



Sex differences in emotional and physiological responses to the Trier Social Stress Test[☆]

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Abstract

Women are more likely than men to be diagnosed with depression and anxiety-related disorders, and it has been hypothesized that this difference is related to sex differences in stress reactivity. Women typically report higher levels of negative affect than men in response to psychosocial stressors, but the evidence for sex differences in physiological reactivity to stressful situations is not consistent. The present study sought to expand this work by evaluating sex differences in reactivity to a social stress challenge across neuroendocrine, autonomic and affective response domains. Participants (32 women, 30 men) completed a standardized psychosocial stress challenge (i.e., the Trier Social Stress Test (TSST)), during which several physiological (e.g., cortisol reactivity, heart rate) and psychological (e.g., depression, irritability, anger, fear) measures were assessed. The findings demonstrated that cortisol reactivity and the magnitude of autonomic responding failed to reliably discriminate between women and men. However, women reported more fear, irritability, confusion and less happiness immediately following the TSST compared to men. The broader implications of these results and how they relate to sex differences in the etiology and clinical presentation of anxiety and mood disorders are discussed.

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1. Introduction

Women are diagnosed with anxiety- and mood-related pathology at higher rates than men, with many epidemiological studies indicating that the female-to-male ratio is approximately 2:1 for mood and anxiety disorders (Gater et al., 1998; Weissman et al., 1996). Differences in a variety of neurobiological (e.g., hormonal and genetic) (Seeman, 1997; Silberg et al., 1999) and psychosocial (e.g., social reinforcement contingencies, emotion regulation strategies) (Chambless & Mason, 1986; Craske, 2003; Thomsen, Mehlsou, Viidik, Sommerland, & Zachariae, 2005) processes have been proposed to contribute to this gender-based divergence.

Psychological stress has been clearly linked to the development of clinical depression and anxiety, with associations reported between increased stress responses, hypothalamic–pituitary–adrenal (HPA) axis hyperactivity and dysregulation and the occurrence of psychopathology (Feijó de Mello, Feijó de Mello, Carpenter, & Price, 2003). The results of human stress studies have largely indicated that men and women do not differ in their physiological responses to acute stress, showing, for instance, little difference in cortisol reactivity (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Kirschbaum, Wüst, & Hellhammer, 1992; Stoney, Davis, & Matthews, 1987). However, there is some evidence that younger men have elevated adrenocorticotropin (ACTH) stress responses compared to younger women (Kirschbaum et al., 1999; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004a). Similarly, there has been a lack of clear evidence for sex differences on measures of autonomic responding to acute stress, with several studies demonstrating no differences in physiological reactivity (Hedlund & Chambless, 1990; Katkin & Hoffman, 1976; Kelly, Forsyth, & Karekla, 2006; Sgoifo et al., 2003), although age-related sex differences in heart rate responses have been observed in response to laboratory social stressors (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004b).

Despite conflicting evidence on physiological reactivity to human stress challenge paradigms, studies have consistently demonstrated that women report more distress to fear-producing and stressful experiences than men (Kelly et al., 2006; Kudielka et al., 2004a). In addition, research has shown that women tend to report more depressive and anxiety-related symptoms (Fujita, Diener, & Sandvik, 1991; Thomsen et al., 2005; Turgeon, Marchand, & Dupuis, 1998), demonstrating a female tendency to experience negative emotions at a greater frequency and intensity than men (Barlow, 2001; Craske, 2003; Nolen-Hoeksema, Larson, & Grayson, 1999).

These links suggest the hypothesis that sex differences in stress reactivity may contribute to the female predominance of diagnoses of major depression and anxiety disorders. It is therefore valuable to extend this research using experimentally controlled procedures known to induce stress and anxiety (e.g., a social stress challenge), while concurrently assessing autonomic, neuroendocrine and affective responses. Moreover, as existing mood and anxiety disorders are known to influence stress reactivity, it is important to study individuals without current psychopathology in order to identify potential risk factors.

In light of these considerations, the aim of the present study was to evaluate sex differences in autonomic, neuroendocrine and self-report responses to a social stress task (i.e., Trier Social Stress Test (TSST)) in a healthy, nonclinical sample. We hypothesized that women would manifest greater subjective distress than men in response to the task.

