Published in final edited form as:

Psychosom Med. 2002 ; 64(2): 294–301.

Defensiveness and Individual Response Stereotypy in Asthma

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Abstract

Objective—Previous literature has shown that the psychological trait of defensiveness is related to elevated sympathetic reactivity to stress and to several cardiac risk factors. The aim of this study was to examine whether these previous findings on defensiveness extend to an asthmatic population.

Methods—Defensiveness was measured by the Marlowe-Crowne Social Desirability Scale using a quartile split: high (upper 25%) and low (bottom 75%). Twenty-two defensive and 66 nondefensive participants with asthma were exposed to laboratory tasks (initial baseline rest period, reaction time task, and a shop accident film).

Results—During the tasks there was evidence of lower skin conductance levels and greater respiratory sinus arrhythmia amplitudes among defensive patients with asthma. After exposure to the tasks, defensive patients with asthma showed a decline on spirometry test measures compared with nondefensive asthmatic patients, who displayed an increase.

Conclusions—These data confirm individual response stereotypy and suggest that defensiveness may be characterized by sympathetic hypoarousal and parasympathetic hyperarousal among patients with asthma. Future studies are needed to determine whether defensiveness is a risk factor for stress-induced bronchoconstriction.

Keywords

defensiveness; asthma; stress; pulmonary function; respiratory sinus arrhythmia; skin conductance level

INTRODUCTION

“Individual response stereotypy” (1) refers to a pattern that occurs among some individuals who tend to react to various kinds of stress with increased activation in a specific physiological system. It is also a hypothesized risk factor for stress-related disease. For example, individuals with a parental history of hypertension demonstrate exaggerated cardiovascular reactivity to stress (2). There is evidence that some people with asthma may display individual response stereotypy in the respiratory system in that they, but not healthy subjects, have been found to react to distressing movies with bronchoconstriction (3–6). Furthermore, patients with asthma tend to show a unique autonomic response pattern that is characterized by diminished sympathetic activity during stress (6) and acute exacerbation of asthma (7). In general, people

with asthma display attenuated β -adrenergic sensitivity and hyperreactive cholinergic responsiveness (8).

The relationship between stress and decline in pulmonary function in asthma may be mediated cholinergically (9). In one study, children with asthma who demonstrated increased airway reactivity to methacholine showed greater physiological and emotional reactions while viewing the “sad” scenes in a movie and decreased pulmonary function test values after the film (4). Respiratory sinus arrhythmia (RSA), a vagally mediated oscillation in heart rate, has been shown to be elevated in patients with asthma (10,11). An analysis of an overlapping portion of the data set reported in this article found that RSA and palmar skin conductance, both of which are cholinergically mediated, measured during exposure to laboratory stressors, were correlated with airway sensitivity to methacholine (12).

Defensiveness is a coping style in which people tend to present themselves in a socially desirable manner. It is characterized by avoidance of threatening stimuli and minimization of negative affect. A large body of research on healthy participants suggests that defensiveness is related to elevated stress-related cardiovascular reactivity and some measures of cardiac risk. There is evidence that defensive individuals react to stress with increased heart rate (13–17), diastolic blood pressure (15,16), systolic blood pressure (16–18), electrodermal activity (13, 19,20), and muscle tension (13). Defensiveness also has been associated with elevated salivary cortisol (21) and cholesterol levels (22) and lower RSA (23). Autonomic effects, such as chronic sympathetic arousal, may directly contribute to the onset or exacerbation of disease among defensive individuals.

These findings demonstrate that the trait of defensiveness may cause people to experience somatic stress and be a risk factor for the development or exacerbation of stress-related illness. A recent 3-year prospective study has linked defensiveness to increased risk of developing hypertension (24). Studies of cardiac patients have also demonstrated that defensiveness is associated with higher baseline levels and greater reactivity of systolic blood pressure (25) and more frequent and severe myocardial ischemia (26). Based on the notion of individual response stereotypy, it may be concluded that defensive patients with cardiovascular disease react to stress with increased β -adrenergic arousal, which can lead to disease exacerbation.

