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Is there less to social anxiety than meets the eye? Emotion experience, expression, and bodily responding

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Emotions are widely held to involve changes in experiential, behavioural, and physiological systems. It is not clear, however, just how tightly coupled these changes are during emotional responding. To examine this issue, we induced social anxiety in 47 high trait social anxiety (HTSA) and 50 low trait social anxiety (LTSA) participants using an impromptu speech paradigm. We assessed anxiety experience, behaviour, perceived physiological activation, and actual physiological activation. HTSA participants felt more anxious, perceived greater physiological activation, and exhibited more anxiety behaviour than LTSA participants. Unexpectedly, the two groups did not differ in objectively measured physiological responding. Internal analyses indicated that for both HTSA and LTSA participants, anxiety experience was associated with perceived physiological activation, but not with actual physiological responding. These results suggest that anxiety experience and perceived physiological activation may be less tightly coupled with actual physiological responses than is typically thought.

Nowhere do our mental processes seem to be more clearly embodied than they are in the emotions. Take anxiety, for example. Anyone who has ever been anxious will tell you that anxiety has a highly salient bodily component, and that the greater the anxiety becomes, the more obvious the racing heart, the sweating palms, and the shortness of breath. This intuitive link between emotion and bodily responding was memorably captured by William James more than a century ago, when he asked: ''What kind of emotion of fear would be left, if the feelings neither of quickened heart-beats nor of shallow breathing, neither of

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trembling lips nor of weakened limbs, neither of goose-flesh nor of visceral stirrings, were present?'' (James, 1884, pp. 193–194). His answer, of course, was that: ''We'd have nothing left behind, no 'mind-stuff' out of which the emotion can be constituted'' (p. 193).

Although James's views have been revised multiple times (e.g., Laird & Bresler, 1990), it is all but axiomatic in contemporary emotion research that peripheral physiological responses constitute an important component of the emotional response. Indeed, many view the close coordination of experiential, behavioural, and physiological responses as an essential function of emotion (e.g., Ekman, 1992; Frijda, 1986; Levenson, 1994). However, as Reisenzein (2000) has cogently argued, empirical support for this ''syndromal view'' of emotion is surprisingly weak. In the following sections, we first review theory and research concerning associations among the experiential, behavioural, and peripheral physiological aspects of an emotional response.[1] We find that the modest observed linkages seem to belie theoretical expectations for greater response coupling. We next consider the empirical literature on response linkages in social anxiety, an affective state in which bodily responding and experience are expected appear to be particularly tightly coupled. Even here, however, we find mixed evidence for a coupling of response systems. We then present a laboratory study designed to further investigate the links between emotion experience and bodily responding in the context of social anxiety.

Response system coupling in emotion

The study of emotion seems to be a magnet for controversy. Indeed, vigorous debates have sprung up over just about every topic imaginable. There is one point, however, upon which many researchers now seem to agree. That is the proposition that emotions consist of organised patterns of experiential, behavioural, and physiological responses (e.g., Frijda, 1986). To say that these response components are organized is to suggest, as Levenson (1994) has argued, that emotions impose a greater degree of coherence among experiential, behavioural, and physiological response systems than is typical at rest. From an evolutionary perspective, this ''emotion as organisation'' perspective makes perfect sense. Emotions must unify disparate response systems and coordinate their action in order to optimise the likelihood that the individual will successfully resolve adaptive challenges.

There's just one problem: There is relatively little empirical evidence that emotions involve closely coordinated changes in experiential, behavioural, and physiological response systems. Indeed, psychophysiologists interested in stress

[1] For the sake of brevity, throughout this paper we use ''physiological responding'' instead of the longer term ''autonomic peripheral physiological responding'', unless otherwise noted.

have long emphasised the weak correlations across response systems (e.g., Weinstein, Averill, Opton, & Lazarus, 1968) and even among measures within the same response system (e.g., Lacey, 1967). More recent studies have similarly found relatively modest correlations among measures of emotional responding in the context of specific emotional states, such as fear (Lang, 1988; Rachman, 1978), exhilaration (Ruch, 1995), and surprise (Reisenzein, 2000). Generally, links between emotion experience and facial behaviour have been strongest (e.g., Adelmann & Zajonc, 1989; Blumberg & Izard, 1991; Rosenberg & Ekman, 1994). However, even these links are often modest (e.g., Fernandez-Dols, Sanchez, Carrera, & Ruiz-Belda, 1997; Fridlund, 1994) and inconsistent (e.g., Casey, 1993; Chovil, 1991; Gross, John, & Richards, 2000). As Reisenzein (2000) notes, there are at least two ways to think about this discrepancy between theoretical expectation and empirical reality. One possibility is that emotions actually involve changes that are far more loosely coupled than is typically thought. A second possibility is that methodological limitations have obscured the highly coordinated response coupling that is indeed present.

Ruch's (1995) analysis shows that different experimental designs affect the resultant estimates of linkages between amusement experience and expression. This finding suggests that methodological factors may indeed play an important role (see also Rosenberg & Ekman, 1994). Nonetheless, even with state-of-the-art methodology, Reisenzein (2000) found only modest correlations among cognitive, experiential, and behavioural components of surprise. He used a meticulously designed procedure to elicit surprise repeatedly, and assessed its cognitive, experiential, and behavioural consequences. Data were carefully coded and analysed, and precautions were taken to correct for measurement error and to detect nonlinear associations. However, even with these precautions, the average correlations among the components of surprise were generally low to moderate.

Clearly, methodological sophistication alone does not guarantee high levels of association among response components. Why not? One possibility is that our theories overstate the coupling of response components. Another possibility, however, is that the highly cognitive emotion of surprise may not be the best place to find the sort of correspondences we have been looking for. Perhaps we would have better luck with anxiety, which seems to so obviously involve coordinated changes in emotion experience, behavioural expression, and physiological responding. To examine this possibility, we first turn to a review of the empirical literature on emotional responding in anxiety, with a focus on social anxiety. Social anxiety is a particularly well-suited emotion to test the syndrome view because introspection and clinical observation suggest that in this emotion we should expect clear correspondences between how we feel and how our body is responding. Given our particular interest in the role of physiological responding in emotion experience, we focused on those studies which measured physiological responding and at least one other response domain.

Response system coupling in social anxiety

Table 1 summarises studies that have measured physiological responses and at least one other emotion response component in the context of social anxiety-provoking tasks. The typical design of these studies is to compare low and high trait socially anxious participants, and to see whether these groups differ in experiential, behavioural, or physiological responses. As shown in column 3 of Table 1, there is clear agreement that individuals who say they are *typically* anxious report feeling greater anxiety in an anxious-making situation than individuals who report that they do not typically feel anxious. Also, as can be seen in column 4, studies that measured anxiety behaviour showed that high socially anxious individuals also exhibit more anxiety behaviour than low socially anxious individuals. However, as noted in column 6, the data are inconclusive as to whether high socially anxious individuals exhibit higher physiological activation than low anxious individuals. This is consistent with a meta-analysis by Patterson and Ritts (1997), which found large effects of social anxiety on cognitive and behavioural measures but smaller effects on physiological measures. One interesting puzzle is that all five empirical studies that measured *self-perceived* physiological activation found that high socially anxious participants report higher physiological activation than low socially anxious participants, independent of whether they actually evidenced heightened physiological activation (see column 5). Those studies that also report *correlations* among response systems (e.g., Borkovec, Stone, O'Brien, & Kaloupek, 1974; Edelmann & Baker, 2002) indicate low to moderate coherence between self-reports and physiological activation. Even in the case of social anxiety, it seems, it is not yet clear how tightly emotion response components are coupled.

