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Fearful imagery in social phobia: Generalization, comorbidity, and physiological reactivity

Lisa M. McTeague, Ph.D.[*], **Peter J. Lang, Ph.D.**[*], **Marie-Claude Laplante, Ph.D.**[*], **Bruce N. Cuthbert, Ph.D.**[+], **Cyd C. Strauss, Ph.D.**[*], and **Margaret M. Bradley, Ph.D.**[*]

[*]University of Florida

[+]University of Minnesota

Abstract

Background—Social phobia has been characterized as a disorder of exaggerated fear of social threat and heightened sensitivity to imagery of social failure.

Methods—To assess the physiological basis of this description, social phobia patients (n=75) and demographically-matched controls (n=75) imagined neutral and fearful events while acoustic startle probes were occasionally presented and eye-blink responses (orbicularis occuli) recorded. Changes in heart rate, skin conductance level, and facial expressivity were also indexed. In addition to comparing controls and social phobia patients, the influences of diagnostic subtype (circumscribed, generalized), comorbid depression, and chronicity were assessed.

Results—Patients exceeded controls in startle reflex and autonomic responding during imagery of social threat whereas the groups evinced commensurate reactivity to contents depicting commonly shared fears (survival threat). Individuals with circumscribed performance phobia were similar to controls, with the exception of more robust reactions to idiographic, performance fear imagery. In contrast, generalized phobic patients were characterized by longer disorder chronicity and demonstrated heightened sensitivity to a broader range of fear contents. Those with generalized phobia plus comorbid depression showed attenuation of fear-potentiated startle and reported the most protracted social anxiety.

Conclusions—Subtypes of social phobia can be objectively distinguished in patterns of physiological reactivity. Furthermore, subtypes vary systematically in chronicity and defensive engagement with the shortest disorder duration (circumscribed phobia) associated with the most robust and focal physiological reactivity, followed by broader defensive sensitivity in more chronic generalized phobia, and finally attenuation of the formerly exaggerated fear potentiation in the comorbidly depressed—the most chronic form.

Corresponding author: Peter J. Lang, PhD Center for the Study of Emotion & Attention University of Florida PO Box 112766 Gainesville, FL 32611 **email:** plang@phhp.ufl.edu Telephone: (352) 392-2439 Fax: (352) 392-6047.

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Keywords

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Introduction

Chronic, distressing imagery of social failure is a prominent symptom of social phobia (1) and a potent mechanism in the disorder's maintenance and exacerbation (2). Controlled exposure to clinically relevant fear via mental imagery is an effective component of non-pharmacological treatments of social phobia, to activate fear memories and ultimately extinguish their emotional impact (3). Here we elucidate the complex psychophysiology of emotional imagery in this disorder by comparing clinical fears with fears that are broadly shared, considering differences in fear processing between circumscribed and more generalized phobic patients, and examining the effects of comorbid depression.

Pitman and colleagues (4-7) employed fearful imagery in an extensive research program to confirm diagnostic status and delineate the pathophysiology of posttraumatic stress disorder (PTSD). Physiological reactions to emotional imagery have also been studied in specific phobia (8), borderline personality disorder (9), depression (10), and self-mutilation (11). The ecological validity of imagery as an assessment tool is corroborated by findings that elicited physiology is akin to the arousal evidenced upon anticipation of actual exposure to a feared stimulus (12), serving to mobilize the autonomic nervous system (e.g., heart rate, skin conductance); to activate expressive facial musculature (e.g., corrugator "frown" muscle); and to ready the somatic system for action (e.g., potentiation of the startle reflex) (13-14). Furthermore, animals confronting survival threat react with a similar reflex pattern, mediated by the brain's defense circuit (centered on the amygdala) (15-16). Neuroimaging studies strongly suggest that a comparable neural circuit (17-18) determines the physiology of human fear.

Physiological hyperarousal upon exposure to feared stimuli features prominently in the diagnostic criteria of social phobia (19). Investigations of reactivity during passive face perception (20-23), fear conditioning (24-25) and in vivo exposure (26-28) indicate that social phobia patients, consistent with their subjective reports of distress and the diagnostic nosology, show physiological hypersensitivity to a range of socially-relevant external challenges. Based on previous research (25,29) it was expected that patients with social phobia would demonstrate heightened defense circuit activation when confronting social threat (i.e., potentiating startle and enhancing skin conductance, heart rate, and facial muscle action [corrugator]) but show similar reactivity to controls during imagined fear contexts in which defensive mobilization is normal and adaptive (e.g., facing an attacking animal). All participants were expected to demonstrate the greatest reactivity to their personal "worst" fear scenes, although patients would nevertheless exceed controls, reflecting hypersensitivity when imagining situations that had prompted treatment seeking.

In subsequent analyses, patient subgroups were defined according to current diagnostic convention (19). Patients with focused fearfulness (i.e., circumscribed performance phobia) were expected to be most similar to controls with the exception of more robust reactions, specifically to their idiographic clinical contents (26-27,30) (i.e., imagery of performance). In contrast, patients with generalized phobia were expected to show broader defensive reactivity, marked by augmented responding to all fearful scenes (31). Finally, generalized patients were

subclassified by comorbid depression, with the expectation that defensive reactivity would be dampened in the depressed relative to non-depressed (32-33).

Subtype predictions reflected expectations concerning the duration of social fearfulness and hence the extent of sensitization of defensive systems (e.g., the startle circuit). Circumscribed phobia patients with specific, exaggerated reactivity may report shorter disorder chronicity than broadly reactive generalized patients (34). Comorbidly depressed generalized patients showing relatively impaired reactivity may endorse the lengthiest chronicity of all subtypes with depression onset typically following social phobia onset (35).

Method

Participants

Participants (81% Caucasian) were assessed at the University of Florida Fear and Anxiety Disorders Clinic: 75 treatment-seeking adults with principal diagnoses of social phobia, and 75, community controls matched for gender, age, race and education—all without psychosis or major physical disease.

Diagnostic Classification

Diagnostic groups were established using the Anxiety Disorder Interview Schedule for DSM-IV (ADIS-IV) (36). In cases of multiple Axis I disorders, diagnostic primacy was determined according to clinician-rated severity (ranging from 0, *No features present*, to 5, *Diagnosis present; severe*) reflecting both distress and interference. Controls denied receiving current or lifetime diagnoses of psychiatric illness and/or treatment and did not receive any clinician-severity ratings that indicated more than subsyndromal symptoms (i.e., severity rating=2). Inter-rater reliability (via videotape) was calculated for 25% of the social phobics, yielding agreement for principal diagnosis among three masters or doctoral-level clinicians at a kappa coefficient of 0.88.

