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Association of airflow limitation with trauma exposure and posttraumatic stress disorder

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Abstract:

Trauma exposure and posttraumatic stress disorder (PTSD) are associated with self-reported asthma and chronic obstructive pulmonary disease. However, these conditions have not yet been related to objective measures of lung function.

1772 adults from the general population were assessed regarding their medical histories and spirometric lung function. Additionally, they were administered a PTSD interview, and assigned to three groups: no trauma; trauma, but no PTSD; and trauma with PTSD.

Adjusting for sociodemographic, clinical and life-style factors, subjects with PTSD had significantly higher odds ratios (ORs) for most asthma-related symptoms than PTSD negative participants (ORs ranging from 3.2 to 8.8). The mean ratio of forced expiratory volume in one second (FEV1) to forced vital capacity (FVC) was lowest in the PTSD group and highest in those without trauma exposure. Traumatic stress was independently associated with FEV1 and FEV1/ FVC. Participants with PTSD compared to those without had a significantly increased risk for airflow limitation independent of its definition (ORs ranging from 4.2 to 7.8).

This is the first study relating traumatic stress and PTSD, respectively, to objective parameters of lung function. Our findings suggest an association of trauma exposure and PTSD with airflow limitation, which may be mediated by inflammatory processes.

Key words:

Airflow limitation; asthma, chronic obstructive pulmonary diseases, posttraumatic stress disorder; pulmonary function testing; trauma

Introduction

There is growing evidence for a close association between exposure to traumatic stress, e.g. childhood maltreatment or combat experience, and poor physical health, particularly pulmonary diseases such as asthma and chronic bronchitis [1-3]. For instance, studies of adult general population samples found a graded relationship between the numbers of childhood adversities and the risk for chronic obstructive pulmonary disease (COPD) [4-6] and asthma [7]. Likewise, traumatic experiences in adulthood are related to the development or worsening of asthma as indicated by research among adults exposed to the September 11, 2001 World Trade Center terrorist attack [8-10].

Posttraumatic stress disorder (PTSD) as the most common psychological sequel of traumatic experiences has also been linked to respiratory dysfunction [11-15]. For example, large community studies found that subjects with PTSD had a three times higher risk for asthma and COPD than those without PTSD [3, 11]. Similar results were found in primary care patients [16]. Likewise, PTSD positive male combat veterans had an increased risk to report chronic pulmonary diseases compared to PTSD negative ones [15]. Among female veterans, those with PTSD had 1.6 times higher risk for self-reported asthma than those without PTSD [13]. Another veteran study indicated that the strongest association between PTSD and other diseases including cardiovascular and digestive ones concerned asthma [14]. Finally, the association between PTSD symptoms and asthma remained after adjustment for familial, genetic and other confounders in a twin study [17].

In sum, consistent findings strongly suggest an association of trauma and PTSD with respiratory conditions characterized by airflow limitation. However, this relationship might be compromised by several methodological problems. First, the diagnoses of asthma or COPD were ascertained by self-report, but not corroborated by medical records or physicians' assessment. Due to the use of self-report of respiratory disorders, it remains unknown whether the observed association is solely the result of self-report bias, particularly as asthmatic and

