
Psychological triggers and hyperventilation symptoms in asthma

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Background: Anecdotal accounts have identified hyperventilation as one route through which psychological factors can trigger bronchoconstriction. However, little is known about the empirical association between psychological and other trigger factors and hyperventilation in asthma exacerbations.

Objective: To study the cross-sectional association between perceived triggers and hyperventilation symptoms in 1 British and 1 German sample of patients with asthma who were recruited from the community and from primary care clinics.

Method: Patients completed relevant language versions of the Asthma Trigger Inventory and the Asthma Symptom Checklist.

Results: After controlling for demographics and asthma severity, perceived asthma triggers measured by subscales of the Asthma Trigger Inventory explained 12.5% to 37.3% of the variance in Asthma Symptom Checklist hyperventilation-hypocapnia symptoms. Psychological triggers accounted for 10.6% to 26.7% of the variance alone and 4.3% to 11.0% of the variance over and above other trigger factors. In contrast, perceived animal and pollen allergen triggers did not contribute unique variance to the hyperventilation symptom report. Psychological triggers did not explain variance in classic airway obstruction symptoms, thus arguing against a general bias toward inflated symptom reports in patients with psychologically induced asthma.

Conclusion: Differences in perceived asthma triggers are substantially associated with hyperventilation symptoms, and patients with more frequent psychological triggers also tend to report that they experience more hyperventilation symptoms during their asthma symptom episodes.

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INTRODUCTION

Observational and experimental studies^{1–3} have established a role for psychosocial factors, such as life events, distress, or various mood states or emotions, in asthmatic symptom episodes and lung function decline; reviews are also available.^{4–7} The potential mechanisms of such influences continue to be the focus of scientific inquiry. Anecdotal accounts and clinical observations have identified hyperventilation as one route through which psychological factors can trigger bronchoconstriction.^{8–10} Indeed, experimental studies^{11,12} have demonstrated reductions in PCO₂ levels or hypocapnia in aversive psychological states, such as anxiety, panic, and phobic responses. Central nervous system pathways underlying such associations could be linked to fight-flight responses,¹³ sensations of dyspnea,^{14,15} or voluntary influences on the respiratory pattern.^{15,16} Hypocapnia has also been shown to constrict the airways in patients with asthma and in healthy control subjects.^{17–19} However, little is known about the actual

empirical association between patients' reports of psychological triggers of their asthma on the one hand and the presence of hyperventilation symptoms in periods of increases in patients' asthma symptoms on the other hand. Although experimental induction of negative affective states typically leads to mild airway obstruction, this has not been linked consistently to changes in ventilation.^{20–23} It is unknown to what extent in daily life psychologically induced increases in asthma symptoms are accompanied by hyperventilation. We assumed that if such a relationship existed, this would also be reflected in a positive association between psychological triggers as perceived causes of asthma symptom episodes and hyperventilation symptoms as particular characteristics of such episodes.

Thus, we studied the cross-sectional association between the reported prevalence of psychological triggers of asthma symptoms and the prevalence of hyperventilation as one of the triggered asthma symptoms. In studying this association, we controlled for the role of other trigger factors that could be linked to hyperventilation in asthma, such as exercise or physical exhaustion, and tested the role of psychological triggers against allergic triggers. In addition, we examined whether there was a unique role of psychological triggers for hyperventilation symptoms or whether reports of psychological triggers were nonspecifically associated with elevated asthma symptoms in general. We expected that self-reported psychological asthma triggers would be positively correlated with perceived hyperventilation symptoms in asthma symptom episodes, that this association was specific for psycho-

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Table 1. Demographic Characteristics, Disease History, and Manifestation in the British Sample and the German Sample

Variable	British sample (n = 172) ^a	German sample (n = 156) ^a	P value for sample differences ^b
Age, y ^c	41.4 (14.9)	40.3 (15.3)	.25
Female sex	68.6	57.7	.04
Education: A levels, degree, or university entrance diploma	40.1	54.5	.01
Marital status of married or living together	52.9	50.6	.74
Family history positive for asthma	66.9	70.5	.55
Childhood dermatitis	25.0	24.4	.90
Allergic rhinitis	34.3	41.0	.21
Chronic bronchitis	19.8	26.8	.15
Age at asthma onset, y ^c	22.3 (18.5)	20.1 (16.8)	.44
Seasonal variations in asthma	52.3	48.7	.91
Symptoms mostly during nighttime	23.5	24.4	.90

^a Data are given as percentage of each group unless otherwise indicated.