For subtype assignment, circumscribed phobia was operationalized as disabling and disturbing anxiety about negative evaluation limited to specific performance contexts[1]. In contrast generalized phobia was defined as significant disturbance in at least two domains: formal performances, informal speaking and interaction, observation of behavior, assertive interaction (6,37)[2].

Of the 75 patients, 25 were classified as circumscribed. Twenty-seven generalized patients met criteria for a comorbid mood disorder (major depression, dysthymic disorder, or depressive disorder not otherwise specified). Only four with circumscribed phobia were diagnosed with an additional mood disorder, precluding meaningful statistical analysis.

Procedure

The University of Florida Gainesville Health Science Center Institutional Review Board (IRB-01) approved the study and participants provided informed consent at the outset of the

[1]For example, idiographic performance situations included taking examinations, singing in choir, athletic participation, performing in a band or orchestra, interviewing, delivering a televised report.

[2]Circumscribed phobia patients endorsed fear (ADIS-IV Fear Severity Rating, 4 and above) and/or avoidance (Avoidance Severity Rating, 4 and above) of at least one formal, structured performance situation (i.e., public speaking, participating in meetings and classes, or idiographic situations). In addition, these individuals exceeded the same threshold for distress and/or functional interference regarding apprehension/avoidance of performance situations, but did not similarly rate other social contexts. In contrast generalized phobics were defined as those meeting the same cutoffs in least two of the following domains: formal performances, informal speaking and interaction, observation of behavior, assertive interaction

assessment. Participants were then administered questionnaires (Table 1) and interview in the morning followed by psychophysiological assessment and clinical debriefing in the afternoon.

Experimental Stimuli—Twenty-four narrative imagery scripts were used (41). Analyses focus on the following scripts: two social threats (social confrontation, public speaking failure), four survival threats (physical attack by animal or human), and two neutral events. Additionally, two idiographic, "personal" fear scripts were constructed representing each patient's primary clinical fear[3]; controls were asked to describe their "worst fear" for these scenarios. Scenes were depicted in ~20 words that quickly revealed the affective tone and reflected participation in the ongoing scene. A female recorded the scenes using minimal prosody for presentation over earphones (Telephonics TDH-49).

Imagery Assessment—Participants were seated in a quiet, dimly lit room. Electrodes were placed and participants instructed to listen to the auditory scripts with eyes closed, and upon offset, vividly imagine the events described, as if actively involved. Throughout the recording session, soft tones cued the participants to relax, breathe slowly, and silently repeat the word "one" to stabilize physiological activity between trials (42). Imagery scripts were interspersed every 30 seconds in the tone series, with content pseudorandomized so that no more than 2 stimuli of the same hedonic valence (pleasant, neutral, unpleasant), or content category (e.g., social threat) were presented consecutively. The script series was repeated in a counterbalanced order.

Each imagery trial consisted of a 1-second baseline, followed by a 6-second auditory script, 12 seconds of imagery, and a tone-cued return to relaxation. Startle probes (50-ms 95 dB(A) white noise, instantaneous rise-time) were presented during imagery at 4-5.5 or 10–11.5 seconds post-onset, or both, and on 25% of the inter-trial intervals, at 21.5–22.8 seconds post-offset of imagery.

Following imagery assessment participants read the scenes and rated each for hedonic valence and emotional arousal (43).

Experimental Control and Data Collection

Stimulus presentation and data acquisition were controlled by a PC-compatible computer running VPM software (44). Using Coulbourn bioamplifiers, electromyograph (EMG) potentials from the left orbicularis oculi (amplified by 20,000; 90-250 Hz bandwidth; 125 ms time constant) and corrugator supercilii (amplified by 20,000; 100-1,000 Hz bandwidth; 500 ms time constant) regions were recorded. Skin conductance level (SCL) and electrocardiogram (ECG) were recorded as previously reported (31). Signals were digitized at 20 Hz throughout each trial with the exception that orbicularis oculi was sampled at 1,000 Hz from 50 ms prior to 250 ms after the startle probe.

Data Reduction and Analysis

Group differences in demographic and questionnaire data were assessed with univariate ANOVAs and Tukey Honestly Significant Difference (HSD) tests for planned comparisons.

EMG and SCL were reduced into half-second bins. The ECG r-r intervals were converted to beats-per-minute and reduced into half-second bins. Reactions were determined by subtracting

[3]For patients with circumscribed phobia, personal scenes described their most fearful performance situations, whereas for those diagnosed with generalized phobia, the scenes described their most fearful scenarios in these domains: formal performances, informal speaking and interaction, observation of behavior, assertive interaction.

amplitude in the 1 second prior to script presentation from response averages during the 12-second imagery period. SCL change was normalized (log[SCL+1]).

The startle blink from orbicularis oculi EMG was scored as the magnitude difference between onset and peak muscle potential (45). Blink magnitude was standardized within subject in relation to the mean and standard deviation of inter-trial probe responses (31).

Using SPSS Version 11.0. to determine diagnostic differences, omnibus repeated measures ANOVAs were performed separately for each physiological measure, with diagnostic status (control, patient) as the between-subjects factor and imagery content (neutral, social threat, survival threat, idiographic fear) as the within-subjects factor. Significant overall group effects were followed up with between-group tests by contents. Given Group × Content effects or prior hypotheses, within-group comparisons were used to delineate response patterns. Analyses were repeated for subtypes (i.e., circumscribed/generalized, presence or absence of comorbid depression). Wilks' lambda was used to address sphericity issues (46).

Results

Social Phobia and Control Groups

Baseline Physiology—No reliable differences emerged between patients and controls in blink magnitude for intertrial startle probes or for SCL or orbicularis EMG activity in the 1-second baseline prior to script onset. However, patients showed greater baseline corrugator EMG tension, $F(1, 148)=9.00$, $p<.001$, and more rapid heart rate (nearly 8 bpm faster than controls), $F(1, 148)=18.29$, $p<.001$[4].

Evaluative Judgments—Ratings of unpleasantness (Table 2) varied over imagery contents, $F(3, 145)=252.30$, $p<.001$, with both groups rating personal fear images most, and neutral scenes least unpleasant. A Content × Diagnosis interaction, $F(3, 145)=15.96$, $p<.001$, showed that patients found social threat scenes second-most unpleasant, and more aversive than controls, $p<.001$, while controls placed survival threat second, rating it more aversive than did patients, $p<.05$.

