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Exaggerated perception of normal physiological responses to stress and hypercapnia in young women with numerous functional somatic symptoms

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Abstract

Objective: This study tested whether functional somatic symptoms are associated with exaggerated increases in self-reported anxiety and somatic complaints in response to stress and CO_2 -enriched air breathing, and whether this association exists in parallel to or in the absence of exaggerated physiological responses. **Methods:** Out of 499 young somatically healthy undergraduate women, 18 participants high in functional somatic symptoms (HSS group) and 18 participants low in symptoms (LSS) were selected. They were submitted to mental stress, mild physical exercise and relaxation during conditions of normal breathing, breathing compressed normal air, and breathing compressed 5% CO_2 -enriched air. In all

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Introduction

Individuals reporting numerous functional somatic symptoms¹ have been frequently described in clinical psychology [1-5]. Two main theoretical frameworks currently exist to explain functional somatic symptoms: one that is primarily physiological and one that is primarily psychological.

The physiological framework is purporting that functional somatic symptoms reflect an underlying biological disposition for exaggerated physiological responses to stress. Indeed, respiratory and autonomic abnormalities have repeatedly been found in subjects with anxiety and psychoconditions, self-reported anxiety and somatic symptoms and respiratory and autonomic responses were assessed. **Results:** HSS participants reported, as compared to LSS, more tenseness, anxiety, and somatic symptoms at baseline and increased responses to mental stress and during 5% CO₂ breathing, but not in response to exercise. However, no evidence was found for a corresponding exaggerated respiratory or autonomic response. **Conclusion:** A young, female, and nonclinical population with numerous functional somatic symptoms and high levels of anxiety is characterized by an exaggerated perception of a normal physiological response.

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somatic disorder [6-13]. Based on this framework, a scenario may be outlined of exaggerated physiological stress reactivity as a primary agent in the aetiology of functional somatic symptoms. In this scenario, the perception of the physiological responses to stress, for instance, sensations of increased respiration or autonomic arousal, may cause (or at least enhance) functional somatic symptoms. Put otherwise, individuals plagued by functional somatic symptoms may correctly perceive an exaggerated physiological response that is misinterpreted as physical illness. If such vulnerable subjects are frequently exposed to mental and physical stressors, the repeated sensation of physiological arousal may blur into the chronic experience of somatic symptoms that characterizes individuals reporting numerous functional somatic symptoms.

Alternatively, the psychological framework is purporting that functional somatic symptoms are associated with enhanced tendencies of anxious and distressed individuals

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¹ The term *functional somatic symptoms* refers to psychosomatic complaints that (after medical examination) cannot be explained by a somatic disorder. See Appendix for a list of the specific symptoms used.

to focus their attention on bodily sensations and to appraise these in a negative and catastrophic manner [3,7,14–16]. Such misperception may occur most often in high trait anxious individuals and may even be a core element of anxiety. High levels of functional somatic symptoms have been associated with negative affectivity [3,14,15]. Based on the psychological framework, functional somatic symptoms may arise exclusively from an exaggerated perception of a normal physiological response. Thus, instead of normal perception of an exaggerated physiological response, individuals with functional somatic symptoms may suffer from an exaggerated perception of a normal physiological response.

The two different theoretical frameworks can be reconciled by adding the element of time. A young and nonclinical population with high levels of anxiety may be characterized by normal physiological responses to stress but perceive these responses to be excessive and, as a consequence, report more somatic symptoms. In due course, mental and physical stressors may actually start to evoke exaggerated and even altered reactivity due to the added anticipatory anxiety of experiencing somatic symptoms. This scheme fits the general idea in stress theory that inadequate coping tends to increase physiological arousal in response to stressors.