^b From a *t* test or a χ^2 test.

^c Data are given as mean (SD).

logical triggers compared with other trigger factors, and that this association would still be demonstrated after controlling for elevated asthma symptom report in general.

METHODS

Patients

Data from 2 larger questionnaire studies that had included research versions of questionnaires on perceived asthma triggers and symptoms of asthma were used. Evaluations of the psychometric properties of these instruments in the present sample are reported elsewhere.^{24–26} The first sample (n = 172) was drawn from 2 large primary care health centers in London, England. The second sample (n = 156) consisted of patients recruited from the community to participate in research studies at a hospital-based research center in Hamburg, Germany (Pulmonary Research Institute, Hospital Grosshansdorf). Samples were largely comparable with regard to demographics and asthma manifestation (Table 1); however, the German sample had more men and participants with a higher education level, probably reflecting differences in the populations from which we had recruited patients (primary care patients vs volunteers for hospital-based research). Patients initially filled out a questionnaire package as part of their participation in these studies and returned it in a sealed envelope. Physicians and other health care personnel directly treating the patients did not have access to information from individual completed questionnaires. Participation was completely voluntary, and patients were assured that refusal to participate would not affect their regular care in any way. The projects were approved by local ethics committees in London and Hamburg.

Instruments

Asthma Trigger Inventory. The English-language²⁶ and German-language²⁵ versions of the Asthma Trigger Inventory (ATI) included a 32-trigger item checklist in which patients rated on a 5-point scale how frequently they personally had

experienced that a particular trigger was associated with asthma symptoms or exacerbations (response format, 0–4: “never,” “rarely,” “sometimes,” “often,” and “always”). The items constituted 6 subscales that were thematically associated with psychological factors, animal allergens, pollen allergens, physical activity, infection, and air pollution or irritants. Typical items for the psychological trigger subscale were “stress at home,” “feeling tense,” “depressed mood,” and “being excited.” The subscales showed good to excellent internal consistency (English version, $\alpha = .81-.94$; and German version, $\alpha = .77-.89$) and low to moderately positive intercorrelations. In both language versions,^{25,26} positive associations between the animal allergy scale and skin prick test results for cat and dog allergens were found. In the English-language version,²⁶ the psychological trigger subscale was associated with airway obstruction during pleasant and unpleasant film clips.

Asthma Symptom Checklist. The Asthma Symptom Checklist (ASC) is a 36-item checklist of symptoms originally devised by Kinsman et al.²⁷ Based on a principle component analysis of the instrument using data from the UK survey, 6 subscales are formed related to panic-fear, irritability, obstruction-dyspnea, obstruction-congestion, hyperventilation, and fatigue, with internal consistencies ranging between $\alpha = .81$ and $\alpha = .94$.²⁴ The German 25-item version²⁸ yielded a 5-factor structure that was identical to the British version but with only a single obstruction subscale. Consistent with these findings, we used the 6-factor structure of the ASC for the British sample and the 5-factor structure for the German sample. Internal consistencies for subscales of this version ranged between $\alpha = .72$ and $\alpha = .90$. The ASC has proved its utility in a number of studies^{29,30} predicting frequency of medication use and health care use.