The present study

Several important limitations of prior studies seem noteworthy. One limitation is that very few studies have simultaneously examined experiential, behavioural, perceived physiological, and actual physiological responses in a highly emotional situation. If we are to fully appreciate linkages across systems, this sort of multimethod approach is essential. A second limitation of prior studies is that many of the methodological advances suggested by Reisenzein (2000) and others have not been implemented in the context of studies of social anxiety, an affective state which seems to be a particularly promising candidate for strong associations among response components. For example, although there are both ethical and practical constraints governing how many times one can make participants intensely anxious in a given experimental session, it seems desirable to have at least one low level and one high level induction period. A third limitation is that when an extreme-groups approach (selecting for emotion traits) has been used in the anxiety literature, the emotion induction procedure is not

TABLE 1
Self-reported anxiety experience, other-rated anxiety behaviour, self-perceived physiological activation, and actual physiological activation in high trait socially anxious as compared to low trait socially anxious groups during social stressors

Study	*N*	*Anxiety experience*[a]	*Anxiety behaviour*[a]	*Perceived physiological activation*[a]	*Actual physiological activation*[a]
Baggett, Saab, & Carver (1996)	55	↑	–	–	↔
Beatty & Behnke (1991)[b]	60	↑	–	–	↔
Beidel, Turner, & Dancu (1985)	26	↑	↑	–	↑
Borkovec, Stone, O'Brien, & Kaloupek (1974)	46	↑	↑	↑	↑
Bruch, Gorsky, Collins, Berger (1989)	84	↑	↑	–	↑
Davidson, Marshall, Tomarken, & Henriques (2000)	28	↑	–	–	↑
Dimberg, Fredrikson, & Lundquist (1986)	24	–	–	–	↑
Eckman & Shean (1997)	52	↑	–	↑	↔
Edelmann & Baker (2002)	54	–	–	↑	↔
Grossman, Wilhelm, Kawachi, & Sparrow (2001)	55	↑	–	↑	↔
Hofmann, Newman, Ehlers, & Roth (1995)	52	↑	–	–	↑
Knight & Borden (1979)	16	↑	–	–	↑
Levin et al. (1993)[c]	50	↑	↑	–	↑
McKinney, Gatchel, & Paulhus (1983)	40	↑	↔	–	↔
Mulkens, de Jong, Dobbelaar, & Bögels (1999)	57	↑	–	↑	↔
Panayiotou & Vrana (1998)	55	↑	–	–	↔
Puigcever, Matinez-Selva, Garcia-Sanchez, & Gomez-Amor (1989)	40	↑	–	–	↔
Stein, Asmundsdon, Chartier (1994)	30	↑	–	–	↑
Turner & Beidel (1985)[c]	52	↑	–	–	↑
Turner, Beidel, & Larkin (1986)	69	↑	–	–	↑
Twentyman & McFall (1975)	31	↑	↑	–	↑

[a] ↔, no group differences between HTSA and LTSA participants; ↑, HTSA participants show higher reactivity than LTSA participants in self-reported anxiety (col. 3), anxiety behaviour (col. 4), perceived physiological activation (col. 5), or actual physiological activation (col. 6) in at least one measure either during or in anticipation of a stressful event; –, no measure was reported.

[b] ↔, no differences were found under ''high intensity conditions''; under ''low intensity conditions'', HTSA participants exhibited higher reactivity than LTSA participants; [c] ↑, Higher reactivity was measured only in subgroups of HTSA participants.

always matched to the participants' particular concerns. This is important because we can hardly expect high levels of coherence if the intended target emotion is not successfully elicited. Fourth, prior studies typically have reported only group means but not correlations among response systems. While coherent mean differences are one prediction the syndrome view makes (e.g., higher

anxiety experience goes together with higher physiological activation), this view also makes the prediction that responses in the experiential, the behavioural, and the physiological systems will be correlated positively and that correlations are larger in more intense emotional responding (e.g., Davidson, 1992; Rosenberg & Ekman, 1994). To test these more specific predictions, correlations among response systems need to be reported.

To address these limitations, we conducted an experiment in which participants watched a neutral nature film (baseline) and then gave an impromptu speech (high anxiety period). We used an impromptu speech paradigm because a high proportion of socially anxious individuals report fear of speaking in front of others (e.g., Hazen & Stein, 1995), and this paradigm has been shown to be a reliable and valid method of inducing high levels of anxiety (e.g., Beidel, Turner, Jacob, & Cooley, 1989a). Inducing high levels of anxiety is particularly important since only high levels of anxiety might impose the kind of coherence among response systems expected from emotion theories. Participants' experiential, behavioural, perceived physiological, and actual physiological responses were assessed repeatedly throughout the session.

We carefully matched subjective and objective indicators of four physiological responses commonly reported during anxiety, namely, racing heart, blushing, sweating palms, and shortness of breath (Amies, Gelder, & Shaw, 1983; Gorman & Gorman, 1987). Whereas physiological responses were measured continuously, and behavior responses were coded throughout the low and high anxiety periods, we assessed emotion experience and perceived physiological responding only selectively so as to avoid disrupting the session. To ensure the relevance of our anxiety induction procedures, we preselected low and high socially anxious individuals using both their scores on a general social anxiety inventory and on a specific scale designed to measure anxiety concerning public speaking. To reduce between-groups variability that could obscure relations among response components, we enrolled only female participants.

Our hypotheses were as follows:

Hypothesis 1: Levels of anxiety experience, behaviour, perceived physiological responding, and actual physiological responding will be greater during the high anxiety period (speech) than during the film viewing baseline. During the baseline, differences between low and high trait socially anxious participants (LTSA and HTSA) will be minimal. During the high anxiety period, HTSA participants will show higher levels of responding across response domains than LTSA participants.

Hypothesis 2: Changes in each of these response domains (from the baseline to the high anxiety period) will be positively correlated with one another. On the basis of priori theorising, we hypothesised that the magnitude of correlations

among response domains would be higher for the HTSA participants than for the LTSA participants.

METHOD

Overview

A total of 97 female undergraduates were selected as low trait socially anxious (LTSA) or high trait socially anxious (HTSA) based on their scores on the Social Phobia and Anxiety Inventory (SPAI). In individual laboratory sessions, these participants gave impromptu speeches under conditions designed to maximise anxiety. Dependent measures sampled the following domains: anxiety experience, anxiety behaviour, perceived physiological activation (racing heart, blushing, sweaty palms, shortness of breath), and objective physiological activation (measures selected to match perceived activation were heart rate, facial blush, skin conductance level, and respiratory rate; additional measures were mean arterial blood pressure, cardiac output, total peripheral resistance, finger pulse amplitude, skin conductance fluctuations, respiratory tidal volume, respiratory sinus arrhythmia, and somatic activity).

Participants

Participants were selected from a pool of 840 female undergraduates at Stanford University using the Social Phobia and Anxiety Inventory (SPAI), a questionnaire validated for identifying individuals with a trait of social phobia or social anxiety and predicting their distress levels (Beidel, Turner, Stanley, & Dancu, 1989b). The high trait socially anxious (HTSA) group was selected from the top 25% of the distribution of scores on the Social Phobia subscale of the SPAI, and the low trait socially anxious (LTSA) group was selected from the bottom 25% of the distribution. In addition, participants had to be above (for the HTSA group) or below (for the LTSA group) the mean of a subscale comprised of the six SPAI items relating to fear of speaking in front of others (items 5, 6, and 22 a–d).

Students selected for the study were telephoned and asked to take part in an investigation of emotion for which they would receive either course credit or money. A total of 115 of these students came to the laboratory for testing, and filled out the SPAI a second time so that their social anxiety status could be verified. Test-retest reliability was high, $r(115) = .94$, $p < .001$. Eleven of the participants were excluded because their overall SPAI scores or their speech fear scores changed markedly from the questionnaire session to the laboratory session. In addition, because of technical malfunctions that affected at least one channel of the physiological recording, seven more participants were excluded from the analyses.

This left 97 participants for analysis, 47 of them high socially anxious (average age = 19.3 years, *SD* = 1.1) and 50 of them low socially anxious (average age = 19.1 years, *SD* = 1.1). Mean SPAI scores (completed at the laboratory) were 105.5 for the HTSA group (*SD* = 19.3) and 33.5 (*SD* = 16.3) for the LTSA group, with a range of possible scores from 0 to 672. Mean scores for fear of speaking in front of others were 27.7 for the HTSA group (*SD* = 5.2) and 8.0 for the LTSA group (*SD* = 4.3), with a range of possible scores from 0 to 36. These 97 participants returned to the laboratory for a second laboratory session, which is the focus of this report.