Emphasis so far has been on the stress-related increase in respiratory activity as a possible explanatory mechanisms for functional somatic symptoms [6-13]. Hyperventilation, however, may have been overrated as a source of functional somatic symptoms [12,17,18] at the cost of attention to stress-related autonomic responses. Withinsubject psychophysiological human studies have demonstrated that mental [19-25] and physical stress [26] usually decrease respiratory sinus arrhythmia (RSA) as an index of (parasympathetic) vagal control of heart rate, whereas increased RSA is associated with conditions of relaxation [27]. Additionally, mental stress usually decreases the pre-ejection period (PEP) as an index of the (sympathetic) cardiac inotropic drive [22,24,25]. Shortterm decreases in RSA and PEP are regarded characteristic features of the fight-flight stress response [22-25]. Clinical studies have further demonstrated that chronically low RSA and/or an excess in task-related decreases in RSA are associated with anxiety [28-30] and depression [31,32]. Thus, both respiratory and autonomic reactivity to mental or physical stress may be exaggerated in individuals with functional somatic symptoms.

The aim of the present study was to test whether individuals high in functional somatic symptoms have exaggerated respiratory or autonomic reactivity to mental stress or mild physical exercise. Additionally, hypercapnic conditions (acidosis stress induced by inhalation of a CO_2 enriched air mixture) were included. Hypercapnia has been reported to evoke more symptoms in psychosomatic patients [33], and, to evoke deviant respiratory [34] and autonomic [35] reactivity in panic patients with related symptoms.

A young and nonclinical sample of women with numerous recent functional somatic symptoms was selected from a large group of somatically healthy undergraduates and contrasted to young female undergraduates without any such symptoms. Self-reported symptoms related to respiratory or autonomic irregularities, heart period, RSA, PEP, respiratory rate and depth, and end-tidal partial pressure of CO₂ (PetCO₂) responses to mental stress and mild physical exercise (as compared to relaxation) were measured during conditions of normal breathing, breathing compressed normal air, and breathing compressed CO₂-enriched air. The hypotheses tested were (1) whether individuals with numerous functional somatic symptoms are characterized by exaggerated increases in self-reported anxiety and somatic symptoms in response to stress, exercise, or 5% CO_2 breathing and (2) whether this occurred in parallel to or in the absence of exaggerated respiratory or autonomic responses.

Methods

Participants

Women with numerous recent functional somatic symptoms (n=18) and women without such symptoms (n=18), all without chronic disease, were recruited from a sample of 499 undergraduate psychology students. Participants were selected that scored equal or below the 20th percentile (value 9) and that scored equal or above the 80th percentile (value 26) on the Hyperventilation Symptom Questionnaire (HSQ, see below) during a group test session. All of the selected individuals that agreed to participate were invited and they completed this questionnaire (for a second time) during the experimental procedure; and all of these participants remained below (low in somatic symptoms, or LSS group) versus above (high in somatic symptoms, or HSS group) the normative median (value 16). All participants underwent spirometry to avoid asthma and airflow obstruction. The study was presented to them as an investigation of breathing patterns. The participants were told they could win up to 100 Dutch guilders (\$40), but none of them did. Nonetheless, they all received a similar amount of 30 Dutch guilders (\$12) after the experiment. All participants signed an informed consent. The study had been approved by the Ethics Committee of the Department of Psychology, University of Amsterdam. None of the participants used medication excepting oral contraceptives in 25 women. The participants were instructed to refrain from eating, drinking (except for water), smoking, or physical exercise within 1 h before the experiment. Technical problems resulted in the loss of physiological data from one participant.

Questionnaires

A Dutch version of the HSQ was used to select participants with numerous recent functional somatic symptoms and participants very low in such symptoms. This questionnaire has 31 items (see Appendix) and assesses the frequency of symptoms during the past month. Ratings were made on a four-point scale (range 0-3) comprising the categories "did not occur," "one or more times a month," "one or more times a week," and "daily." For the purpose of this study, we omitted the three psychological symptoms, leaving 28 somatic symptoms (see Appendix). The total score ranges from 0 to 84 points.

A Dutch translation of the Spielberger State–Trait Anxiety Inventory (STAI) was used to measure trait anxiety [36,37]. The trait questionnaire has 20 items, and the total score ranges from 20 to 80 points.