PTSD positive subjects were found to inaccurately perceive and report symptoms and distress, possibly due to negative emotional states such as anxiety and depression often associated with both conditions [18-20]. Second, asthma-related symptoms resemble certain features of panic attacks (e.g., chest tightness, shortness of breath.) making it difficult to distinguish between these two conditions. Correspondingly, male combat veterans with PTSD did not have more commonly physician-diagnosed pulmonary diseases than those without PTSD [21], and the agreement between patients' and physicians' reports of respiratory dysfunction was only low in Vietnam veterans independent of their PTSD status [22]. Third, traumatic experiences and PTSD, respectively, have not yet been related to objective measures of lung function. Finally, many of the above mentioned investigations focused on either specific traumatic events or selected populations, e.g. childhood maltreatment [4, 6] or combat veterans, respectively [13-15, 17, 23], making it difficult to compare findings across various studies and raising the question of generalizability. Thus, general population studies, which include a wide variety of traumatic experiences, are important. In light of these considerations, the objective of our study was twofold: (i) to investigate the differential relationship of traumatic stress and PTSD, respectively, with self-reported symptoms and conditions of pulmonary disease in a general population sample controlling for potential confounders, and (ii) to assess the association between trauma exposure, PTSD and objective measures of lung function, i.e.forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and their ratio. Additionally, traumatic stress and PTSD were related to airflow limitation as determined by spirometry. However, because the prevalence of airflow limitation strongly depends on its definition, which is currently under intense debate [24], we applied different operationalizations [25-27].

Methods

Procedure and Subjects

Participants were recruited as part of the Study of Health in Pomerania (SHIP), which is an ongoing population-based project in northeastern Germany involving the three cities of Greifswald, Stralsund, and Anklam and 29 surrounding communities. The population density of this predominantly rural area is low and ranges between 50 and 1485 inhabitants per square kilometre. From the total population of 212157 inhabitants, a representative sample totalling 7008 persons aged 20 to 79 years was selected from population registries considering the inclusion criteria of German citizenship and residency in West Pomerania. In Germany, each inhabitant is obliged to be registered. Information on name, birthday and residence address is collected from the day of birth and afterwards. Any relocation has to be advertised. This data base can be used for scientific purposes, and the completeness of information allows drawing population samples with high grade of representativeness. The two-stage cluster sampling method was adopted from the WHO MONICA Project Augsburg, Germany, and yielded twelve 5-year age strata for both sexes, each including 292 individuals [28]. A total of 4310 individuals (2193 women), 68.8% of all eligible subjects, took part in the baseline study (SHIP-0), which was performed between October 1997 and May 2001. The study was approved by the local Ethics Committee. The detailed objectives and design of SHIP are published elsewhere [29]. In brief, data were collected in two medical centres specifically established for this study and located within the local hospitals. Participants were offered free transportation to the examination centres and back home, a meal and 15 Euros as incentives. The data collection comprised four parts: a health- and risk factor-related self-report questionnaire, an oral health examination, a medical examination, and a computer-assisted health-related interview. The latter was conducted by trained interviewers; additionally, there was a continuous quality monitoring [29].

The present study was part of the first five-year follow-up investigation (SHIP-I) performed between December 2002 and December 2006 [30], i.e. a cross-sectional study nested in a

long-term community-based cohort study. For SHIP-I, there were 130 passive non-responders due to migration, and 231 deceased subjects. Of the remaining 3949 eligible persons, 649 were active non-responders. Thus, a total of 3300 participants of the original study were followed up (83.6% response rate). The non-responders were significantly older, more often single, less educated, and unemployed [31]. Pulmonary function testing was offered to any subject volunteering in SHIP-I, and was finally carried out on a random subsample comprising 1809 subjects [32, 33]. Those performing spirometry were younger, less often separated, divorced or widowed, had a higher educational level, were less often current smokers, and had lower frequencies of trauma exposure and PTSD, respectively, than non-performers (results not presented in detail). Of the 1809 participants with spirometry data, 37 subjects had to be excluded for the following reasons: one individual (0.05%) did not complete the interview, another 23 (1.3%) exhibited cognitive impairment as defined by a Mini Mental State Examination score of 23 or below, one subject (0.05%) suffered from lung cancer, and 9 (0.5%) had incomplete pulmonary function testing data. Thus, 1772 adults living in the community were analyzed in the present study.