Ratings of emotional arousal paralleled the aforementioned unpleasantness judgments, Content $F(3, 145)=336.32$, $p<.001$; Category × Diagnosis $F(3, 145)=8.48$, $p<.001$. Controls rated personal fear scenes as most arousing followed by survival threat, social threat and neutral scenes. Patients differed in rating social threat as secondary to personal fear, and relative to controls, social threat was judged more arousing, $p<.001$.

Fearful Imagery: Potentiation of the Startle Reflex—Blink magnitude (Table 2, Figure 1) was larger during fearful contents compared to neutral, Content $F(3,134)=13.44$, $p<.001$, all fear-neutral comparisons, $p<.01$; and overall, patients were more reactive than controls, Diagnosis $F(1, 136)=7.55$, $p<.01$. Moreover, patients and control reactions differed over contents, Content × Diagnosis $F(3, 134)=4.42$, $p<.01$: Controls showed reflex potentiation during survival threat and personal fear relative to neutral imagery, $ps<.01$, but did not evidence potentiation during social threat. Patients showed reliable potentiation during all three fear contents, $ps<.05$, with significantly larger reflexes than controls for both social threat and personal fear scenes, $ps<.01$, with the latter response even augmented above survival threat, *post hoc*, $p<.05$.

[4]Analyses for heart rate change were calculated on residuals secondary to removing baseline effects via linear regression. For corrugator results on residuals and change scores were equivalent and thus, change scores were reported here.

Fearful Imagery: Autonomic and Facial Responses—As illustrated in Figure 2, greater skin conductance reactivity was found for patients than controls, Diagnosis $F(1, 144) = 8.55$, $p<.01$, carried primarily by the patients' larger response during social threat, $p<.01$, and personal fear imagery, $p<.05$. The pattern of response over contents was similar for the two groups, Content $F(3,142)=21.89$, $p<.001$; Content × Diagnosis $F(3, 142)=2.36$, *ns*, except for the predicted stronger reaction to social threat, relative to neutral, for the patients, $p<.001$, with no such difference for controls.

Heart rate change was also modulated by imagery scene, Content $F(3,144)=16.39$, $p<.001$, varying between diagnostic groups, Content × Diagnosis $F(3, 144)=6.00$, $p<.01$. Thus, controls showed heart rate increase only to personal fear, differing from their deceleration to neutral content, $p<.001$; patients showed acceleration to both personal fear and social threat, differing from neutral content, *ps*<.001. In between-group tests, patients showed greater acceleration to social threat, $p<.001$, and more deceleration to neutral content, $p<.01$, than controls.

Corrugator tension was largest for personal fear and least for neutral imagery in both diagnostic groups, Content × Diagnosis $F(3, 146)=2.02$, *ns*, and all fear contents prompted more activity than neutral, Content $F(3,146)=9.94$, $p<.001$, subtest *ps*<.01. There was, however, a group difference in overall muscle contraction magnitude, Diagnosis $F(1,148)=4.26$, $p<.05$, due to significantly greater tension during survival threat imagery in controls, $p<.05$.

Social Phobia Subtypes

Circumscribed & Generalized Symptomatology—When circumscribed and generalized subgroups were distinguished, blink magnitude, Group $F(2, 135)=4.40$, $p<.05$, as well as affective modulation varied between-groups, Category × Group $F(6, 266)=3.65$, $p<.01$. The circumscribed subgroup and controls showed a similar pattern over categories except that whereas controls showed the largest response to survival threat, the circumscribed subgroup responded most to personal fear—exceeding their responses to all other neutral and fearful imagery, *ps*<.05. Conversely, patients with generalized phobia showed broader defensive reactivity with equivalent fear potentiation across all fear contents, exceeding controls in response magnitude during social, $p<.01$, and neutral imagery, $p<.05$. Interestingly, an overall group difference was observed for personal fear reactivity, Group $F(2, 135)=4.09$, $p<.05$, owing to the pronounced responding of the circumscribed subgroup compared to controls, $p=.05$. Furthermore, a posthoc analysis of personal fear versus neutral processing, Group $F(2, 135)=3.49$, $p<.05$, revealed that patients with circumscribed phobia reacted more robustly to idiographic fear scenes than controls, $p<.05$, with the same trend for the generalized subgroup, $p=.06$.

Skin conductance change varied with imagery content, Category $F(3,141)=22.90$, $p<.001$, consistently across groups, Category × Group $F(6, 282)=1.46$, *ns*. However, group differences emerged for social threat scenes, Group $F(2, 145)=4.31$, $p<.01$: the generalized subgroup exceeded controls, $p<.05$, and the circumscribed subgroup showed a similar trend, $p=.06$. Only circumscribed patients showed larger sympathetic increases than controls to personal fear imagery, $p<.05$, *one-tailed*.

Baseline heart rate increased linearly with symptomatology, Group $F(2,147)=10.60$, $p<.001$, i.e., from controls to circumscribed to the generalized subgroup, linear trend, $p<.001$. However, heart rate changes during imagery varied similarly over contents in both social anxiety subgroups, consistent with findings for the entire patient sample. Thus, both groups showed greater deceleration than controls for neutral imagery, *ps*<.05, and greater acceleration than controls for social threat imagery, *ps*<.01. The circumscribed subgroup showed the greatest heart rate increase during personal fear imagery, exceeding all other contents including social threat scenes, which evoked the second largest response.

Corrugator change was similar across patient and control groups. Based on the preceding results for controls and all patients, a posthoc analysis was performed on reactivity during survival threat imagery, Group $F(2,147)=3.40$, $p<.05$. Pairwise comparisons revealed that relative to controls, the generalized subgroup showed a blunted response to survival threat imagery, $p<.05$, whereas the circumscribed patients were similar to controls.

Subjective pleasure and arousal did not vary between patient subgroups.

Age of onset for circumscribed (M=23.6, SD=9.3) was later than generalized patients (M=17, SD=11.6), Group $F(1, 73)=5.31$, $p<.01$. Chronicity of social phobia disorder was thus longer in generalized (M =13.3, SD=12) relative to circumscribed patients (M=8.5, SD=14.2; Group $F(1, 73)=6.16$, $p<.05$).