Mental stress, relaxation, and exercise manipulations

The program used for the mental stress task was running on an MS-DOS computer. An intelligence test and a reaction-time task were simultaneously presented by this program. Intelligence test questions were presented one by one in the middle of the screen. The maximum time for each question was 60 s and the elapsed time was visible on the screen. The participants were instructed to select one of five multiple-choice responses (1-5) and to press the corresponding key on the keyboard. The reaction-time task consisted of random-timed falling red and green coins on the left and right sides on the screen. The participants were instructed to press the left button (located at the left side of the keyboard) when a green coin was falling on the left side and to press the right button (located at the right side of the keyboard) when a green coin was falling on the right side. The computer acknowledged each response (or lack of response) with a brief auditory signal: a musical tone indicating a correct response and a low-frequency buzz indicating error. The combined score on intelligence and reaction-time tasks was expressed in Dutch guilders on the screen. The initial amount was 100 Dutch guilders (\$40), which gradually diminished as a result of the errors made. Real bank notes were placed in front of the participants before the task started, and withdrawn when lost. Two research assistants observed the participants and their performance at close distance to increase the stressfulness of the task. For ethical reasons, participants were debriefed regarding the true reason of this task immediately after it was finished.

Participants were instructed to sit quietly and read a magazine for the relaxation task. No research assistants were present in the experimental room during this task. The program used for the exercise task was also running on the MS-DOS computer. The participants were instructed to cycle on a bicycle home-trainer, which was set at minimal resistance, while watching the computer screen. A feedback procedure was used to ensure that the same increase in heart rate was obtained (for each participant) during exercise as during mental stress. The participants were instructed to cycle faster or slower in such a way that the top of the bar on the screen was as close as possible to a set-point indicated by a line. The height of the bar represented their mean heart rate over the previous 10 s, and it was updated every 4 s. Participants were kept unaware that the height of the bar reflected their current heart rate, and that the line reflected their (previous measured and saved) mean heart rate during the corresponding part of the mental stress task. The participants' body posture during this exercise task was fairly similar to their posture during the mental stress and relaxation task.

Compressed normal air and 5% CO₂-enriched air breathing

Compressed normal air and CO2-enriched air were stored in two cylinders, which were located in a room adjacent to the experimental room. One cylinder contained medical air and the other a mixture of medical air and CO₂. Each cylinder had its own flow regulation as well as a moisturizing device. The air flow from both cylinders was connected by a T-piece to a single silicon tube with an inner diameter of 7 mm, and a length of 4 m, of which 1 m came out in the experimental room. This end was fed into a silicon air reservoir, in turn connected (via a silicon tube of 32-mm inner diameter and a length of 50 cm) to a silicon half-face mask (Dräger Combitox Nova RA). This nonleaking mask, commonly used among fire workers, had two valves that separated incoming and exhaled airflow. The flow of both cylinders could be adjusted to create a part with compressed normal air and a part with a compressed air mixture with 5% CO₂.

Self-reported somatic symptoms and tenseness-anxiety

The symptoms breathlessness, dizziness, nausea, heart pounding, tenseness, and anxiety were selected as symptoms related to respiratory or autonomic irregularities and/or that will most likely be experienced as a result of CO₂enriched air inhalation [33]. These symptoms were measured (repeatedly) on seven-point scales, ranging from 1 (*not at all*) to 7 (*very much*). The words were presented with the MS-DOS computer, and participants had to rate the degree they experienced the presented symptom (at that moment) and they had to type the corresponding number on the keyboard. The scores for breathlessness, dizziness, nausea, and heart pounding were pooled and named "somatic symptoms," the scores for tenseness and anxiety were pooled and named "tenseness–anxiety."

Physiological recordings

Interbeat intervals (IBIs, as measure of heart period), systolic time intervals [38], respiratory rate, and a raw estimate of changes in respiratory depth (tidal volume) were measured with the Vrije Universiteit Ambulatory Monitoring System (VU-AMS version 4.3, TD-FPP, Vrije Universiteit, Amsterdam, The Netherlands). This device uses six Ag/AgCl electrodes to record the electrocardiogram and thoracic impedance (dZ). Details on the measurement procedure with the VU-AMS can be found in de Geus et al. [39] and Willemsen et al. [40].