Psychological Assessment

The health-related interview of SHIP-I included the PTSD module of the Structured Clinical Interview for DSM-IV (SCID) [34], frequently administered by traumatic stress professionals [35], the Composite International Diagnostic-Screener (CID-S) [36], and the Mini Mental State Examination [37]. The PTSD interview begins by directly asking about the exposure to events included as traumas in DSM-IV (criterion A1). If a participant answered “no” to each of the trauma questions, the module was terminated. Otherwise the interview was continued to assess the DSM-IV PTSD symptoms including fear, helplessness, or horror as initial reaction (criterion A2), five re-experiencing symptoms (criterion B), seven avoidance symptoms (criterion C), and five arousal symptoms (criterion D). If participants did not pass the required

diagnostic threshold, the interview was terminated. Because there is evidence for a graded relationship between the number of adverse childhood experiences and the risk of obstructive airway disease [38], traumatic stress was not only treated as binary variable (absent = 0 vs. present = 1), but was also defined by the number of traumatic events meeting both DSM-IV A1 and A2 criteria.

The Composite International Diagnostic-Screener (CID-S) [36]was used to estimate the 12-months prevalence rate of panic attacks, anxiety and depression (27). It comprises items reflecting the so-called stem questions of the Composite International Diagnostic Interview assessing the core symptoms of the respective disorders. Panic attacks were classified in case subjects endorsed the corresponding item. Endorsement of at least one of six items reflecting anxiety and depression was used as proxy measure of negative emotional states. The sensitivity of the CID-S was reported to be 85.3% [36].

Cognitive functioning was assessed by means of the MMSE [37] before administering the other measures. MMSE scores of 23 or below were considered to indicate cognitive impairment, and participants not exceeding this cut-off were excluded.

Medical History of Lung Disease and Pulmonary Function Testing

Personal medical history was assessed by a computer-assisted personal interview including several asthma-related questions modified according to the interview applied within the European Community Respiratory Health Survey [39]. Finally, respondents were also asked if they had suffered from a physician-diagnosed chronic bronchitis or asthma in the year prior to the study. Medication was recorded according to the Anatomical Therapeutic Chemical (ATC) classification [40] and drugs of interest (ATC code R03) were treated as binary variables (no use = 0 vs. use = 1). Participants also underwent routine medical examination including anthropometric measurements to assess height and weight. Smoking status was classified as never smoking (= 0), former smoking (= 1), or current smoking (i.e. one or more

cigarettes per day; = 2). Marital status was subdivided into three categories: never married (= 0), married (= 1), and divorced, separated or widowed (= 2). Corresponding to the German school system, education was treated as dichotomous variable (10 years or less = 0 vs. 11 years or more = 1).

Pulmonary function testing was conducted using a bodyplethysmograph equipped with a pneumotachograph (VIASYS Healthcare, JAEGER, Hoechberg, Germany) that meets the American Thoracic Society criteria [41, 42]. The volume signal of the equipment was calibrated with a 3.0 liter syringe connected to the pneumotachograph in accordance with the manufacturer's recommendations, and this was performed at least once on each testing day. Barometric pressure, temperature and relative humidity were registered every morning. Calibration of the volume was examined under Ambient Temperature Pressure conditions and the integrated volumes were Body Temperature Pressure Saturated corrected [41, 42]. The participants performed at least three lung function maneuvers in order to obtain a minimum of two acceptable and reproducible values [26]. Immediate on-screen error codes indicating the major acceptability (including start, minimal duration and end of test) and reproducibility criteria supported the attempt for standardized procedures. Prior to the tests, the required maneuvers were demonstrated by the operator and the individuals were encouraged and supervised throughout the performance of the tests. The best results for FVC and FEV1 were taken. We also calculated percentage of predicted values of FEV1 and FVC based on sex-specific equations of the ECCS [26] and of the NHANES III [27]. Additionally, three definitions of airflow limitation were applied: (i) a fixed ratio of FEV1/ FVC $\leq$ 70% according to the recommendation of the Global Initiative for Chronic Obstructive Lung Disease [25], (ii) FEV1/ FVC values below the 5^{th} percentile according to the equation of the ECCS [26], and (iii) FEV1/ FVC values below the 5^{th} percentile derived from the equation of the NHANES III [27].