The ethnic composition of the sample was mixed: 9% African American, 10% Asian American, 57% Caucasian, 7% Latino, 2% Native American, and 15% other. HTSA and LTSA groups were matched by ethnicity. All participants were native English speakers. Written informed consent was obtained after the procedures had been fully explained.

Procedure

Core elements of the procedure are summarised in Figure 1. Upon arrival at the laboratory, the participant was greeted by a male experimenter and seated in a well-lit and comfortable 9 × 12 ft room held between 70° and 73.5°F. Time of day was matched between the two groups. Participants had at least 15 min to habituate to their surroundings before the procedure began. After physiological sensors were attached, the experimenter communicated with participants by intercom. Participants remained seated during the whole procedure.

Participants underwent testing in four stages: film viewing baseline (3 min), preparation for the speech (3 min), speech (3 min), and recovery (2 min). The session began with a neutral videotape of seascapes (cf. Piferi, Kline, Younger,

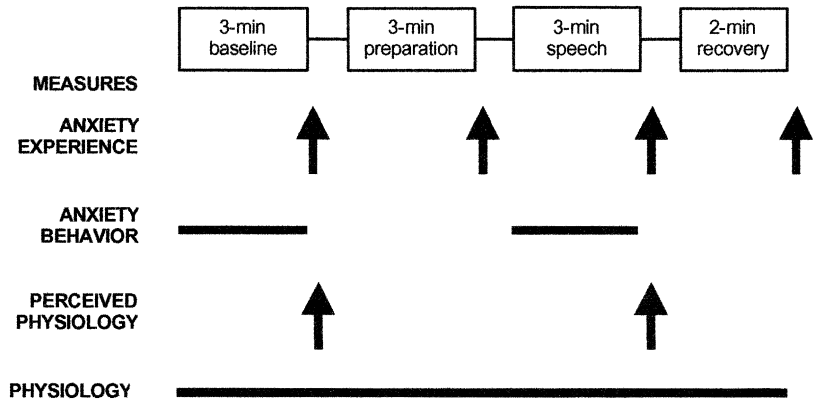

Figure 1. Profile of experimental tasks and dependent variables.

& Lawler, 2000). At the end of this low anxiety baseline, the participant filled out a set of emotion ratings, after which she underwent three short procedures not relevant to this report. Before starting the next procedures, the experimenter entered the subject room and directed the participant to put on a headset-microphone unit. The experimenter then informed the participant that for the next 2 min she would be presented with short bursts of white noise through the headset in order to get accustomed to the tones she would be hearing during later parts of the study. These white noise bursts were designed to increase task apprehension, task anxiety, and task difficulty. They were presented every 15 seconds during the preparation and the speech periods.

After this, participants rested for a minute, and again watched the seascapes video as they had during the film viewing baseline. The experimenter then informed the participant that:

> In the next phase of the study, you'll be giving a speech on a topic that we'll provide. During your speech, we'll be watching you closely. We'll also be videotaping you, and will be showing this videotape to a panel of judges, who will rate how convincing a job you do.

The experimenter then entered the subject room to set up the video camera on a tripod, which was ostentatiously positioned directly in front of the participant. Unbeknownst to participants there was another video camera, which was hidden behind darkened glass and ran continuously throughout the session. They were then informed that the topic of the speech would be: ''Is it wrong for the government to execute people?'' To increase anxiety, participants were also told:

> Your speech will be videotaped and later scored by a panel of judges who will rate and compare your speech to others given under the same circumstances. The judges will rate your speech on its overall persuasiveness, so it's very important that you try to be as thorough and persuasive as possible, talking for the full three minutes.

After the 3 min preparation period, participants were asked to orally rate their current anxiety on a 11-point Likert scale, ranging from 0 (none at all) to 10 (extremely). They were then given 15 s to collect their thoughts before starting the speech. After 3 min of speech, they were given 2 min to recover. The experimenter then asked participants again to orally rate their current anxiety and to fill out a second set of emotion ratings regarding the speech.

Self-report measures

Trait measures. As described above, participants filled out the Social Phobia and Anxiety Inventory (Beidel et al., 1989b) two times and also answered several demographic questions. On average, there was one week time

lag between filling out the SPAI and coming to the laboratory session. After the laboratory session, participants were contacted via e-mail and asked to fill out the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960). Eighty-nine of our participants (91.8 %) filled out this questionnaire. This measure was used for secondary analyses.

Anxiety experience. Participants rated their anxiety on an 11-point Likert scale, ranging from 0 (none at all) to 10 (extremely) for the baseline, preparation, speech, and recovery periods.

Perceived physiological responses. Participants rated the extent to which they felt a racing heart, blushing, sweaty palms, and shortness of breath using four separate 11-point Likert scales, ranging from 0 (none at all) to 10 (extremely). Participants made these four ratings for the film viewing baseline and the speech.

Behavioural measures

Two trained judges rated the 3 min videotape segments of the film viewing baseline and speech periods, making minute-by-minute ratings of behavioural signs of anxiety in each period. Judges were blind to whether participants were low or high in trait anxiety. Tapes showed participants' head and upper chest, since movement of their body and extremities was limited due to physiological sensors. Each minute, the raters coded overall behavioural anxiety on a 5-point Likert scale, ranging from 1 (not at all) to 5 (extremely). Coding was done for the speeches with the sound muted in order to take into account only behavioral anxiety and not measures of speech quality or speech fluency. Judges were given specific indicators of anxiety, such as rigid posture, fearful facial expression, and rigid facial expression, but their ratings also reflect their overall impressions of the speakers (cf. Glass & Arnkoff, 1989; Harrigan & O'Connell, 1995). Interrater reliabilities were adequate: .65 for the baseline and .78 for the speech. Means of the two judges' ratings were averaged across both 3 minutes periods for subsequent analyses of behavioural data.

Physiological measures

During the experimental session, physiological channels were sampled continuously at 400 Hz using laboratory software. Later, customised analysis software (Wilhelm, Grossman & Roth, 1999) was applied to physiological data reduction, artifact control, and computation of average physiological scores for each participant for the baseline, preparation, speech, and recovery periods. Measures of physiological activation were chosen to match the measures of

perceived physiological activation and to comprehensively assess response systems known to be involved in emotional responding.

Measures to match perceived physiological activation. We obtained four measures to match our measures of perceived physiological responding. *Heart rate* (to match ''racing heart'') was calculated from respiratory rate (RR) intervals in the electrocardiogram (ECG). Values from ectopic or other kinds of abnormal beats were deleted and replaced by linearly interpolated values. *Facial blush* (to match ''blushing'') was measured with a plethysmograph transducer that was attached to the participant's left cheek right below the cheek bone. Customised software was used to calculate average blood volume as the area under this curve to index facial blush. *Skin conductance* (to match ''sweaty palms'') was derived from a signal using a constant-voltage device to pass 0.5 V between Beckman electrodes (using an electrolyte of sodium chloride in Unibase) attached to the palmar surface of the middle phalanges of the first and second fingers of the nondominant hand. Skin conductance level was indexed by the mean level after movement and electrode contact artifacts had been edited out using a customised detection procedure (Wilhelm & Roth, 1996). *Respiratory rate* (to match ''shortness of breath'') was measured using an inductive plethysmography device (Respitrace Corporation, Ardsley, NY) connected to bands containing coils of insulated wires placed around the abdomen and chest. Calibration against 800 ml fixed-volume bags was accomplished by the least-squares method (Chadha et al., 1982). Respiratory rate was calculated breath-by-breath using customised programmes.