The arterial partial pressure of CO_2 was estimated (to assess hypo- or hypercapnia) by measuring the PetCO₂ in the exhaled air at the end of a normal expiration. This was measured with the Capnogard etCO₂ Monitor (Novametrix, Wallingford, CT, USA) and expressed in millimeters mercury (mm Hg). A small tube was inserted in each of the participants' nostrils.

Procedure

All experimental sessions took place between 11 a.m. and 4 p.m. and lasted approximately 2.5 h. Experimental tasks took place in an experimental room that was sound shielded and dimly lit. Waiting periods took place in an adjacent waiting room. After general instructions, the recording electrodes were attached and connected to the VU-AMS. Next, the experiment consisted of three 15-min waiting periods and three experimental tasks that were conducted in fixed order: (1) waiting, (2) the mental stress task, (3) waiting, (4) the relaxation task, (5) waiting, and (6) the mild physical exercise task. The three experimental tasks consisted of three parts of 4 min each, again conducted in fixed order: (a) breathing normally, (b) breathing compressed normal air through the face mask, and (c) breathing compressed 5% CO2-enriched air through the face mask. Each time after entering the experimental room, the VU-AMS was connected to the MS-DOS computer and the participants were attached to the PetCO₂ recording equipment. They were disconnected for each waiting period. Participants had to rate their experienced symptoms at the onset of each experimental task and after each part (i.e., 4 times during each task, 12 times in total). After the

Smoking, sporting, oral contraceptive use, age, height, weight, HSQ, and STAI-Trait scores in LSS and HSS individuals

	LSS $(n=18)$	HSS $(n=18)$	Significance
Percentage			
Smoking	44.44	55.56	ns
Sporting	44.44	27.78	ns
Oral contraceptives	66.67	72.22	ns
Mean (S.D.)			
Age	20.61 (2.43)	19.78 (1.22)	ns
Height	170.50 (6.48)	174.17 (6.11)	ns
Weight	63.50 (6.93)	64.78 (8.54)	ns
HSQ	7.61 (4.15)	33.28 (9.35)	***
STAI-Trait	31.17 (5.06)	48.94 (9.68)	***

ns=not significant.

*** P<.001 (two-tailed).

Table 2

Mean (S.D.) values for the self-reported somatic symptoms, tensenessanxiety, and the physiological variables at baseline (i.e., the relaxation, no-mask condition) for LSS and HSS individuals

	Mean (S.D.)		
	LSS	HSS	Significance
Somatic symptoms (scale 1–7)	1.03 (0.00)	1.54 (0.92)	**
Tenseness-anxiety (scale 1-7)	1.08 (0.19)	1.78 (1.00)	**
IBI (s)	0.91 (0.15)	0.92 (0.11)	ns
PEP (ms)	91.20 (9.03)	87.06 (4.67)	ns
RSA (HF power in $\log ms^2$)	3.23 (0.39)	3.32 (0.34)	ns
Respiratory rate (Hz)	0.25 (0.04)	0.24 (0.04)	ns
Respiratory power (AU)	0.62 (0.27)	0.52 (0.28)	ns
PetCO ₂ (mm Hg)	36.90 (2.38)	36.47 (1.93)	ns

ns=not significant.

** P < .01 (two-tailed).

exercise task was finished, all equipment was disconnected, electrodes were removed, participants were debriefed, paid, and sent home.

Data analysis

The heart period data of each participant were analyzed in segments representing 128 s. An artifact preprocessing was performed on the IBI data by detecting outliner IBI values with three methods: (a) by absolute values (>1800 or <300 ms), (b) a moving average filter (>3 S.D. from the moving mean), and (c) by visual inspection. Since artifacts cannot simply be deleted because the continuity of time would be lost, spuriously short IBIs were summed and missing beats were "created" by splitting spuriously long IBIs. The IBI mean values were computed from these corrected data. Next, uniformly spaced samples were created and the segments were discrete Fourier transformed. Heart period power values were computed for the high frequency (HF) band (0.125-0.5 Hz). Changes in these HF power values were used to estimate changes in RSA. The power values were log¹⁰ transformed to obtain normal distributions.