The autonomic response pattern associated with defensiveness has not been previously studied in asthma. Consistent with the previous literature, we predicted that defensive participants would show high stress-related responses. However, in the present sample of patients with asthma, it was hypothesized that defensiveness would not be associated with the well-documented finding of elevated sympathetic nervous system activity. Rather, because of the stereotyped pattern of response to stress exhibited by some people with asthma, this group of defensive individuals was predicted to display reduced sympathetic responsiveness and heightened vagal activity. In turn, it was predicted that this unique autonomic response among defensive asthmatics would be associated with decrements in pulmonary function.

The present study is a reanalysis of data that have been previously reported on an overlapping sample of adults with asthma (12). Data were collected during 1993 to 1995.

METHODS

Participants

The sample consisted of 88 adults with asthma (53 women and 35 men, 66 nondefensive and 22 defensive), who were recruited through radio and newspaper advertisements, posted announcements, and notices to physicians. Each subject was paid \$150 for participation in the study. The mean age of patients was 25.9 years (SD = 6.7), with a range of 18 to 40 years.

A board-certified pulmonary physician (S.M.H.) selected patients according to the following criteria: 1) history of recurrent asthma (wheeze that responds to albuterol) within the prior 12 months and 2) below-normal spirometry values ($FEV_1 < 80\%$ expected or $FEF_{50\%} < 60\%$ expected) with no indication of restrictive lung disease (assessed by history and a diffusion capacity test). Thus, diagnosis of asthma required the presence of asthma symptoms, impaired pulmonary function, and evidence of elevated airway reactivity and/or reversibility of pulmonary restriction. However, it should be noted that these specific criteria differ from those recommended in the most recent *Guidelines for the Diagnosis and Management of Asthma* (27), and it is possible that some patients may not have met formal criteria for this disease according to current standards.

Exclusion criteria were 1) history of chronic bronchitis or sinusitis; 2) emphysema or chronic respiratory disease other than asthma, as determined by history of symptoms and physical examination; 3) cigarette smoking within the past 2 years; and 4) cardiovascular, neurological, or psychiatric disease requiring psychoactive medication.

Behavioral Tasks

Half of the participants were exposed to tasks in the following order: mental arithmetic, reaction time, shop accident film, and thoracic surgery film. The other half received the tasks in reverse order. They were instructed that the tasks were designed to increase arousal for the purpose of studying the physiological effects of stress. Data from the mental arithmetic task and thoracic surgery film are not reported in this article because the time intervals were not long enough to allow for analysis of heart rate variability. Descriptions of these two tasks and the absence of significant effects for order have been reported elsewhere (12).

Reaction Time—A computer screen displayed a numerical countdown, by seconds, starting with 6. In place of “0,” a series of asterisks appeared, which signaled the subject to press a button with the dominant hand as quickly as possible. The task consisted of 40 trials of 6-second duration with 10-second interstimulus intervals.

Shop Accident Film—Participants viewed 5 minutes of the film “It Didn’t Have to Happen.” This video depicts scenes of accidents that occur in a workshop and is designed to increase physiological arousal.

Tension Ratings—After each task, participants completed ratings on a 9-point Likert scale indicating how relaxed or tense they felt during the preceding task.

Taylor Manifest Anxiety Scale

The Taylor Manifest Anxiety Scale (TMAS) is a 50-item true-false scale of various symptoms that reflect trait anxiety. The scale has demonstrated good internal consistency (0.82) and test-retest reliabilities ($r = .81-.89$) and correlates highly with other scales of negative affect (28–30).