Statistical Analysis

All analyses were computed using the 'Statistical Package for the Social Sciences' (SPSS, version 14.0). We applied χ^2-test for categorical variables and analyses of variance for continuous variables, followed by post-hoc pairwise comparisons according to the method of Bonferroni. To determine the relationship of trauma and PTSD with self-reported respiratory symptoms and conditions we performed logistic regression analyses with these binary variables (present vs. absent) as dependent variable and trauma exposure and PTSD as independent variables; sex, age, height, marital status, education, smoking, panic attacks and negative emotional states were considered as potential confounders. The differential impact of traumatic stress and PTSD, respectively, on lung function parameters were analyzed by linear regressions with absolute and percentages of predicted values of FEV1, FVC, and the ratio of FEV1/ FVC as dependent variables. Significance level was set at $p < .05$.

Results

The study population comprised 909 women (51.3%) and 863 men (48.7%) with a mean age of 52.1 years (SD = 13.6; range: 25 – 86 years). Of the 1772 community residents included in the present study, 915 subjects (51.6%) had been exposed to at least one traumatic event. Twenty-eight participants met criteria for PTSD (1.6% of the total study population, and 3.6% of those with trauma exposure). Participants were assigned to one of the following groups: no trauma exposure (no trauma; n = 857), trauma, but no PTSD (trauma; n = 887), and trauma with the development of PTSD (PTSD; n = 28). These three subsamples differed significantly with respect to age, marital status, the frequency of panic attacks and negative emotional states as well as height (cf. Table 1).

Please insert Table 1 about here

Associations between trauma, PTSD and asthma-related variables are depicted in Table 2. PTSD positive participants reported the highest proportions, followed by those with trauma exposure, but without PTSD; those without traumatic experiences had the lowest proportions. Traumatized subjects had an increased likelihood to report awakening due to shortness of breath and an attack of coughing compared to participants without trauma exposure. Compared to residents without PTSD, the PTSD group had a significantly higher risk for almost all asthma-related symptoms.

Please insert Table 2 about here

Between-group comparisons revealed that non-traumatized participants had significantly higher values for both FEV1 and FVC than the two other groups, which did not differ from each other (cf. Table 3). The FEV1/ FVC ratio was significantly lower in traumatized subjects compared to the group of non-traumatized participants, while those with PTSD did not differ from the two other groups. There were no significant differences in the percentages of predicted values of FEV1 and FVC based on the ECCS equations between the groups. With respect to percentages of predicted values according to the NHANES equation, PTSD positive participants had significantly lower FEV1 values than the two other groups, and traumatized subjects had significantly lower FVC values than non-traumatized ones, while the PTSD group did not differ from the others. When defining airflow limitation according to the ECCS or by a fixed FEV1/ FVC ratio $\leq$ 70%, the PTSD group had a significantly increased risk for airflow limitation compared to residents without PTSD (OR = 7.8; 95% CI: 1.8-33.2, and OR = 4.2; 95% CI: 1.0-17.4, respectively). Traumatized participants did not have a higher risk for airflow limitation than those without trauma exposure.

Please insert Table 3 about here

To determine the associations of traumatic stress and PTSD, respectively, with lung function independent of other determinants, we performed linear regressions with absolute and percentages of predicted values of FEV1, FVC, and the ratio of FEV1/ FVC as dependent variables (cf. Table 4). Traumatic stress as defined by the numbers of traumatic events was significantly related to absolute FEV1 and the ratio of FEV1/ FVC; furthermore, it was associated with predicted values of FEV1 and FVC (ECCS equation) and with predicted FVC values (NHANES). There were no associations of PTSD with these parameters. Re-analyses of the data treating traumatic stress as binary variable (absent vs. present) did not yield substantially different results: PTSD was not associated with any measure of lung function; traumatic stress was related to the absolute value of FEV1 and the predicted values of FVC, but no longer to the FEV1/ FVC ratio nor to the predicted values of FEV1 (results not shown).