The thoracic impedance (dZ) data (sampled at 10 Hz) were band-pass filtered by a discrete wavelet transform filter with a cubic spline function as base (0.125-0.5 Hz). Next, the respiratory power values were computed from this filtered thoracic impedance (dZ) data by computing the variance of this filtered time series. Changes in the respiratory power values were used as a (raw) estimation of changes in respiratory depth (tidal volume). The respiratory power values were also \log^{10} transformed to obtain normal distributions. The mean respiratory rate values were estimated from the band pass filtered thoracic impedance (dZ) data by counting the number of up-going zero crossings and dividing this value by the time of a segment. This procedure is comparable to the method

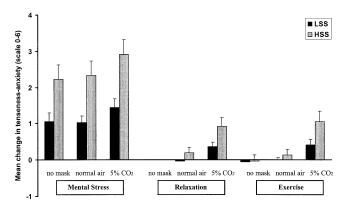


Fig. 1. Group differences in mean \pm S.E.M. change values (related to the relaxation, no-mask condition) for tenseness–anxiety in response to the tasks and the breathing manipulations between LSS and HSS individuals.

0,1 LSS BHSS Mean change in interbeat interval (s) -**n** --0,2 -0,3 mask normal air 5% CO no mask normal air 5% CO: no mask normal air 5% CO: Mental Stress Relaxation Exercise -0,4

Fig. 3. Group differences in mean \pm S.E.M. change values (related to the relaxation, no-mask condition) for IBI in response to the tasks and the breathing manipulations between LSS and HSS individuals.

used by de Geus et al. [39] who computed the mean total respiratory cycle time as the mean interval between the initiating moments of inspiration.

The dZ/dt values (sampled at 250 Hz around each Rwave) were ensemble averaged over 60 s. The B-points were manually determined for each ensemble averaged segment, and the PEP values were determined by summing a fixed Q-to-R interval of 48 ms to the R–B interval time. The 1-min ensemble averaged PEPs were pooled over two succeeding values to obtain a value for each two min period, similar to the other measures.

For each measure, nine repeated observations were available for each participant (three tasks with three different breathing parts of 4 min). Baseline differences between the groups were tested for the relaxation task during normal breathing (i.e., the conditions without breathing through the face mask). Next, within-subject effects of task (3), breathing manipulation (3), and interactions with group (2) were tested with repeated measures MANOVA tests using Wilks' lambda. Main effects of either task or breathing manipulation were followed by post hoc tests of all three possible contrasts.

Results

Group differences for the background variables

No significant group differences were found for smoking, exercise behavior, use of oral contraceptives, age, height, or weight (see Table 1). However, participants of the HSS group scored (as expected) significantly higher on the HSQ, and they also scored significantly higher on the STAI-Trait. A significant and high correlation was found between the HSQ and STAI-Trait scores (r=.86, n=36, P<.001).

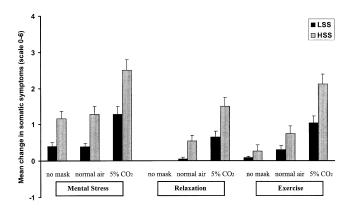


Fig. 2. Group differences in mean \pm S.E.M. change values (related to the relaxation, no-mask condition) for somatic symptoms in response to the tasks and the breathing manipulations between LSS and HSS individuals.

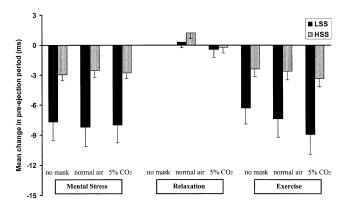


Fig. 4. Group differences in mean \pm S.E.M. change values (related to the relaxation, no-mask condition) for PEP in response to the tasks and the breathing manipulations between LSS and HSS individuals.

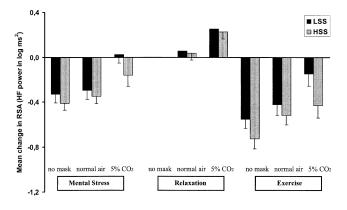


Fig. 5. Group differences in mean \pm S.E.M. change values (related to the relaxation, no-mask condition) for RSA (HF Power) in response to the tasks and the breathing manipulations between LSS and HSS individuals.