Marlowe-Crowne Social Desirability Scale

The Marlowe-Crowne Social Desirability Scale (MC) is a 33-item true-false scale that is commonly used to measure defensiveness (31). It asks the respondent about common negative traits (eg, jealousy) and positive characteristics of unusual levels of responsibility and general virtue. The items were chosen to be unrelated to psychopathology. The MC has good internal consistency ($KR-20 = 0.88$) and test-retest reliability ($r = .89$).

The MC was originally designed to measure the tendency of individuals to respond to questions in a socially desirable way. Later studies have revealed that the MC loads on both self-deception

and impression management factors, with a larger loading on the latter (32,33). However, Weinberger and Davidson (34,35) have suggested that individuals who score high on the MC use coping strategies more consistent with self-deception than impression management.

Consistent with previous research in this area (13), the present study utilized a quartile split of the MC (high, upper 25%; low, bottom 75%) and a median split of the TMAS. The cutoff score for both the MC and TMAS was ≥ 18 to define high defensiveness and trait anxiety, respectively. Although techniques for measuring defensiveness have included median, tercile, and quartile splits, the cutoff scores for the MC in previous studies have typically ranged from 17 to 19 (13,15,18,21,22,25,26).

Weinberger et al. (13) created a 2×2 matrix based on dichotomous splits for the MC and TMAS. The MC was used to discriminate between individuals that truly experience low levels of anxiety and those who simply report low levels of anxiety but experience high physiological arousal. Participants who scored high on defensiveness and low on trait anxiety were classified as having a repressive coping style. Traditionally, individuals who score high on both measures (ie, defensive high anxious) have been excluded from analyses because few people score in this category. More recent studies, though, have analyzed the effects of defensiveness and anxiety separately and found that defensiveness may be independently linked to autonomic stress reactivity (19,25). Therefore, in the present study, we examined the main effects for the MC and TMAS, as well as the interaction between the two, to determine whether defensiveness or repressive coping per se would account for our findings.

Palmar Skin Conductance

Skin conductance was analyzed as a marker of sympathetic nervous system activity and to differentiate cholinergic from parasympathetic autonomic effects. It is controlled by the sympathetic nervous system but is cholinergically mediated (36). Silver/silver chloride electrodes, with a 0.05-M solution of NaCl as the electrolyte, were attached to the hypothenar eminence of the nondominant hand, placed approximately 1.5 cm apart. Skin conductance level (SCL) was recorded at a sensitivity of 1 $\mu\text{mho/cm}$ using a Lykken coupler, which applies 0.5 V across the two sites. Data were lost for one nondefensive participant because of equipment failure.

Respiratory Sinus Arrhythmia and Heart Rate

RSA refers to the cyclical variations in heart rate that accompany the respiratory cycle (ie, heart rate usually increases during inspiration and decreases during expiration). For adults with normal respiration rates (approximately 9–24 breaths/min), RSA is considered to reflect vagal cardiac nerve traffic (37). Measurement and interpretation of RSA can be altered by respiration, which has been shown to mediate changes in RSA without affecting mean heart rate (38). This finding has been demonstrated under conditions of complete β -adrenergic blockade (39). The relationship between RSA amplitude and respiration rate, between 6 and 24 breaths/min, is an inverse one (38–40). For these reasons, a consensus panel (37) has recommended controlling for respiration to utilize RSA as a measure of vagal nerve activity.

An electrocardiogram (ECG) was recorded using a three-lead placement (with active leads on the right arm and left leg). The signal was amplified through an AC coupler from a Beckman Type RM Dynograph at a time constant of 1.0. Data for heart rate were produced from the digitized output of a Beckman cardiometer input coupler (Type 9857). Cardiac interbeat interval was detected from ECG R-wave spikes using a Delta-Biometrics vagal tone monitor, which digitizes the raw ECG signal at a rate of 1000 Hz and creates an ASCII file of R-R intervals calculated to the nearest millisecond.