In addition, due to conflicting evidence of sex differences in physiological reactivity to stress challenge studies, the present study evaluated potential sex differences in cortisol and autonomic responses to the TSST and whether these results would correspond to sex differences in evaluative responses to the stress challenge procedures.

2. Method

2.1. Participants

Study candidates (adults aged 18–65) were recruited from the community and invited to participate in one of the four thematically and methodologically similar studies on stress reactivity. Sixty-two participants (32 women, 30 men; $M = 28.03$ years of age, $SD = 11.31$) meeting inclusion criteria gave voluntary written informed consent on forms approved by the Butler Hospital Institutional Review Board. Participants were administered the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) (First, Spitzer, Gibbon, Williams, 2002) and a medical history questionnaire, and were excluded if they reported any of the following: (a) current Axis I psychiatric disorder, (b) significant current acute or chronic medical conditions, (c) participation in current psychological or psychopharmacological treatment or (d) current use of any medication which might influence HPA axis function, including psychotropics, beta blockers, angiotensin-converting enzyme inhibitors, ketoconazole, metyrapone, corticosteroids and oral contraceptives.

2.2. Materials and apparatus

2.2.1. Pre-experimental questionnaires

A battery of psychometrically sound measures was administered during the first session. This battery included the following measures: (a) the Inventory for Depressive Symptomatology, Self-report version (IDS-SR; Rush et al., 1986; Rush, Gullion, Basco, Jarrett, & Trivedi, 1996), a 30-item questionnaire designed to assess symptoms of depression; (b) the Childhood Trauma Questionnaire (CTQ; Bernstein & Fink, 1998), a 70-item self-report instrument that assesses history of childhood maltreatment; (c) the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983), a 14-item instrument that assesses the degree to which recent situations in one's life are stressful; and (d) the Spielberger State–Trait Anxiety Inventory Form-Y (STAI-S–T; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), which consists of two 20-item questionnaires used to assess state and trait levels of anxiety in clinical and nonclinical populations.

2.2.2. Experimental biochemical and physiological measures

Plasma cortisol concentrations were determined using the GammaCoat cortisol I-125 coated-tube radioimmunoassay (RIA) kit (INCSTAR Corp., Stillwater, MN). The intra- and inter-assay coefficients of variation observed for quality assessment samples (5 and 20 $\mu\text{g}/\text{dl}$) were less than 5% and 10%, respectively. Continuous heart rate recordings were obtained with a wireless PolarTM cardiac monitor and heart rate data were averaged across 10-min epochs during the TSST protocol.

2.2.3. Experimental self-report measures

Participants were asked to complete paper-and-pencil visual analogue rating scales 30 min prior to, immediately following and again 45 min after the TSST. These scales included the following emotions: (a) calm, (b) fear, (c) happiness and (d) irritability. All scales were anchored from 0 = *not at all* to 100 = *most ever*.

The Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1984) was used to assess participants' mood states at baseline (i.e., 30 min before the TSST), immediately following the TSST and once again 45 min after the TSST. The POMS consists of 72 items, and 6 subscales are derived measuring tension–anxiety, depression, anger–hostility, fatigue, confusion and vigor. Each item is scored on a 5-point Likert scale ranging from 0 = *not at all* to 4 = *extremely*.

2.3. Procedure

Participants were seen in two sessions, with the first visit involving the clinical assessment described above to determine eligibility. If eligibility requirements were met, participants were invited to a second session for the experimental portion of the study. All experimental sessions were scheduled to take place between 1200 and 1600 h to minimize confounds from diurnal variation in hormone levels. Physical exams were conducted in order to rule out acute or unstable medical illness. Fifty minutes before the stress challenge, a wireless cardiac monitor was placed on the subject's wrist and chest, and an indwelling IV catheter was inserted at the antecubital fossa. The TSST is a valid and reliable protocol used to induce moderate psychosocial stress, with associated HPA axis and autonomic nervous system arousal, in laboratory settings (Kirschbaum, Pirke, & Hellhammer, 1993; cf. Dickerson & Kemeny, 2004, for a review). The TSST protocol consisted of a 10-min preparatory period, a 5-min speech and a 5-min mental arithmetic task. Total plasma cortisol was measured at baseline (i.e., 15 min prior to the social stress challenge), immediately prior to the onset of the challenge, and at five time points after the TSST (15, 30, 45, 60 and 75 min after the onset of the challenge). Mean heart rate responses were assessed every 10 min beginning 20 min prior to the TSST and continuing until 20 min following the challenge. Participants completed the self-report measures at baseline, immediately following the TSST, and 45-min post-challenge.