Group differences for self-reported somatic symptoms and tenseness–anxiety

Table 2 displays mean values of the self-reported somatic symptoms and tenseness–anxiety at baseline (i.e., for the relaxation task during normal breathing). Figs. 1 and 2 show the changes over these baseline values in response to the various experimental tasks.

The HSS participants reported, as expected, significantly more baseline somatic symptoms [t(17.45)=3.08, P<.01] and tenseness–anxiety [t(18.24)=2.88, P<.01].

A significant task by group interaction was found for somatic symptoms [F(2,33)=4.92, P<.05] and tenseness–anxiety [F(2,33)=3.49, P<.05]. Follow-up tests revealed that the HSS participants reported significantly more somatic symptoms and tenseness–anxiety than LSS participants during mental stress as compared to relaxation [F_{som} sym(1,34)=10.11, P<.01; $F_{\text{ten-anx}}(1,34)=7.12$, P<.01], and they reported significantly more somatic symptoms and tenseness–anxiety somatic symptoms and tenseness–anxiety more somatic symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to relaxation symptoms and tenseness–anxiety during mental stress as compared to symptoms and tenseness–anxiety during mental stress as compared to symptoms and tenseness–anxiety during mental stress as compared to symptoms and tenseness–anxiety during mental stress as compared to

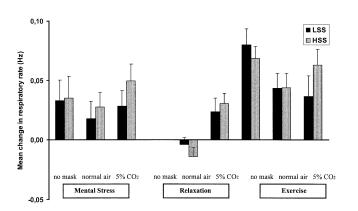


Fig. 6. Group differences in mean \pm S.E.M. change values (related to the relaxation, no-mask condition) for respiratory rate in response to the tasks and the breathing manipulations between LSS and HSS individuals.

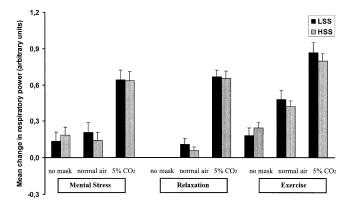


Fig. 7. Group differences in mean \pm S.E.M. change values (related to the relaxation, no-mask condition) for respiratory power in response to the tasks and the breathing manipulations between LSS and HSS individuals.

to exercise [$F_{\text{som sym}}(1,34) = 4.19$, P < .05; $F_{\text{ten-anx}}(1,34) = 5.98$, P < .05].

A significant breathing manipulation by group interaction was found for somatic symptoms [F(2,33)=5.89, P<.01]. Follow-up tests revealed that HSS participants reported significantly more somatic symptoms than LSS participants during compressed 5% CO₂-enriched air breathing as compared to normal breathing [F(1,34)=9.67, P<.01] and during compressed 5% CO₂-enriched air breathing as compared to compressed normal air breathing [F(1,34)=4.53, P<.05]. Additionally, HSS participants reported significantly more somatic symptoms than LSS participants during compressed normal air breathing as compared to normal breathing [F(1,34)=8.27, P<.01].

Group differences for the physiological variables

Table 2 displays mean values of the physiological variables at baseline (i.e., for the relaxation task during normal

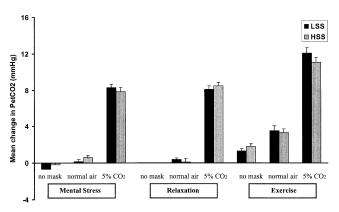


Fig. 8. Group differences in mean \pm S.E.M. change values (related to the relaxation, no-mask condition) for PetCO₂ in response to the tasks and the breathing manipulations between LSS and HSS individuals.

