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Violence exposure, a chronic psychosocial stressor, and childhood lung function

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Abstract

Background—Chronic psychosocial stressors, including violence, have been linked to neuropsychological and behavioral development in children as well as physiologic alterations that may lead to broader health effects.

Methods—We examined the relationship between violence and childhood lung function in a prospective birth cohort of 313 urban children 6 and 7 years of age. Mothers reported on their child's lifetime exposure to community violence (ETV) and interparental conflict in the home [Conflict Tactics Scale (CTS)] within one year of the lung function assessment.

Results—In linear regression analyses, adjusting for maternal education, child's age, race, birthweight, tobacco smoke exposure, and medical history, girls in the highest CTS verbal aggression tertile had a 5.5% (95% CI: −9.6, −1.5) decrease in percent predicted FEV₁ and a 5.4% (95% CI: −9.7, −1.1) decrease in FVC compared to girls in the lowest tertile. The CTS verbal aggression subscale was associated with lung function among boys in the same direction, albeit this was not statistically significant. Boys in the highest ETV tertile had a 3.4% (95% CI: −8.0, 1.1) lower FEV₁ and 5.3% lower (95% CI: −10.2, −0.4) FVC compared to boys in the lowest tertile. The ETV score was not a significant predictor of girl's lung function.

Conclusions—Interparental conflict, specifically verbal aggression, and exposure to community violence were associated with decreased childhood lung function independent of socioeconomic status, tobacco smoke exposure, birthweight and respiratory illness history. Gender differences were noted based on the type of violence exposure which may warrant further exploration.

Keywords

lung function; violence exposure; children

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Background

Early life factors contributing to lung growth are of interest given that childhood lung function is important to the development of chronic obstructive pulmonary disease (COPD) in later life (1). The public health implications are substantial as COPD is projected to be the fourth leading cause of death worldwide by the year 2020 (2). Longitudinal studies of the natural history of lung function to date have shown that a number of early life risk factors including asthma (3), active and passive smoking prenatally and during childhood (4, 5), atopy (6), lower respiratory infections (7), and perinatal factors including birthweight, gestational age, and nutrition (8, 9) are associated with reduced lung function over the life course. However, these factors account for a relatively small proportion of the risk suggesting that as yet unidentified risk factors deserve further exploration.

Overlapping research from a number of areas suggest that negative emotion and psychological stress influence respiratory processes and lung function, having both transient (10, 11) and more long-term effects (12). These findings have been described in asthmatics and normal controls as well as both children and adults, albeit studies demonstrating more long-term effects have focused largely on adults.

The mind-body paradigm linking psychological stress and affective states to key physiological mechanisms (e.g. neuroendocrine and immune functioning, oxidative stress, autonomic response) and ultimately to disease expression provides a framework to explore plausible physiologic mechanisms through which psychological stress may operate to influence lung function (13). Factors known to induce chronic persistent inflammation (e.g. tobacco smoke exposure, asthma, air pollution, viral respiratory infections) are recognized risk factors for reduced lung function (14, 15). Notably, imbalance of physiologic processes that lead to chronic inflammation (e.g. hypothalamic-pituitary-adrenal (HPA) axis function, the proteases-antiproteases, the oxidants-antioxidants) can occur both in the face of these known risk factors (15) and under chronic stress (13). In humans, altered cortisol expression has been negatively associated with lung function (16). Chronic caregiver stress in the postpartum and early childhood period have been associated with persistent wheeze in early childhood (17) as well as factors that may initiate or potentiate inflammation in the lung (e.g. IgE expression, enhanced nonspecific and allergen-specific lymphocyte proliferation, differential cytokine expression) (18, 19). Both persistent wheeze and atopy have been linked to reduced lung function in childhood (20). Chronic psychological stress has also been linked to other risk factors that are associated with reduced lung function including asthma (13), respiratory infections (21, 22), and smoking behaviors. Other studies have associated lung growth and development with broader social constructs known to be associated with increased psychosocial stress and more negative affect (e.g., lower socioeconomic status) (23–25).

