



Social-evaluative threat: Stress response stages and influences of biological sex and neuroticism

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ARTICLE INFO

Keywords:

Social-evaluative threat
Gender
Cortisol
Anxiety
Social evaluation
Threat

ABSTRACT

Social-evaluative threat (SET) – when the self could be negatively judged by others – can cause pronounced responses in the different stress systems: threat/challenge appraisal, the sympathetic (SNS) and parasympathetic (PNS) nervous systems, experienced motivation and affect, and the hypothalamus-pituitary-adrenal (HPA) axis. Here, we utilize a four-stage stress response model to shed light on the complex associations between different stress responses, where earlier stages are hypothesized to predict later stages. Additionally, we take into account important moderators, such as biological sex (controlling for menstrual cycle phase), personality traits (neuroticism and extraversion), and baseline stress levels. Thirty-seven men and 30 women in their luteal phase participated in an impromptu public speaking task to induce SET. Stress responses in four different stages were measured using: self-reported appraisal (threat or challenge, stage 1: S1), cardiovascular measures (pre-ejection period as SNS index, respiratory sinus arrhythmia as PNS index, S2), self-reported motivation and affect (state approach motivation, state anxiety, S3) and endocrine measures (cortisol as HPA index, S4). Stress reactivity was calculated by subtracting individual peaks from baseline. Results showed that SET induced pronounced stress reactivity in stages two to four. Against expectations, self-reported appraisal (S1) or motivation and affect (S3) did not predict later stress reactivity. As hypothesized, increased SNS (but not PNS) reactivity (S2) predicted increased HPA reactivity (S4). Bayesian model comparison confirmed the absence of sex differences in stress reactivity, likely due to controlling for menstrual cycle phase and sex differences in neuroticism levels. Higher trait neuroticism predicted blunted SNS (S2) and HPA (S4) reactivity, while higher baseline stress levels predicted blunted stages two and three reactivity overall. In conclusion, this rigorously controlled experiment partly supports and partly contradicts previous findings regarding associations between stress response stages, and offers new insight into the causes of blunted HPA responses in women.

1. Introduction

Social-evaluative threat (SET) – when the self could be negatively judged by others – can occur frequently in daily life (Dickerson and Kemeny, 2004; Smith et al., 2012). SET can threaten the universal need to belong, an evolutionary powerful motivator not to be socially rejected, that causes distress when not met (Baumeister and Leary, 1995). Therefore, SET is one of the strongest psychological laboratory stressors (Dickerson and Kemeny, 2004), causing responses in each stress system after threat/challenge appraisal, notably in the sympathetic (SNS) and parasympathetic (PNS) nervous systems, experienced motivation and affect, and the hypothalamus-pituitary-adrenal (HPA) axis. These stress responses are typically experimentally provoked using a public

speaking task (PS; Bliss et al., 1956) or the Trier social stress test (TSST; Kirschbaum et al., 1993; the combination of PS with a mental math test).

It has long been assumed that the stress systems are associated (Darwin, 1872/Darwin, 1965; Lindsley, 1951). However, empirical research into these associations suggests that different stress response systems interact in a complex manner (e.g., Campbell and Ehlert, 2012; Denson et al., 2009; Dickerson et al., 2004; Dickerson and Kemeny, 2004; Joëls and Baram, 2009). Indeed, many theoretical models exist that try to explain these complex associations (see Table A.1 in the Appendix). Based on the biopsychosocial (BPS) model of arousal regulation (Blascovich, 2013; Blascovich and Tomaka, 1996), we propose the utilization of a four-stage stress response model to guide the

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<https://doi.org/10.1016/j.psyneuen.2019.104378>

Received 19 December 2018; Received in revised form 9 July 2019; Accepted 9 July 2019

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analyses of these complex interactions, where former stages are hypothesized to influence the latter stages. We will discuss each stage in detail, and summarize empirical support for their interactions.

At the initial appraisal stage (S1), goal-relevant situations are appraised in terms of the demands of the situation and the available resources (Lazarus and Folkman, 1984). A threat is perceived when demands outweigh resources, while a challenge is perceived when resources outweigh demands. Previous research has suggested that threatening appraisal leads to smaller SNS and combined SNS/PNS responses (S2) after a math task (e.g., Quigley et al., 2002; Tomaka et al., 1993), but to larger negative affective responses (S3) after a math task (Gramer and Saria, 2007; Tomaka et al., 1993), after the TSST (Gaab et al., 2005), and during PS (Gramer and Saria, 2007; Gramer and Sprintschnik, 2008), as well as to larger HPA responses (S4) after the TSST (Gaab et al., 2005; Schlotz et al., 2011).

The second stage (S2) is characterized by increased activity of the SNS to prime the body for action. Additionally, though not explicitly mentioned in the BPS model, the PNS – involved in preparing the body for rest – usually reduces in activity (Porges, 1995). Combined effects of both the SNS and PNS are reflected in, e.g., increased heart rate (HR) and blood pressure (BP) (Glick et al., 1965). Larger responses in combined SNS/PNS seem to be related to larger negative affective responses (S3) after PS, according to a meta-analysis (Feldman et al., 1999) and to other PS studies (Feldman et al., 2004; Gonzalez-Bono et al., 2002), as well as during and after the TSST (Hellhammer and Schubert, 2012; Rimmele et al., 2007). Additionally, larger responses in combined SNS/PNS seem to be related to larger increases in HPA responses (S4) after the TSST (Gold et al., 2003; Kirschbaum et al., 1996) and PS (Al'Absi et al., 1997; Cohen et al., 2000), though this effect seems to be driven by SNS increases and not PNS decreases (Bosch et al., 2009; Evans et al., 2016).

The third stage (S3) is characterized by motivational and affective responses (Blascovich and Tomaka, 1996). Motivational responses represent a motor preparation to approach challenges and a conflict to both approach and avoid goal-relevant threats (Blascovich, 2013; Frijda et al., 1989). Additionally, the subjective emotional experience, i.e., affect, represents elicited feelings and serves to regulate the stress response (Blascovich and Tomaka, 1996). Although motivation is typically not measured, more negative affective responses (both undifferentiated negative affect and specific emotions such as anxiety) during and after the TSST seem to be related to larger increases in HPA responses (S4) (e.g., Childs et al., 2010; Hellhammer and Schubert, 2012).

Finally, the fourth stage (S4) represents an increased activity of the HPA axis, reflected in the release of the “stress hormone” cortisol (Hellhammer et al., 2009). As a promoter of prolonged energy mobilization, the HPA depends on a slow cascade to produce cortisol, with the peak activation in saliva being around 13.5 min behind that of SNS (Engert et al., 2011) and around 16 min behind that of affect (Schlotz et al., 2008).

The BPS model (Blascovich and Tomaka, 1996) differs with our operationalization in three ways: First, we added the measurement of PNS in stage 2 (Porges, 1995), since there is some evidence that associations between often-used combined SNS/PNS indices (e.g., heart rate) and other stress response indices (e.g., cortisol) are driven by SNS activity (Bosch et al., 2009; Evans et al., 2016). Second, we placed HPA axis activity in stage 4 instead of grouping it together with “physiological arousal” during stage 2, based on its known timing difference with SNS activity (Engert et al., 2011). Third, we simplified the model in several ways, such as: by not distinguishing between benign and malignant patterns of stage 2 SNS activation (i.e., with vascular resistance and cardiac performance); by not taking cognitive or interpersonal moderators of appraisal into account; and by not measuring cognitive and behavioral responses. Despite these simplifications, the theoretical framework of the BPS model (Blascovich and Tomaka, 1996) could prove to be a useful tool to help break down the complex interactions

between stress responses in order to guide hypothesis-driven analyses by using multiple regressions. Based on the BPS model (Blascovich and Tomaka, 1996) and the aforementioned empirical support, we specifically hypothesized that increased threat appraisal (S1) would lead to smaller SNS increases and PNS decreases (S2), larger anxiety increases (S3), and larger cortisol increases (S4). Moreover, we expected that larger SNS increases and PNS decreases (S2) would be associated with larger anxiety increases (S3), and that larger SNS (but not PNS) responses (S2) would be associated with larger HPA increases (S4). Finally, we expected that larger anxiety increases (S3) would be associated with larger cortisol increases (S4). Besides our hypothesis-driven analyses, additional exploratory analyses will be discussed.