Additional physiological measures. In order to gain a more complete understanding of participants' physiological activation, we obtained eight additional measures. *Blood pressure* was obtained from the third finger of the nondominant hand by means of the Finapres™ 2300 (Ohmeda, Madison, WI) system. Beat-to-beat stroke volume was measured using Wesseling's pulse-contour analysis method (BEATFAST, TNO-Biomedical Instrumentation, Amsterdam), which has been previously validated (Stok et al., 1993). *Cardiac output* was calculated as stroke volume × heart rate. *Total peripheral resistance* was calculated as mean arterial pressure × 80/cardiac output. *Finger pulse amplitude* was measured with a plethysmograph transducer attached to the tip of the participant's second finger. Customised software was used to calculate beat-to-beat mean blood volume. From this curve, local maxima minus local minima were calculated to index finger pulse amplitude. *Skin conductance fluctuations* were detected as changes in skin conductance level (SCL) from a zero-slope baseline exceeding 0.2 μSiemens. Fluctuations likely to stem from electrode contact artifacts were identified and excluded. *Respiratory tidal volume* was measured using the same procedures as respiratory rate by indexing breath-to-

breath air volume inhaled. In order to index *respiratory sinus arrhythmia* (RSA), the RR interval series from the ECG was converted into a time series of instantaneous RR intervals with a resolution of 4 Hz. RSA, or vagal control of heart rate, was estimated as the magnitude of the transfer function relating RR interval oscillations to lung volume oscillations (resampled to 4 Hz) at the peak respiratory frequency (Saul et al., 1991). The peak respiratory frequency was automatically detected as the greatest local maximum in the lung volume power spectral density. Spectral coherence at this frequency was required to be at least 0.5. Epochs with peak respiratory frequency below the 0.15–0.50 Hz band were excluded. *Somatic activity* was measured by a piezo-electric device attached to the subject's chair. This device generates an electrical signal proportional to the subject's overall body movement in any direction.

RESULTS

Statistical analyses

We performed two sets of analyses to address our hypotheses. To test *Hypothesis 1*, we used multivariate analyses of variance (MANOVAs) for each response domain that had multiple response measures, with period as a repeated measure, and group as a between-participants factor. Univariate tests were used to follow up on significant multivariate tests; they also were used for response domains with single response measures. The univariate tests focused on the low anxiety baseline and the high anxiety speech. Significant effects of period were followed up with pairwise comparisons to assess whether anxiety experience, behaviour, perceived physiological activation, or actual physiological activation were higher during the speech than during the baseline. Significant effects of anxiety group were followed up with groupwise comparisons to assess whether HTSA participants exhibited higher activation than LTSA participants during the baseline or the speech.[2] To test *Hypothesis 2*, we first transformed change scores (speech minus baseline) for all variables into *z*-scores in order to control for differences in variances between the two groups. Pearson product-moment correlations were then calculated for those pooled *z*-transformed change scores for all measures, first, between all participants and, second, between participants within each anxiety group.[3] We then used the *r* to *z* transform to test for differences in the magnitude of correlations between the two groups.

[2] In order to be consistent across response domains we performed these analyses on raw scores. It should be noted, however, that analyses using change scores from baseline yield results identical to those using raw scores.

[3] Patterns of correlations were identical if correlations were calculated using raw scores. However, we chose to report change scores in order to control for individual differences in baseline activation.

Testing Hypothesis 1: Group differences in anxiety experience

As predicted, a 2 × 4 (Group × Period) analysis of variance revealed significant main effects of anxiety group, $F(1, 94) = 54.50$, $p < .001$, $\eta^2 = .36$, and period, $F(3, 94) = 249.22$, $p < .001$, $\eta^2 = .85$, as well as a significant two-way interaction of anxiety Group × Period, $F(3, 94) = 25.57$, $p < .001$, $\eta^2 = .30$. As evident in Figure 2, a paired *t*-test revealed that all participants reported significant increases in anxiety from baseline to the speech period ($p < .001$, two-tailed, $\eta^2 = .79$). Four *t*-tests for independent groups showed that HTSA participants reported higher levels of anxiety than LTSA participants for all periods except for the baseline (for baseline $p = .053$; $\eta^2 = .04$; all other ps $< .001$ two-tailed, η^2s $> .18$). In addition, HTSA participants reported significantly higher *increases* in anxiety than LTSA participants from baseline to speech, as evidenced by significantly greater change scores from baseline to speech for HTSA participants as compared to LTSA participants ($p < .001$, two-tailed, $\eta^2 = .30$). Adjustment for familywise errors with the Bonferroni method does not change the significance of these results.

Testing Hypothesis 1: Group differences in anxiety behaviour

A 2 × 2 (Group × Period) analysis of variance revealed significant main effects of anxiety group, $F(1, 95) = 7.70$, $p < .007$, $\eta^2 = .08$, and period, $F(1, 95) = 138.06$, $p < .001$, $\eta^2 = .60$. Unexpectedly, the Group × Period interaction was not significant for anxiety behaviour, $F(1, 95) = 1.28$, $p = .26$, $\eta^2 = .02$. All participants exhibited increased levels of anxiety behaviour during the speech

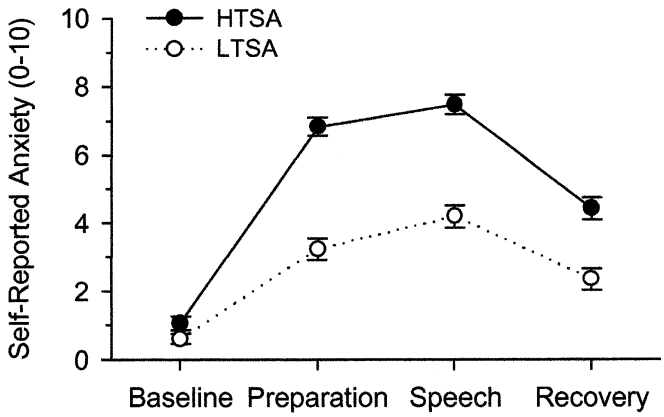

Figure 2. Self-reported anxiety for baseline, preparation, speech, and recovery for high trait socially anxious (HTSA) vs. low trait socially anxious (LTSA) participants.

(M = 3.0, SD = 0.89, for LTSA participants, and M = 3.5, SD = 0.88 for HTSA participants), as compared to the baseline (M = 1.9, SD = 0.57, for LTSA participants, and M = 2.1, SD = 0.79 for HTSA participants). Two t-tests for independent groups revealed that there were no group differences during the baseline (p = .15, two-tailed, η^2 = .02) but that during the speech HTSA participants exhibited higher levels of anxiety behaviour than LTSA participants (p = .005, two-tailed, η^2 = .08). A groupwise t-test showed that HTSA participants did not exhibit significantly greater increases in anxiety behaviour from baseline to speech than LTSA participants (p = .26, two-tailed, η^2 = .01).

Testing Hypothesis 1: Group differences in perceived physiological responding

As expected, the 2 × 2 (Group × Period) multivariate analysis of variance revealed significant main effects of anxiety group, $F(4, 92) = 4.93$, p = .001, η^2 = .18, and period, $F(4, 92) = 20.76$, $p < .001$, η^2 = .47, as well as a significant two-way interaction of Anxiety group × Period, $F(4, 92) = 4.30$, p = .003, η^2 = .18. As evident in Figure 3, Panels (a) through (d), paired t-tests revealed that participants reported increased levels of physiological activation during the speech as compared to the baseline (all ps < .001, two-tailed, η^2s > .12). Eight t-tests for independent groups revealed that after adjusting for familywise errors with the Bonferroni method there were no significant group differences during the baseline (all ps > .025, two-tailed, all η^2s < .06) but that during the speech HTSA participants reported higher levels of physiological activation (all ps < .005, two-tailed, all η^2s > .08). HTSA participants reported significantly greater increases in perceived physiological activation than LTSA participants from baseline to speech, as evidenced by significantly higher change scores (all ps < .022, two-tailed, all η^2s > .06). Adjustment for familywise errors with the Bonferroni method does not change the significance of these results.