Recent research considers exposure to violence, conceptualized as a specific chronic psychological stressor, as a major cause of childhood morbidity in urban communities, including respiratory outcomes (26). The notion that experiencing episodic violence leads to chronic psychological stress is grounded in trauma theory (27). In general, stressors are thought to influence the pathogenesis of disease by causing negative affective states [e.g., anxiety, depression, and post-traumatic stress disorder (PTSD)] which in turn exert direct effects on biological processes or behavioral patterns that influence disease risk (28). When environmental demands are perceived to be threatening and exceed one's abilities to cope or when one feels that events are unpredictable or that one has no control, individuals experience stress. Children and families living with violence are likely to view their world and their lives to be out of their control. Living in an unpredictable or uncontrollable environment associated with a chronic pervasive atmosphere of fear and perceived threat

predisposes these populations to greater deleterious effects of stress (26, 27). A growing body of research, conceptualizing violence exposure as a chronic psychosocial stressor, documents the adverse psychological, behavioral and physiological consequences on children growing up in chronically violent neighborhoods and homes (26, 29–32).

Frequency of experiencing and witnessing serious and lethal violence among inner-city youth are high, particularly among lower-SES urban communities in the United States (33). In addition, estimates of domestic violence against women and children indicate widespread exposure to violence in the home (34). Given this, research focused on understanding the possible broader pediatric health effects of growing up with violence is of great public health importance.

We examined the association between violence exposure, conceptualized as a chronic psychological stressor, and lung function among urban children adjusting for SES, in utero and second hand tobacco smoke (SHS) exposure, birthweight and history of respiratory illness including asthma, allergies, and lower respiratory tract infections which may mediate this relationship.

Methods

Study Population

Participants were drawn from the Maternal-Infant Smoking Study of East Boston, a prospective cohort designed to study the effects of pre- and postnatal tobacco smoke exposure on childhood lung growth and development and respiratory health [for details see (4)]. In brief, pregnant women receiving prenatal care (< 20th week of gestation) at an urban community health center in Boston, Massachusetts between March 1986 and October 1992 were eligible for enrollment. Women who did not speak English or Spanish, who did not plan to have pediatric follow-up at the clinic, or who were less than 18 years of age were excluded. One thousand women were eligible and enrolled, of whom 848 continued participation and delivered a live-born infant. In 1996–1997, new study initiatives were implemented, including the assessment of social stressors, at which time 500 women and their children continued active follow-up. The study was approved by the human studies committees at the Brigham & Women's Hospital and the Beth Israel Deaconess Medical Center.

Violence Exposure Assessment

Violence exposure was ascertained using a modified version of the *Survey of Children's Exposure to Community Violence (ETV)* (29). The ETV questionnaire gathers data on both direct victimization and witnessing violence as well as factors known to influence the impact of violence (e.g. familiarity with the perpetrator or victim, events occurring more than once and whether the events occurred at home). The survey measured lifetime exposure to specific events including hearing gunshots, and witnessing and/or experiencing shoving, hitting or punching, knife attacks, or shootings. Mothers were asked to report on their child's lifetime exposure to violence. Among subjects continuing participation in 1996–1997, 412 mothers completed the violence assessment. Those who did not participate in the violence assessment were more likely to be non-Hispanic (52% non-responders vs. 45% responders) and current smokers (42% non-responders vs. 28% responders). In addition, 171 children who were at least eight years of age reported on their own lifetime community violence exposure on the ETV. Acceptable two week test-retest reliability in a random sample of mothers and their children has been demonstrated for the violence survey in this cohort (35).