Additional measures. Demographics and information on disease manifestation and treatment were collected on a separate questionnaire. To characterize the sample, we analyzed

the following variables: sex, age, marital status (0 indicates unmarried; and 1, married), educational status (a yes or no response to UK A levels or degree for the British sample and university entrance diploma or higher for the German sample), age of disease onset, family history of asthma and allergy, childhood diseases (dermatitis, allergic rhinitis, and chronic bronchitis), and seasonal and nighttime symptoms. Asthma severity was rated by study personnel following either the British Thoracic Society scheme (UK sample³¹), which is based on medication information; or National Heart, Lung, and Blood Institute/World Health Organization guidelines (German sample²³), which uses information on symptom frequency and lung function. Medical record review and patient self-report were used for classifications.

Statistical Analysis

A multivariate analysis of variance with the 6 ATI trigger subscales as dependent variables was calculated to explore differences in levels of reported asthma triggers between British and German samples. In case of an overall sample difference, univariate analyses of variance were used to determine the contributions of individual ATI subscales. Hierarchical multiple regressions were used to calculate the following: (1) the extent to which the ATI psychological trigger subscale predicted ASC hyperventilation symptom report, after controlling for demographics, asthma severity, and other trigger factors; and (2) the association of the ATI psychological trigger subscale with other symptom dimensions of the ASC, after controlling for demographics, asthma severity, and other trigger factors. The second set of analyses sought to clarify whether psychological factors were nonspecifically associated with elevations on any of the ASC symptom scales. In these analyses, the dependent variable was the ASC hyperventilation subscale score. The order of predictors entered was as follows: (1) sex, age, marital status, and education level in the first step (relevance of these variables in asthma trigger and symptom reports is demonstrated by earlier analyses^{24–26,33}); (2) British Thoracic Society steps 1 and 5 (tested against steps 2–4) in the British sample or National Heart, Lung, and Blood Institute/World Health Organization severity (rated as intermittent, mild persistent, moderate persistent, and severe persistent) in the German sample in the second step; (3) the 5 ATI nonpsychological subscales in the third step; and (4) the ATI psychological trigger subscale in the fourth step. To determine the overall amount of variance in hyperventilation symptoms explained by the ATI psychological trigger subscale alone without controlling for the other 5 ATI trigger subscales, the analysis was repeated, entering only the ATI psychological trigger scale in the third step. In addition, to compare the predictive power of ATI psychological triggers with that of allergic triggers, the same analysis was calculated with only the 2 ATI allergic trigger subscales entered in the third step; there was also 1 analysis with the 4 ATI nonspecific trigger subscales (physical activity, air pollution, infection, and psychological factors) in the third step and with the 2 ATI specific trigger scales (pollen and animal

allergens) in the fourth step. Additional exploratory hierarchical multiple regression analyses were calculated with the other ASC scales as dependent variables to study the specificity of the association between psychological triggers and hyperventilation. For each multiple linear regression analysis, we ensured that the assumption of normal distribution of the residuals was confirmed using the Kolmogorov-Smirnov test (skewness <1) and that criteria for extreme outliers (Cook distance >1.0) and multicollinearity (variance inflation factors >4) were not met.³⁴ Semipartial r^2 was used as an estimator of effect size.

RESULTS

Differences in the levels of reported asthma triggers were found between the German and the British sample ($F_{6,321} = 271.90$, $P < .001$). The British sample reported significantly more frequent nonallergic triggers (including psychological triggers) and less frequent animal allergic triggers ($P < .02$ -.001 for all).

In the British sample, after controlling for demographics and asthma severity, perceived asthma triggers measured by ATI subscales explained 37.3% of the variance in ASC hyperventilation symptoms. Psychological triggers alone accounted for 26.7% of the variance ($F_{1,166} = 66.45$, $P < .001$) and 11.0% of the variance over and above other trigger categories (Table 2). Other trigger scales yielding a unique contribution in explaining variance in hyperventilation symptoms in the final regression model were the ATI infection and air pollution trigger subscale.