Please insert Table 4 about here

Discussion

To the best of our knowledge this is the first study relating traumatic stress and PTSD, respectively, to objective parameters of pulmonary function testing, thus extending prior research suggesting an association between trauma, PTSD and self-reported pulmonary disease [3, 8-15]. We found that PTSD was much more strongly linked with almost all asthma-like symptoms than mere trauma exposure alone, which is in line with other studies [3, 11-15]. Pulmonary function testing revealed that non-traumatized participants had significantly higher FEV1 and FVC values than those with trauma exposure and PTSD, respectively. The FEV1/ FVC ratio was significantly lower in the traumatized group

compared to those without traumatic experiences and PTSD; the same was true for the predicted FVC values according to the NHANES equation. Finally, PTSD positive participants had significantly lower predicted FEV1 values according to the NHANES equation than the two other groups that did not differ from each other. Regardless of its definition, airflow limitation was significantly more frequent in the PTSD group compared to those without PTSD even when controlling for relevant confounders. However, PTSD was not independently related to lung function parameters as revealed by linear regression analyses. In contrast, traumatic stress was associated with absolute values of FEV1 and predicted values of FVC.

Before discussing the mechanisms possibly linking traumatic stress and PTSD with airflow limitation, it has to be emphasized that our results are puzzling in that they do not follow a consistent pattern. While PTSD, but not trauma exposure was associated with airflow limitation when defined categorically, traumatic stress, but not PTSD was related to dimensional measures of lung function indicating airway obstruction. These inconsistencies deserve some considerations. From a statistical point of view, categorizations of continuous variables should be avoided [43]. Thus, our approach using the dimensional measures of lung function as outcome might be considered superior to the use of the dichotomous variable airflow limitation. However, a clear-cut distinction between normal and pathological values is preferred from a clinical perspective suggesting that airflow limitation as outcome is clinically more meaningful than dimensional spirometry values. Future studies will need to clarify which outcomes are adequate before it will be possible to resolve the controversial debate about the differential impact of traumatic stress and PTSD on physical health in general and airway obstruction in particular [3, 44, 45].

Several pathways to poor physical health have been suggested for both trauma exposure and PTSD including biological changes, engagement in poor health behaviour, and dysfunctional coping [1, 14, 45, 46]. All of these lead to increased allostatic load, defined as the cumulative

burden that an organism experiences due to repeated cycles of adaptation, eventually causing somatic changes that promote disease [47]. With respect to biological mechanisms, chronic traumatic stress and PTSD are characterized by changes in the hypothalamic-pituitary-adrenal axis and the sympathetic-adrenal-medullary system [48]. It was suggested that these alterations lead to a pro-inflammatory state [1, 14, 49, 50]. Thus, inflammation may be the link between trauma exposure and PTSD on the one hand and airflow limitation on the other which itself is associated with inflammatory processes [51].

Our study holds some major strengths including the population-based design, the controlling for potential confounders, the exclusion of individuals with cognitive impairment, the assessment of traumatic events and PTSD with a psychometrically sound interview [35] as well as the use of spirometric lung function testing. However, some methodological limitations merit discussion. First, because our investigation was cross-sectional, the reported associations do not allow any causal inferences. More specifically, information about the temporal relationship of respiratory symptoms and lung function findings relative to trauma exposure and PTSD onset was not available. However, there is evidence that PTSD positive subjects do not have a higher risk of physical illness before trauma exposure than those without PTSD [16] suggesting that traumatic experiences are likely to have predated airflow limitation in most cases. Nevertheless, we are in need of prospective studies to confirm this. Second, the lifetime prevalence of trauma exposure found in our study (51.6%) may appear high for a general population sample thus questioning whether it is representative. However, epidemiological studies in adult community populations have reported lifetime prevalences for traumatic events ranging between 20% and 90%, depending on the definition and assessment of traumatic experiences, the country where the study was conducted as well as the age range of the sample [30, 52]. In line with our results, a study of the adult general population in the Netherlands, found that 52.2% reported the experience of at least one traumatic event [53]. In contrast, the lifetime prevalence of PTSD in our investigation (1.6%