However, there is also considerable empirical evidence not supporting the BPS model at each stage. For associations with the first stage, threat/challenge appraisal was often found to be unrelated to combined SNS/PNS responses (S2) during PS (e.g., Feldman et al., 2004; Gramer, 2003), as well as to PNS responses after a math task (Quigley et al., 2002). Additionally, a meta-analysis found that both estimates of threat and (more so) challenge appraisals were related to larger HPA responses (S4) (Denson et al., 2009). For associations with the second stage, many studies have failed to find relationships between combined SNS/PNS responses and affective responses (S3) after the TSST (e.g. Denson et al., 2012; Ditzen et al., 2007) and during and after PS (e.g., Al'Absi et al., 1997; Gramer and Reitbauer, 2010). For associations with the third stage, more studies failed to find relationships between negative affective responses and HPA responses (S4) than those that did, for example during and after the TSST (e.g., Gold et al., 2003; Nierop et al., 2008), as well as during and after PS (e.g., Cohen et al., 2000; Jansen et al., 2000), and one study even found a negative correlation after the TSST (Het et al., 2012). Compellingly, a review and a meta-analysis confirmed there to be no significant associations between changes in negative affect after the TSST and HPA responses (Campbell and Ehlert, 2012; Dickerson and Kemeny, 2004). Instead, there might only be associations with specific affective phenomena, such as fear of losing social approval after PS, as supported by a meta-analysis (Denson et al., 2009), and shame after the TSST, as proposed by a review (Dickerson et al., 2004). In light of this conflicting evidence against the BPS model, our hypotheses become more uncertain.

However, the conflicting evidence might also be explained by recurrent methodological issues. Conflicting associations involving SNS and PNS responses (S2) might be due to using only combined SNS/PNS indices (Bosch et al., 2009). The conflicting associations involving negative affect (S3) might be explained by multiple issues, predominantly: using only a single time point (instead of change from baseline); measuring global mood states instead of specific emotions (Denson et al., 2009); and measuring affective responses retrospectively (Barrett, 1997). The latter point is especially problematic, since recall of negative affect is only moderately accurate (Barrett, 1997), and negative affect measured after the cessation of stress is greatly reduced as compared to negative affect measured during stress (or its anticipation) (Ditzen et al., 2008; Hellhammer and Schubert, 2012). Therefore, it is not surprising that only affective responses using measurements during stress (anticipation) seem to be associated with responses in other stress systems (Hellhammer and Schubert, 2012). Finally, differences between tasks might be explained by response specificity to stressor type (Dickerson et al., 2004). While PS is a social stressor and a math task is mainly a cognitive stressor, the TSST cannot distinguish between them (Benschop and Schedlowski, 1999; Denson et al., 2009). We argue that PS is most suitable for studying SET responses, since PS is perceived as more threatening and anxiety-inducing than a math task (Gramer, 2003; Gramer and Saria, 2007), and more likely to reveal associations between stress responses (Al'Absi et al., 1997). Moreover, the TSST and math tasks may induce sex differences in stress responses due to stereotype threat to mathematics (Steele and Aronson, 1995; Vick et al., 2008). Thus, the current study aims to avoid these methodological issues by repeatedly measuring multiple stress systems in response to PS

and exploring the associations in reactivity.

Furthermore, important moderators can influence stress responses and their associations, which most of the previously mentioned studies disregarded. According to the BPS model (Blascovich and Tomaka, 1996), physiological processes and biological factors define the dynamic range of physiological arousal systems; while cognitive factors, such as affective states, moderate the affective stress response. For example, higher baseline stress levels are well-known to cause blunted stress responses (Kudielka et al., 2004b; Oswald et al., 2006). Besides baselines, the arguably most important moderator is biological sex. Women seem to be more prone to threat appraisal than men due to increased attention for negative social stimuli (Benenson et al., 2013), and they report higher negative affect after the TSST (Kelly et al., 2008) and PS (Carrillo et al., 2001). Importantly, women might respond with larger HR increases than men to the TSST (Childs et al., 2010; Kudielka et al., 2004a) and to a math task (Tersman et al., 1991), and a review has confirmed that women often show larger HR reactivity while men show larger blood pressure reactivity (Ordaz and Luna, 2012). Additionally, reviews and a meta-analysis have shown that men respond with larger cortisol increases than women (Kajantie and Phillips, 2006; Kudielka and Kirschbaum, 2005; Liu et al., 2017; Ordaz and Luna, 2012). Indeed, associations between stress systems may also differ between sexes (Carrillo et al., 2001; Rattel et al., 2019 Submitted). Importantly, however, sex differences in stress responses might be caused mainly by sex hormones rather than biological sex itself (Kajantie and Phillips, 2006; Ordaz and Luna, 2012). For example, when statistically adjusting for sex hormone baseline levels, women and men exhibit similar cortisol responses to the TSST (Juster et al., 2016). Additionally, comparisons of different menstrual cycle phases (utilizing naturally fluctuating sex hormones) revealed that women have smaller cortisol responses to the TSST than men only in the follicular phase and when taking oral contraceptives (e.g., Kirschbaum et al., 1999; Liu et al., 2017). In contrast, in the luteal phase (when estrogen and progesterone levels are both high), they have similar cortisol responses (e.g., Kirschbaum et al., 1999; Rohleder et al., 2001); although other studies have still found higher cortisol responses to the TSST in men than women in either the luteal or follicular phase (e.g., Bouma et al., 2009; Childs et al., 2010). Moreover, women show larger negative affective responses after the TSST in the luteal phase than in the follicular phase and than men, as well as larger SNS responses and combined SNS/PNS responses during PS and a math test (e.g., Childs et al., 2010; Sita and Miller, 1996); although other studies found no differences in combined SNS/PNS responses between menstrual cycle phases after PS and the TSST (Childs et al., 2010; Stoney et al., 1990). Finally, associations between stress systems seem to differ between menstrual cycle phases, with negative associations between cortisol responses and negative affect after the TSST during the follicular phase and positive associations during the luteal phase (Duchesne and Pruessner, 2013; Tiwari et al., 2015). Thus, by only testing in the luteal phase, the current study expects to find no sex differences in HPA responses, but possibly larger anxiety and smaller SNS/PNS responses in women, as well as positive associations between state anxiety and HPA responses. Uniquely, we will test for evidence for our null hypothesis of no sex differences by using Bayesian statistics.

Personality traits are also important moderators of stress responses. According to the BPS model (Blascovich and Tomaka, 1996), intrapersonal factors, such as resilient personality traits, moderate the appraisal of the situation. Particularly, higher neuroticism and lower extraversion are related to lower resource appraisal and higher negative affect after PS (Penley and Tomaka, 2002). Additionally, both higher neuroticism and extraversion have been found to be associated with blunted responses in combined SNS/PNS indices and the HPA to PS (Bibbey et al., 2013; Evans et al., 2016); although some studies have found larger SNS increases after PS with higher neuroticism (Evans et al., 2016) and some studies found no associations between personality and stress responses after the TSST (Laceulle et al., 2015) or after

PS (Chida and Hamer, 2008). This divergence might be due to sex differences: when analyzing men and women separately, blunted HPA responses to the TSST were associated with higher neuroticism levels in women but with lower extraversion levels in men (Oswald et al., 2006) and baseline cortisol was positively correlated with neuroticism in men but negatively in women, especially after controlling for oral contraceptive use (DeSoto and Salinas, 2015). Menstrual cycle might also influence the relationship between neuroticism and stress responses, as there seem to be negative correlations to BP responses during a math test in the follicular phase but no significant correlations in the luteal phase (Schallmayer and Hughes, 2010). Thus, we hypothesize to find decreased stress responses for all stages with higher levels of extraversion and neuroticism, and a potential interaction with sex.

In conclusion, the current study measures responses to PS in multiple stress systems and follows a four-stage stress response model to investigate their associations. Additionally, we aimed to avoid common methodological pitfalls and to take into account important moderators, such as biological sex (controlling for menstrual cycle phase), personality traits (neuroticism and extraversion), and baseline stress levels.

2. Method

2.1. Participant characteristics

Forty-two men and 43 women were invited to participate in this experiment as part of a larger study. Participants were 18–35 years of age, right-handed (important for measurement of electroencephalography (EEG); reported elsewhere), had normal or corrected-to-normal vision, were currently studying at college or university, were heterosexual (which can influence cortisol: Juster et al., 2015), free of psychiatric and endocrinological disorders, not taking medication that could influence cognition, emotion, or hormones,¹ and were not a regular smoker or drinker (which can influence cortisol: Foley and Kirschbaum, 2010). Additionally, female participants did not use oral hormonal contraception or an intrauterine device for at least the last three months, were not currently pregnant or breast-feeding, and had a regular menstrual cycle (see section 2.1.1 Luteal phase calculation). To ensure participants fulfilled all inclusion criteria, as well as to assess a history of psychiatric disorders (two participants) or current medical/endocrinological disorders (none), we used a custom health questionnaire. Participants were compensated with cash or course credit. The experiment was approved by the ethical committee of the University of Salzburg.

Eighteen participants were excluded in total, due to: ambidexterity (two men), technical difficulties (missing recording timing markers: three men and one woman; dreadlocks making EEG measurement impossible: one woman), or not being in the luteal phase (11 women; see section 2.1.1 Luteal phase calculation). Thus, the final sample contained 37 male participants (age: $M = 22.8$, $SD = 2.6$) and 30 female participants (age: $M = 22.9$, $SD = 2.8$)².