Testing Hypothesis 1: Group differences in actual physiological responding

We conducted a 2 × 4 (Group × Period) multivariate analysis of variance (MANOVA) with all 12 physiological measures. This analysis revealed a significant main effect of Period, $F(36, 47) = 32.1$, $p < .001$, η^2 = .96, but no significant effect of Group, $F(12, 71) = 0.2$, p = .997, η^2 = .04, and no significant Group × Period interaction, $F(36, 47) = 1.1$, p = .419, η^2 = .06. As evident in Figure 3, Panels (e) through (h) and Figure 4, Panels (a) through (h), paired t-tests revealed that participants exhibited increased levels of physiological activation during the speech as compared to the baseline (all ps < .016, two-tailed, all η^2s > .07), with the exception of respiratory rate, which was statistically equal during speech and baseline (p = .288, two-tailed, η^2 = .02). Participants thus exhibited higher heart rate, higher facial blush, higher skin conductance

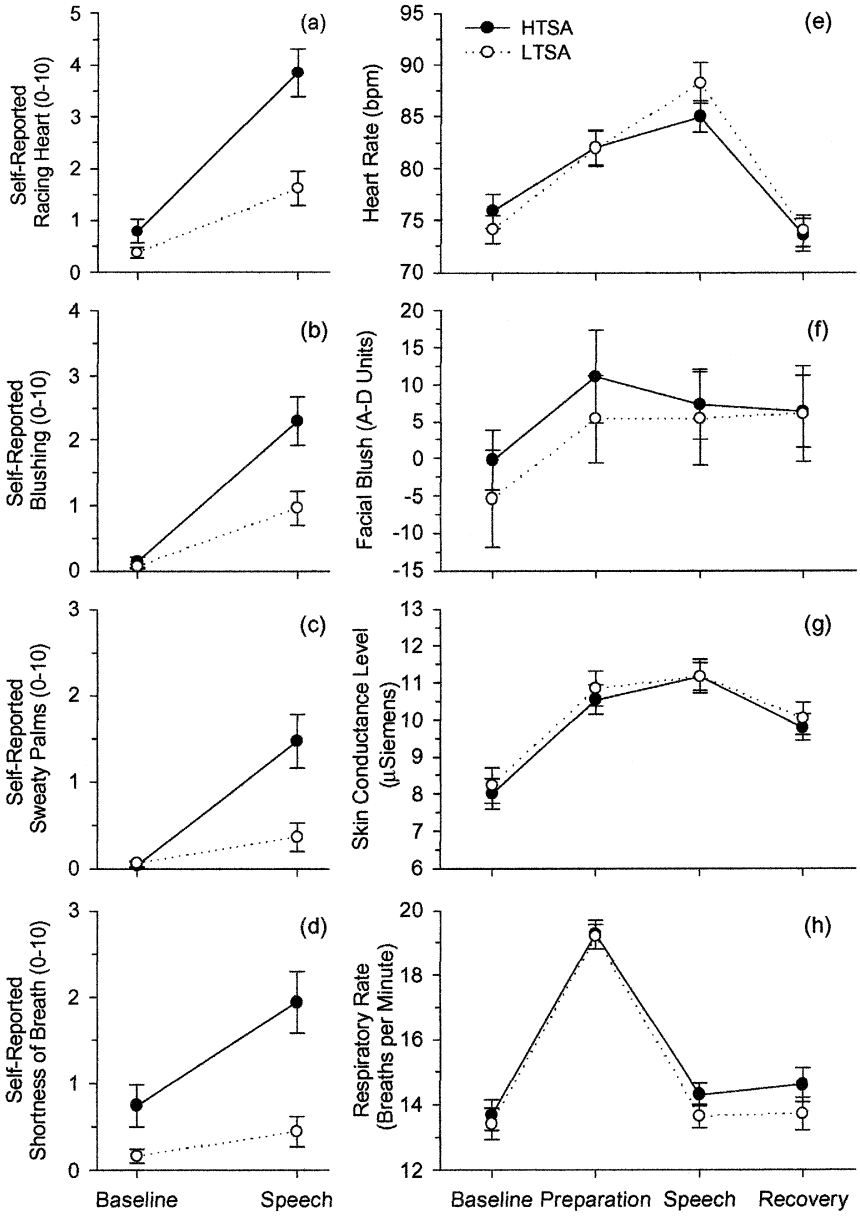

Figure 3. (a)–(d): Self-reported racing heart, blushing, sweaty palms, and shortness of breath for baseline and speech for high trait socially anxious (HTSA) vs. low trait socially anxious (LTSA) participants. (e)–(h): Actual physiological activation of HTSA vs. LTSA participants as measured by heart rate, facial blush, skin conductance level, and respiratory rate for baseline, preparation, speech, and recovery.

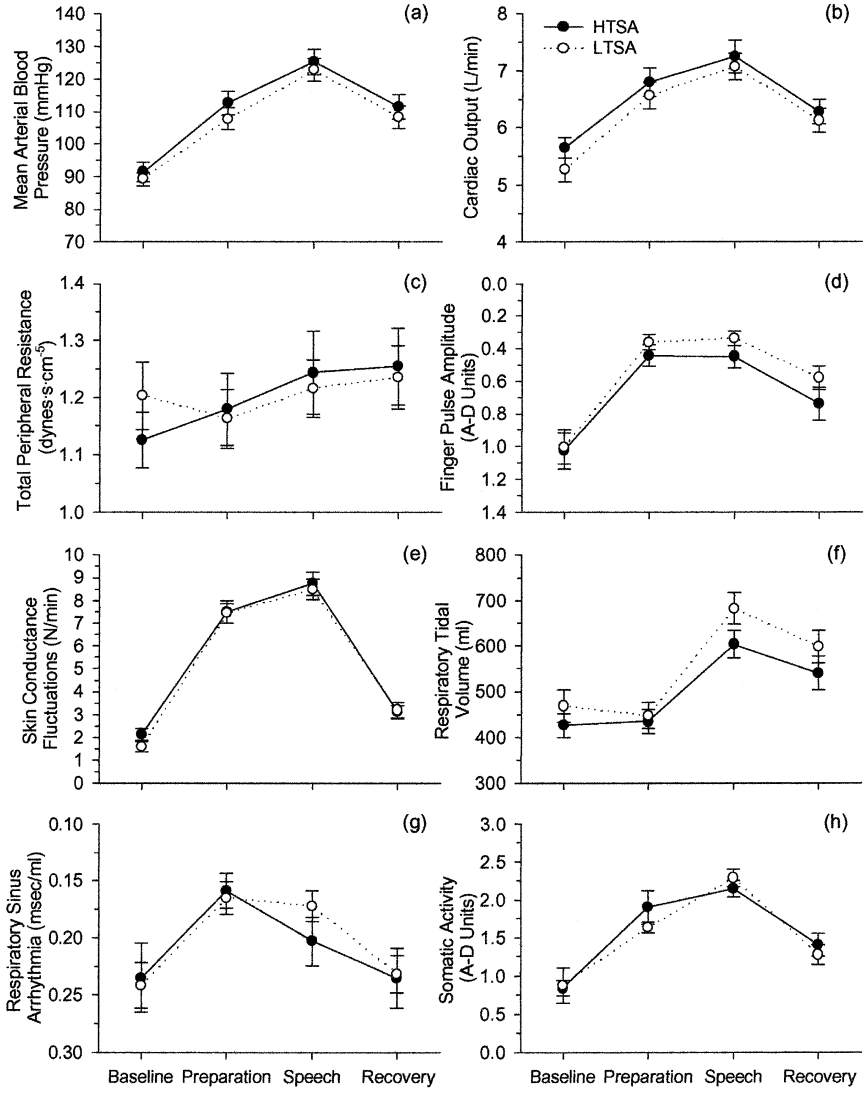

Figure 4. (a)–(h): Actual physiological activation of high trait socially anxious (HTSA) vs. low trait socially anxious (LTSA) participants as measured by mean arterial pressure, cardiac output, total peripheral resistance, finger pulse amplitude, skin conductance fluctuations, respiratory tidal volume, respiratory sinus arrhythmia, and somatic activity for baseline, preparation, speech, and recovery.

levels, higher mean arterial pressure, higher cardiac output, higher total peripheral resistance, lower finger pulse amplitude, more skin conductance fluctuations, higher tidal volume, lower respiratory sinus arrhythmia (RSA), and more somatic activity during the speech than during the resting baseline. Contrary to our hypothesis, groupwise *t*-tests showed that LTSA and HTSA participants did not differ in their actual physiological responding at any point in the session (all *p*s > .110, two-tailed, all η^2s < .03).[4] Adjusting for familywise errors with the Bonferroni method does not change the significance of the results for all physiological measures.