Cardiac interbeat interval data were manually edited for artifact and faulty R-wave detection using the MXEDIT program (41). Records were rejected for calculation of RSA if editing was required for more than 10% of the data points or if epochs were less than 2 minutes. Using these standards, data for RSA were lost for four nondefensive and two defensive participants. The Log-a-Rhythm program (42) calculated RSA using the fast Fourier transform method (43), with the high-frequency (HF) peak defined as 0.15 to 0.40 Hz.

It has also been recommended that the selection of the HF band should include all respiratory frequencies and avoid significant overlapping variance by low-frequency (LF) waves (37). Therefore, RSA was computed a second time by adjusting the band width upward or downward for each participant to include the entire HF peak. These analyses on the adjusted HF bands were used to confirm the results of the unadjusted HF band analyses (0.15–0.40 Hz) for the subset of participants with distinguishable HF and LF peaks. Because identical results were obtained for the adjusted HF band analyses, only the unadjusted HF band analyses are reported.

Respiration Rate

Respiration rate was measured by attaching a Pneumotrace respiration belt (UFI, Morro Bay, CA) around the abdomen of the patient, between the umbilicus and the xiphoid process. The signal was recorded through an external bridge circuit, with power supplied through a Beckman Type 9853A coupler, and was digitally sampled after analog-to-digital conversion. A computer program designed in our laboratory detected respiratory peaks and calculated respiration rate (ie, breaths per minute).

Because of late equipment arrival, strain gauge recordings were available for only 52 nondefensive and 15 defensive participants. Where data were not collected with the respiration belt, respiration rate was estimated from the HF peak, which almost always is the component of heart rate variability that occurs at the frequency of respiration (38). To ensure the accuracy of the respiratory frequency parameters used, comparisons were made between the HF peak and the computer-generated strain gauge recordings (where available). For cases where the difference between these two values was ≥ 0.05 Hz, the strain gauge data were visually inspected and respiration rate was determined manually. Respiration rate could not be calculated for five nondefensive and two defensive participants because strain gauge data were not available and a HF peak could not be clearly identified.

Spirometry

A forced vital capacity (FVC) maneuver involves maximal inspiration followed by a forceful and rapid expiration into a spirometer. Thus, FVC refers to the total volume of air exhaled during this procedure. FEV_1 is the volume of air expired during the first second of the FVC maneuver. The FEV_1/FVC ratio is also commonly used as an indicator of obstructive impairment because FVC may be normal in asthma, but FEV_1 tends to be reduced during an asthma flare. $FEF_{50\%}$ is the flow that occurs at 50% of FVC and the measurement consists of flow from the smaller/lower airways. This measurement also tends to be reduced during asthma exacerbation.

Pulmonary function was measured by a pneumotachometer-based spirometer (PneuMedics Mega 4000, Milford, CT). Participants performed three FVC maneuvers, although an additional test was required if the two highest FVC or FEV_1 values were not within 5%. The curve with the largest sum of FEV_1 and FVC (the “best test”) was selected for data analyses, as is recommended for pulmonary evaluations of midexpiratory flow (44). Although the American Thoracic Society currently recommends scoring the highest FEV_1 and FVC values, even if they originate from different curves, the “best test” curve is still considered valid because of minimal differences between the two methods (45). Spirometry variables used in

the analyses were calculated on the basis of the percentage expected in the normal population for age, gender, height, and weight. Spirometry measures were lost for one nondefensive participant because of equipment failure.

Procedure

Participants were asked not to take bronchodilator medication for 12 hours before their scheduled arrival in the laboratory and not to drink caffeine on the day of the testing session. If an upper respiratory infection occurred within the previous month, then testing was delayed.

After arriving at the laboratory, participants received verbal and written descriptions of the procedure and signed an informed consent statement. They then completed the MC, TMAS, and spirometry testing. Next, the respiration belt was connected and participants were seated in a semireclined position. Electrodes were attached for measuring electrocardiographic activity and palmar skin conductance. During the initial baseline period, participants rested for 3 minutes. They were then exposed to the tasks and allowed to rest for 3 minutes after each task, at which time participants reported how relaxed or tense they felt during the task. After the final rest period, postspirometry testing was performed. The appropriate institutional review boards approved the study protocol.