2.1.1. Luteal phase calculation

Female participants were tested during the luteal phase of their menstrual cycle. The luteal phase ranged from the third day post-ovulation to three days before the onset of the next menses (days 17–26 for a 28-day cycle). A regular menstrual cycle was defined as a 21–35 day

¹ Current medication intake included: antibiotics (one participant) and mild pain medication (one participant). History of medication intake was not assessed.

² A post-hoc statistical power calculation conducted with G*Power (Faul et al., 2007) showed that the final sample size offered 80% power to detect sex differences with an effect size of $d = 0.7$ or larger, and to detect stress system associations of $d = 0.33$ or larger. Additional use of Bayesian model comparison offered the distinction between nonsignificant results due to lack of power or due to support for the null hypothesis.

cycle duration, with an intra-cycle variability of < 7 days (Fehring et al., 2006). In case of a mildly irregular cycle, defined as inter-cycle variability 3–7 days, ovulation tests for urinary LH testing (Pregnafix®) were used to confirm the ovulation (instead of calculating the expected ovulation date) before scheduling the experiment (13 participants, out of which nine participants are in the final sample). Participants reported the onset of their next menses after testing and were excluded in case the experiment was completed on < 2 days or > 12 days before the onset of next menses.

For the women in the final sample, the average day of testing within the menstrual cycle was 20.5 ($SD = 3.6$), with an average cycle duration of 27.6 days ($SD = 3.2$). As an extra check of the luteal phase, free salivary progesterone levels were analyzed. A combination of saliva measurements one, four and seven were taken as the average progesterone level. The same analysis procedure was used as described in section 2.3.5 *Endocrine physiology*, but for one time point (with duplicates). Progesterone levels were in a plausible range of 42.6 and 527.5 pg/ml, with an average of 194.7 ($SD = 121.9$).

2.2. Social-evaluative threat manipulation

To induce social-evaluative threat (SET), an impromptu speaking task was used. We adjusted a protocol of the Leiden public speaking task (Westenberg et al., 2009) – validated in young adolescents – for a young adult sample, which uses a pre-recorded audience. The advantage of using a pre-recorded audience is that the audience composition and behavior is consistent for all participants, while still causing a considerable stress response in salivary cortisol levels, comparable to that of the TSST (van den Bos et al., 2014; Westenberg et al., 2009). In brief, participants were first told in the lab that they would give a five-min speech about their positive and negative personality characteristics. The speech would be given in front of a life-size-projected pre-recorded audience (eight PhD-students (50% male) and one male teacher from Salzburg University, all with neutral facial expressions) and a camera. We told participants that their video would later be evaluated by that same audience on 10 aspects concerning speech delivery, content, and quality. In reality, however, the videos were not evaluated and participants were informed about this deception during the debriefing. Participants were given five min to prepare their speech, without taking notes. During the speech, the video of the neutral pre-recorded audience was shown, including a small timer counting up to five min while a camera recorded their speech. The camera viewer was flipped so that participants could see themselves during the speech, to increase self-focus. In the case that a participant stopped talking for longer than 20 s, a prompt was given by the experimenter. The entire SET manipulation lasted about 18 min, including the speech in the final five min.

A flowchart of the SET protocol is displayed in Table 1 and a screenshot of the video audience is shown in Figure S2 (Supplementary Materials; full instructions and the pre-recorded audience video are available upon request from the first author).

2.2.1. Procedure

Participants were unaware of the upcoming tasks during the baseline measurements and only were told they would perform tasks relating to “brain activity, hormones, and heart rate” before signing the informed consent. Only after completing an approach-avoidance task (AAT; reported elsewhere) were participants first instructed about the upcoming public speaking task. After giving their speech, participants completed another AAT. Participants remained seated throughout the experiment, so as not to influence the cardiovascular measures. The experiments were only performed between 12am and 6pm, when cortisol levels are low and sensitive to external stimulation (Nicolson, 2008). Participants were instructed not to eat or drink anything (except for water) or exercise within 30 min prior to the start of the experiment, and did not smoke on the same day (except for one male participant, who was not excluded), which was checked using a custom health

questionnaire.

The experiment was held in English, both for practical reasons as well as to increase the stressfulness of the speech. No participants spoke English as a native language (mostly German), but all participants were competent enough to successfully participate (We checked English language competence; see section 2.3.1 *English competence*).

2.3. Assessments and measures

2.3.1. English competence

A general English language competence test (www.cambridgeenglish.org/test-your-english/general-english/) was used to assess whether lower English language competence might have an influence on SET reactivity, since the speech was held in English. It contained 25 items ($\alpha = .74$) with each three or four response options and one correct answer (e.g., “It was only ten days ago then/since/after that she started her new job.”).

2.3.2. Traits

The Big Five Aspects Scale (DeYoung et al., 2007) was used to measure trait neuroticism (e.g. “I get upset easily.”) and extraversion (e.g. “I make friends easily.”) (both scales: $\alpha = .89$).

Descriptions of additional questionnaires that were assessed but not used for the analyses in the current manuscript can be found in the Supplementary Materials.

2.3.3. Self-reported appraisals

Resource and demand appraisals (stage one of the stress response; Tomaka et al., 1993) were both assessed with single questions. Demand appraisal was measured with: “How demanding do you expect the upcoming task to be?” and resource appraisal with: “How able are you to cope with the upcoming task?”. Responses were given on a visual analogue scale (VAS), with scores ranging from *not at all* (0) to *extremely* (100).

Since resources and demands were correlated ($r(65) = -0.29$, $p = 0.016$), a continuous composite measure of resources and demands was calculated for parsimony and to aid interpretation (according to e.g., Tomaka et al., 1993). Demands were subtracted from resources, yielding positive values in case of higher resources than demands (challenge) and negative values in case of higher demands than resources (threat).

2.3.4. Cardiovascular physiology

Cardiovascular physiology was recorded to measure the following indices of stage two of the stress response: heart rate (HR; index of combined SNS/PNS), mean blood pressure (BP; index of combined SNS/PNS), pre-ejection period (PEP; index of SNS), and respiratory sinus arrhythmia (RSA; index of PNS), as well as respiratory rate (RR) as a covariate in RSA analyses (Grossman and Taylor, 2007).

Electrocardiography (ECG), impedance cardiography (ICG), and respiration were recorded continuously using a 64-channel amplifier (TMSi, Oldenzaal, the Netherlands) and the recording software package Polybench 1.22 (TMSi, Oldenzaal, the Netherlands) with a sampling rate of 1024 Hz. Lead-II ECG was recorded using two spot electrodes and ground. ICG was recorded using an eight-spot pairwise electrode configuration over the neck and abdomen following guidelines by Sherwood et al. (1990). Respiration was recorded using inductive plethysmography (SleepSense, S.L.P. Inc, IL, USA) at the thoracic diaphragm. Systolic and diastolic BP were measured from the left upper arm using an automated blood pressure monitor (Ecomed BU-90E, Medisana AG, Neuss, Germany).

The ECG and ICG signals were analysed using ANSLAB (Autonomic Nervous System Laboratory V2.6; Blechert et al., 2015), according to standard analysis protocols (i.e., manual artefact correction, resampling to 4 Hz, subsequent ensemble-averaging). Respiratory sinus arrhythmia was analysed by spectral analysis as the high frequency component of

Table 1
Flowchart of the SET protocol.

Time (min) respective to start SET	Time (min) respective to end SET	Activities	Measurements
-77	-95	General instructions; questionnaires (incl. informed consent)	
-62	-80	Watching nature video	Attaching (electro)physiological assessments ^a
-27	-45	Practice measurement	Blood pressure 1
-25	-43	Baseline (viewing fixation cross)	(Electro)physiology (first and last 2.5 min (baseline))
-20	-38		Blood pressure 2 (baseline)
-19	-37		VAS ^b 1 (baseline)
-18	-36		Saliva ^c 1 (baseline)
-14	-32	AAT ^d 1	EEG ^a
0	-18	Start of SET: Instructions speech task	
4	-14		Blood pressure 3
5	-13	Anticipation (viewing fixation cross)	(Electro)physiology (first and last 2.5 min)
10	-8		Blood pressure 4
11	-7		VAS 2 and appraisal
13	-5	Speech	Video recording
18	0	End SET	
19	1		VAS 3
20	2		Saliva 2
23	5		Blood pressure 5
24	6	Recovery (viewing fixation cross)	(Electro)physiology (first and last 2.5 min)
29	11		Blood pressure 6
30	12		VAS 4
31	13		Saliva 3
33-53	15-35	AAT 2	EEG
35	17	After AAT 2 practice	VAS 5
36	18		Saliva 4
40	22	After AAT 2 block 1	VAS 6
41	23		Saliva 5
45	27	After AAT 2 block 2	VAS 7
46	28		Saliva 6
50	32	After AAT 2 block 3	VAS 8
51	33		Saliva 7
53	35		Blood pressure 7
54	36	Second recovery (viewing fixation cross)	(Electro)physiology (first and last 2.5 min)
59	41		Blood pressure 8
60	42	Debriefing	Detaching (electro)physiological assessments
70	52	Questionnaires	
100	82	End	

^aImpedance cardiography (ICG), electrocardiography (ECG), respiration, and electroencephalography (EEG; reported elsewhere) were measured continuously.