Testing Hypothesis 2: Correlations among measures

As shown in Table 2, across all participants, anxiety experience was modestly positively correlated with anxiety behaviour.[5] Also as expected, anxiety experience was positively correlated with each of the measures of perceived physiological activation. These positive intercorrelations, which may be seen in the left half of Table 2, suggest a fair degree of coherence between anxiety experience and perceived physiological activation and, to a lesser degree, with anxiety behaviour.

More surprising is the lack of correlation between anxiety experience, behaviour, and perceived physiological activation, on the one hand, and actual physiological activation on the other. As may be seen in the right half of Table 2, both anxiety experience and anxiety behaviour were not significantly correlated with any of the measures of actual physiological responding. It is also noteworthy that of the 10 albeit mostly positive correlations between perceived physiological responding and actual physiological responding, only two were

[4] One possible alternative explanation for physiological activation is that, in the case of giving a speech, physiological activation reflects overall body movement or active task engagement rather than affective state (e.g., Obrist, Webb, Sutterer, & Howard, 1970). In other words, not anxiety but body movement or preparation for body movement might be responsible for most of the variability in the autonomic activation observed. To test whether this was the case we correlated overall body movement with the eight physiological measures. None of the relevant correlations reached statistical significance, indicating that physiological activation was not primarily under control of body movement. We also checked whether length of the speeches affected physiological responding. Although all participants had received instructions to ''talk for the full three minutes'', a few ended early (N = 17, with an average of 15 s). The eight correlations between length of speeches and measures of physiological responding were all not significant (all *p*s > .06), indicating that length of speech did not affect physiological responding.

[5] In Tables 2 and 3, we only present correlations for heart rate, facial blush, skin conductance level, and respiratory rate because these measures were matched with perceived physiological responding. However, the results found also hold for blood pressure, cardiac output, total peripheral resistance, finger pulse amplitude, skin conductance fluctuations, respiratory tidal volume, RSA, and somatic activity. There are also no group differences in correlations for these measures.

TABLE 2
Correlations between z-transformed change scores (speech minus baseline) for self-reported anxiety, anxiety behaviour, self-perceived physiological activation, and corresponding physiological measures for all participants ($N = 97$)

			Self-perceived physiological activation				*Actual physiological activation*			
	Anxiety experience	*Anxiety behaviour*	*Racing heart*	*Blushing*	*Sweaty palms*	*Shortness of breath*	*Heart rate*	*Facial blush*	*SCL*[a]	*RR*[b]
Anxiety experience		.21*	.52*	.40*	.34*	.43*	−.03	.16	.14	.18
Anxiety behaviour			.03	.13	.22*	.11	−.10	−.06	.02	−.01
Self-perceived physiological activation										
Racing heart				.54*	.42*	.63*	.22*	.18	.21*	.16
Blushing					.61*	.52*	.07	.18	.16	.17
Sweaty palms						.47*	.07	.10	.16	−.01
Shortness of breath							−.01	.18	.04	.12
Actual physiological activation										
Heart rate								.17	.04	.19
Facial blush									−.02	.16
SCL[a]										−.09
RR[b]										

[a] SCL = skin conductance level; [b] RR = respiratory rate; * $p < .05$ (two-tailed).

significant. Only one of these was the expected match (heart rate), and even this was small in size (.22). Also, as has been observed repeatedly in prior studies, our whole-period measures of actual physiological responding were not significantly correlated with each other.

Might findings observed in Table 2 be due to the fact that the low levels of anxiety evident in the LTSA participants imposed relatively low levels of coherence among responses, and this masked the high levels of coherence evidenced among the HTSA participants? To test this possibility, we computed correlations among response measures separately for each of the two participant groups. As may be seen in Table 3, the picture that emerges from these correlations matches that observed in Table 2 nearly exactly. For both groups, anxiety experience was most tightly coupled with perceived physiological responses, and less tightly coupled with actual physiological responses. Once again, actual physiological activation generally did not predict perceived physiological responding well, and the various measures of actual physiological responding were poorly correlated. We had hypothesised that changes among response components would be more tightly coupled in HTSA than in LTSA participants. This hypothesis was not supported. Indeed, the correlation between anxiety experience and heart rate was significantly higher for the LTSA than for the HTSA group ($p = .030$), a finding opposite to expectation.

Secondary analyses

The finding that LTSA and HTSA participants did not differ in physiological activation was surprising, particularly because participants in these two groups clearly differed in self-reported physiological activation. Likewise, the low and sometimes even negative correlations among physiological activation on the one hand, and anxiety experience, behaviour, and perceived physiological responding on the other hand, were somewhat unexpected. Several factors suggest that these null findings should be accepted at face value. These include the adequately large sample size, the single sex sample, the care with which we manipulated and measured emotional responding under controlled conditions, and the large number of physiological measures we included. In particular, we were able to rule out the possibility that group differences would appear in parasympathetic activation (RSA) or in measures indicative of a threat versus challenge appraisal (heart rate, cardiac output, total peripheral resistance; cf. Blascovich & Tomaka, 1996; Lazarus & Folkman, 1984). In addition, we performed three additional analyses to exclude important alternative explanations for our findings.

A first interpretation of the lack of group differences that we thought bore examination in a secondary analysis was the effect of social desirability. It is well known that individuals who report low levels of trait anxiety either may be

TABLE 3

Correlations between *z*-transformed change scores (speech minus baseline) for self-reported anxiety, anxiety behaviour, self-perceived physiological activation and corresponding physiological measures for low trait socially anxious (light grey, left bottom, $N = 50$) and high trait socially anxious participants (dark grey, right top, $N = 47$)

			Self-perceived physiological activation				*Actual physiological activation*			
	Anxiety experience	*Anxiety behaviour*	*Racing heart*	*Blushing*	*Sweaty palms*	*Shortness of breath*	*Heart rate*	*Facial blush*	*SCL*[a]	*RR*[b]
Anxiety experience		**.21**	**.45***	**.24**	**.15**	**.35***	**−.15**	**−.03**	**.19**	**.10**
Anxiety behaviour	*.14*		**−.13**	**.12**	**.28**	**−.04**	**.11**	**.07**	**.03**	**−.07**
Perceived physiological activation										
Racing heart	*.41**	*.12*		**.52***	**.30***	**.61***	**.19**	**.16**	**.16**	**.01**
Blushing	*.40**	*.08*	*.46**		**.57***	**.52***	**.02**	**.03**	**.15**	**.04**
Sweaty palms	*.31**	*.07*	*.44**	*.57**		**.36***	**.24**	**.13**	**.20**	**−.04**
Shortness of breath	*.47**	*.31**	*.59**	*.40**	*.62**		**.02**	**.20**	**−.04**	**.11**
Actual physiological activation										
Heart rate	*.30**	*−.22*	*.46**	*.29**	*.05*	*.10*		**.14**	**.09**	**.02**
Facial blush	*.34**	*−.12*	*.26*	*.22*	*.16*	*.25*	*.18*		**−.24**	**.24**
SCL[a]	*.09*	*05*	*26*	17	*.08*	*.14*	*.03*	*12*		**−.12**
RR[b]	*.24*	*.03*	*.32**	*.34**	*−.01*	*.12*	*.36**	*.12*	*−.07*	

Correlations for low anxious participants are in the left bottom (italics), correlations for high trait socially anxious participants are in the right top (bold face) of the table. Correlations for the high trait socially anxious group are statistically no different from correlations for the low trait socially anxious group (all *p*s > .05), with the exception of anxiety experience and heart rate ($p = .03$). [a] SCL = skin conductance level; [b] RR = respiratory rate; * $p < .05$ (two-tailed).

truly low anxious, or may, for defensive reasons, assert they are low anxious when they really are not. In particular, individuals high in social desirability have been shown to under-report negative emotions. That is, they say that they experience little anxiety but they exhibit heightened physiological reactivity (e.g., Weinberger, Schwartz, & Davidson, 1979). This observation suggests an alternative explanation of our findings, namely that a subgroup of high social desirability individuals within our LTSA group might report being LTSA while exhibiting much higher physiological activation than true LTSA participants. On average, this would obscure true differences in physiological activation between the two groups.