Mother's also completed the *Conflict Tactics Scale (CTS) Form R* (36) designed to measure the extent to which her spouse or partner engaged in psychological and physical attacks

against her in the past year. Internal consistency, test-retest reliability, and validity have been established for the CTS including the Spanish translation (37). The CTS was scored according to published guidelines - subscales were calculated for verbal aggression and physical violence as follows (38). The verbal aggression subscale consisted of seven items (e.g., insulting or swearing at a partner, destroying a belonging, threatening to hit a partner). Minor physical violence consisted of three items (e.g. throwing something at a partner, pushing or slapping). Severe physical violence included six items (e.g. did your partner kick, hit, choke or beat you or use a knife or gun against you). Participants indicated whether a behavior had occurred never, once, twice, 2–5, 6–10, 11–20, or more than 20 times in the past year. Subscales were calculated by converting the frequency categories to a 0 to 6 scale and summing the items within each of the three subscales. Among those reporting any of the major violence events (n=91), nearly all (n=83) also reported minor violence consistent with prior research (38). Because of the significant overlap between the minor and major violence subscales these were combined into a composite scale for any physical violence. Scores on the CTS subscales ranged from 0 to 30, [mean $\pm$ standard deviation (SD)] (12.3 ± 7.8) on the verbal aggression subscale and from 0 to 39 (3.3 ± 6.1) on the physical violence subscale.

Lung Function Measures

Lung function was measured using a Morgan spirometer (P.K. Morgan, Andover, MA) when children were between 6 and 7 years of age, within one year of the violence assessment. Standing height without shoes was measured prior to spirometry. The lung function protocol was developed by an experienced pulmonologist and pediatric pulmonary function technologist. The children were not naïve to spirometry as they had been participating in lung function testing from the time they were 4 years old in anticipation of formal testing at age 6 to 7 years. A trained pulmonary function technologist instructed children during testing and monitored the flow-volume curve to ensure good effort. Maneuvers were repeated to obtain three acceptable curves; data from each technically acceptable effort were stored following established guidelines (39, 40). Forced expiratory volume in one second (FEV₁), forced vital capacity (FVC) and forced mid-expiratory flow rate (FEF_{25–75%}) were measured from the best acceptable blow (39).

Covariates

Demographic characteristics, as well as smoking and medical history were ascertained through standardized questionnaires. Maternal education level was categorized as less than high school, high school graduate or technical school graduate, or some college. Low birthweight was defined as less than 2500g.

At each clinic visit during pregnancy, mothers were asked about their smoking status and a urine specimen was obtained for determination of a creatinine-corrected cotinine (4). Mothers were classified as non smokers if they always reported not smoking and each of their urine cotinine levels were < 200 ng/mg creatinine (4). If the report of nonsmoking by the mother was contradicted by the urine cotinine, the mother was classified as a current smoker for that interval. Maternal reported postnatal SHS exposure was assessed monthly through age 26 months, every 6 months between 26 months and 4 years, and annually thereafter. Children were considered to be exposed to SHS at each follow-up interval if the mother reported active smoking or smoking by any other person living in the household. Postnatal SHS was categorized as early only (occurring from birth to 25 months of age) and both early and late as there were few children (n= 20) in the late only category (26 months or older). Also, prenatal smoke exposure was highly correlated with postnatal SHS exposure (i.e., of 66 children exposed to in utero tobacco smoke, only one was not exposed to SHS after birth).

Information on respiratory illness history was ascertained using the standardized ATS-DLD-78-C questionnaire (41). History of childhood asthma was determined based on parental report of physician diagnosed asthma. Mother's were also asked if their child was ever diagnosed with hay fever, eczema or if they received allergy shots up until the time that pulmonary function testing occurred. Presence of allergies was dichotomized as none, or one or more (eczema, hay fever or allergy shots). In addition they were asked whether the children had had a lower respiratory tract infection (croup, bronchitis, bronchiolitis or pneumonia) on the monthly follow-up questionnaires administered from birth to the time the child was two years old. Lower respiratory infections were categorized as having had none, one to two and three or more in that interval.

Statistical Analysis

Lung function measurements were available for 330 children who also had information on violence exposure. Among the children for whom violence exposure data were available, there were no significant differences between those who participated in the lung function tests and those who did not based on socio-demographics, medical history, tobacco smoke exposure or violence exposure. Seventeen children were missing data on, birthweight, mother's education level or in utero tobacco exposure, leaving 313 children for analyses.