^bVAS = visual analogue scale measuring state anxiety and state approach motivation.

^cSaliva sampling was used to assess cortisol, as well as estradiol and testosterone (reported elsewhere).

^dAAT = approach-avoidance task (reported elsewhere).

Note. Light shaded areas represent the SET manipulation, and dark shaded areas represent the speech.

variation in IBIs (i.e., high frequency heart-rate variability) within the 0.14–0.5 Hz frequency band using fast Fourier transform with the Welch algorithm. RSA values were transformed by natural logarithm. RR was calculated as 60 s divided by the continuously measured breath

duration. BP was calculated as mean arterial pressure ($2/3$ diastolic + $1/3$ systolic)(Sesso et al., 2000).

For each measure except BP, the last 150 s of the resting state was used as a baseline (and the second measurement for BP), as

recommended (Blascovich and Tomaka, 1996). To assess the response, the first and last 150 s of anticipation, first recovery, and second recovery were used (and measurements before and after these periods for BP). During the speech itself, cardiovascular physiology was not measured due to excessive movement artefacts.

2.3.5. Self-reported affective and motivational states

Affective and motivational responses (stage three of the stress response) were measured using state anxiety and state approach motivation, respectively (Harrewijn et al., 2016; Poppelaars et al., 2018; Rinck et al., 2013). State anxiety was measured with the single question: “How anxious do you feel right now?”, and state approach motivation was measured with the single question: “How much are you looking forward to the next part of the study?”, both on a VAS, with scores ranging from *not at all* (0) to *extremely* (100).

2.3.6. Endocrine physiology

In order to assess free salivary cortisol (stage four of the stress response as an index of HPA activity), seven saliva samples of 2 ml each (as an adequate amount for analyzing multiple hormones) were collected by spitting directly in the tube, at -36, 2, 13, 18, 23, 28, and 33 min after the end of the SET manipulation. All samples were immediately stored in a freezer at -20°C until further analysis. After thawing, solid particles were removed by centrifugation twice (3000 rpm for 15 min, and again at 3000 rpm for 10 min) and the remaining saliva was refrozen until further analysis. Analysis was performed at the University of Salzburg using ELISA (DeMediTec Diagnostics, Kiel, Germany). Cortisol levels were quantified using two duplicate measures for each saliva sample to increase reliability, and samples with intra-assay coefficients of variability above 25% were repeated (undergoing an additional freeze/thaw cycle).

2.4. Statistical analyses

Statistical analysis was performed in RStudio version 1.1.423 (RStudio Team Inc., 2016) with R version 3.1.3 (R Core Team, 2018). Extreme outlier data points based on the Grubbs test and the mean-shift outlier test were removed for three participants. Missing data due to outliers and missing data points due to artefacts or technical difficulties were managed using multiple imputation with the MICE package (Van Buuren and Groothuis-Oudshoorn, 2011), allowing the use of all non-excluded 67 participants in the statistical analyses. Details of both approaches can be found in the Supplementary Materials, and both the data and the used analysis scripts are freely available via Mendeley Data (Poppelaars et al., 2019).

Significance of SET reactivity (see section 2.4.1 SET reactivity) differing from zero was tested using one-sample *t*-tests (two-tailed), for men and women separately (i.e., 37 men and 30 women). Sex differences were tested using two-sample *t*-tests (two-tailed; variances not assumed equal). Significance of and sex differences in RSA were tested using a linear regression (with and without RR as covariate). The regression coefficients were then converted into *t*-values. To provide confirming evidence of the null hypotheses, Bayes factors were calculated from *t*-values using the BayesFactor package with default non-informative priors.

For all analyses, alpha was set at 0.05, and false-discovery rate (FDR) correction was performed to correct for multiple comparisons. Uncorrected *p*-values are reported for transparency, with FDR-corrected significance indicated by superscript symbols.

2.4.1. SET reactivity

SET responses were computed with a reactivity measure of individual peak minus baseline (Seraganian et al., 1985), henceforth identified as Δ (also known as peak reactivity; Khoury et al., 2015). The peak represents the individual maximum or minimum value (depending on the measure) during or right after SET (i.e., either early or late

anticipation, or early or late first recovery). This is a straightforward and easily interpretable measure of Δ that is sensitive to individual differences and captures Δ due to increases, independent of speed of reactivity or recovery dynamics.

Instead of calculating the area under the curve (AUC) for the cortisol response, as is commonly done, we opted to use Δ to increase comparability with other stress systems (but using all measurement points as possible peaks due to its delayed response). Individual Δ cortisol was strongly correlated with AUC with respect to the increase (Pruessner et al., 2003), $r = 0.93$, $p < .001$.

2.4.2. Associations between stress response systems

Firstly, multiple linear regressions were performed to predict later stages of the stress response by earlier stages of the stress response. As stress responses, for stage one, the resources and demands composite score was taken as appraisal. For stage two, Δ PEP and Δ RSA were taken as the most direct cardiovascular measure of SNS and PNS response, respectively (Cacioppo et al., 1994). For stage three, Δ state approach motivation was taken as a measure of the motivational response, and Δ state anxiety was taken as a measure of the subjective experience of the affective response; for stage four, Δ cortisol was taken as the most direct noninvasive measure of the peripheral HPA response (Hellhammer et al., 2009). Δ RR was added to correct for Δ RSA; however, this did not change results and was subsequently left out for parsimony. Secondly, multiple linear regressions were performed to predict each stage of the stress response by baseline states (SNS, PNS, motivation, affect, HPA) and traits (extraversion and neuroticism). Interactions with sex and English language competence were tested for each model separately.

As an additional measure of the associations between all measures, Pearson correlations were performed. The results can be found in Tables S1-4 of the Supplementary Materials.

For ease of interpretation, two effect sizes were calculated: partial Pearson correlations (correlation between dependent and independent variable, correcting for other independent variables in the model) and Cohen's *d*.

3. Results

3.1. English competence

Participants showed adequate English language competence, scoring an average of 17.57 ($SD = 3.2$) correct answers on the test, corresponding to an average of 70.3% correct answers. Men ($M = 17.68$, $SD = 2.7$) and women ($M = 17.43$, $SD = 3.7$) had comparable means of English language competence, $t(49) = 0.30$, $p = 0.768$, $BF = 0.26$. Importantly, English language competence did not interact with SET reactivity at any stage, and was therefore left out of the main analyses (Section 3.4: Associations between stress response systems) for parsimony.

3.2. Sex differences in traits and stress response indices at baseline

Men had lower trait neuroticism than women, and higher mean BP at baseline (see Table 2). Men and women had similar means of baseline state anxiety, state approach motivation, PEP, RR, and cortisol. The results on trait extraversion, baseline HR, and baseline RSA were inconclusive, according to the Bayes factors.

3.3. Stress responses and sex differences

Descriptives and tests of sex differences of the timing of individual peaks can be found in Table S5 of the Supplementary Materials. Fig. 1 shows the SET response for all measured variables. Results are shown in Table 3.

Table 2
Sex differences in traits and stress indices at baseline.

Trait / stress index at baseline	Sex	Mean	SD	t (df)	p	BF
Extraversion [20-100]	Male	71.70	10.8	1.59 (51)	0.117	0.74 inc.
	Female	66.67	14.3			
Neuroticism [20-100]	Male	51.00	11.2	3.24 (61)	0.002*	18.09 H1
	Female	59.80	10.9			
State anxiety [0-100]	Male	14.84	20.2	0.73 (56)	0.469	0.32 H0
	Female	18.77	23.2			
State approach motivation [0-100]	Male	67.89	19.5	0.40 (62)	0.687	0.27 H0
	Female	69.73	17.7			
Mean BP [mmHg]	Male	88.28	8.6	4.08 (59)	< .001**	1.81*10 ² H1
	Female	79.44	9.0			
HR [beats/min]	Male	68.66	11.5	2.15 (63)	0.035	1.74 inc.
	Female	74.19	9.5			
PEP [ms]	Male	121.36	21.7	0.53 (54)	0.601	0.28 H0
	Female	124.48	25.4			
RSA [ms ²]	Male	7.54	1.3	0.74 (61)	0.463	0.32 H0
	Female	7.77	1.2			
RR [breaths/min]	Male	15.04	3.1	0.11 (52)	0.917	0.25 H0
	Female	14.95	3.8			
Cortisol [ng/ml]	Male	3.75	2.2	0.38 (55)	0.706	0.27 H0
	Female	3.97	2.6			

Note. SD = standard deviation; BF = Bayes factor; BP = blood pressure; HR = heart rate; PEP = pre-ejection period; RSA = respiratory sinus arrhythmia (natural logarithm of high-frequency spectral power); RR = respiratory rate. ** = significant at $\alpha = 0.01$ after FDR correction; * = significant at $\alpha = 0.05$ after FDR correction; H0 = evidence in support of equal group estimates; H1 = evidence in support of different group means; inc. = inconclusive evidence in support of neither equal nor different group means.