To test this alternative explanation, we used scores on the Marlowe-Crowne Social Desirability Scale to assess social desirability, and thereby to identify participants who might have falsely reported being LTSA. We divided participants into low and high social desirability groups with a median split (*Mdn* = 14). In the low social desirability group the mean score was 8.8 (*SD* = 3.1); in the high social desirability group the mean score was 18.2 (*SD* = 3.3). If individual differences in social desirability accounted for our null findings, using only participants low in social desirability should yield differences in physiological responding between anxiety groups. Repeating the same analyses as before using only low social desirability participants did not yield significant main effects or interactions involving anxiety for the physiological measures. Moreover, recalculating the correlations among self-reported anxiety, behaviour, perceived, and actual physiology while partialing out social desirability scores did not change the pattern of significance of the correlations. These results indicate that individual differences in social desirability did not account for our findings.

We next examined the low correlations among response domains shown in Tables 2 and 3 more closely. One explanation for the low correlations is that nonlinear relationships among anxiety measures might represent a better fit for the data than the Pearson product-moment correlations we reported. However, performing logarithmic, quadratic, and cubic curve estimation using physiological measures as predictors and self-reported anxiety, anxiety behaviour, and perceived physiological measures as dependent variables allowed us to ascertain that nonlinear relationships did not better describe relationships among the measures (all *p*s > .11).

We also tested the alternative hypothesis that physiological responding during certain shorter key periods within the speech (e.g., during maximal activation or at the end of the speech) would cohere more tightly with anxiety experience, behaviour, or perceived physiological activation. To test this hypothesis, we divided the speech period into nine 20 second segments, calculated change scores from baseline for each segment, and performed multiple regressions, using those scores as the predictors, and anxiety experience, behaviour, and perceived physiological activation as the dependent

variables. After adjusting for familywise errors with the Bonferroni method, none of the resulting coefficients were significant. Thus, using shorter segments within the speech produced estimates of the coherence among response domains comparable to the ones based on the whole-speech period.

DISCUSSION

Our study examined links among emotion response components by inducing anxiety in participants selected to be either low (LTSA) or high trait socially anxious (HTSA). Results indicated that both LTSA and HTSA participants showed higher levels of anxiety experience, behaviour, perceived physiological responding, and actual physiological responding during the high anxiety period (giving a speech) than in the baseline period (film viewing). Also as predicted, compared to LTSA participants, HTSA participants showed greater increases in anxiety experience, anxiety behaviour, and perceived physiological activation from the baseline to the high anxiety period. There were two surprises, however. First, we found that despite their differences across the other response channels, low and high trait anxiety participants did not differ in their actual physiological responses during the high anxiety period. Second, we found that in both participant groups, correlations among response channels were modest at best. In particular, anxiety experience, behaviour, and perceived physiological responding, on the one hand, and actual physiological responding on the other, were not significantly correlated.

In the light of prior reports of physiological differences between low and high trait socially anxious participants (see Table 1), as well as expectations for greater response coherence during high anxiety, how can one make sense of our findings? It is of course possible that we failed to observe physiological differences between low and high trait socially anxious participants because our participants were poorly selected, our methods were insensitive, or our measures were incomplete. However, we think this is unlikely for a number of reasons. We selected only participants who scored in the top or bottom quartile of the SPAI distribution *and* were above or below the mean, respectively, on a speech-anxiety scale. Furthermore, we used strict exclusion criteria for participants whose anxiety reports were not stable over our two administrations of the SPAI. Our social challenge task was carefully selected to have high ecological validity. It has been widely used (Beidel et al., 1989b), and, replicating prior findings, we found large increases in indices of anxiety across domains for both groups. We also think it is unlikely that we either measured the wrong response systems, or measured the right response systems at the wrong times. After all, we assessed each of the major domains of emotional responding (experiential, behavioural, and physiological) during baseline, preparation, speech, and recovery, and took special pains to obtain a particularly wide array of physiological measures

continuously throughout the session. The physiological measures obtained also allowed us to exclude the possibility that group difference occurred in parasympathetic activation or in threat versus challenge patterns of physiological activation. We were further able to exclude a number of alternative explanations for our results, including effects of social desirability, nonlinear relationships among measures, and correlations among shorter segments of our measures.

Of course, participants were selected for their self-description of being anxious in a variety of social situations (the SPAI measures the psychological trait social anxiety), not on the basis of behavioural observation of signs of anxiety or a proven record of physiological responsivity in social situations. Because of this selection bias that favours one response system, one would expect that self-report during a social challenge test would be strongly consistent with this general self-description while responses in the other domains may be less indicative. Nevertheless, one would expect both behavioural and physiological domains to reflect the acute increase in self-reported anxiety during a social challenge in the trait socially anxious group. This was not the case in the present study.

It bears noting that the group differences in physiological responding found in prior studies of social anxiety were typically only relatively minor. Group differences in physiological responding were typically shown in only one measure (e.g., only systolic blood pressure or only finger pulse volume), only during one aspect of the task (e.g., only during anticipation, recovery, *or* habituation), or only between certain subgroups (e.g., physiological responders vs. cognitive responders). Importantly, these findings are often not consistent across studies, and even group differences that *were* shown more reliably are small in comparison to differences within groups as well as differences in self-reported measures. Finally, given the well known ''file-drawer'' problem, we suspect that null findings are under-represented in the published literature. These arguments lead us to believe that, consistent with the results of our study: (a) differences in physiological responding between low and high trait social anxiety groups are either nonexistent or very small; and that (b) coherence among response domains is low in social anxiety.

Implications for emotion theory

The lack of differences in physiological responding between LTSA and HTSA participants—together with the lack of association between perceived and actual physiological activation—leads us to reject a general theory of anxiety that postulates that increased levels of peripheral physiological responding always co-occur with higher levels of anxiety experience. More generally, while our focus on social anxiety does not allow for a test of all emotions, our findings do

permit us to reject an emotion theory that requires coherence between experience and physiology for all emotions and in all emotional situations. The fact that physiological activation increased from the baseline to the high anxiety speech in all participants leaves open the possibility that physiological activation might be necessary for the experience of anxiety, but such activation is clearly not sufficient to explain interindividual variation in anxiety experience.

Our results suggest that emotion responses across domains really are less tightly coupled in anxiety than prior analyses have led us to expect. Indeed, like Reisenzein (2000), we find no evidence of tight response coupling even at higher levels of emotion intensity. These findings are consistent with cognitive and social constructivist theories of emotion as well as with some evolutionary accounts (e.g., Averill, 1980; D'Andrade, 1984; Öhman, 1986; Smith & Ellsworth, 1985). According to cognitive and social constructivist theories, individuals experience a specific emotion only after interpreting an emotional situation and the perceived physiological activation evoked by it (e.g., Cacioppo, Berntson, & Klein, 1992; Rimé, Phillipot, & Cisamolo, 1990; Schachter & Singer, 1962). The perception of physiological symptoms might play a key role in emotional experience, but this perception is assumed to follow cognitive schemata of emotions and is not necessarily based on *actual* physiological responding (cf. Mandler, 1975; Sarason, 1985). This interpretation is supported by studies carried out by Pennebaker and colleagues. In several studies, they found that people, in particular women, base their judgements of physiological sensations on external signals, such as emotional slides, rather than on the actual physiological response (e.g., Pennebaker, 1981; Pennebaker & Roberts, 1992).

Our findings are also consistent with evolutionary accounts of phobias such as the one proposed by Öhman (1986). According to his theory, different types of fear might involve different evolutionary evolved systems, which are flexibly adapted to specific situational demands and appraisals. Social anxiety might involve activation of the '''submissiveness system'' to avoid psychosocial harm, such as humiliation, while specific phobias might involve activation of the ''predatory defence system'' to support effortful motor responses. Indeed, Öhman's findings indicate that there is less autonomic nervous system activation in social phobia as compared to animal phobias (see also Lang, Levin, Miller, & Kozak, 1983).

Implications for clinical science

Although our sample was drawn from a nonclinical population, we believe that our findings have several implications for clinical science. In this context, it is important to note that the social anxiety scores among our high trait anxiety participants were higher than those in clinical samples from

other studies (e.g., $M = 105.5$ in our sample vs. $M = 96.8$ in Beidel et al., 1989b).