We implemented Rasch modeling techniques to summarize responses to the community ETV questionnaire into a continuous score as previously described (35, 42). The continuous ETV measure was obtained by modeling the conditional probabilities of responding yes to each violence question given the extremity of each question and the true, but unobserved, violence exposure level of each person. The model was generalized to account for salient features of each event (e.g., whether the event occurred once or more than once, whether the child knew the victims or perpetrators) (43) and to include both the mother and child's report of events in a multi-informant model (44). The Rasch model produces two scores, one based on the parent's responses to the violence survey, the other on the child's responses to the survey. If a child is missing his or her self-report the model predicts the rasch score based on the existing correlation between the parent and child responses in the remainder of the cohort, so that all children have two scores. The final violence score is the average of the two rasch scores. This is a desirable approach here as children are unlikely to be able to recall events occurring during very early childhood at the same time that parents may be more likely to underreport events that occur when children are older and violence occurs outside of the supervision of parents (45, 46). As violence was assessed within one year of lung function testing, some subjects reported experiences that may have occurred after lung function was measured. For each item on the ETV asking about a particular event, subjects and their mothers were asked a follow-up question to report how old the child was when each reported event had occurred. Only those events reported prior to the lung function measurement were included in the Rasch model. In prior research, construct validity using Rasch modeling to summarize the multi-item survey of exposure to community violence demonstrates that the summary measure is moderately to strongly associated with aggressive behavior, delinquency, and psychological comorbidities (e.g., anxiety, depression) in a positive direction as expected (47, 48). We previously reported the inverse relationship between socio-demographic factors and the Rasch ETV in this cohort (35), corroborating other research (46, 48). As a further validity check, we assessed the relationship between the Rasch ETV scale and the parent-report version of the Checklist of Children's Distress Symptoms (CCDS) (49) which had been administered contemporaneously with the violence assessment in this sample. The CCDS is a 24-item measure of children's distress symptoms (including posttraumatic stress symptoms) occurring in the previous 6 months, with higher scores indicating greater distress. In these data, the continuous ETV measure was positively correlated with scores on the CCDS (Spearman $r=0.31$ $p<0.0001$).

Estimated effects of lung function are reported using percent predicted lung function (based on height, age, gender and race/ethnicity) as the independent variable. To obtain the predicted lung function values for each child we regressed the log transformed lung function measures against log height, age, gender and race/ethnicity. The effect of the violence measures on lung function were estimated using multiple linear regression adjusting for mother's education level, child's age, race/ethnicity, birthweight, pre- and postnatal smoking exposure. All models were stratified by gender. The Rasch community ETV measure and the CTS subscales were entered as continuous measures (per one standard deviation change) and also considered tertiles to assess for a threshold effect or a dose-response relationship (50). To evaluate whether lower respiratory infections and having a history of asthma or allergies would attenuate the association between violence exposure and lung function we conducted additional regressions further adjusting for these variables. Model assumptions were assessed using regression diagnostics. All analyses were conducted in SAS version 9.0 (SAS Institute, Cary, NC).

Results

Fifty percent of the children were females, 51% Hispanic, 39% of the mothers had not completed high school and 21% of the mothers smoked during pregnancy (Table 1). The average age for both boys and girls (mean $\pm$ SD) at the time of lung function testing was 6.2 $\pm$ 0.3. The mean $\pm$ SD lung function results for girls were FEV₁ 1.25L $\pm$.20, FVC 1.35L $\pm$.22 and FEF_{25-75%} 1.81L/sec $\pm$.40 and for boys were FEV₁ 1.31L $\pm$.21, FVC 1.42L $\pm$.25 and FEF_{25-75%} 1.84L/sec $\pm$.44. There were no significant differences in the level of exposure on the violence scales when comparing boys and girls. Scores on the continuous scales (mean $\pm$ SD) comparing boys and girls respectively were 1.08 $\pm$ 0.09 vs. 1.11 $\pm$ 1.1 on the ETV, 1.44 $\pm$ 1.0 vs. 1.63 $\pm$ 0.9 on the CTS verbal aggression subscale, and 0.46 $\pm$ 0.9 vs. 0.61 $\pm$ 1.1 on CTS physical violence subscale.