Statistical Analyses

A mixed model design was applied for analyses of SCL, RSA, and heart rate. Mixed models incorporate components of a repeated-measures ANOVA and a regression analysis (46). In contrast to repeated-measures ANOVAs, this approach does not assume sphericity. The mixed model design is particularly useful for analyzing repeated measures with time-dependent covariates. Task was treated as a within-subjects factor with three levels (initial rest period, reaction time task, and shop accident film), and defensiveness (MC) and trait anxiety (TMAS) were between-subjects factors. Age and gender were treated as between-subjects covariates in the analyses for SCL, RSA, and heart rate. Changes in RSA have been shown to occur as a function of age (47–50) and gender (49). Analyses for RSA were conducted with and without respiration rate as a time-dependent covariate to determine if changes in RSA were mediated by between-groups differences in respiration rate. The natural logarithm was calculated for RSA to normalize the distribution of these data.

Changes in pulmonary function were analyzed by using repeated-measures ANOVAs with pretask and posttask spirometry test measures as levels of the within-subject factor. Defensiveness and trait anxiety were treated as between-subjects factors. Secondary analyses examining simple main effects treated FEV_1/FVC , $\%FEV_1$, and $\%FEF_{50\%}$ as separate families, and familywise error rate ($p = .05$) was divided by the number of members in each family. Thus, the α level was defined as 0.025 for the two individual tests in each family.

Pearson correlations were used to assess the relationship between changes in pulmonary function and autonomic variables. Finally, a repeated-measures ANOVA was carried out for self-report of tension ratings during the tasks.

Secondary analyses were conducted using paired-samples t tests if a main effect of task was present on any of the variables described above. A Bonferroni correction was used; thus, an α level of 0.025 was applied for the two individual tests that were required for each variable. An α level of less than 0.05 was considered significant for all other analyses.

RESULTS

No significant between-groups differences were present for age ($t(86) = 1.41, p = .162$), gender ($\chi^2(1,88) = 1.91, p = .167$), or any of the spirometry measures taken before the tasks: %

FEV_1/FVC ($t(85) = 0.45, p = .657$), $\%FEV_1$ ($t(85) = 0.31, p = .755$), and $\%FEF_{50\%}$ ($t(85) = 0.09, p = .927$). Table 1 shows the means for each of these variables.

Skin Conductance Level

There was a significant main effect for defensiveness ($F(1,74) = 5.61, p = .021$) on SCL. Defensive participants showed lower SCL (least square mean = $8.99 \mu mho$, adjusted for age and gender, $SE = 0.81$), across the tasks than nondefensive patients (least square mean = $10.97 \mu mho$, $SE = 0.44$). The main effect for trait anxiety and interactions for defensiveness by trait anxiety, defensiveness by task, and trait anxiety by task were not significant.

There was a main effect for task ($F(2,152) = 21.75, p < .001$) on SCL. Paired-samples t tests showed that SCL increased from baseline (mean = $9.78 \mu mho$, $SE = 0.41$) to the reaction time task (mean = $10.30 \mu mho$, $SE = 0.41$; $p = .004$) and from baseline to the film (mean = $10.95 \mu mho$, $SE = 0.41$; $p < .001$).

Respiratory Sinus Arrhythmia and Heart Rate

Defensive subjects displayed higher RSA (least square mean = $2.32 ms^2$, adjusted for age and gender, $SE = 0.19$) across the tasks than nondefensive participants (least square mean = $1.81 ms^2$, $SE = 0.10$) ($F(1,81) = 5.43, p = .022$). These results remained significant after a covariance adjustment for respiration rate in the analyses ($F(1,81) = 5.37, p = .023$). All other relevant main effects and interactions were not significant.