3.3.1. Self-reported appraisal (stage one)

Self-reported appraisal was not significantly higher (i.e., resources > demands: challenge) or lower (i.e., resources < demands:

threat) than zero, although there was inconclusive evidence to support neither a true difference nor no difference from zero, according to the Bayes factors. Importantly, men and women had comparable appraisals.

3.3.2. Cardiovascular indices (stage two)

For both men and women, mean BP, HR, and RR significantly increased after SET, while PEP, and RSA (both corrected and uncorrected for RR) significantly decreased after SET (with lower PEP values indicating higher SNS activity, and lower RSA values indicating lower PNS activity). Additionally, men and women had comparable means of Δ cardiovascular indices.

3.3.3. Self-reported states (stage three)

For both men and women, state anxiety significantly increased after SET, while state approach motivation significantly decreased after SET. Additionally, men and women had comparable means of Δ self-reported states.

3.3.4. Endocrine indices (stage four)

For both men and women, cortisol significantly increased after SET. Δ Cortisol did not differ significantly between men and women, although based on Bayes factors there was inconclusive evidence to support neither equal nor different group means.

To explore whether the tendency toward higher Δ cortisol in men was due to lower neuroticism, we regressed Δ cortisol on sex and neuroticism. Interestingly, the inconclusive sex difference in Δ cortisol disappeared, and men and women showed comparable means, as indicated by the Bayes factor favoring the null hypothesis. This indicates that the sex difference in Δ cortisol may be largely attributable to sex differences in sex hormones (due to menstrual cycle phases) and trait neuroticism.

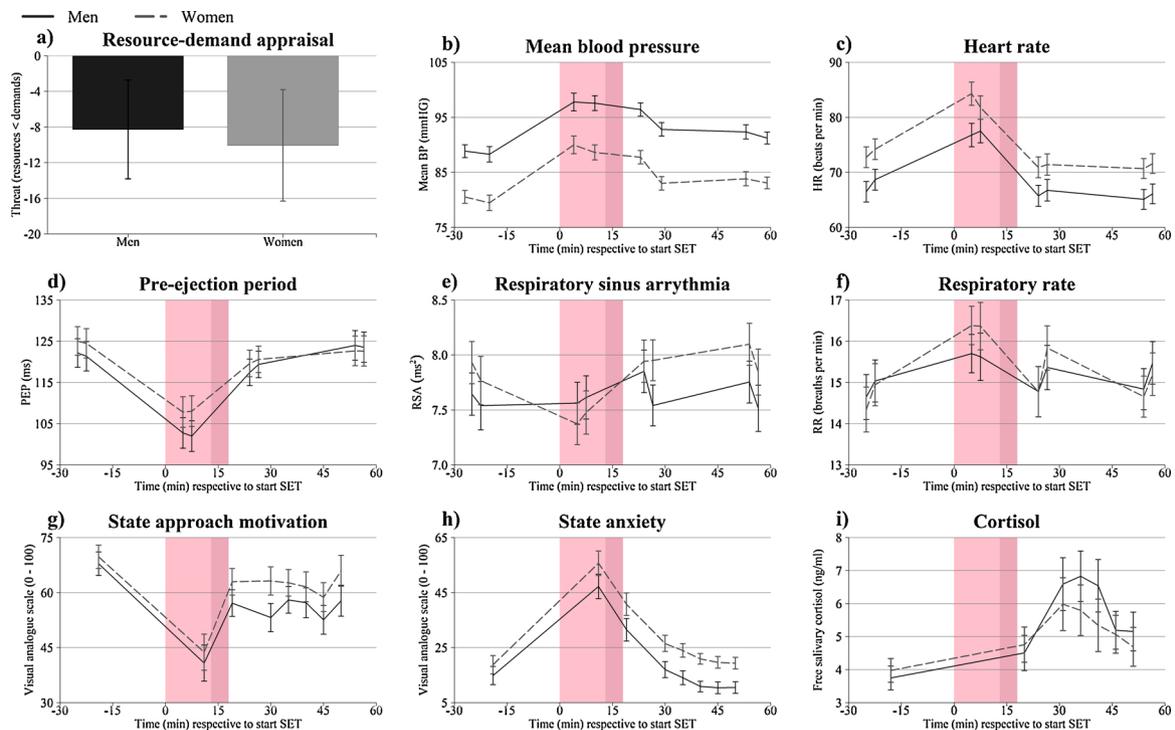


Fig. 1. Social-evaluative threat (SET) responses for men and women separately measured as: a) resource-demand appraisal (S1; negative values indicate threat appraisal, i.e., resources < demands), b) mean blood pressure (S2), c) heart rate (S2), d) pre-ejection period (S2), e) respiratory sinus arrhythmia (S2), f) respiratory rate (S2), g) state approach motivation (S3), h) state anxiety (S3), and i) cortisol (S4). Note. Light shaded areas represent the SET manipulation, and dark shaded areas represent the speech. Time zero represents the start of the SET manipulation. Error bars represent standard errors of the mean.

Table 3
Stress reactivity results for men and women and differences between sexes.

Reactivity index	Sex	Stress reactivity		Sex difference in reactivity																																																																																																																																																																	
		Mean	SD	t (df)	p	BF	t (df)	p	BF																																																																																																																																																												
Resource-demand appraisal [-100 to 100]	Male	-8.27	33.8	1.49 (34)	0.145	0.49 ^{inc.}	0.21 (60)	0.831	0.26 ^{H0}																																																																																																																																																												
	Female	-10.07	34.3	1.61 (27)	0.119	0.61 ^{inc.}				State anxiety [0-100]	Male	37.97	30.8	7.51 (34)	< .001***	1.78*10 ⁶ H1	0.66 (63)	0.510	0.30 ^{H0}	Female	42.37	23.4	9.90 (27)	< .001***	1.20*10 ⁸ H1	State approach motivation [0-100]	Male	-31.68	24.4	7.90 (34)	< .001***	5.31*10 ⁶ H1	0.69 (53)	0.495	0.31 ^{H0}	Female	-26.97	30.4	4.86 (27)	< .001***	6.27*10 ² H1	Mean BP [mmHg]	Male	12.91	5.9	13.36 (34)	< .001***	3.92*10 ¹² H1	0.45 (49)	0.653	0.27 ^{H0}	Female	13.72	8.3	9.07 (27)	< .001***	1.92*10 ⁷ H1	HR [beats/min]	Male	10.52	8.8	6.91 (31)	< .001***	3.26*10 ⁵ H1	0.23 (51)	0.816	0.26 ^{H0}	Female	9.93	10.5	4.93 (25)	< .001***	7.54*10 ² H1	PEP [ms]	Male	-20.47	14.6	7.96 (29)	< .001***	6.22*10 ⁶ H1	0.42 (53)	0.678	0.27 ^{H0}	Female	-18.90	14.5	6.81 (25)	< .001***	8.69*10 ⁴ H1	RSA [ms ²]	Male	-0.44	0.6	4.19 (32)	< .001***	1.51*10 ² H1	0.71 (43)	0.479	0.31 ^{H0}	Female	-0.59	0.9	2.91 (55)	0.006**	6.25 ^{H1}	RSA [ms ²] (corrected for RR)	Male			3.04 (56)	0.004**	8.42 ^{H1}	0.69 (57)	0.491	0.31 ^{H0}	Female			3.04 (56)	0.004**	8.09 ^{H1}	RR [breaths/min]	Male	2.31	2.7	4.88 (31)	< .001***	9.89*10 ² H1	0.32 (54)	0.751	0.26 ^{H0}	Female	2.53	2.6	4.99 (24)	< .001***	8.80*10 ² H1	Cortisol [ng/ml]	Male	3.87	4.7	5.00 (34)	< .001***	1.41*10 ³ H1	1.37 (59)	0.175	0.56 ^{inc.}	Female	2.28	4.6	2.64 (26)	0.014*	3.59 ^{H1}	Cortisol [ng/ml] (corrected for neuroticism)	Male			4.24 (63)	< .001***	1.71*10 ² H1	0.34 (60)	0.738	0.26 ^{H0}	Female	
State anxiety [0-100]	Male	37.97	30.8	7.51 (34)	< .001***	1.78*10 ⁶ H1	0.66 (63)	0.510	0.30 ^{H0}																																																																																																																																																												
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	Female	-0.59	0.9	2.91 (55)	0.006**	6.25 ^{H1}				RSA [ms ²] (corrected for RR)	Male			3.04 (56)	0.004**	8.42 ^{H1}	0.69 (57)	0.491	0.31 ^{H0}	Female			3.04 (56)	0.004**	8.09 ^{H1}	RR [breaths/min]	Male	2.31	2.7	4.88 (31)	< .001***	9.89*10 ² H1	0.32 (54)	0.751	0.26 ^{H0}	Female	2.53	2.6	4.99 (24)	< .001***	8.80*10 ² H1	Cortisol [ng/ml]	Male	3.87	4.7	5.00 (34)	< .001***	1.41*10 ³ H1	1.37 (59)	0.175	0.56 ^{inc.}	Female	2.28	4.6	2.64 (26)	0.014*	3.59 ^{H1}	Cortisol [ng/ml] (corrected for neuroticism)	Male			4.24 (63)	< .001***	1.71*10 ² H1	0.34 (60)	0.738	0.26 ^{H0}	Female			4.24 (63)	< .001***	1.33*10 ² H1																																																																																												
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	Female			3.04 (56)	0.004**	8.09 ^{H1}				RR [breaths/min]	Male	2.31	2.7	4.88 (31)	< .001***	9.89*10 ² H1	0.32 (54)	0.751	0.26 ^{H0}	Female	2.53	2.6	4.99 (24)	< .001***	8.80*10 ² H1	Cortisol [ng/ml]	Male	3.87	4.7	5.00 (34)	< .001***	1.41*10 ³ H1	1.37 (59)	0.175	0.56 ^{inc.}	Female	2.28	4.6	2.64 (26)	0.014*	3.59 ^{H1}	Cortisol [ng/ml] (corrected for neuroticism)	Male			4.24 (63)	< .001***	1.71*10 ² H1	0.34 (60)	0.738	0.26 ^{H0}	Female			4.24 (63)	< .001***	1.33*10 ² H1																																																																																																												
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	Female	2.28	4.6	2.64 (26)	0.014*	3.59 ^{H1}				Cortisol [ng/ml] (corrected for neuroticism)	Male			4.24 (63)	< .001***	1.71*10 ² H1	0.34 (60)	0.738	0.26 ^{H0}	Female			4.24 (63)	< .001***	1.33*10 ² H1																																																																																																																																												
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	Female			4.24 (63)	< .001***	1.33*10 ² H1																																																																																																																																																															