Unadjusted analyses are presented in Table 1. In multivariate models (Table 2) a one standard deviation increase in the verbal aggression scale significantly predicted a 2.8% decrease [95% confidence interval (CI)] (95% CI: -4.6, -1.0) in FEV₁ and a 2.5% decrease (95%CI: -4.4, -0.6) in FVC among girls. Girls exposed to tobacco smoke exposure in-utero had a 6.4% (95%CI: -12.1, -0.7) decrease in FEV₁, those exposed to early second hand smoke up to 25 months of age had a 7.0% decrease (95%CI: -13.1, -0.9) and those exposed to both early and late tobacco smoke postnatally had a 4.5% decrease (95%CI: -9.1, 0.04) in FEV₁ compared to girls not exposed. Similar decreases were noted in FVC among girls. No significant associations were noted with tobacco smoke exposure among boys (data not shown).

In multivariate analyses of the violence categorical variables, girls exposed to the highest tertile of verbal aggression had significantly lower FEV₁ 5.5% [(95% CI: -9.6, -1.5), p for trend .007], and FVC 5.4% [(95% CI: -9.7, -1.1), p for trend 0.01], compared to girls in the lowest tertile (Figure 1); girls in the highest tertile of physical violence exposure at home had also lower FEV₁ [(4.0%, 95%CI: -8.4, 0.4) p for trend 0.05] and FVC [(2.6%, 95%CI: -7.3, 2.2), p for trend 0.13]. The ETV indicator was not significantly related to lung function among girls. Among boys, exposure to the highest tertile of the ETV scale compared to the lowest was associated with decreases in FEV₁ [(3.4%, 95% CI: -8.0, 1.1), p for trend 0.14] and FVC [(5.3%, 95% CI: -10.2, -0.4, p for trend 0.03)] (Figure 2). Higher scores on the verbal aggression CTS subscale were also associated with reduced lung function in boys although these associations did not reach statistical significance. Boys in the 2nd tertile of physical violence exposure had lower FEV₁ (-4.0%, 95% CI: -8.7, 0.7) and FVC (-5.0%, 95%CI: -10.1, 0.1) compared to boys in the lowest category, however boys in the highest tertile had a higher FEV₁ (3.3%, 95%CI: -2.1, 8.7) and FVC (2.1%

95% CI: -3.8, 7.9) than boys in the lowest category of exposure. No significant associations between FEF_{25-75%} and the verbal aggression, physical violence CTS subscale or ETV were found.

Upon further adjustment for asthma diagnosis, lower respiratory infections or presence of allergies the associations previously seen between the violence indicators and lung function did not change. To confirm that the associations seen were not due to the tertile categorization of the violence variables we repeated these analyses categorizing the violence variables by the median split and quartiles; the associations remained qualitatively similar.

We further explored two additional models, 1) jointly modeling the verbal aggression scale and the ETV scale and 2) jointly modeling the ETV scale and the physical violence scale. The independent significant associations noted when examining the verbal aggression scale remained statistically significant even after inclusion of the community ETV scale in the model. The effect estimates seen when independently examining the ETV scale and the physical violence scale did not change after inclusion of these two variables together in the model. Due to concerns with collinearity we did not include all three measures in one model as the verbal and physical violence scales were highly correlated ($r=0.65$, $p < 0.0001$).

Discussion

In this urban cohort, maternal-report of community violence and parental conflict in the home were significantly associated with early childhood lung function even after adjusting for tobacco smoke exposure, race/ethnicity, birthweight, socioeconomic status and relevant respiratory health factors including, asthma, allergies and respiratory infections. Gender differences were observed suggesting differential effects of the type of violence exposure on lung function in that, among girls, indicators of parental conflict in the home had the greatest effect while among boys, the community violence exposure seemed to have the stronger influence.