2.4. Data reduction and statistical analyses

2.4.1. Plasma cortisol

Total plasma cortisol responses were analyzed with a repeated-measures analysis of covariance (ANCOVA). A repeated-measures time factor and a between-subjects sex factor were included in the analysis. Since cortisol responses may be affected by age, this factor was included as a covariate. When appropriate, the Greenhouse–Geisser degrees of freedom adjustment procedure was applied to the repeated-measures time factor to correct for violations of sphericity that often occur with repeated-measures analyses. In addition, net area under the cortisol curve over time (net AUC) was calculated using the trapezoidal method, and the change in cortisol concentration from baseline to peak (peak delta) was measured. Calculated measures of net AUC and peak delta were both analyzed using ANCOVAs (with the between-subjects sex factor and using age as a covariate).

2.4.2. Heart rate

A repeated-measures ANCOVA was used to analyze sex differences in mean heart rate during the TSST, with the repeated-measures time factor and the between-subjects sex factor included in the analysis. Due to previously observed sex differences in heart rate reactivity related to age, this factor was included as a covariate. Again, the Greenhouse–Geisser degrees of freedom adjustment procedure was applied to the repeated-measures time factor to correct for violations of sphericity. An ANCOVA was used to analyze heart rate summary variables (i.e., net AUC, peak delta), with age as a covariate.

2.4.3. Self-report measures

Pre-experimental psychological questionnaires (i.e., IDS-SR, CTQ, PSS, STAI) were analyzed using one-way ANOVAs. In order to control for baseline responses on experimental self-report measures (visual analogue scales, POMS subscales), change scores were calculated (i.e., subtracting baseline responses from values measured immediately following and 45 min after the TSST) and were analyzed separately using one-way ANOVAs. A Holm's modified Bonferroni approach was adopted to control for experimentwise error (Holm, 1979), as this correction is more powerful than more traditional approaches (i.e., Tukey test and the Bonferroni correction) when guarding against artificial inflation of Type I error rates (Jaccard, 1998).

3. Results

3.1. Pre-experimental questionnaires

Means and standard deviations for the pre-experimental questionnaires are presented in Table 1. We first evaluated whether women and men differed on several variables related to negative affect. Women reported significantly higher levels of depressive symptoms, $F(1, 56) = 8.85, p = 0.004$, and trait anxiety, $F(1, 60) = 8.11, p = 0.006$, than men. Women also reported significantly higher levels of perceived stress than men, $F(1, 60) = 4.55, p = 0.037$. No significant differences were observed as a function of sex for measures of state anxiety or childhood trauma. Overall, responses on measures of anxiety and negative affect were within normal limits for a nonclinical population (see Table 1).

Table 1
Means and standard deviations for pre-experimental measures

Variable	Men		Women	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
IDS-SR	6.52 ^a	3.97	10.45 ^a	5.91
CTQ	6.61	1.96	7.51	2.35
PSS	31.57 ^b	6.50	34.94 ^b	5.90
STAI-S	28.38	7.78	30.99	6.93
STAI-T	29.50 ^c	8.61	35.45 ^c	7.84

Note: $N = 62$ (32 women); IDS-SR = Inventory for Depressive Symptomatology, Self-report version, CTQ = Childhood Trauma Questionnaire (total score), PSS = Perceived Stress Scale, STAI-S = State Anxiety Inventory (Form Y), STAI-T = Trait Anxiety Inventory (Form Y). ANOVA sex effect p values: a, $p = 0.004$; b, $p = 0.037$; c, $p = 0.006$.

3.2. Biochemical and physiological measures

3.2.1. Plasma cortisol

Since cortisol responses may be affected by hormonal variations related to the menstrual cycle, a univariate ANOVA was conducted to check for differences in cortisol responses based on self-reported menstrual cycle phase. No significant differences in cortisol reactivity were observed for participants in different phases of the menstrual cycle. A repeated-measures ANCOVA of cortisol responses (controlling for age) demonstrated a significant main effect for time, $F(6, 46) = 4.08$, $p = 0.013$ (see Fig. 1). No significant sex differences were found in the repeated-measures ANCOVA, or in analyses of cortisol summary variables in response to the TSST (i.e., net AUC, peak delta).