Note. SD = standard deviation; BF = Bayes factor; BP = blood pressure; HR = heart rate; PEP = pre-ejection period; RSA = respiratory sinus arrhythmia (natural logarithm of high-frequency spectral power); RR = respiratory rate. *** = significant at $\alpha = 0.001$ after FDR correction; ** = significant at $\alpha = 0.01$ after FDR correction; * = significant at $\alpha = 0.05$ after FDR correction; H0 = evidence in support of no difference from zero / equal group means; H1 = evidence in support of difference from zero / different group means; inc. = inconclusive evidence in support of neither difference nor no difference from zero / equal nor different group means.

3.4. Associations between stress response systems

Details of the regression results can be found in Table 4. The significant associations between the putative stress response stages are shown in Fig. 2. Scatterplots of the significant associations can be found in Figure S1 of the Supplementary Materials.

Interactions with sex were tested for each model. However, since there were no significant interaction effects, they were removed again and men and women were combined for parsimony.

The results showed that, against expectations, threat appraisal (S1) and affect and motivation (S3) were not associated with baseline states and traits, nor with Δ SET during any stage. Associations between stage two and stage four (HPA) could only be found for Δ SNS – where larger increases in SNS activity were associated with larger increases in HPA activity – but not for PNS. As hypothesized, higher trait neuroticism was associated with smaller increases in both Δ SNS and Δ HPA, although trait extraversion was not significantly associated with any Δ in stress systems. Furthermore, as expected, higher baseline levels were associated with decreased stage two and three stress reactivity overall (SNS, PNS, motivation, and affect).

Additionally, exploratory Pearson correlations between trait predictors (extraversion, neuroticism) and baseline state measures, and between different stress response measures showed that higher baseline HPA activity (cortisol) was significantly correlated with higher baseline SNS activity (i.e., lower PEP)³, while there were no significant correlations between stress reactivity indices. Similarly, no correlations within sexes were significant after FDR-correction. Details of the correlation results can be found in Tables S1 – S4 of the Supplementary Materials.

³ The variance inflation factor values were below 1.20, indicating that multicollinearity was not an issue.

4. Discussion

This study aimed to investigate associations between different stress response stages and the moderating influence of biological sex and personality traits on these associations. Results showed significant stress responses to SET in all stress systems of stages two to four in both sexes, including increases in SNS activity (i.e., decreases in PEP; S2), decreases in PNS (i.e., decreases in RSA; S2), increases in combined SNS/PNS cardiovascular indices such as HR and BP (S2), as well as decreases in self-reported state approach motivation (S3), increases in self-reported negative affect (i.e., state anxiety; S3), and, finally, increases in peripheral HPA activity (i.e., cortisol; S4). However, participants did not seem to appraise the public speaking task as either threatening or challenging (S1). Thus, the SET manipulation successfully induced changes in almost all stress response systems in both men and women: a prerequisite for our following statements.

While examining sex differences in baseline stress levels and stress reactivity, we found higher baseline BP in men than women. This is not a surprising finding, as young men seem to have higher resting blood pressure and risk of hypertension (e.g., Hart et al., 2012). Additionally, we found that women reported higher trait neuroticism than men, which is in line with several large-scale studies (e.g., Lynn and Martin, 1997) and a meta-analysis (Feingold, 1994). As expected, we found that men and women did not significantly differ in any stress reactivity indices. We postulate that this is due to the stringent control of menstrual cycle phase in the current study, and is consistent with previous studies that tested women in the luteal phase and found no sex differences in Δ HPA (e.g., Kajantie and Phillips, 2006; Kirschbaum et al., 1999). The present study suggests that this absence of sex differences extends to other stress systems, notably SNS and PNS, as well as self-report indices of state approach motivation and negative affect (state anxiety). Importantly, using Bayesian model comparison and sufficient sample sizes for sex subgroups, responses in all stress systems could for the first time be convincingly qualified as supporting the null hypotheses of no sex differences. Initially, however, in the case of Δ cortisol the evidence was

Table 4
Results of the theory-based regression models (rows represent outcomes, columns represent predictors).

Outcomes	Reactivity predictors				Baseline predictors				Trait predictors			
	Stage 1: Resources and demands appraisal	Stage 2: ΔRPEP	Stage 2: ΔRSA	Stage 2: Δstate approach motivation	Stage 2: Δstate anxiety	Baseline PEP	Baseline RSA	Baseline state approach motivation	Baseline state anxiety	Baseline Cortisol	Trait Extraversion	Trait Neuroticism
Stage 1: Resources and demands appraisal	-	-	-	-	-	b = -.03, SE = .20, p = .884, r = -.02, d = .04	b = 1.92, SE = 3.71, p = .607, r = .07, d = .14	b = .21, SE = .23, p = .362, r = .12, d = .24	b = -.31, SE = .21, p = .139, r = -.19, d = .39	b = -1.74, SE = 2.06, p = .403, r = -.11, d = .22	b = .07, SE = .37, p = .850, r = .02, d = .05	b = .23, SE = .39, p = .566, r = .08, d = .15
Stage 2: ΔPEP	b = -.05, SE = .312, p = .13, r = .26	-	-	-	b = -.27, SE = .08, p = .002*, r = -.42, d = .94	b = -1.75, SE = 1.55, p = .265, r = -.17, d = .34	b < .01, SE = .01, p = .140, r = .20, d = .42	b < .01, SE = .09, p = .981, r < .01	b = -.06, SE = .08, p = .485, r = -.10, d = .19	b = -.78, SE = .79, p = .330, r = -.13, d = .27	b = .35, SE = .15, p = .021, r = .32, d = .68	b = .56, SE = .15, p < .001*, r = .46, d = 1.03
Stage 2: ΔRSA	b < .01, SE < .01, p = .704, r = .05, d = .10	-	-	-	b = .01, SE < .01, p = .140, r = .20, d = .42	b = -.28, SE = .08, p < .001*, r = -.46, d = 1.04	b < .01, SE < .01, p = .140, r = .20, d = .42	b < .01, SE < .01, p = .613, r = -.07, d = .14	b = .03, SE = .04, p = .544, r = .08, d = .17	b < .01, SE = .01, p = .832, r = .03, d = .06	b = .01, SE = .01, p = .360, r = .12, d = .25	b = .01, SE = .01, p = .360, r = .12, d = .25
Stage 2: ΔState approach motivation	b = .10, SE = .10, p = .324, r = .12, d = .25	b = -.02, SE = .26, p = .928, r = -.01, d = .03	b = -1.27, SE = 4.77, p = .791, r = .04, d = .07	-	b = -.11, SE = .15, p = .494, r = -.09, d = .18	b = -4.81, SE = 2.75, p = .086, r = -.23, d = .46	b = -.54, SE = .17, p = .003*, r = -.38, d = .81	b = -.07, SE = .15, p = .634, r = -.06, d = .12	b = -.36, SE = 1.54, p = .380, r = -.12, d = .23	b = -1.36, SE = 1.54, p = .380, r = -.12, d = .23	b = -.12, SE = .27, p = .658, r = -.06, d = .12	b = .30, SE = .29, p = .304, r = .13, d = .27
Stage 2: ΔState anxiety	b = .13, SE = .10, p = .200, r = .16, d = .33	b = -.22, SE = .24, p = .370, r = -.12, d = .23	b = 9.21, SE = 4.54, p = .047, r = .25, d = .52	-	b = .30, SE = .14, p = .038, r = .27, d = .56	b = -.34, SE = 2.54, p = .893, r = -.02, d = .04	b = .10, SE = .16, p = .522, r = .08, d = .17	b = -.65, SE = .14, p < .001**, r = -.51, d = 1.18	b = 2.10, SE = 1.43, p = .147, r = .19, d = .39	b = .26, SE = .25, p = .305, r = -.13, d = .27	b = .29, SE = .27, p = .295, r = -.14, d = .28	b = .29, SE = .27, p = .295, r = -.14, d = .28
Stage 3: Δcortisol	b = -.01, SE = .02, p = .471, r = -.09, d = .19	b = -.13, SE = .04, p = .002*, r = -.40, d = .89	b = .58, SE = .79, p = .463, r = -.10, d = .20	b = .03, SE = .02, p = .163, r = .19, d = .38	b < .01, SE = .03, p = .891, r = -.02, d = .04	b = .47, SE = .47, p = .315, r = -.14, d = .28	b = -.04, SE = .03, p = .201, r = -.17, d = .34	b = .03, SE = .03, p = .222, r = -.17, d = .34	b = -.03, SE = .03, p = .222, r = -.17, d = .34	b = -.22, SE = .27, p = .428, r = -.11, d = .22	b = .09, SE = .05, p = .048, r = -.26, d = .53	b = .17, SE = .05, p < .001*, r = -.42, d = .92