In the German sample, the ATI trigger subscales explained 12.5% of the variance in hyperventilation symptoms over and above all other factors. The ATI psychological triggers alone accounted for 10.6% of the variance in hyperventilation symptoms ($F_{1,150} = 18.76$, $P < .001$) and 4.3% over and above all other trigger factors (Table 3). In this sample, the psychological trigger subscale was the only ATI trigger subscale contributing uniquely to the final regression model. Because the criterion of skewness of the residuals of less than 1 was not met in this analysis, we repeated it after excluding 2 outliers but found no substantial deviation of findings from the initial analysis.

The ATI specific allergic trigger scales played a relatively small role in explaining variance in hyperventilation symptoms. In the British sample, an additional 6.4% ($F_{2,163} = 6.14$, $P = .003$) was explained by ATI pollen and animal allergen subscales if they were entered as the only trigger scales in the third step and only 0.2% ($F_{2,159} < 1$, $P = .75$) if they were entered in the fourth step after accounting for all other nonspecific trigger subscales (including the psychological trigger scale). In the German sample, only an additional 1.4% ($F_{2,148} = 1.14$, $P = .32$) was explained if they were entered alone in the third step and an additional 0.5% ($F_{2,144} < 1$, $P = .67$) if entered after accounting for all other nonspecific trigger subscales (including the psychological trigger scale). Exclusion of the 2 outliers did not substantially change these findings.

Table 2. Results of the Hierarchical Multiple Linear Regression Analysis (Final Model) Testing the Association of ATI Psychological Triggers With ASC Hyperventilation Symptoms in the 172 Patients in the British Sample With Asthma

Predictor according to step ^a	<i>b</i> ^b	SE	β Value ^b	<i>P</i> value	<i>sr</i> ²
Step 1					
Age	0.001	0.004	.018	.81	0
Sex ^c	−0.062	0.111	−.035	.58	0.001
Marital status	0.014	0.105	.008	.90	0
Education level	0.009	0.107	.006	.09	0
Step 2					
BTS step 1	−0.185	0.120	−.108	.12	0.015
BTS step 5	−0.419	0.306	−.084	.17	0.012
Step 3					
ATI pollen allergen	−0.011	0.050	−.016	.83	0
ATI animal allergen	0.034	0.045	.049	.45	0.004
ATI physical activity	−0.038	0.056	−.050	.49	0.003
ATI air pollution	0.174	0.069	.201	.01	0.038
ATI infection	0.244	0.065	.283	.001	0.080
Step 4					
ATI psychological	0.378	0.067	.418	.001	0.168

Abbreviations: ASC, Asthma Symptom Checklist; ATI, Asthma Trigger Inventory; BTS, British Thoracic Society; *sr*², semipartial *r*².

^a For step 1, *R* = 0.272, *R*² = 0.074, and *P* value for change in *R*² = .01; for step 2, *R* = 0.288, *R*² = 0.083, and *P* value for change in *R*² = .45; for step 3, *R* = 0.588, *R*² = 0.346, and *P* value for change in *R*² = .001; and for step 4, *R* = 0.675, *R*² = 0.456, and *P* value for change in *R*² = .001.

^b Reported coefficients refer to the final model.

^c Coded as follows: females, 0; and males, 1.

Table 3. Results of the Hierarchical Multiple Linear Regression Analysis (Final Model) Testing the Association of ATI Psychological Triggers With ASC Hyperventilation Symptoms in the 156 Patients in the German Sample With Asthma

Predictors according to step ^a	<i>b</i> ^b	SE	β Value ^b	<i>P</i> value	<i>sr</i> ²
Step 1					
Age	−0.005	0.005	−.107	.33	0.007
Sex ^c	0.042	0.116	.029	.72	0
Marital status	−0.078	0.132	−.056	.56	0.002
Education level	−0.173	0.125	−.122	.17	0.013
Step 2					
NHLBI severity	−0.015	0.058	−.025	.80	0
Step 3					
ATI pollen allergen	0.040	0.045	.076	.38	0.005
ATI animal allergen	−0.023	0.050	−.040	.65	0.001
ATI physical activity	−0.012	0.058	−.018	.83	0
ATI air pollution	0.046	0.081	.060	.57	0.002
ATI infection	0.077	0.062	.123	.21	0.011
Step 4					
ATI psychological	0.327	0.120	.276	.007	0.049

Abbreviations: ASC, Asthma Symptom Checklist; ATI, Asthma Trigger Inventory; NHLBI, National Heart, Lung, and Blood Institute; *sr*², semipartial *r*².