of the total sample) appears low compared to U.S. figures, e.g. 7.8% in the National Comorbidity Survey [54], but is very similar to the lifetime prevalence of 1.4% reported by another German general population study [55]. Again, differences in methodology and socioeconomic background of the study populations can explain divergent results [30]. Third, our general population sample cannot be considered representative for two reasons. The follow-up sample differed from the baseline sample [31], and there were significant sociodemographic and clinical differences between participants performing spirometry and those who did not. Fourth, panic attacks and negative emotional states were only assessed by a screening instrument, but not by specific measures or structured interviews. Furthermore, the additional assessment of the personality trait negative affectivity, reflecting the tendency to experience negative emotions [56], might have been useful as it is positively related to elevated symptom reporting [19]. Finally, small numbers of participants with PTSD and self-reported pulmonary diseases and objectively defined airflow limitation, respectively, may have compromised statistical power, i.e. the precision of risk estimates was low in some instances. Thus, we may have even failed to find 'true' associations between PTSD and measures of airflow limitation.

Notwithstanding these caveats, our study in concert with prior research strongly indicates an association of traumatic experiences and PTSD with obstruction of the respiratory system. From a clinical point of view, diagnostic and treatment attention should be paid to obstructive pulmonary diseases in traumatized and PTSD positive subjects both in primary care as well as in special medical and mental health settings. It was suggested that treatment of PTSD may reduce allostatic load and thus positively influences physical health [57]. Prospective studies are needed to further clarify the association between trauma, PTSD and respiratory dysfunction as well as the beneficial impact of PTSD treatment on pulmonary diseases.

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Statement of interest:

The authors do not report any conflicts of interest.

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Table 1. Sociodemographic and clinical characteristics of the study population

	No trauma (n = 857)	**Trauma** (n = 887)	**PTSD** (n = 28)	χ^2 / **F**	**p ≤**
Women, %	51.5	50.5	71.4	4.773	.092
Age, years [a]	49.3 ± 12.4	54.8 ± 14.0	54.8 ± 16.2	39.207	.001
Marital status, %				24.446	.001
Never married	16.8	13.2	10.7		
Married	71.6	69.2	53.6		
Separated, divorced, widowed	11.6	17.6	35.7		
School education, %				3.521	.172
≤ 10 years	82.6	80.6	92.9		
≥ 11 years	17.4	19.4	7.1		
Smoking status, %				3.335	.503
Never	43.7	44.1	32.1		
Former	34.4	36.3	39.3		
Current	21.9	19.6	28.6		
Panic attacks, %	7.4	10.6	39.3	34.801	.001
Negative emotional states, %	19.5	28.2	85.7	73.951	.001
Height, cm [b]	170.5 ± 9.0	169.1 ± 9.1	166.9 ± 9.4	6.165	.002
Weight, kg	80.2 ± 15.8	80.4 ± 15.9	79.9 ± 16.2	0.029	.972

PTSD: posttraumatic stress disorder

[a] Post-hoc pairwise comparisons (Bonferroni) indicated that traumatized subjects were significantly older than non-traumatized participants.

[b] Post-hoc pairwise comparisons (Bonferroni) indicated that traumatized subjects were significantly smaller than non-traumatized participants; PTSD positive subjects did not differ from the two other groups.