Note. PEP = pre-ejection period, RSA = respiratory sinus arrhythmia. b = unstandardized regression coefficient, SE = standard error, r = partial Pearson correlation coefficient (correlation between dependent and independent variable, correcting for other independent variables in the model); d = Cohen's d, ** = significant at α = 0.01 after FDR correction; * = significant at α = 0.05 after FDR correction.

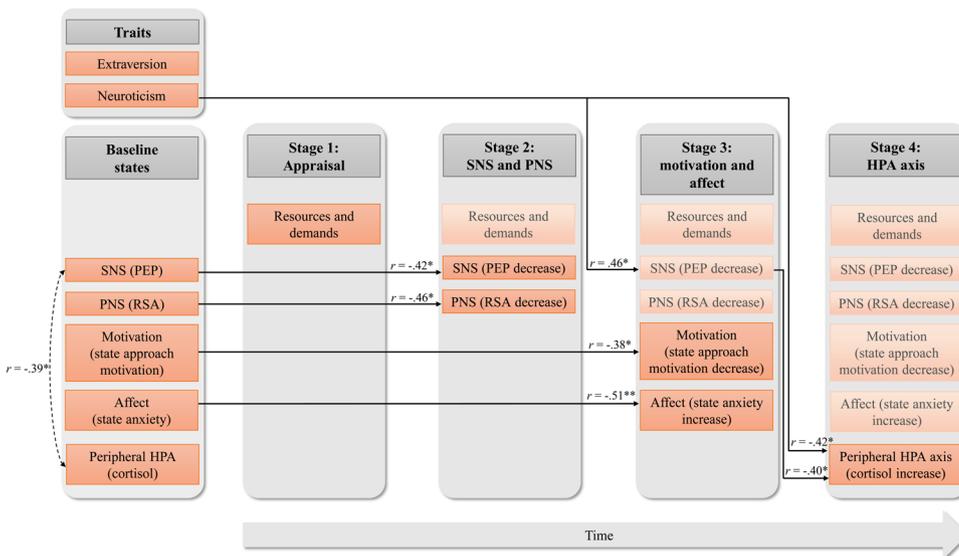


Fig. 2. Results of the linear regression models revealing associations between the different stages of stress response developing over time. Note. r 's (solid arrows) represent significant partial correlation coefficients, and r 's (dashed arrows) represent significant correlation coefficients. ** = significant at $\alpha = 0.01$ after FDR correction; * = significant at $\alpha = 0.05$ after FDR correction. HPA = hypothalamus-pituitary-adrenal axis, SNS = sympathetic nervous system, PEP = pre-ejection period, PNS = parasympathetic nervous system, RSA = respiratory sinus arrhythmia.

inconclusive, suggesting the possibility of small sex differences. We suspect this to be influenced by trait neuroticism, since women reported higher trait neuroticism and higher neuroticism levels predicted blunted Δ HPA. Indeed, when statistically controlling for trait neuroticism, there was evidence for no sex differences in Δ cortisol. Another possibility could have been that neuroticism differentially related to Δ cortisol in men and women. However, as is evident from the scatterplot S1b in the Supplementary Materials, men and women both demonstrate a similar negative slope of neuroticism \times Δ cortisol (for men, $r = -0.37$, for women, $r = -0.27$, nonsignificant difference: $Z = 0.43$, $p = 0.667$), but with a different intercept. Hence, blunted Δ HPA in women (e.g., Liu et al., 2017) may result from a combination of lower sex hormone levels (i.e., during the follicular phase) and higher neuroticism in women (e.g., Lynn and Martin, 1997).

Next, we explored associations between different stress response stages using a four-stage stress response model, based on the biopsychosocial (BPS) model of arousal regulation (Blascovich, 2013; Blascovich and Tomaka, 1996). First of all, we did not find interactions with sex for any stress reactivity index, which we suspect is due to the stringent control of menstrual cycle phase. Conforming to previous literature using PS, the TSST, and math tasks, we expected to find that increased threat appraisal (S1) would lead to smaller SNS increases and PNS decreases (S2), no differences in state approach motivation but larger state anxiety increases (S3), and larger cortisol increases (S4). However, results showed that appraisal (S1) did not predict any stress reactivity index. This might be due to task type, since previous PS studies found no associations between appraisal and combined Δ SNS/PNS (e.g., Feldman et al., 2004; Gramer, 2003) and no studies investigated associations with Δ HPA yet; although associations were found between threat appraisal and Δ negative affect (Gramer and Saria, 2007; Gramer and Sprintschnik, 2008). Another reason could be that we measured appraisal right before the speech, at which point some reappraisal might have already taken place (Quigley et al., 2002). In hindsight, a better alternative would have been to measure it earlier. Additionally, apart from the present results, there are some indications that Δ HPA might not be related to threat/challenge appraisals as such (i.e., the ratio between resources and demands), but only to demand appraisal (e.g., Gaab et al., 2005; Schlotz et al., 2011). This might also explain the conclusion of a meta-analysis that more extreme estimates of both challenge and threat appraisals were related to larger Δ HPA (Denson et al., 2009). Finally, it might be possible that appraisal is influenced by menstrual cycle, as no previous studies investigated this. To untangle these possible reasons, future studies should measure appraisal right after instructions and after preparation to assess

reappraisal, distinguish between resources and demands in their analyses, and compare women in their luteal phase with their follicular phase.

Subsequently, we expected to find that larger SNS increases and PNS decreases (S2) would be associated with larger anxiety increases (S3), and that larger Δ SNS (but not Δ PNS) would be associated with larger HPA increases (S4). Finally, we expected that larger anxiety increases (S3) would be associated with larger HPA increases. Indeed, results showed that increased SNS activity during SET predicted larger Δ HPA with a large effect size ($d = 0.91$), while Δ PNS did not predict Δ HPA, in line with previous studies using PS (Bosch et al., 2009; Evans et al., 2016). Further supporting an association between SNS and HPA activity, we found that baseline values of SNS and HPA activity were positively correlated. However, in contrast to our expectations, we found no associations with self-reported motivation or affect (S3). This might be due to the fact that most conflicting evidence from the literature on stress associations regards negative affect (while motivation is rarely researched), and the majority of studies have not found significant associations. Meta-analyses and reviews have reported positive associations with only small effect sizes for associations with combined Δ SNS/PNS during PS (between $d = 0.26$ and $d = 0.32$; Feldman et al., 1999) and no significant associations with Δ HPA after the TSST ($d = 0.23$; Campbell and Ehlert, 2012; Dickerson and Kemeny, 2004). Since the current study had statistical power to find associations with effect sizes of $d = 0.33$ or larger, associations with Δ negative affect might have remained undiscovered. Another reason might be that associations might mainly exist for specific facets of negative affect elicited by SET. For example, one meta-analysis only found associations between Δ HPA after PS for estimates of surprise, uncontrollable repetitive thoughts, brooding, submissiveness, and the fear of losing social approval (Denson et al., 2009), and a review highlights associations with shame after the TSST (Dickerson et al., 2004). In that case, state anxiety might be too general for examining the complexity of affective reactions to SET. Future studies should extend the current study by measuring associations with several salient facets of negative affect—such as shame and fear of losing social approval—using a well-powered sample.