Our findings are consistent with models of social anxiety that emphasise the role of cognitive processes. While some autonomic activation occurred for all participants when they became more anxious, it did not differentiate between low and high trait socially anxious individuals. Further, the present data indicate that perceived physiological responding is not related to actual physiological responding. Cognitive processes that are implied in the emotion generation in high anxious individuals include heightened self-focused attention, dysfunctional appraisal of social situations, and negative self-schemata (e.g., Clark & Wells, 1995; Hope, Gansler, & Heimberg, 1989; Leary & Kowalski, 1995). In particular, perception and appraisal of physiological activation might play a key role within the cognitive processes involved in social anxiety (e.g., Edelmann & Baker, 2002; Sarason, 1985).

One central question concerning this notion could be tested using our data. Although there were no differences in physiological reactivity between the anxiety groups, high trait socially anxious participants *reported* experiencing significantly higher levels of physiological responding during the speech than low trait socially anxious participants—see Figure 3, Panels (a) through (d). This could be the result of two different processes, which have quite different consequences for models of anxiety. On the one hand, higher reports of physiological activation in the high trait anxious group could be a function of more sensitive self-perception in this group as compared to the low trait anxious group (cf. Blascovich & Katkin, 1982; Reed, Harver, & Katkin, 1990). In this view, one might expect to see relatively high correlations between self-reported physiological activation and actual autonomic activation in high trait socially anxious individuals, whereas one should see relatively low correlations between self-reported physiological activation and actual autonomic activation in low trait socially anxious individuals. This pattern of correlations clearly was not found (see Table 3).

An alternative explanation is that high trait anxious participants' higher self-reports of physiological activation are based on subjective processes and distorted cognitive processing of physiological activation. Following this account, one would expect lower or equal correlations between self-perceived physiological activation and actual autonomic activation for high trait socially anxious individuals as compared to low trait socially anxious individuals. Our data are consistent with this explanation because correlations between perceived and actual physiological activation within the HTSA group are statistically equal to or lower than correlations within the LTSA group (see Table 3). Together, these findings lend support to the notion that anxious participants distort their own physiological responding in a stressful social situation (e.g., Derakshan & Eysenck, 1997; Edelmann & Baker, 2002; Mulkens et al. 1999; Steptoe & Vögele, 1992).

Limitations and future directions

We have stressed the design features of our study that give us confidence in its results. We are keenly aware, however, that any study has limitations, and ours is no exception. In the following section, we note five important limitations of our study, and suggest directions for future research.

One limitation of our study is that we used only female college students as participants. While some studies indicate that our core findings might generalise to other populations (e.g., Baggett et al., 1996; Panayiotou & Vrana, 1998; Puigcerver et al., 1989), there might also be important differences in emotional reactivity as well as coherence among response systems as a function of participants' age, sex, and culture (e.g., Grossman et al., 2001; Matsumoto & Kuppersbusch, 2001; Tsai, Levenson, & Carstensen, 2000). Although the response coherence view in its general form should hold for all groups, it will be important to assess more diverse samples in future studies.

A second limitation is that we focused exclusively on social anxiety. While it is true that our findings are consistent with results recently reported by Reisenzein (2000) for surprise, we believe that it is important to be careful when generalising to other emotional disorders and other emotions. For example, as mentioned above, Öhman (1986) showed different patterns of physiological activation in social anxiety as compared to specific phobias. In a similar vein, emotional disorders such as panic disorder, situational phobias, or major depression often show characteristic autonomic or respiratory abnormalities (e.g., Rottenberg, Wilhelm, Gross, & Gotlib, 2002; Wilhelm, Gerlach, & Roth, 2001; Wilhelm & Roth, 1998). Thus, studies of other emotions, subtypes of anxiety, and emotional disorders are needed to investigate the generalisability of our findings. If they do not generalise, theory-driven explanations of these differences could provide interesting insights into the nature of emotions.

A third limitation of our study follows from our decision to assess just two levels of anxiety (low levels using a neutral film, and high levels using an impromptu speech paradigm) rather than many instances of anxiety. We made this decision to provide continuity with prior research on anxiety, and also because we were not certain that we could elicit sustained, high levels of anxiety in an ethical manner in our study participants. However, this design decision had two downsides. First, it allowed us to assess self-reported anxiety only retrospectively, after each 3 minute task. Although each rating of anxiety was made immediately after each task, this method might have introduced biases relative to more frequent or online ratings (cf. Feldman Barrett, 1997; Fredrickson & Kahneman, 1993). Second, our design did not allow us to assess the question of whether *within a given subject* there was a positive association among response components across trials. As Ruch (1995) and Reisenzein (2000) have noted, this within-subjects design is often more sensitive than the between-subjects design that we employed, and coherence among emotion components might be

higher when performing within-participants analyses (e.g., Pennebaker, 1982; Reisenzein, 2000; Ruch, 1995). The fact that our findings are consistent with those of other investigators who have used precisely such within-subjects designs increases our confidence in our findings (e.g., Edelmann & Baker, 2002). However, while a between-participants approach is important in its own right (cf. Buck, 1979; Underwood, 1975), additional within-participants data with higher time resolution are needed for a more complete understanding of the coherence of emotional components in anxiety.

A fourth limitation concerns the response domains we assessed. Our findings show just how important it is to adopt a multimethod approach to emotion assessment. As we have seen, response indicators that one might expect to cohere (e.g., perceived heart rate and actual heart rate) do not necessarily converge as expected. Although we broadly sampled from a variety of response domains, we did not assess ongoing attentional shifts and cognitive activity, in part because we worried about the interference such an assessment might require. However, it will be important in future studies to more specifically test *which* cognitive processes are involved in the experience of anxiety. A next step might involve assessing how high trait anxious individuals differ in cognitive processes from low-anxious individuals. Clinical studies have already approached this problem suggesting that high trait socially anxious individuals do in fact differ from low trait socially anxious individuals for example in heightened self-focused attention, fearful evaluation of the self and others, and the presence of negative cognitive schemata (e.g., Asmundson & Stein, 1994; Clark & Wells, 1995; Maddux, Norton, & Leary, 1988). Brain imaging studies might additionally be useful to disentangle cognitive processes involved in normal and pathological emotional processing (e.g., Davidson, Marshall, Tomarken, & Henriques, 2000; Ochsner, Bunge, Gross, & Gabrieli, 2002).

A fifth limitation is that although we manipulated anxiety, changes in physiological activation were allowed to naturally vary with other response components. Our data are consistent with the interpretation that high trait socially anxious participants base their ratings on cognitive processes and possibly misperceive and exaggerate their physiological activation (e.g., Mulkens et al., 1999; Steptoe & Vögele, 1992). However, more stringent tests of this hypothesis are needed. An alternative explanation for the group differences we reported could be that low trait socially anxious participants *underestimate* their physiological activation as compared to high anxious participants. This explanation is consistent with some prior studies indicating that more accurate perception of physiological responding might intensify emotional experience (e.g., Wiens, Mezzacappa, & Katkin, 2000). To address this possibility, more studies that experimentally manipulate physiological activation and identify how and where bodily states are represented in the brain are needed (e.g., Critchley, Mathias, & Dolan, 2001).

Concluding comment

Emotions have long been conceptualised by lay people, philosophers, and social scientists alike as being embodied, that is, as being accompanied by bodily changes (e.g., Damasio, 1999; James, 1884; Kövecses, 2000). Emotion theorists have elaborated on this view, and the notion that emotions represent patterned changes across experiential, behavioural, and physiological response systems has gained widespread currency. Surprisingly, evidence for this view has been limited, even in the context of emotions, such as anxiety, which seem to hold promise for showing clear and compelling correspondences between how we feel and how our body responds. Is this discrepancy between emotion theory and empirical findings the result of *theoretical overestimation* of coherence or *empirical underestimation* of coherence? In the case of social anxiety, at least, it appears that our theories may need to be adjusted to accommodate mounting evidence of limited associations between anxiety experience, anxiety behaviour, and perceiving physiological activation, on the one hand, and actual physiological activation on the other.

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