3.2.2. Heart rate

Heart rate responses during the TSST challenge evaluated using a repeated-measures ANCOVA similarly revealed a significant main effect of time, $F(5, 23) = 4.42$, $p = 0.007$ (see Fig. 2). No other effects were significant. Summary measures of heart rate (i.e., net AUC, peak delta) did not vary significantly as a function of sex.

3.3. Self-report measures

3.3.1. Visual analogue scales

Table 2 shows means and standard deviations of the self-report measures (i.e., change scores for the visual analogue scales) assessed before and after the TSST as a function of sex. Immediately following the TSST, women reported greater increases in irritability and

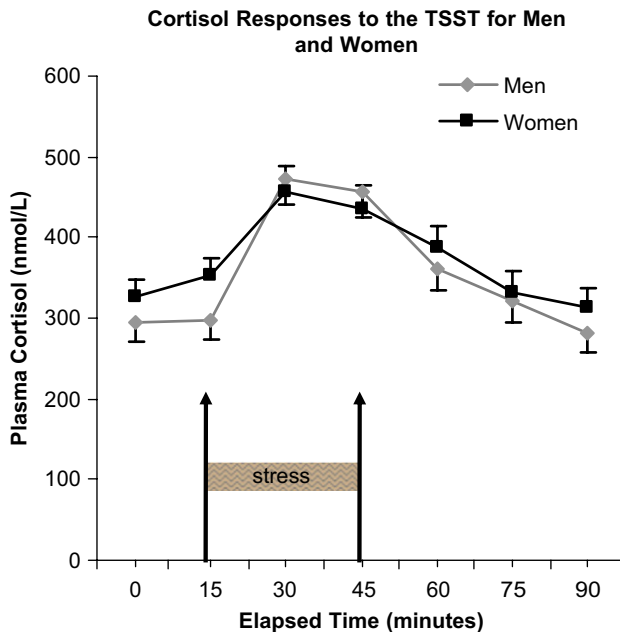


Fig. 1. Mean total plasma cortisol (nmol/l) responses in men and women before and after the TSST.

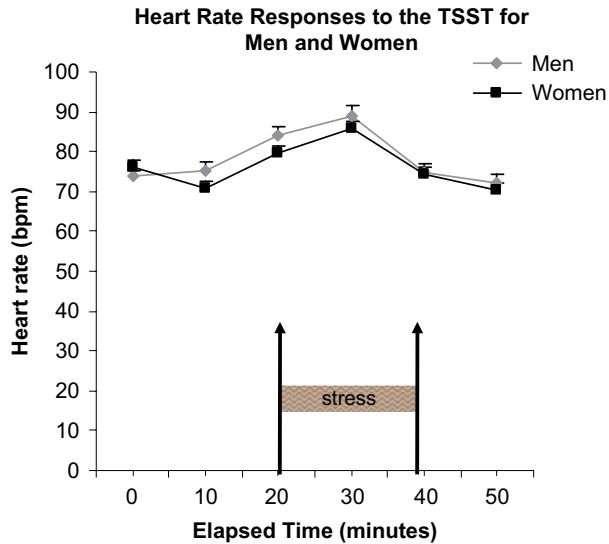


Fig. 2. Mean heart rate, recorded in beats per minute (bpm) across time for men and women before and after the TSST.

Table 2

Means and standard deviations of visual analogue scale change scores assessed immediately following and 45 min after the TSST

	Post-challenge				After 45 min			
	Men		Women		Men		Women	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Calm	-14.30	19.59	-9.31	18.55	-2.74	17.75	0.33	16.90
Fear	0.14 ^a	4.19	4.31 ^a	6.92	-0.96	3.35	-2.69	8.04
Happiness	0.74 ^b	7.17	-3.92 ^b	8.01	4.00	14.22	1.92	10.67
Irritability	3.33 ^c	6.45	8.96 ^c	12.81	-1.41	4.07	1.40	8.52

ANOVA sex effect *p* values: a, *p* = 0.010; b, *p* = 0.033; c, *p* = 0.047.

fear than men ($F(1, 51) = 4.13, p = 0.047$, and $F(1, 51) = 7.07, p = 0.010$). In addition, women had greater decreases in happiness following the TSST, $F(1, 49) = 4.80, p = 0.033$. No other significant effects were observed for the change in visual analogue scales immediately post-challenge. Change scores for the self-report ratings assessed 45 min following the TSST failed to discriminate between women and men.