The observed reductions in childhood lung function in relation to violence exposure measures were of comparable magnitude to effect estimates reported for prenatal tobacco smoke exposure and postnatal environmental tobacco smoke exposure both in our data and from other studies (51, 52). In the Six cities studies, Cunningham et al reported a 1.3% decrease in FEV₁, 0.2% decrease in FVC and 4.9% decrease in FEF_{25-75%} among children who were exposed to prenatal tobacco smoke exposure compared to those not exposed. Among children exposed to postnatal tobacco smoke a decrease of 0.1% FEV₁, 0.1% increase in FVC and a 0.6% decrease in FEF_{25-75%} was reported compared to children not exposed (52).

A number of pathways can be considered when linking violence and lung function. Violence exposure has increasingly been conceptualized as a chronic social stressor (26). Psychological stress has been associated with the activation of the sympathetic and adrenomedullary (SAM) system and the hypothalamic-pituitary-adrenocortical (HPA) axis (13). Negative emotional responses disturb the regulation of the HPA axis and the SAM systems; that is, in the face of stress, physiological systems may operate at higher or lower levels than during normal homeostasis. It is the disturbed balance of these systems that is relevant to disease. Immune, metabolic, and neural defensive biological responses important for the short-term response to stress, may produce long term damage if not checked and eventually terminated (53). Disturbed regulation of both the HPA axis and the SAM system related to chronic stress suggests that immune function, which is modulated by these systems, may also be dysregulated and this may have implications for respiratory processes including lung growth and development (13). Overlapping research in other studies has

demonstrated that exposure to violence is associated with disrupted cortisol response in children (32, 54). And finally, altered cortisol expression has been found to be negatively associated with lung function in adults albeit this has not been studied in children (16).

Additionally, chronic stress, may operate through pathways that have been hypothesized for other environmental toxicants influencing lung function (13). For example, tobacco smoke contains a number of compounds with oxidative potential (55). Spiteri et al (56) proposed that differences in host detoxification provide the basis for either resolution or progression of inflammation in atopic individuals after exposure to an environmental trigger such as allergens or air pollutants. This hypothesis may be extended to other environmental factors such as psychological stress that may augment oxidative toxicity and increase airway inflammation. There are studies to suggest that psychological stress augments oxidative toxicity. Emotionally stressed rats have increased levels of 8-deoxy-hydroxy-guanosine (8-OHdG), a commonly used biomarker of oxidative stress (57). Similarly, staying awake all night increased the levels of thiobarbituric acid reactive substances (an indicator of lipid peroxidation) in humans (58). It is plausible that chronic stress may enhance airway inflammation through similar mechanisms to influence lung function.

Possible explanations for the observed gender differences based on type of violence exposure may be grounded in child development research suggesting that factors may modify the impact of violence events occurring at home and in the community differently for boys and girls. Gender differences in psychological distress associated with marital or family discord have been demonstrated. Specifically, evidence suggests that girls possess a greater propensity than boys to become directly involved in parental conflict situations (59). Girls may be more susceptible than boys to adverse effects of family conflict in part because girls are more sensitive to affective cues and the states of others or because of identification with their mother who is commonly the one being abused. Other research finds that aggressive responses to stressors including community violence exposure are more common among boys than among girls which may lead to a more direct impact of community violence among boys (60). Boys are more likely to confront violent situations alone and with aggression while girls are more likely to respond with greater depressive symptomatology and seek out support in addressing problems (61). Such differential behavioral and psychological responding based on gender may also translate into varied disruption of underlying physiological stress pathways (e.g., HPA axis, nervous system reactivity) that, in turn, may have varied influence on physical health outcomes.

Moreover, the effects of violence exposure on health outcomes may also be mediated through the influence of violence on emotional development. In overlapping research, early life caregiving experiences and trauma exposures have been shown to impact childhood emotional understanding and expression (62, 63) with some studies showing differential gender effects (64). Children exposed to violence and neglect in their environment have been found to express higher levels of negative emotion (anxiety, depressive mood, anger, hostility, and irritability) and more adverse stress reactivity (65, 66). Negative emotion, in turn, has been linked to impaired lung function in both cross-sectional (67) and longitudinal analyses (12) while positive emotion seems to be protective (68).