In the analyses for heart rate, there was a main effect for task ($F(2,152) = 9.04, p < .001$). Paired-samples t tests showed heart rate acceleration from baseline (mean = 73.53 beats/min, $SE = 1.21$) to the reaction time task (mean = 74.48 beats/min, $SE = 1.20$; $p = .003$) and a nonsignificant tendency ($p = .060$) for deceleration from baseline to the film (mean = 72.63 beats/min, $SE = 1.23$). All other relevant main effects and interactions were not significant.

Spirometry

A defensiveness by pretask-to-posttask interaction was present for FEV_1/FVC ($F(1,80) = 6.82, p = .011$), $\%FEV_1$ ($F(1,80) = 4.32, p = .041$), and $\%FEF_{50\%}$ ($F(1,80) = 8.65, p = .004$). These analyses indicated that defensive patients showed greater decrements in pulmonary function after exposure to laboratory tasks compared with nondefensive participants. Figure 1 displays the changes from pretask to posttask for the spirometry test variables.

These significant interactions allowed for examination of the simple main effects. Paired-samples t tests were conducted separately for each group on the spirometry test values to detect whether pretask-to-posttask changes were significant. Among defensive participants, the decrease in FEV_1/FVC was significant ($t(21) = 3.52, p = .002$), and a nonsignificant trend ($t(21) = 1.78, p = .089$) was present for the decline in $\%FEF_{50\%}$. Among nondefensive participants, the increase in $\%FEV_1$ was significant ($t(64) = 2.41, p = .019$).

An interaction was also present for trait anxiety by pretask-to-posttask on $\%FEF_{50\%}$ ($F(1,80) = 6.34, p = .014$). However, the increase among individuals with low trait anxiety from pretask (mean = 49.19% , $SE = 2.96$) to posttask (mean = 51.02% , $SE = 3.20$) and the decrease among the high anxiety group from pretask (mean = 51.41% , $SE = 3.14$) to posttask (mean = 50.60% , $SE = 2.84$) were not significant changes.

Relationship Between Pulmonary Changes and Autonomic Measures

SCL, averaged across the tasks, was directly related to changes in FEV_1/FVC ($r = .23, p = .031$), $\%FEV_1$ ($r = .37, p = .001$), and $\%FEF_{50\%}$ ($r = .26, p = .01$), including all participants in these analyses. These findings indicate that lower levels of SCL were associated with greater

decrements in pulmonary function, whereas higher levels of SCL were linked with increases in pulmonary function. RSA and heart rate were not related to changes in spirometry test variables.

Ratings of Tension During Tasks

There was a main effect for task ($F(2,142) = 4.83, p = .009$, Greenhouse-Geisser $\epsilon = 0.97$) on subjective ratings of tension levels during the tasks. Paired-samples t tests showed that tension was rated as higher during the film (mean = 5.45, SE = 0.19) than at baseline (mean = 4.57, SE = 0.21; $p < .001$). Participants responded that they felt somewhere between “neither tense nor relaxed” and “slightly tense” during the film. The mean response of participants for the baseline period was somewhere between “slightly relaxed” and “neither tense nor relaxed.” Tension levels during the reaction time task (mean = 4.83, SE = 0.20) did not differ from baseline levels. No other relevant effects or interactions were significant.

DISCUSSION

The present study provides evidence that the autonomic nervous system of defensive patients with asthma¹ may be “tuned” differently than that of other defensive individuals. Defensive asthmatics displayed diminished SCL and elevated RSA amplitudes across a series of behavioral tasks compared with nondefensive participants. After exposure to these tasks, defensive participants with asthma displayed a decline in FEV₁/FVC, %FEV₁, and %FEF_{50%} compared with nondefensive asthmatics, who showed increases on these measures.² These changes in pulmonary function were correlated with SCL but not RSA amplitudes.