Finally, we investigated the influence of important moderators of personality traits neuroticism and extraversion, and baseline stress levels. As hypothesized, higher trait neuroticism (though not extraversion) predicted smaller Δ SNS and Δ HPA. This is consistent with previous findings that higher trait neuroticism is associated with blunted Δ HPA in women (Oswald et al., 2006) as well as with blunted Δ HPA in both sexes (Bibbey et al., 2013), although some studies have found

larger SNS increases after PS with higher neuroticism (Evans et al., 2016). Unlike other studies suggesting that the relationship between baseline HPA and neuroticism is influenced by sex (DeSoto and Salinas, 2015), we found no evidence for sex differences. Importantly, the blunting influence of neuroticism on cardiovascular SNS responses might help explain the mechanism of how neuroticism decreases the effect of high blood pressure (i.e., angiotensin-converting enzyme) on long-term mortality risk (O'Suilleabháin and Hughes, 2018). Future studies could address long-term effects of neuroticism on mortality risk via physiological pathways, as well as affective, motivational, and behavioral pathways. Secondly, associations with baseline states showed that baseline levels of SNS activity, PNS activity, motivation, and negative affect predicted blunted stress reactivity during stage two and three, in line with previous studies (Kudielka et al., 2004b; Oswald et al., 2006). This pattern might be partly caused by ceiling and/or floor effects in the case of self-report indices, and by the law of initial values in the case of SNS and PNS through which high levels inhibit further increases (Wilder, 1962). However, it is slightly puzzling that we did not find this association for HPA levels, since the HPA has a *negative feedback loop* for the same purpose (Chrousos and Gold, 1992).

It should be noted that the distinction and ordering of the stress response stages are likely not as clear-cut as our operationalization of the BPS model suggests (Blascovich and Tomaka, 1996). For example, neuroscientific research has shown that all responses to psychological stressors are initiated by the same brain areas (e.g., amygdala and medial prefrontal cortex), leading to coordinated autonomic and neuroendocrine systemic adjustments (Buijs and Van Eden, 2000; Dampney, 2015). For example, the occurrence of changes in SNS/PNS activity before affective and motivational changes might as well occur the other way around or simultaneously. Timing differences in stress response stages as typically measured may in fact be due to inherent boundary conditions of their specific physiological (or psychological) pathways and mechanisms.

Furthermore, the current study has several limitations. Associations between stress response systems were tested using several multiple linear regression models. However, this might have better been tested using structural equation modelling, which would have required a much larger sample size. Considering our modest sample size and stringent use of multiple comparison correction, there might be a high number of false negatives, for example among the associations with negative affect. Additionally, we did not measure or correct for depression levels and subtypes (Bianchi and Laurent, 2016), although it is likely that any subthreshold psychopathology in this healthy sample would already be well captured by neuroticism. Moreover, since there are indications that other personality traits than extraversion and neuroticism, such as openness and agreeableness, also influence stress reactivity (Bibbey et al., 2013; Ó Súilleabháin et al., 2017), future studies should include these measures. Furthermore, our measures of cognitive appraisal, state anxiety, and state approach motivation were changed slightly from their original forms (Harrewijn et al., 2016; Rinck et al., 2013; Tomaka et al., 1993), which might have influenced their validity. Finally, we measured appraisal right before the speech, i.e., 13 min after the speech was announced, when some reappraisal might have already taken place (Quigley et al., 2002), which might have been too late. Finally, we only examined initial increases but not subsequent decreases or any adaptation that might occur over the course of multiple stressors during the experiment (possibly eliciting situation-specific adaptation and habituation/sensitization patterns), some of which might also be influenced by neuroticism (Hughes et al., 2011; Stemmler and Wacker, 2010). In light of these limitations, replications and extensions of our findings are needed.

5. Conclusions

Using a rigorously controlled experiment, theoretically-guided

analyses of a four-stage stress response model based on the BPS model (Blascovich, 2013; Blascovich and Tomaka, 1996) were performed, where earlier stages were used to predict later stages. Our results partly supported and partly contradicted our model. We found no significant associations with appraisal (S1) or affect and motivation (S3), but only that larger Δ SNS (S2) predicted larger Δ HPA (S4). As such, our results do not resemble the stress reactivity associations as in the BPS model, that our analysis plan was inspired by (Blascovich and Tomaka, 1996). Rather, our results indicate incoherent reactivity of the different stress response systems, with only the SNS and HPA being associated. As proposed in the stress coherence/compensation model (Andrews et al., 2013), it is possible that after an initial coherent stress increase in each system, the systems interact with and compensate one another to reach an optimal response. However, our results suggest that even this initial response of (most) stress systems does not show a significant linear association, although a pattern might become discernible when examining more complex (non-linear) relationships. Future research is needed to investigate this possibility further.

Secondly, this study expanded the literature by offering insight into the causes of blunted stress responses in women, where we provided first evidence of the absence of sex differences in stress reactivity during the luteal phase of the menstrual cycle (while controlling for the sex difference in neuroticism in the case of HPA reactivity) through the use of Bayesian model comparison. We postulate that previously found blunted HPA responses in women are caused by the combination of low sex hormone levels (i.e., during the follicular phase) and higher neuroticism in women.

Conflict of interest statement

The authors report no conflict of interest.

CRediT authorship contribution statement

Eefje S. Poppelaars: Conceptualization, Methodology, Software, Formal analysis, Investigation, Data curation, Writing - original draft, Writing - review & editing, Project administration, Funding acquisition. **Johannes Klackl:** Conceptualization, Methodology, Writing - review & editing, Project administration, Supervision, Funding acquisition. **Belinda Pletzer:** Methodology, Formal analysis, Writing - review & editing, Resources, Supervision, Funding acquisition. **Frank H. Wilhelm:** Methodology, Software, Writing - review & editing, Resources, Supervision, Funding acquisition. **Eva Jonas:** Writing - review & editing, Project administration, Supervision, Funding acquisition.

Acknowledgements

This work was financially supported by the Doctoral College "Imaging the Mind" of the Austrian Science Fund (FWF; W1233-G17; PhD student Eefje S. Poppelaars; supervising faculty members Eva Jonas, Frank Wilhelm, Johannes Klackl, and Belinda Pletzer) and by Salzburg University's promotion stipend (to Eefje S. Poppelaars). (All funding sources had no further involvement.)

We thank Tilmann Hüppauff, Maximilian Lüdke, Benjamin Mayer, Tim Schöttelndreier, and Magdalena Zacher for their assistance with data collection. We thank Aditi Arora, Andreas Böhm, Sara Fernández Rodríguez-Cabello, Adrian Lüders, Michael S. Prentice, Elizaveta Prentice, Elie E. Rassi, Frank van Schalkwijk, and Lavinia C. Uscatescu for being part of the pre-recorded video audience.

We thank the editor and five anonymous reviewers for their helpful comments to improve previous drafts of this paper.

Appendix A

Table A1

Table A1

Overview of several theoretical models about stress responses and their interactions.

Reference	Model name	Main idea
(Dienstbier, 1989)	The physiological mediation model	'Toughening' and 'aging' manipulations cause temperament and performance, mediated by physiological stress responses.
(Lazarus, 1991)	The cognitive-motivational-relational theory	Personality influences appraisal, which in turn causes action tendencies, affective responses, and physiological responses simultaneously, followed by different types of coping.
(Blascovich and Tomaka, 1996)	The biopsychosocial model of arousal regulation	Appraisal of motivated-performance situations influences physiological stress responses, which in turn influence affective responses. Biological/personal/cognitive variables moderate the associations/responses.
(Cacioppo et al., 1998)	The reactivity hypothesis	HPA axis stress reactivity is negatively associated with immune response reactivity, leading to higher disease risk.
(McEwen, 1998)	The allostatic load model	Stress response systems can become overstimulated or not perform normally in a number of situations, e.g., during chronic stress or when low reactivity in one system leads to high reactivity in another system.
(Boyce and Ellis, 2005)	The evolutionary–developmental theory of the origins and functions of stress reactivity	Stress reactivity is curvilinearly related to psychosocial stress and adversity during early development.
(Del Giudice et al., 2011)	The adaptive calibration model of stress responsivity	Stress reactivity depends on the stressfulness of the developmental context, with different contexts leading to one of four reactivity patterns, differing between men and women.
(Andrews et al., 2013)	The stress coherence/compensation model on the interaction of the stress systems	Stress response consist of two stages, coherence and compensation; where the reactivity of and associations between subjective stress, SNS, and HPA axis systems differ depending on the stage.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.psyneuen.2019.104378>.

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