Table 2. Association between self-reported respiratory symptoms and conditions, trauma exposure and PTSD

Self-reported respiratory symptoms and conditions, %	**No trauma** (n = 857)	**Trauma** (n = 887)	**PTSD** (n = 28)	χ^2	p	**Trauma** OR (95% CI) [a]	**PTSD** OR (95% CI) [a]
Wheeze without having a cold	3.4	4.6	17.9	14.678	.001	1.1 (0.9-1.4)	2.8 (1.0-8.3)#
Woken with chest tightness	4.6	4.7	28.6	33.104	.001	1.1 (0.9-1.4)	3.2 (1.3-8.0)*
Woken with shortness of breath	2.0	2.9	25.0	52.461	.001	1.5 (1.1-1.8)**	4.9 (1.8-13.8)**
Woken by an attack of coughing	5.8	9.2	39.3	44.454	.001	1.2 (1.0-1.4)*	3.7 (1.6-8.5)**
Asthma attack	0.7	1.2	14.3	42.780	.001	1.2 (0.8-1.8)	8.8 (2.4-32.5)***
Medication (ATC code R03)	2.8	5.9	17.9	20.881	.001	1.2 (1.0-1.5)#	3.6 (1.2-10.5)*
Asthma	1.6	3.2	10.7	11.767	.003	1.2 (0.9-1.5)	3.2 (0.9-11.9)*
Chronic bronchitis	3.7	5.0	17.9	13.016	.001	1.1 (0.9-1.3)	2.6 (0.9-7.7)#

PTSD: posttraumatic stress disorder; OR: odds ratio; CI: confidence interval, ATC: Anatomical Therapeutic Chemical classification

[a] Adjusted for age, sex, height, marital status, education, smoking, panic attacks, and negative emotional states

$p \leq .10$ * $p \leq .05$ ** $p \leq .01$ *** $p \leq .001$

Table 3. Comparison of pulmonary function testing between the three subsamples

	No trauma (n = 857)	**Trauma** (n = 887)	**PTSD** (n = 28)	**F/ χ^2**	**p ≤**
FEV1, l [a]	3.45 ± 0.88	3.17± 0.88	2.84 ± 0.90	25.411	.001
FVC, l [a]	4.04 ± 1.03	3.76 ± 1.01	3.38 ± 0.95	20.207	.001
FEV1/ FVC, % [b]	85.4 ± 6.0	84.4 ± 6.5	83.2 ± 9.3	6.877	.001
FEV1 % pred (ECCS) [c]	109.4 ± 15.5	107.7 ± 16.7	102.2 ± 19.1	4.544	.011
FVC % pred (ECCS) [c]	106.4 ± 14.4	104.7 ± 15.4	101.7 ± 17.7	3.525	.030
FEV1 % pred (NHANES) [d]	102.4 ± 14.2	100.9 ± 15.3	95.4 ± 17.3	4.775	.009
FVC % pred (NHANES) [b]	94.4 ± 12.5	92.7 ± 13.1	89.4 ± 15.2	5.480	.004
AL (fixed ratio ≤ 70%), %	1.4	2.7	10.7	13.031	.001
AL (ECCS), %	1.1	2.0	10.7	16.422	.001
AL (NHANES III), %	0.9	1.6	7.1	8.491	.014

PTSD: posttraumatic stress disorder; FEV1: forced expiratory volume in one second; FVC: forced vital capacity; FEV1 % pred (ECCS): % of predicted FEV1 values according to the ECCS equation; FVC % pred (ECCS): % predicted FVC values according to the ECCS equation; FEV1 % pred (NHANES): % of predicted FEV1 values according to the NHANES equation; FVC % pred (NHANES): % of predicted FVC values according to the NHANES equation; AL (fixed ratio): airflow limitation as defined by the fixed ratio of FEV1/ FVC ≤ 70%; AL (ECCS): airflow limitation as defined by FEV1/ FVC values below the 5th percentile according to the equation of the European Community for Coal and Steel (ECCS); AL (NHANES III): airflow limitation as defined by FEV1/ FVC values below the 5th percentile according to the equation of the National Health and Nutrition Examination Survey (NHANES III)

[a] Post-hoc pairwise comparisons (Bonferroni) indicated that non-traumatized subjects had significantly higher values than the two other groups, which did not differ from each other.