3.3.2. POMS

Table 3 shows means and standard deviations for change scores derived from the POMS before and after the TSST. Immediately following the TSST, women had significantly greater increases in confusion than men, $F(1, 52) = 8.30, p = 0.006$. No other significant

Table 3

Means and standard deviations of POMS change scores assessed immediately following and 45-min after the TSST

	Post-challenge				After 45 min			
	Men		Women		Men		Women	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Tension–anxiety	2.48	4.63	2.85	4.53	−0.92	2.00	−2.46	3.51
Depression	−0.54	2.42	0.16	1.72	−0.36	0.76	−0.24	0.70
Anger–hostility	−1.14	2.22	0.71	3.76	−0.17	0.76	−0.23	0.76
Fatigue	−0.53	1.02	−0.72	3.47	−0.64	1.47	−0.03	3.78
Confusion	0.00 ^a	1.79	1.86 ^a	2.80	−0.74	2.02	−0.81	1.33
Vigor	−0.93	3.38	−1.19	4.21	−0.43	3.46	−1.89	3.14

Note: POMS = profile of mood states. ANOVA sex effect *p* value: a, *p* = 0.006.

effects were observed for the POMS subscales shortly after the TSST or 45 min following the TSST.

4. Discussion

The purpose of this study was to examine, in a nonclinical healthy sample, possible sex differences in response to a psychological stress challenge. Results showed that healthy women reported greater increases in negative affect, fear and confusion and decreases in happiness immediately following a social stress challenge. Sex differences in self-reported negative affect corresponded to pre-experimental differences in depressive symptomatology and trait anxiety between men and women, even though these baseline differences were not clinically significant. Moreover, as the analyses involved change scores, the differences in emotional reactivity to the stress test are not simply due to baseline differences in affective state. These differences are also not accounted for by sex differences in autonomic or cortisol reactivity during the challenge procedure, or by a history of adverse childhood experiences.

The lack of observed sex differences in HPA axis reactivity to laboratory stress and biological challenges is consistent with several previous studies (Kirschbaum et al., 1999, 1992; Stoney et al., 1987). It has been hypothesized that sex differences in cortisol reactivity to stress may be modulated by hormonal variation occurring during the menstrual cycle (Weiss, Longhurst, & Mazure, 1999) and it has been suggested that reproductive hormones, particularly androgens and estrogen, may potentiate psychiatric symptoms (Seeman, 1997; Yonkers, 1994), modify HPA stress reactivity and predispose individuals to the development of affective and anxiety disorders. In this study, we did not focus our investigation on differences in acute stress reactivity due to the phase of menstrual cycle, although in preliminary analyses, we did not find differences in HPA stress reactivity based on menstrual cycle phase. However, our results do not rule out a more pervasive influence of gonadal hormones on affect and cognition in response to stress (Rehman, & Masson, 2005; Sanders, Sjodin, & de Chastelaine, 2002; Steiner, Dunn, & Born, 2003). Moreover, sex differences in stress responses have been found with other measures of stress reactivity. For instance, studies of sex differences in catecholamine reactivity to stress have found

larger epinephrine responses to stressors in men compared to women (Lundberg, 2005). In addition, though no sex differences were found in the present study on the peripheral physiological measures of stress reactivity we examined, these results do not necessarily exclude the possibility of differences between men and women on more central mechanisms related to stress reactivity and depression.