The findings of the present study are consistent with the notion of individual response stereotypy (1) and suggest that defensiveness may not have a unique autonomic response associated with it. Rather, an interaction between defensiveness and disease may determine an individual’s physiological response to stress. Defensive individuals whose parasympathetic system is particularly reactive may show a predominantly parasympathetic response to stress. Asthma may be characterized by parasympathetic hyperreactivity to stress. Other individuals characterized by parasympathetic symptoms (eg, acid indigestion, vascular headaches, and faintness) may show a similar pattern.

Previous findings showing a link between defensiveness and increased sympathetic arousal may be specific to the populations examined in those studies: patients with (or at risk for) cardiovascular disease. In contrast to these patients, defensiveness among asthma sufferers may be characterized by cholinergic hyperresponsiveness as well as β -adrenergic hyporesponsiveness. In the present study, defensive patients with asthma exhibited an attenuated sympathetic response during the laboratory tasks, as indicated by lower SCL and decreased pulmonary function after the tasks. These data seem to be consistent with previous findings among people with asthma showing a diminished adrenaline response during stress (6) and acute asthma exacerbation (7).

¹We are labeling patients with asthma despite qualifications stated in the description of participants.

²We note that changes in midexpiratory flow and the ratio FEV₁/FVC may be compromised if substantial change in FVC has occurred (51). For example, if FVC markedly increases, then the point at which FEF_{50%} is calculated may be at a lower lung volume and therefore FEF_{50%} will seem to be reduced simply because of the increase in FVC (52). Similarly, decreases in FEV₁/FVC may not be indicative of poorer lung function if changes are the result of increases in the denominator of this ratio. We examined these possibilities among defensive patients, and it does not seem that decreases in FEF_{50%} or FEV₁/FVC were simply attributed to increases in FVC. Defensive asthmatics did not show a significant increase from pretask FVC (mean = 4.04 liters, SE = 0.21) to posttask FVC (mean = 4.14 liter, SE = 0.23) ($p = .22$). Only 3 of the 22 defensive individuals showed a clinically significant increase in FVC (ie, 12% and 200-ml increase) (51). Among these three participants, only one showed a decrease in FEF_{50%}, and two displayed a decline in FEV₁/FVC.

Defensive asthmatic patients showed elevated RSA amplitudes across the tasks, which is also in contrast to data on defensive healthy people showing lower RSA (23). Although respiration was controlled in the present study, it should be noted that RSA may not be a definitive estimate of cardiac vagal tone. There was no between-subjects difference in heart rate, and correlation coefficients between RSA and heart rate revealed no clear relationship. This may reflect sympathetic influence on heart rate or a decoupling of chronotropic from tonic vagal influence on heart rate (53).

The findings of this study seem to be attributed to defensiveness rather than a “repressive coping style,” which involves a low level of self-reported anxiety in addition to defensiveness. Other studies that analyzed defensiveness and anxiety separately have similarly found that measures of anxiety add little power to defensiveness in predicting autonomic stress reactivity (19,25). In the present study, trait anxiety was not related to any of our autonomic or pulmonary function measures with the exception of %FEF_{50%}.

Our findings could not be explained by participants’ reactions to particular stressors used in the study. The data did not reveal defensiveness by task interactions, and findings in the same direction were obtained for all variables when excluding the behavioral tasks and analyzing only the initial baseline period. This may be attributed to the mild nature of the stressors, as indicated by the self-report ratings of tension produced by the tasks. Alternatively, the short duration of the baseline period (3 minutes) and the knowledge of participating in a study of psychological stress may have evoked a physiological stress response (ie, anticipatory anxiety) among participants during baseline. Tension ratings for the baseline period revealed that participants did not feel particularly relaxed.

Observed changes in pulmonary function may nonetheless have been stress-related. Some support for this notion was provided by elevations in SCL and heart rate and the significant correlations between SCL and changes in pulmonary function. Sympathetic hypoarousal may be detrimental to patients with asthma because of the attenuation of adrenaline’s relaxant effects on airway smooth muscle. Conversely, the increase in pulmonary function among nondefensive participants may be related to increased sympathetic arousal during exposure to the laboratory tasks.