Influence of Comorbid Depression—Comorbid depression in the generalized subgroup modulated startle magnitude, Group $F(2, 110)=6.62$, $p<.01$, and affective modulation patterns, Category × Diagnosis $F(6, 216)=2.18$, $p<.05$. The heightened sensitivity to social threat demonstrated by these patients was attributable to the non-depressed subgroup, which showed exaggerated reactivity relative to neutral for both social threat, $p<.05$, *one-tailed*, and personal fear scenes, $p<.05$. Conversely, depressed patients showed reliable augmentation only to personal fear contents. Furthermore, between-group differences emerged for all contents except survival threat, with the non-depressed exceeding the control, $p<.001$, and depressed groups. $p<.05$, *one-tailed*, during social threat processing[5].

For skin conductance, heart rate, and facial action, overall response amplitude and content modulation showed little difference between patients with or without comorbid depression.

Comorbid depression typically developed subsequent to social phobia, mean age of onset difference=9.6 years (SD=13.2), $t(24)=3.63$, $p<.001$.

Confluence of Fear, Anxiety, & Depression—Given the reliability of startle potentiation to clinically-relevant contents and resting heart rate in distinguishing across groups, follow-up analyses were conducted to explore the simultaneous effects of all subtypes. A repeated-measures ANOVA was calculated using all groups entered as a between-subjects factor (controls, circumscribed, generalized non-depressed, generalized depressed) and social threat category (standard, idiographic) as a within-subjects factor. Defensive reactivity clearly varied across groups (Figure 3), Group $F(3, 134)=4.42$, $p<.01$, Quadratic trend, $p<.05$, with broad and exaggerated defensive reactivity evinced by generalized non-depressed patients, $p<.001$ compared to controls.

Reliable covariation by subgroups emerged in baseline heart rate (Figure 4), Group $F(3, 146)=7.83$, $p<.001$, Linear trend, $p<.001$; controls had the lowest, followed by circumscribed, generalized, and then generalized depressed phobia. The same pattern was observed in rated distress as indexed in frequency of comorbid anxiety disorders, questionnaire measures of fear, trait anxiety, and depression (Table 2), and clinician-rated social phobia severity. Furthermore, as resting heart rate and symptom severity increased among patients, clinician-rated prognosis worsened, age of onset decreased, and chronicity increased.

Medication and Drug Use

Twenty-seven of the 75 patients indicated current use of prescribed medications for alleviating mental health symptoms. Most frequently, these medications were selective serotonin-reuptake

[5]The same pattern of results was found when comorbid depression was examined across the entire sample of patients, including the four depressed circumscribed phobics in the depression group.

inhibitors (SSRIs; 15%) and/or benzodiazepines (21%). The effects of these and less frequently endorsed compounds (e.g., norepinephrine-dopamine reuptake inhibitors, 4%; beta blockers, 4%) were assessed by comparing resting and imagery reactivity among the medicated and non-medicated patients both for patients as a whole and within subtypes. Considering either general psychotropic usage or more specific classes of drugs, no reliable effects emerged, perhaps due to the relatively small proportion of the sample on a heterogeneous array of medications[6]. However these null findings are consistent with prior psychophysiological studies of depression (47-50).

Reported usage of both prescription and over-the-counter medications for promoting physical health, as well as recreational substance use were also collected but low frequencies of endorsement precluded statistical analysis[7].

Discussion

Patients diagnosed with social phobia as a whole exceeded controls in defensive reactivity when imagining either idiographic fear or standard social threat scenes, but did not differ when imagining contents presumed to be fearful for all participants. This pattern emerged in startle potentiation and in autonomic responding implicating a concordant, general hypersensitivity of the defense system to clinically relevant imagery. This psychophysiological pattern is consistent with activation of subcortical fear structures as observed by Stein and colleagues (22) who found that greater medial temporal (amygdala) activation in social phobia patients than controls, specific to angry faces.

This specificity for clinically pertinent stimuli did not extend to facial expressivity. Patients showed modulation of corrugator tension similar to controls—greater for fear than neutral contents. The only difference that emerged was a smaller increase to survival threat imagery among those with generalized social phobia compared to controls, whereas activity to all other contents was equivalent across groups. This overall normal response was unexpected, as studies have shown that the socially fearful respond with exaggerated corrugator action to angry faces (23,52-53). Of course the present findings reflect imagined scenarios, not reactions mirroring direct perception of an angry face, and unlike the startle reflex, facial expressions can be modulated through top-down control. Thus, a stoic impassive response could be, for example, a protective reaction in situations of social threat, disguising an intense, internal experience of interpersonal failure.

Social Phobia Subtypes

Patients with circumscribed phobia responded similarly to controls with the exception of a stronger reaction to personal fear scenarios. Unlike controls, their startle potentiation and

[6]Medication usage did not reliably differ among patient subtypes. The low rates of usage largely precluded statistical analysis but in the interest of completeness sample proportions and group comparisons for the most frequently endorsed psychotropics by medication and social phobia subtype follow. SSRIs: circumscribed (20%; n=5), generalized non-depressed (9%; n=2), generalized depressed (30%; n=8), Group, X^2 (2)=3.40, *ns*; Norepinephrine-reuptake inhibitors: circumscribed (4%; n=1), generalized non-depressed (4.3%; n=1), generalized depressed (3.7%; n=1), Group, X^2 (2)=0.01, *ns*; Benzodiazepines as needed: circumscribed (20%; n=5), generalized non-depressed (4.3%; n=1), generalized depressed (26%; n=7), Group, X^2 (2)=4.22, *ns*; Regularly-scheduled, daily benzodiazepines: circumscribed (4%; n=1), generalized non-depressed (4.3%; n=1), generalized depressed (3.7%; n=1), Group, X^2 (2)=0.01, *ns*; Beta-blocker as needed for anxiety: circumscribed (4%; n=1), generalized non-depressed (0%; n=0), generalized depressed (0%; n=0), Group, X^2 (2)=2.03, *ns*; Prescription sleep aids: control (1.3%; n=1), circumscribed (4%; n=1), generalized non-depressed phobics (0%; n=0), generalized depressed (7.4%; n=2), Group, X^2 (2)=3.70, *ns*.

[7]As previously demonstrated (51) social phobics were more likely (16%) than controls (5%) to be current cigarette smokers, X^2 (1)=4.59, $p<.05$, but no differences were revealed between patient subtypes. Further, no physiological effects were observed comparing resting and imagery reactivity of smokers and non-smokers. For example, in predicting resting heart rate smoking status neither interacted with group membership (controls and patient subtypes), $F(3, 142)=1.18$, *ns*, or exerted a main effect, $F(1, 142)=0.26$, *ns*. All participants denied current cigar or pipe smoking or use of smokeless tobacco.

autonomic response exceeded reactions to all other scenes, including standard social threat. Essentially, defensive hyper-reactivity in these patients was limited to their performance fear.