The results of the current study draw attention to the debate about sex differences in response to specific stressors (e.g., achievement vs. social stressors) and the use of emotion regulation strategies (Stroud, Salovey, & Epel, 2002). Though the current stress challenge has been characterized as a “social stress” paradigm, it has elements that tend to be achievement- or instrumentally oriented (e.g., arithmetic tasks, public speaking tasks). Results of studies examining sex differences in response to similar achievement-oriented tasks have been either unsuccessful in demonstrating sex differences in physiological reactivity, or have demonstrated elevated reactivity in men rather than women (Kirschbaum et al., 1999, 1992; Kudielka et al., 2004a; Stroud et al., 2002). Thus, since achievement and instrumental stressors may not be, on the average, as salient for women as for men, such stressors may not be expected to elicit HPA axis hyperactivity in women. However, studies that use stressors that are associated with an increased risk of depression in women, namely interpersonal stressors (e.g., social rejection tasks and marital conflict), have successfully identified elevated cortisol reactivity, as well as increases in subjective distress and negative affect in women (Fehm-Wolfsdorf, Groth, Kaiser, & Hahlweg, 1999; Stroud et al., 2002). Thus, further research that employs the use of interpersonal stress challenges may be particularly useful in eliciting HPA axis hyperactivity and negative affect that is more closely associated with the pathogenesis of depression in women.

Sex differences in irritability and fear in response to a social stress challenge are consistent with previous studies that demonstrate that women are more likely to endorse negative affect than men in response to interpersonal stressors (Rudolph, 2002; Rudolph & Hammen, 1999; van Os & Jones, 1999). Previous research has demonstrated that women report more stressful life events than men, especially interpersonal stressors, and that women are more likely to become depressed in response to these stressors than men (McGonagle & Kessler, 1990). However, it does not appear that women differ from men in their actual exposure to acute stressors (Bebbington, 1996; Maciejewski, Prigerson, & Mazure, 2001). Thus, a female tendency to perceive events as more stressful and to be more sensitive to interpersonal stress than their male counterparts could constitute a vulnerability for the development of mood- and anxiety-related disorders. It is notable that women in our study reported significantly more perceived stress than men, as this supports the idea that women may be perceiving similar life events as more negative compared to men, and contributing to their tendency to report higher trait anxiety and depressive symptoms.

Sex differences in the use of emotion regulation strategies (e.g., suppression, reappraisal, rumination) during and following episodes of acute stress (Craske, 2003; Gross, 2002; Nolen-Hoeksema et al., 1999) may also explain sex differences in the prevalence of mood and anxiety pathology. Women are more likely than men to ruminate when they are experiencing negative affect (Butler & Nolen-Hoeksema, 1994; Thomsen et al., 2005), which may, in turn, lead to the feeling of less perceived control and, subsequently, more depressive and anxious symptomatology (Nolen-Hoeksema et al., 1999). Interestingly, women reported more confusion than men following the TSST, which may support the notion that women may be engaging in rumination and are more distracted by the

emotional nature of the social stress challenge, causing interference with their cognitive processing of the task.

Further research into the association between stress, anxiety and depression, especially studies of high-risk or clinical populations, would help elucidate causes for a female predisposition to report higher levels of negative affect in stressful situations. Of particular need are more prospective and longitudinal studies of sex differences in stress reactivity that examine the interplay between biological and psychosocial mechanisms in the development of depression and anxiety. For instance, differences in coping styles may provide a psychological basis for the observed sex differences (Nolen-Hoeksema et al., 1999). Additionally, other vulnerabilities (i.e., social roles genetic predispositions, hormonal factors, emotion regulation strategies) may play a substantial role in how women and men differentially respond to acute stressors. In addition, more research is necessary to elucidate the potential benefits of heightened emotional sensitivity and expressiveness in women, which may include more willingness to seek out emotional experiences, the facilitation of social roles and the ability to experience amplified positive emotions as well as negative emotions (see Grossman & Wood, 1993). Empirical studies focused on the factors involved in sex differences in the clinical presentation of affective and anxiety disorders would also provide direction in the creation of more efficacious interventions and prevention approaches that target processes involved in the differential development of anxiety and mood disorders in men and women.

In summary, the results of the present study demonstrate that women are more likely than men to report higher levels of negative affect and fear to social stress challenges. In contrast, they do not significantly differ in the extent of autonomic arousal and cortisol reactivity. The findings are consistent with most previous studies and further support the notion that, on average, women tend to perceive stressful life events as more stressful than men. These basic sex differences in the perception and response to stress could constitute a vulnerability to the subsequent development of depression and anxiety in women, especially in situations of chronic stress. The nature of the observed sex differences in the self-report of negative affect warrant further investigation as they may lie at the heart of the sex differences in occurrence of anxiety and depression and may provide an approach to understanding etiology and to improving treatment.

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