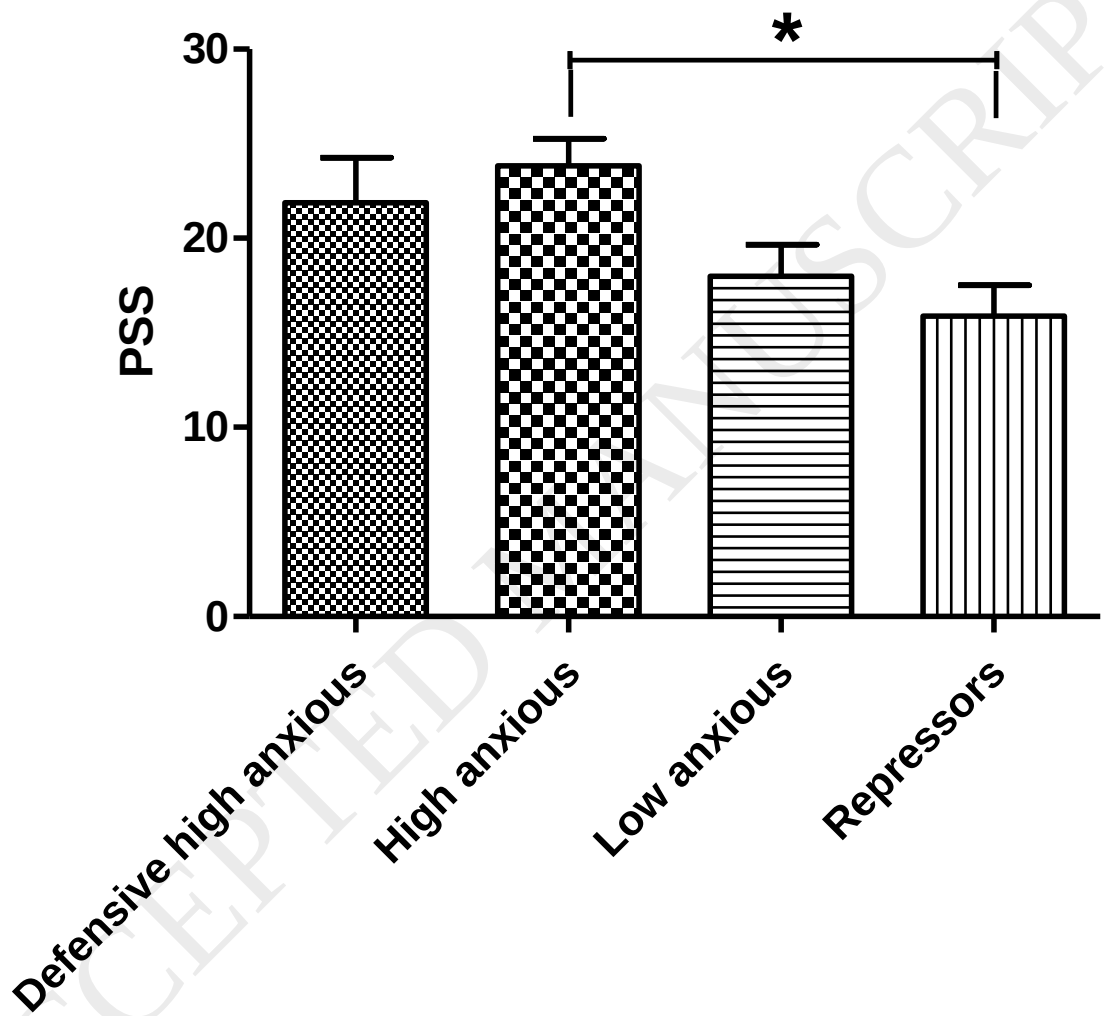

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Table 1

Variable		Correlations											
	M (SD)	Cortisol recovery	STAI-T	MC-SDS	PSS	Pre-stress	Pre-arousal	Post-stress	Post-arousal	Age	Menstrual cycle phase	Smoking status	BMI
Cortisol reactivity	5.45 (6.81)	.423**	.298*	.016	.214	-.025	.197	.200	.184	.381**	-.098	-.049	-.119
Cortisol recovery	4.15 (4.68)		.070	.113	.150	.047	.205	.169	.107	.313*	-.181	.088	-.002
Trait anxiety (STAI-T)	44.92 (9.85)			-.341**	.698**	.351**	-.196	.238	-.164	.108	.136	.129	-.103
Defensiveness (MC-SDS)	16.83 (4.73)				-.235	-.250*	.086	.002	.089	-.061	-.070	-.117	.231
Perceived Stress (PSS)	20.01 (7.42)					.245	-.280*	.251*	-.197	.024	.079	.018	.160
Pre-stress (SACL)	1.14 (.47)						-.030	.228	-.122	.024	.106	-.064	-.053
Pre-arousal (SACL)	.78 (.51)							-.107	.457**	.103	-.028	-.055	-.059
Post-stress (SACL)	1.03 (.55)								-.144	.081	.115	-.141	.067
Post-arousal (SACL)	.99 (.56)									-.016	-.067	-.021	.149
Age	20.45 (3.49)										-.010	.116	.215
Menstrual cycle phase % luteal	35.94											.012	.005
Smoking status % non-smoker	87.50												-.005
BMI	21.64 (3.92)												

*p <.05, **p <.001

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Table 2

Variable M (SD)	Repressor (*n* = 17)	Defensive high anxious (*n* =8)	High anxious (*n* = 23)	Low anxious (*n* = 16)	
Trait anxiety (STAI-T)	35.53 (7.35)	51.13 (2.10)	53.74 (6.11)	39.13 (4.18)	**
Defensiveness (MC-SDS)	21.82 (1.67)	21.63 (2.26)	13.57 (2.90)	13.81 (3.12)	**
Perceived Stress (PSS)	15.88 (6.24)	21.88 (7.95)	23.84 (7.17)	17.98 (6.06)	*
Pre-stress (SACL)	0.96 (0.55)	1.03 (0.45)	1.33 (0.40)	1.11 (0.40)	*
Pre-arousal (SACL)	0.86 (0.56)	0.83 (0.38)	0.72 (0.52)	.76 (.54)	
Post-stress (SACL)	0.82 (0.54)	1.29 (0.61)	1.15 (0.47)	.96 (.58)	*
Post-arousal (SACL)	0.99 (0.49)	1.08 (0.66)	0.92 (0.59)	1.03 (.58)	
Age	19.47 (1.62)	19.25 (1.28)	20.83 (3.93)	21.50 (4.66)	
BMI	22.22 (3.42)	22.14 (2.97)	20.60 (3.63)	22.27 (5.10)	
Menstrual cycle phase % luteal	18.00	63.00	39.00	38.00	
Smoking status % non-smoker	88.00	100.00	78.00	94.00	

*p <.05, **p <.001