In contrast, patients with generalized phobia showed strong startle potentiation (compared to controls) for all social threat contents—both idiographic and standard. There was also a substantial response to survival scenarios, and even a significant blink response to neutral scenes (compared to controls or their own inter-trial response), implying a "generalization" of hyper-excitability in startle circuits to non-threatening as well as threatening cues. In light of the more extended disorder chronicity, however, a longitudinal examination is warranted to examine whether hypersensitivity to survival threat and even innocuous, non-social imagining is present at the outset of generalized social phobia or results from enduring dysfunction.

These subtype findings differ notably from the mixed results of prior in vivo exposure studies of social phobia. Some researchers have found increased heart rate in circumscribed, compared to generalized phobia during speech or performance challenge (26-27,30,54) while many have found no differences (55-58). Additionally, interpersonal interaction exposure has not discriminated those with social phobia from controls either as a whole or accounting for subtypes (57-59). These inconsistent findings may be attributable to the fact that all humans must mobilize physiologically to be successful in social exchanges, particularly amidst strangers, perhaps obscuring any fear-specific physiology. Assessment of social imagery may be a more sensitive index of the persistent, pathognomic thoughts and images of social failure that characterize the disorder.

Comorbid Depression

Considering comorbid depression revealed the defensive hyper-reactivity and reliable affective modulation of the generalized groups was due to the subgroup without depression. Non-depressed patients demonstrated fear potentiation during social threat imagery that was heightened compared to controls or depressed patients. Furthermore, participants with generalized phobia plus depression showed dampened affective modulation with augmentation of probe responses only for the most arousing scenes. Kaviani and colleagues (32) reported a similar result for major depressive disorder patients during picture viewing. High levels of anhedonia and depression were associated with reduced affective startle modulation, whereas anxiety, independent of mood symptoms, yielded augmented fear potentiation. Further underscoring the generalizability of this effect, Melzig et al. (33) found that startle potentiation during threat of shock was stronger in non-depressed panic patients than in controls, whereas panic patients with comorbid depression showed no reliable threat-induced potentiation.

Importantly, the attenuated response magnitude and weak affective modulation seen here by depressed patients was uniquely indexed by the startle reflex. Regardless of depression status, social phobia patients showed exaggerated autonomic increases and subjective distress during social threat imagery. Thus, it is unlikely that the reduced startle response was due to inattention or avoidance of the emotional task. Given that the startle effectors are somatic, reduced potentiation may reflect the psychomotor retardation and behavioral inhibition associated with depression, while autonomic outputs from the defense system are intact or even augmented. Similar response system discordance was observed by Melzig et al. (33) in depressed, panic disorder patients under threat of shock.

In our sample with generalized symptomatology, depression typically developed secondary to social phobia, implicating longstanding interpersonal apprehension and functional interference in the development of comorbid depression and attenuated startle responsivity. That chronic anxiety and depression may be related to somatic hypo-reactivity has been increasingly supported by animal research varying stressor duration (60). For example, rats exposed to brief (10 days) and/or less threatening stress demonstrate hypervigilance and hyperarousal, whereas

rats exposed to longer duration stress (20-30 days) develop more generalized anxiety and depressive-like symptoms, including passivity and reduced movement and communication behaviors.

Baseline heart rate

Resting heart rate increased from control to circumscribed to generalized without depression, and finally the generalized phobia with comorbid depression subgroup—the same pattern observed in measures of social phobia chronicity, as well as dimensional symptom reports of fearfulness, anxiety and depression. Given the equivalent resting skin conductance levels across groups, these heart rate findings suggest an attenuated parasympathetic tone in social phobia that is exacerbated with chronic, broadly based negative affect. Systematic investigations of heart rate variability in depression (61-62) have similarly revealed higher heart rates and diminished parasympathetic influence.

Importantly, neither psychotropic nor recreational drug use exerted a discernible impact on baseline or physiological reactivity patterns (47-50).

Summary

Individuals with social phobia demonstrated defensive physiological hyper-reactivity to internally generated images depicting scenarios of interpersonal failure and performance anxiety. In light of the tendency to mentally perseverate on perceived social failures among the socially anxious (63), these results implicate accompanying physiological hyperarousal as a contributor to the exacerbation and maintenance of social anxiety. In addition, although accumulating evidence based on subjective symptom reports has suggested limited utility in maintaining diagnostic specifiers for social phobia in the upcoming revision of the diagnostic canon (64-65), the present findings undergird conventional diagnostic practice—demonstrating that subtypes can be objectively distinguished in patterns of physiological reactivity. Furthermore, these findings suggest that social phobia subtypes systematically vary in fear specificity, chronicity and defensive propensities with the shortest disorder duration (circumscribed phobia) associated with the most robust and focal defensive enhancement, followed by broader defensive sensitivity in the more chronic generalized phobia, and finally comorbid depression and attenuation of the formerly exaggerated fear potentiation in the most enduring form.