It is also the case that methodological issues could partially explain the observed differences, in that boys may be more likely (relative to girls) to report experiences of violence, however we found no difference in mean levels of violence scores based on gender in these analyses. We also noted more robust associations among girls than boys, particularly when continuous exposure indicators were utilized. Girl's lung function was associated with conflict in the home which was measured over the past year, boy's lung function was associated with neighborhood violence which was measured over the lifetime.

It is possible there is less accurate recall among lifetime measures of exposures leading to more random error which could lead to weaker associations. The observed U-shape relationship between witnessing physical violence at home and lung function among boys, was unexpected. While this is possibly a spurious association, existing literature has documented inverted U-shaped associations between stress and task performance consistent with Yerkes-Dodson (1908) model of stress and learning (69, 70). However, it is difficult to conclude that higher levels of violence exposure lead to better lung function in this case. If such a relationship holds in future research it will likely be more informative to also explore other characteristics of individuals showing enhanced functioning in the setting of violence exposure (e.g., resiliency factors, stress buffers, emotion regulation).

The present findings may be relevant to other empirical studies demonstrating an inverse association between childhood social status indicators and pulmonary function. Childhood SES predicted both baseline lung function and rates of decline among young adults in the Coronary Artery (Disease) Risk Development in (Young) Adults (CARDIA) study (24) as well as in older women in the British Women's Heart and Health Study (25), when controlling for current SES, childhood tobacco smoke exposure and childhood asthma. Poor housing and neighborhoods are associated with increased exposure to environmental factors that may affect lung growth and development (e.g. chronic dampness, vermin, cockroaches, tobacco smoke, air pollution) (71, 72). Marginalized populations of lower SES disproportionately exposed to these physical environmental toxicants may also experience elevated psychosocial stressors such as violence exposure (26). Differential exposure to life stressors including violence may partially explain socioeconomic disparities in health (73) and thus warrants further attention.

We acknowledge a number of strengths and weaknesses in these analyses. The rigorous protocol used to assess lung function is a particular strength in this cohort. We were able to control for a number of covariates that have been associated with both violence exposure and lung function in prior studies [(i.e. socioeconomic status, race/ethnicity and tobacco smoke exposure) as well as prenatal factors and child health indicators (i.e. birthweight, history of asthma, and/or allergies, respiratory infections)]. The use of cotinine validation of in utero tobacco exposure is an additional strength. However it is possible that the observed associations may be due to unmeasured confounding by home characteristics or other exposures which may affect children's lung function and be associated with violence exposure given that both may disproportionately impact lower-income urban populations (e.g. air pollution). The availability of both child self-report of violence exposure as well as parents report for their children can be seen as a strength given that we were trying to examine the relationship between violence experienced prior to lung function assessment at a young age (approximately 6 to 7 years). Children are unlikely to recall very early life experiences with violence while parents may be more accurate reporters for the early preschool years. While the concordance between parent-child reporting of violence exposure has been shown to be moderate, with parents consistently underreporting (35), the discordance increases as children get older and are no longer under direct parental supervision, particularly in the preadolescent years (i.e., 12 and older) (45). Thus, discordance in reporting for earlier events as in this analysis is less of a concern.

While we account for various forms of home and community violence exposure, we do not have information on sexual violence or more direct measures of child abuse and neglect potentially underestimating violence exposure. Moreover, if children were exposed to other violent events between the time of the initial assessment and the lung function measurements (as the violence could have been assessed up to one year prior to lung function testing), there may be misclassification of our sample based on violence exposure which would be expected to drive the results toward the null. Studies that both expand on

the measure of different violence exposure types and ascertain exposure at specific critical periods of development may offer further insight into the characteristics of violence exposure that most impact lung development. Other considerations for future directions based on these findings include the assessment of emotion dysregulation and affective states as well as neuroendocrine biomarkers of purported stress pathways that may be mediating these effects. It will also be important to examine whether the effects of exposure to violence in early childhood and lung function hold as these children age into adolescence and early adulthood.