In contrast to other studies’ finding that stress-induced asthma exacerbation may be cholinergically mediated (4,9), RSA amplitudes and changes in pulmonary function were not related to each other in the present study. The autonomic mechanism for the pulmonary decline among defensive asthmatics thus requires further clarification. Markers of other airway mediators (eg, inflammatory cells, nonadrenergic noncholinergic nerves, and neuropeptides) were not collected; thus, it would be speculative to discuss their possible role.

Only a small proportion of the participants actually exhibited pulmonary function changes that are considered to be clinically significant. Approximately 14% of defensive patients demonstrated a decrease in FEV₁ of 15% or greater, the criterion recommended for assessing exercise-induced bronchoconstriction (54). Conversely, 14% of nondefensive participants experienced a clinically significant increase (ie, increase in FEV₁ of at least 12% and 200 ml) (51). Nevertheless, the contrasting pulmonary function responses of the two groups and the mild nature of the stressors used suggest that these findings should be explored further.

To date, this is the only study that has examined autonomic and pulmonary function among adult defensive patients with asthma. Future directions for research in this area should focus on replicating these findings on a larger sample of people with asthma, as well as extending the literature on defensiveness to other medical conditions. If these findings are replicated, then it will be important to understand the mechanism that may explain stress-induced asthma

among defensive patients. Immunological, behavioral, and autonomic pathways should all be explored, as should the probable interactions that exist among them.

In conclusion, defensive asthmatics showed lower SCL and higher RSA amplitudes during exposure to laboratory stressors and pretask-to-posttask decreases in pulmonary function compared with nondefensive asthmatics. These results are especially interesting because they contrast with previous findings in other populations demonstrating a link between exaggerated sympathetic nervous system activity and defensiveness. We interpreted these data to be consistent with the theory of individual response stereotypy in asthma and hypothesize that this tendency may interact with defensiveness in producing stress-related asthma symptoms.

Glossary

ANOVA	analysis of variance
ECG	electrocardiogram
FEF_{50%}	forced expiratory flow at 50% of vital capacity
FEV₁	forced expiratory volume at 1 second
FVC	forced vital capacity
HF	high frequency
LF	low frequency
MC	Marlowe-Crowne Social Desirability Scale
RSA	respiratory sinus arrhythmia
SCL	skin conductance level
TMAS	Taylor Manifest Anxiety Scale

Acknowledgments

This work was supported by Grant HL-44097 and Grant HL-58805 from the National Heart, Lung, and Blood Institute, National Institutes of Health. The authors are indebted to Dr. Robert Hamer for his assistance with conducting mixed models analyses; to Dr. Erich Labouvie for statistical advice; to Dr. Stephen Porges for MXEDIT, a PC-based program used to edit interbeat interval data; to Dr. William Craelius for his Log-a-Rhythm program, which was used to calculate RSA using the fast Fourier transform method; and to Dr. Mahmood Siddique for comments on earlier versions of this manuscript.

This work was completed as partial fulfillment of the requirements for the degree of Master of Science in the Department of Psychology at Rutgers University by the first author.

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Fig. 1.
Comparison between nondefensive and defensive patients on changes from pretask to posttask in FEV₁/FVC, %FEV₁, and %FEF_{50%}.

TABLE 1

Participants' Characteristics

	Defensive	Nondefensive
Gender, % female	72.73	56.06
Age, y		
Mean	27.18	24.91
SD	7.08	6.35
MC		
Mean	20.71	11.70
SD	2.26	3.42
FEV ₁ /FVC, pretask		
Mean	0.70	0.71
SD	0.11	0.10
FEV ₁ , pretask		
Mean (% predicted)	82.91	84.42
SD	20.50	15.44
FEF _{50%} , pretask		
Mean (% predicted)	49.95	50.32
SD	22.43	18.04