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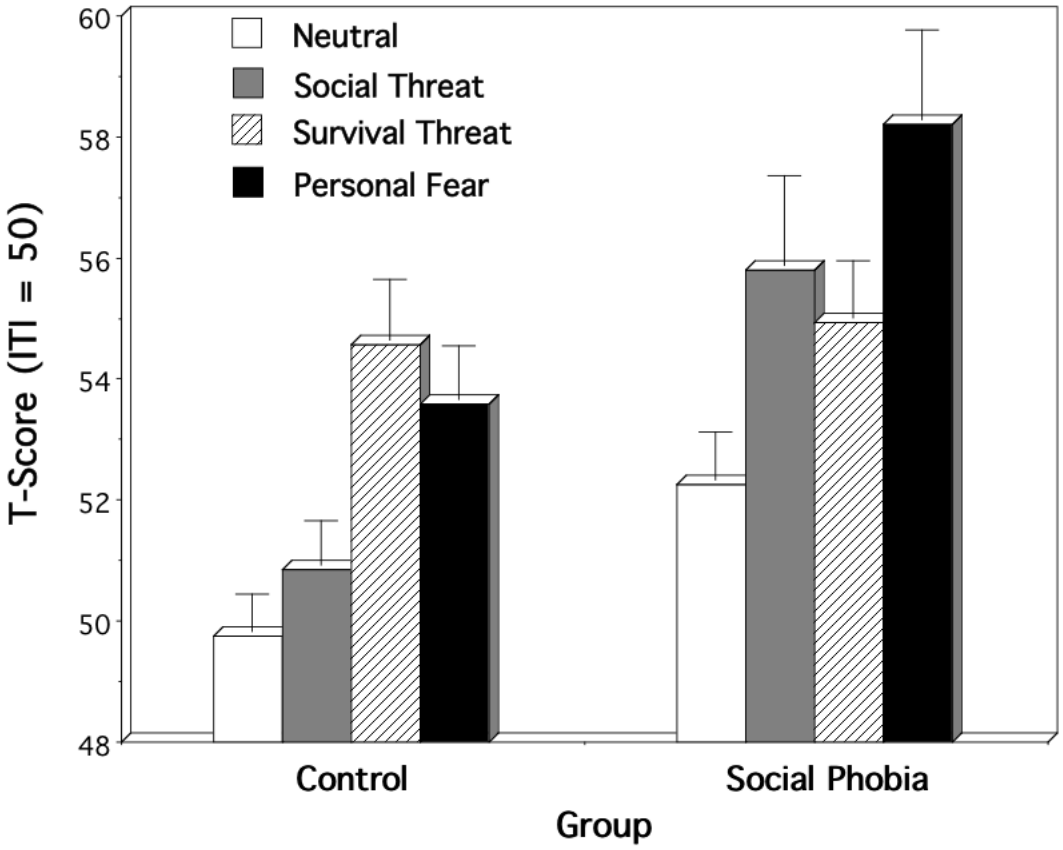

Figure 1.
Mean startle reflex responses during neutral, social threat, survival threat, and personal fear imagery for control and social phobia groups (standardized to the distribution of responses during intertrial intervals). Error bars refer to standard error of the mean.

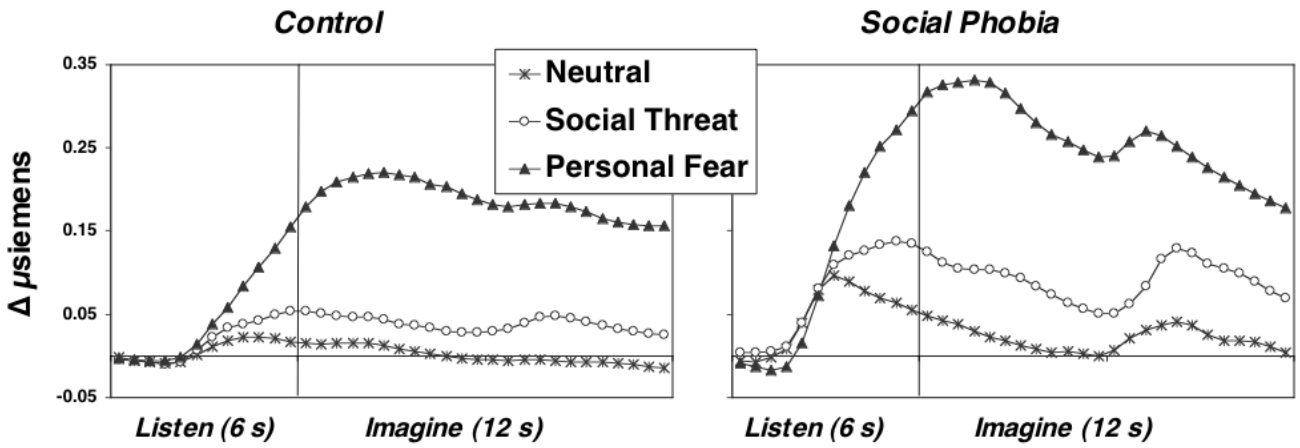

Figure 2.
Skin conductance level change in half-second averages during neutral, social threat, and personal fear script perception and imagery for control (left panel) and social phobia (right panel) groups. Comparing the log transformed means for each epoch revealed that phobic patients were reliably more reactive than controls to all contents during listen, *ps*<.05, and to social threat, *p*<.01, and personal fear, *p*<.05, during imagery.