Table 3 Task and breathing manipulation effects

		F	Significance		F	Significance
	IBI			PEP		
Task manipulation		134.03	***		20.12	***
Relaxation-stress	Ţ	129.04	***	Ļ	40.17	***
Relaxation-exercise	ļ	200.67	***	ļ	31.50	***
Exercise-stress	1 1	56.38	***	_	_	ns
Breathing manipulation		13.75	***		_	ns
No-mask–normal air	Ļ	6.01	*	-	_	ns
No-mask-5% CO ₂	Ļ	27.14	***	-	_	ns
Normal air-5% CO ₂	Ļ	13.82	**	-	-	ns
	HF power			Respirato	ry rate	
Task manipulation	-	50.02	***	-	33.38	***
Relaxation-stress	\downarrow	58.79	***	↑	10.53	**
Relaxation-exercise	\downarrow	102.82	***	1	66.85	***
Exercise-stress	1	41.06	***	\downarrow	12.06	***
Breathing manipulation		30.71	***		12.45	***
No-mask-normal air	1	13.30	***	\downarrow	16.31	***
No-mask-5% CO ₂	1	62.64	***	_	-	ns
Normal air -5% CO ₂	Ť	31.53	***	\downarrow	14.15	***
	Respirato	ry power		$PetCO_2$		
Task manipulation	-	30.57	***		55.64	***
Relaxation-stress	_	-	ns	_	-	ns
Relaxation-exercise	Î	51.16	***	↑	105.77	***
Exercise-stress	Ļ	23.46	***	Ļ	91.07	***
Breathing manipulation		173.14	***		734.37	***
No-mask-normal air	1	39.06	***	1 1	49.44	***
No-mask-5% CO ₂	1	339.38	***	1	1262.53	***
Normal air-5% CO ₂	Ť	308.36	***	\uparrow	1473.44	***

ns=not significant.

* P < .05 (two-tailed).

** P<.01 (two-tailed).

*** P<.001 (two-tailed).

breathing). Figs. 3-8 show the changes over these baseline values in response to the various experimental tasks.

The groups did not differ significantly on any of the expected physiological values at baseline, and the expected interaction effects of Group × Task and Group × Breathing Manipulation were also not found. Note that although the figure depicting change in HF power suggests a trend for reduced values for the HSS participants during mental stress and mild physical exercise while breathing 5% CO₂-enriched air, the MANOVA test did not yield significance. The only significant interaction involving group was a group by task effect for PEP [F(2,32)=3.82, P < .05]. Follow-up tests revealed an unexpected direction: HSS participants had significantly less PEP reduction than LSS participants during mental stress [F(1,33)=7.81, P < .01] and exercise [F(1,33)=5.38, P < .05] as compared to relaxation.

Task and breathing manipulation effects

The absence of the expected group effects for the physiological variables did not reflect a failure to obtain effects of task or breathing manipulations per se. The direction and significance of task and breathing manipulation effects are summarized in Table 3. Significant task and breathing manipulation effects were found for IBI, HF heart period variability power, respiratory rate, respiratory power, and PetCO₂.

Discussion

Young, nonclinical female participants with numerous functional somatic symptoms showed significantly higher self-reported somatic symptoms and tenseness-anxiety than female participants with only few such symptoms. These group differences in self-reported somatic symptoms and tenseness-anxiety sharply increased during mental stress and during 5% CO2-enriched air breathing, but not during exercise. However, no clear group differences were apparent for the heart period (IBI), RSA, PetCO₂, respiratory depth and respiratory rate at baseline, or in response to mental stress, exercise, or 5% CO2-enriched air breathing. The results of the current study clearly indicate that numerous functional somatic symptoms and high levels of anxiety, suggestive of hyperventilation, are not necessarily characterized by respiratory or autonomic abnormalities at baseline or by exaggerated respiratory or autonomic

responses to stressors. The increase in self-reported functional somatic symptoms related to respiratory or autonomic irregularities must have been a result of a differential *perception* of physiological reactivity because, as was shown here, respiratory or autonomic reactivity was not excessive itself. It is, therefore, concluded that a young, female, and nonclinical population with numerous functional somatic symptoms and high levels of anxiety is characterized by an exaggerated perception of a normal physiological response.