[b] Post-hoc pairwise comparisons (Bonferroni) indicated that traumatized subjects had significantly lower values than non-traumatized participants; PTSD positive subjects did not differ from the two other groups.

[c] Post-hoc pairwise comparisons (Bonferroni) did not reveal any group differences.

[d] Post-hoc pairwise comparisons (Bonferroni) indicated that PTSD positive subjects had significantly lower predicted values than non-traumatized participants who did not differ from the traumatized group.

Table 4. Differential association of trauma exposure and PTSD with absolute and percentage of predicted values of FEV1, FVC and FEV1/ FVC (linear regression analyses)

	FEV1, l [a]	**FVC, l [a]**	**FEV1/ FVC [a]**	**FEV1 % pred (ECCS) [b]**	**FVC % pred (ECCS) [b]**	**FEV1 % pred (NHANES) [b]**	**FVC % pred (NHANES) [b]**
	β	**β**	**β**	**β**	**β**	**β**	**β**
Age, y	-.448 ***	-.403 ***	-.211 ***	-	-	-	-
Sex (reference: women)	.262 ***	.285 ***	-.081 *	-	-	-	-
Height, cm	.446 ***	.477 ***	-.077 *	-	-	-	-
Marital status (reference: never married)							
married	.025	.021	.014	.039	.001	-.004	-.098 **
divorced	.020	.024	-.023	.052	.055	-.006	-.074 *
Education	.009	.009	-.010	-.002	.005	.012	.031
Smoking (reference: never)							
former	-.041 **	-.041 **	-.020	-.101 ***	-.146 ***	-.049	-.058 *
current	-.070 ***	-.031 *	-.161 ***	-.146 ***	-.090 ***	-.118 ***	-.021
Asthma/ bronchitis	-.040 **	-.029 *	-.037	-.065 *	-.056 *	-.060 *	-.053
Medication	-.064 ***	-.032 *	-.166 ***	-.146 ***	-.078 **	-.154 ***	-.091 ***
Panic attacks	.008	.019	-.039	.025	.067 **	.015	.050 *
Negative emotional states	-.022	-.020	-.006	-.035	-.013	-.050 *	-.042

Table 4. Differential association of trauma exposure and PTSD with absolute and percentage of predicted values of FEV1, FVC and FEV1/ FVC (linear regression analyses) - continued

Traumatic stress	-.034 *	-.019	-.063 **	-.062 **	-.077 ***	-.036	-.054 *
PTSD	-.003	-.008	.005	-.015	-.014	-.017	-.015
Model estimates							
F	337.64	362.89	18.06	11.62	8.18	9.80	5.79
$p \leq$	.001	.001	.001	.001	.001	.001	.001
R^2	.727	.741	.119	.062	.043	.052	.029

FEV1: forced expiratory volume in one second; FVC: forced vital capacity; FEV1 % pred (ECCS): % of predicted FEV1 values according to the ECCS equation; FVC % pred (ECCS): % predicted FVC values according to the ECCS equation; FEV1 % pred (NHANES): % of predicted FEV1 values according to the NHANES equation; FVC % pred (NHANES): % of predicted FVC values according to the NHANES equation; PTSD: posttraumatic stress disorder; R^2: explained variance

[a] Linear regression analysis adjusted for age, sex, height, marital status, education, smoking, asthma/ bronchitis, medication, panic attacks and negative emotional states

[b] Linear regression analysis adjusted for marital status, education, smoking, asthma/ bronchitis, medication, panic attacks and negative emotional states

* $p \leq .05$ ** $p \leq .01$ *** $p \leq .001$