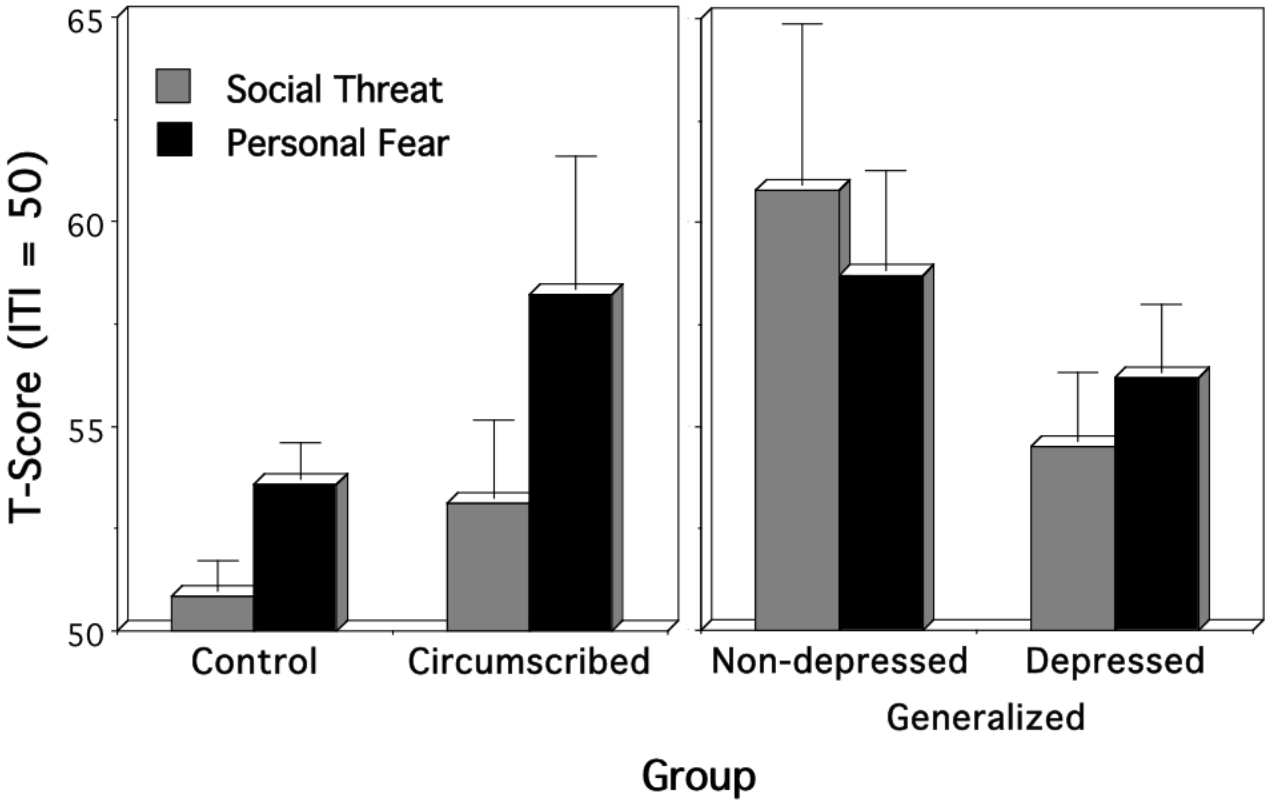

Figure 3.
Mean startle reflex responses during social threat and personal fear imagery for control, circumscribed social phobia, generalized social phobia without comorbid depression, and generalized social phobia with comorbid depression groups (standardized to the distribution of responses during intertrial intervals). Error bars refer to standard error of the mean.

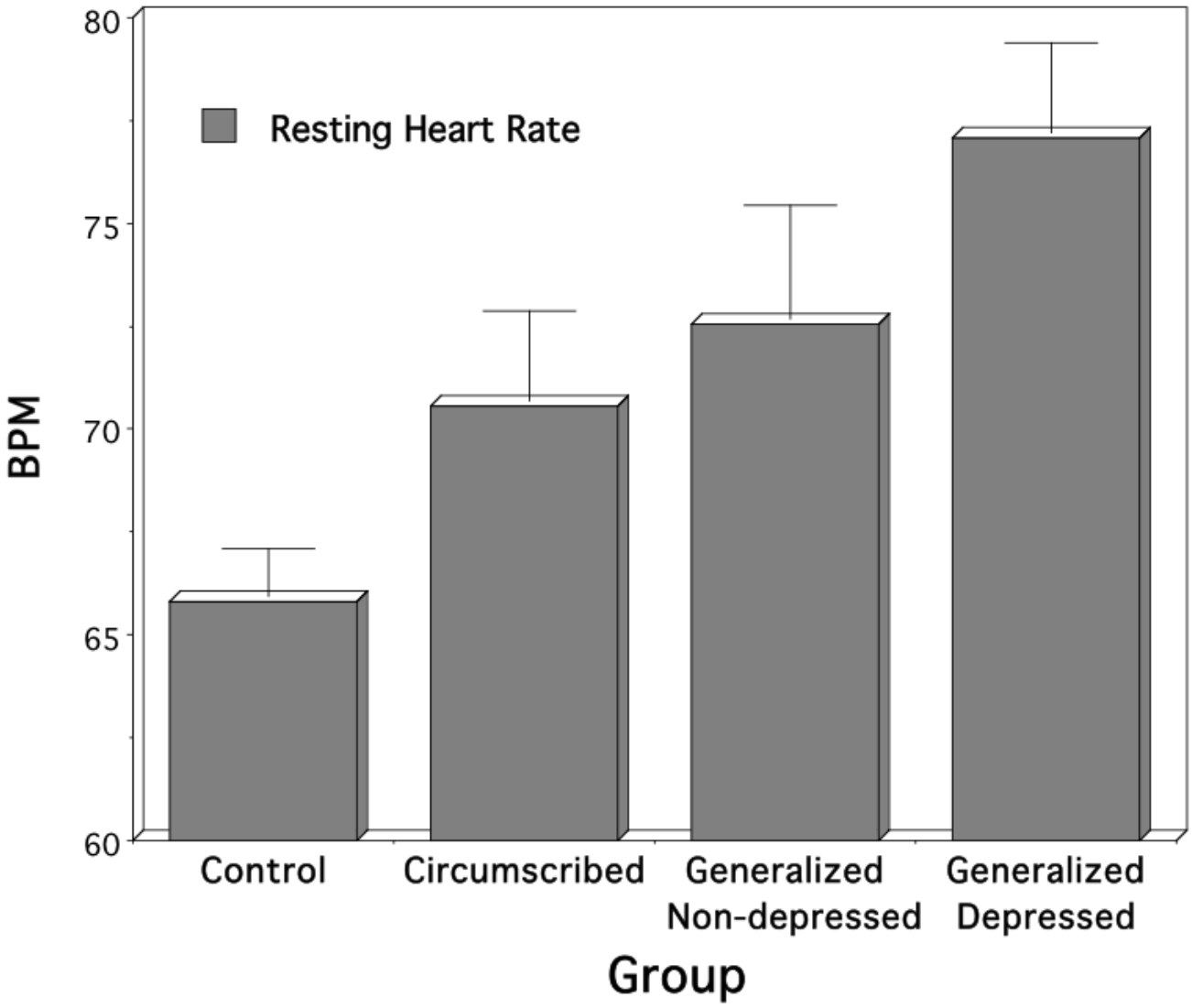

Figure 4.
Mean heart rate in the 6-second resting interval prior to script onset for control, circumscribed social phobia, generalized social phobia without comorbid depression, and generalized social phobia with comorbid depression groups. Error bars refer to standard error of the mean.

Table 1

Demographic, interview, and questionnaire responses (means and standard deviations) for Controls and Social Phobia Subtypes