Our results replicate and extend similar findings by Wientjes and Grossman [9] and Wientjes et al. [10-12], who studied psychological and physiological factors in young nonclinical males high in functional somatic symptom reporting. Their results indicated that trait anxiety explained nearly one-third of the symptom variance, whereas only 4% was explained by a physiological factor: a relatively small reduction in partial CO₂ pressure at rest [9]. In another study, autonomic and respiratory responses were compared in response to a mental task and moderate physical exercise [12]. Again, a small reduction in partial CO₂ pressure values was found during baseline conditions (rest), which was within normocapnic limits, but differences in autonomic and respiratory responses to stressors from a control group were not found or very small. Although the reduction in partial CO₂ pressure at rest as observed by Wientjes et al. [12] was not found in our study, their conclusion that little evidence is available to support the idea that the symptoms are a direct consequence of (stressinduced) hyperventilation in young, nonclinical men can now be extended to young, nonclinical women.

The absence of baseline and stress-related aberrations of autonomic and respiratory responses for individuals high in functional somatic symptoms is entirely consistent with the results of some recent psychophysiological studies that have failed to find (general) physiological correlates of anxiety and social phobia under socially threatening situations, despite increases in reported anxiety and somatic complaints [41,42]. Nonetheless, our results run against a large body of research suggesting respiratory and autonomic abnormalities in panic and anxiety patients. Papp et al. [43], Klein [44], Coryell et al. [34], and Bystritsky et al. [35] have reported respiratory and autonomic abnormalities in a clinical population of panic patients with similar symptoms. Although the participants of our study were much younger and not selected from a clinical population, they reported high scores on the used HSQ similar to the scores of the patients measured by Hornsveld et al. [17]. They also experienced a pronounced increase in somatic symptoms during the tasks and air mixture manipulations of the current study, significantly above that of the control group. Thus, although the conclusions based on the results of the current study are limited to our particular population of young, undergraduate, nonclinical, and female subjects, these results do (in line with other studies) demonstrate that exaggerated respiratory or autonomic reactivity does not

necessarily accompany exaggerated stress-induced functional somatic symptoms and anxiety. They leave open the possibility, therefore, that aberrant respiratory or autonomic functioning is not primary to anxiety disorders, but instead may develop as a consequence of these disorders.

Our study confirms the relation between self-reported somatic symptoms and the tenseness-anxiety ratings. The scores on the HSQ were also highly correlated with scores on the trait anxiety questionnaire. The association between anxiety and functional somatic symptoms is attributed to attention, selective search for information or perceptual learning [14]. For example, it has been demonstrated that highly anxious individuals attend more to bodily symptoms, and that attention to bodily symptoms increases the perceived intensity of these symptoms [14,15,45,46]. Thus, increased anxiety and tenseness of the participants with numerous functional somatic symptoms may have resulted in increased awareness of physiological reactivity, and as a result in increased self-reported symptoms.

An unexpected reduction in PEP reactivity to stress was found for participants with numerous functional somatic symptoms compared to the control group. Such reduced PEP reactivity may reflect an increased tendency to respond to stress with increases in peripheral vascular resistance, which was shown to be a stable individual trait [47]. Increased peripheral vascular resistance may mask a true increase in cardiac sympathetic drive by attenuating PEP reactivity through afterload effects [48]. Alternatively, participants with numerous functional somatic symptoms may be characterized by an attenuation of beta-adrenergic influences on the heart. Bum-Hee et al. [49] demonstrated that tension-anxiety ratings (measured by the Profile of Mood States) are negatively correlated with beta-receptor density, even in patients who do not have psychiatric illness. Possibly, low beta-receptor density also characterizes individuals with numerous functional somatic symptoms. Exploration of this intriguing idea must first await independent replication of the present results in a study that can rule out effects of afterload (which we could not do in this study blood pressure was not measured).

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Appendix

The symptoms of the HSQ are muscle cramps, muscle stiffness, stiffness around the mouth, sudden feeling of muscle weakness, trembling, dizziness, blurred vision, headaches, faintness, paresthesias, warm feeling in the head, hot flashes, cold hands or feet, shivering, inability to take a deep breath, tightness in the chest, lump in the throat, sudden fast or deep breathing, breathlessness, rapid heartbeat, pounding heart, irregular heartbeat, chest pain, abdominal pain or cramps, bloated stomach, tiredness, sweating, nausea (i.e., the additional psychological symptoms unrest/tension, anxiety/panic and feelings of unreality were omitted).

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