^a For step 1, *R* = 0.187, *R*² = 0.035, and *P* value for change in *R*² = .25; for step 2, *R* = 0.210, *R*² = 0.044, and *P* value for change in *R*² = .23; for step 3, *R* = 0.355, *R*² = 0.126, and *P* value for change in *R*² = .02; and for step 4, *R* = 0.412, *R*² = 0.169, and *P* value for change in *R*² = .001.

^b Reported coefficients refer to the final model.

^c Coded as follows: females, 0; and males, 1.

When testing the specificity of the association between psychological triggers and hyperventilation symptoms, both the British and German samples showed an additional prominent role for the psychological trigger scale in explaining ASC mood symptoms of panic-fear and irritability (Table 4),

whereas no specific role was observed in explaining classic obstruction symptoms. In general, perceived triggers explained a sizeable proportion of the variance in symptom reports (9%–31%) in both samples. Only ATI nonspecific trigger subscales explained unique variance in ASC phys-

Table 4. Explained Variance in ASC Asthma Symptoms (Beyond Hyperventilation) by ATI Trigger Subscales (Without ATI Psychological Subscale) and Additional Variance Explained by ATI Psychological Trigger Subscale

Dependent variable	Significant ATI subscales in the final model	Change in R^{2a}	P value	Additional change in R^2 for the ATI psychology subscale ^b	P value	β Value	t Value	R^2 for the final model
British sample								
Panic-fear	Psychology	0.116	.001	0.131	.001	.457	5.67	0.351
Irritability	Psychology and infection	0.160	.001	0.097	.001	.393	4.74	0.313
Dyspnea	Infection and physical activity	0.168	.001	0.004	.37	.079	0.90	0.229
Congestion	Infection	0.313	.001	0	.87	-.012	-0.17	0.434
Fatigue	Psychology, infection, and physical activity	0.228	.001	0.028	.01	.211	2.61	0.348
German sample								
Panic-fear	Psychology	0.167	.001	0.125	.001	.469	5.05	0.299
Irritability	Psychology	0.116	.002	0.129	.001	.477	5.09	0.291
Obstruction	Infection and air pollution	0.164	.001	0	.85	.019	0.20	0.214
Fatigue	Air pollution	0.151	.001	0.003	.47	.073	0.73	0.185

Abbreviations: ASC, Asthma Symptom Checklist; ATI, Asthma Trigger Inventory.

^a The R^2 change in step 3 for the 5 ATI trigger subscales (animal allergens, pollen allergens, physical activity, air pollution, and infection), after controlling for sex, age, marital status, and education level in step 1; and British Thoracic Society treatment levels or asthma severity according to the National Heart, Lung, and Blood Institute in step 2.

^b Results in R^2 change in step 4 for the ATI psychological trigger subscale.

ical symptom scales of obstruction and fatigue in the final models.

DISCUSSION

In this study, we found evidence for an association of perceived psychological triggers of asthma symptoms with hyperventilation symptoms. Patients who reported that psychological triggers were more commonly (or more frequently) involved in periods of increased asthma symptoms also reported that hyperventilation-hypocapnia symptoms occurred during symptom episodes. The findings of this association were strengthened by relatively comparable results in 2 patient samples that differed with respect to a number of characteristics, such as nationality, sex composition, and educational background. The 2 samples also varied in the overall level of perceived triggers on most subscales of the ATI, which could be because of cultural idiosyncrasies or differences in the specific patient subpopulations sampled (eg, primary care outpatients vs volunteers for hospital-based research). Thus, earlier assumptions regarding the role of hyperventilation as a pathway for psychosocial stress effects on asthma⁸⁻¹⁰ generally seem to find sufficient support on the level of self-reports from patients.