Measure	Controls	Circumscribed	Generalized Non-depressed	Generalized Depressed	Group Effect
Questionnaire Measures					
FSS Social	40.29 (12.89)[b c d]	56.4 (15.73)[a c d]	74.41 (16.22)[a b d]	87.67 (13.29)[a b c]	$F(3, 147) = 91.21$, $p < .001$
FSS Total	156.47 (37.42)[c d]	173.80 (36.90)[c d]	218.24 (61.07)[a b d]	262.39 (55.75)[a b c]	$F(3, 147) = 40.92$, $p < .001$
STAI-Trait	30.80 (8.57)[b c d]	45.28 (10.80)[a c d]	53.35 (6.13)[a b]	59.23 (9.36)[a b]	$F(3, 145) = 87.37$, $p < .001$
BDI Total	3.51 (4.55)[b c d]	10.20 (7.27)[a d]	11.61 (5.72)[a d]	21.15 (9.71)[a b c]	$F(3, 147) = 52.89$, $p < .001$
Interview Measures					
Social phobia severity (1-5)		3.48 (0.59)[c d]	4.00 (0.67)[b]	4.22 (0.70)[b]	$F(2, 72) = 8.67$, $p < .001$
Age of onset (Years)		23.64 (9.29)[d]	18.61 (13.26)	15.70 (9.71)[b]	$F(2, 72) = 3.53$, $p < .05$
Chronicity (Years)		6.71 (11.22)[d]	11.52 (11.15)	14.74 (12.46)[b]	$F(2, 72) = 3.13$, $p = .05$
Prognosis (1-5)		1.80 (0.58)[d]	2.13 (0.48)	2.30 (0.61)[b]	$F(2, 72) = 5.20$, $p < .001$
Comorbid anxiety disorder (%)		16.00[d]	26.09[d]	62.96[b c]	$X^2(2) = 13.84$, $p < .01$
Demographics					
Age (Years)	31.79 (11.61)	32.16 (13.84)	30.13 (13.22)	30.44 (9.41)	$F(3, 147) = 0.21$, *ns*
Gender (% Female)	65.79	44.00	47.83	59.26	$X^2(3) = 2.70$, *ns*
College graduate (%)	61.84	56.00	43.48	51.85	$X^2(3) = 4.91$, *ns*

Note. FSS Social = Social Fearfulness subscale of the Fear Survey Schedule (38); FSS Total; = Fear Survey Schedule total score (38); STAI-Trait = Trait scale of State Trait Anxiety Inventory (39); BDI =Beck Depression Inventory (40); Severity = clinician-rated severity (6-point scale ranging from 0, *No features present*, to 5, *Diagnosis present; severe*) reflecting both distress and interference; Age of onset = patient-reported onset of social phobia diagnosis; Chronicity = years from patient-reported onset of social phobia diagnosis to assessment; Prognosis = clinician-rated estimate of treatment prognosis (4-point scale ranging from 1, *Excellent*, to 4, *Poor*); Superscripts = Results of Tukey HSD pairwise comparisons

[a] Posthoc between-group comparison to control is significant at $p < .05$

[b] Posthoc between-group comparison to circumscribed at $p < .05$

[c] Posthoc between-group comparison to generalized is significant at $p < .05$.

[d] Posthoc between-group comparison to generalized depressed is significant at $p < .05$.

Table 2

Mean Responses and Standard Deviations to Imagery Scenes by Control and Social Phobia Groups

Imagery scene/ Response Modality	Control	Social Phobia	Group Effect
Neutral			
Startle Reflex (T-score)	49.76 (5.01)	52.25 (6.72)	$F(1, 136) = 6.07$, $p < .05$
SCL Δ (log (μS + 1))	-0.006 (0.036)	-0.004 (0.045)	$F(1, 144) = 2.51$, *ns*
Heart Rate Δ (bpm)	-0.49 (1.95)	-1.35 (1.67)	$F(1, 147) = 8.36$, $p < .01$
Corrugator EMG Δ (μV)	-0.07 (0.79)	0.002 (0.77)	$F(1, 148) = 0.32$, *ns*
Pleasure (1-9)	6.93 (1.53)	6.49 (1.28)	$F(1, 148) = 3.55$, *ns*
Arousal (1-9)	2.31 (1.54)	2.42 (1.20)	$F(1, 148) = 0.21$, *ns*
Social Threat			
Startle Reflex (T-score)	50.85 (6.08)	55.81 (12.32)*	$F(1, 136) = 8.91$, $p < .01$
SCL Δ (log (μS + 1))	0.007 (0.044)	0.038 (0.081)*	$F(1, 144) = 8.56$, $p < .01$
Heart Rate Δ (bpm)	-0.20 (1.36)	0.87 (1.89)*	$F(1, 147) = 15.82$, $p < .001$
Corrugator EMG Δ (μV)	0.61 (1.34)*	0.33 (0.57)*	$F(1, 148) = 2.74$, *ns*
Pleasure (1-9)	3.39 (1.00)*	2.63 (1.15)*	$F(1, 147) = 18.39$, $p < .001$
Arousal (1-9)	6.29 (1.68)*	7.28 (1.22)*	$F(1, 147) = 16.82$, $p < .001$
Survival Threat			
Startle Reflex (T-score)	54.57 (8.22)*	54.93 (7.93)*	$F(1, 136) = 0.07$, *ns*
SCL Δ (log (μS + 1))	0.004 (0.022)*	-0.009 (0.044)	$F(1, 144) = 0.89$, *ns*
Heart Rate Δ (bpm)	-0.07 (1.03)	-0.13 (1.18)*	$F(1, 147) = 0.10$, *ns*
Corrugator EMG Δ (μV)	0.95 (1.97)*	0.33 (0.68)*	$F(1, 148) = 6.57$, $p < .05$
Pleasure (1-9)	2.68 (0.99)*	3.10 (1.07)*	$F(1, 148) = 6.16$, $p < .05$
Arousal (1-9)	6.69 (1.53)*	6.84 (1.27)*	$F(1, 148) = 0.38$, *ns*
Personal Fear			
Startle Reflex (T-score)	53.58 (7.33)*	58.22 (12.32)*	$F(1, 136) = 7.17$, $p < .01$
SCL Δ (log (μS + 1))	0.045 (0.086)*	0.076 (0.0971)*	$F(1, 144) = 4.23$, $p < .05$
Heart Rate Δ (bpm)	0.93 (2.12)*	0.92 (2.09)*	$F(1, 147) = 0.002$, *ns*
Corrugator EMG Δ (μV)	1.16 (2.55)*	0.65 (1.14)*	$F(1, 148) = 2.53$, *ns*
Pleasure (1-9)	2.65 (1.41)*	2.52 (1.38)*	$F(1, 148) = 0.36$, *ns*
Arousal (1-9)	7.69 (1.67)*	7.87 (1.41)*	$F(1, 148) = 0.55$, *ns*

Note. Δ = change; EMG = electromyographic; SCL = skin conductance level; bpm = residual beats per minute after removing prescript onset baseline effects; μV = microvolt; μS = miscrosiemen; Pleasure rated on the Self-Assessment Manikin (39) 1= Completely unhappy, 9=Completely happy; Arousal rated on the Self-Assessment Manikin[39] 1= Completely relaxed, 9=Completely aroused

*Within-group comparison to neutral condition is significant at $p < .05$.