The association between psychological triggers and hyperventilation was not because of a general tendency of some patients being prone to report more triggers than others. Overall, across both samples, reported triggers accounted for substantial portions of the variance in symptom reports, ranging from 9% to 37% after controlling for standard demographics and asthma severity. However, psychological triggers still accounted for an additional 4% to 10% of the variance in hyperventilation symptoms after controlling for

reports of other trigger factors. Specific trigger factors, such as pollen and animal allergens, did not account for unique variance in hyperventilation symptoms or other symptom subscales of the ASC. Only reports of nonspecific trigger factors were more uniquely and consistently linked to symptom report, particularly the domain of infectious triggers. Because perceived symptoms are major determinants of quality of life,^{35,36} greater attention toward the management of nonspecific trigger avoidance may, therefore, result in palpable improvements in patients' well-being. Addressing the management of psychological triggers would involve psychosocial interventions to modify the experiential and behavioral pathways that affect the disease process. This could include the recommendation for breathing retraining that targets the patients' tendency to hyperventilate directly.³⁷

Psychological triggers were also not associated indiscriminately with any type of asthma-related symptoms and, thus, did not suggest an association with generally inflated symptom reports. Classic obstruction symptoms measured by the ASC showed no unique association with psychological trigger reports. However, the ATI psychological trigger scale was also strongly related to reports of mood symptoms of panic-fear and irritability during asthma symptom episodes. Considering that anxiety and panic states have been linked to reductions in end-tidal carbon dioxide (P_{CO_2}) suggestive of hyperventilation^{10,11,38} and that anger has been shown to increase ventilation and affect lung function adversely,^{21,39-41} a considerable overlap between these mood symptom domains and hyperventilation symptoms in asthma can be expected. Indeed, the empirical association typically found between ASC hyperventilation and mood symptoms is moderate to high.²⁷ Thus, self-report of psychological asthma triggers is

probably more generally associated with emotional response and distress during asthmatic symptom episodes.

Emotional arousal, particularly negative affect, has been shown to be sufficient to increase airway obstruction that reaches clinically relevant levels in many patients with asthma.^{4,42} Thus, emotional responses to an asthmatic symptom episode could constitute an additional factor contributing to the exacerbation, potentially leading to a vicious circle of lung function decline and distress.²⁷ A cross-sectional survey such as ours is not sensitive enough to inform about the exact temporal relationship between asthma triggers and symptoms. It may be that patients reporting psychological triggers also referred to mood symptoms, such as panic-fear and irritability, that are elicited by initial airway obstruction and contribute to the exacerbation as secondary triggers. This would be less likely for hyperventilation symptoms because there is no overt thematic overlap between this type of symptom and psychological trigger items of the ATI. More research is needed to determine the importance of the role of hyperventilation and emotional triggers in the causal chain of events leading to asthmatic symptom episodes or more severe asthma exacerbations.

Another limitation of our study is the pure self-report nature of the data. Although there is evidence that psychological trigger reports are also associated with stronger airway obstruction to experimentally elicited emotional states,²⁶ little is known about the association of reported hyperventilation symptoms with actual changes in blood gas levels of patients with asthma. It is not known whether those who report such symptoms would also respond to experimental bronchial provocation and daily life asthma exacerbations with hypocapnic breathing. Experimental and ambulatory studies that include monitoring of anxiety, symptoms, lung function, and P_{CO_2} ⁴³ would be needed to address this issue successfully. Nevertheless, the current findings suggest that an exploration of the link between psychological asthma triggers and hyperventilation with more advanced techniques is indicated and may help in shaping adjunctive behavioral interventions for this disease.

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