Chronic psychosocial stressors, including violence, have been linked to neuropsychological and behavioral development in children as well as physiologic alterations that may lead to broader health effects. However, the effects of violence exposure on children's respiratory health are largely unknown. This study demonstrates a relationship between higher-level violence exposure, both at home and in the community, on childhood lung function which was independent of tobacco smoke exposure, childhood socioeconomic status and respiratory illness history. Understanding early childhood risk factors that contribute to lung function growth and decline and may be amenable to prevention and intervention is an important area of research given the relationship between childhood lung function and later development of chronic obstructive pulmonary disease (74). Studies such as ours may inform new interventions. Moreover, further exploration of specific environmental stressors, such as violence that disproportionately burden lower income populations, may also help to explain socioeconomic disparities in lung function.

Abbreviations

ETV	Exposure to Violence
CTS	Conflict Tactic Scale
FEV₁	Forced Expiratory Volume
FVC	Forced Vital Capacity
FEF_{25-75%}	Forced Mid-Expiratory Flow Rate
UTS	In-Utero Tobacco Smoke
SHS	Second Hand Smoke

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References

1. Spurzem JR, Rennard SI. Pathogenesis of COPD. *Semin Respir Crit Care Med.* 2005; 26:142–153. [PubMed: 16088433]
2. Mannino DM. Epidemiology and global impact of chronic obstructive pulmonary disease. *Semin Respir Crit Care Med.* 2005; 26:204–210. [PubMed: 16088437]
3. Lange P, Parner J, Vestbo J, Schnohr P, Jensen G. A 15-year follow-up study of ventilatory function in adults with asthma. *N Engl J Med.* 1998; 339:1194–1200. [PubMed: 9780339]
4. Hanrahan JP, Tager IB, Segal MR, Tosteson TD, Castile RG, Van Vunakis H, Weiss ST, Speizer FE. The effect of maternal smoking during pregnancy on early infant lung function. *Am Rev Respir Dis.* 1992; 145:1129–1135. [PubMed: 1586058]

5. Gold DR, Wang X, Wypij D, Speizer FE, Ware JH, Dockery DW. Effects of cigarette smoking on lung function in adolescent boys and girls. *N Engl J Med*. 1996; 335:931–937. [PubMed: 8782500]
6. Weiss ST. Atopy as a risk factor for chronic obstructive pulmonary disease: epidemiological evidence. *Am J Respir Crit Care Med*. 2000; 162:S134–S136. [PubMed: 10988168]
7. Gern JE, Rosenthal LA, Sorkness RL, Lemanske RF Jr. Effects of viral respiratory infections on lung development and childhood asthma. *J Allergy Clin Immunol*. 2005; 115:668–674. quiz 75. [PubMed: 15805982]
8. Barker DJ, Godfrey KM, Fall C, Osmond C, Winter PD, Shaheen SO. Relation of birth weight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease. *BMJ*. 1991; 303:671–675. [PubMed: 1912913]
9. Shaheen S. The beginnings of chronic airflow obstruction. *Br Med Bull*. 1997; 53:58–70. [PubMed: 9158284]
10. Affleck G, Apter A, Tennen H, Reisine S, Barrows E, Willard A, Unger J, ZuWallack R. Mood states associated with transitory changes in asthma symptoms and peak expiratory flow. *Psychosom Med*. 2000; 62:61–68. [PubMed: 10705912]
11. Ritz T, Alatupa S, Thons M, Dahme B. Effects of affective picture viewing and imagery on respiratory resistance in nonasthmatic individuals. *Psychophysiology*. 2002; 39:86–94. [PubMed: 12206299]
12. Kubzansky LD, Sparrow D, Jackson B, Cohen S, Weiss ST, Wright RJ. Angry breathing: A prospective study of hostility and lung function in the Normative Aging Study. *Thorax*. 2006; 61:863–868. [PubMed: 16950835]
13. Wright RJ, Cohen RT, Cohen S. The impact of stress on the development and expression of atopy. *Curr Opin Allergy Clin Immunol*. 2005; 5:23–29. [PubMed: 15643340]
14. Moshhammer H, Hoek G, Luttmann-Gibson H, Neuberger MA, Antova T, Gehring U, Hrubá F, Pattenden S, Rudnai P, Slachetova H, Zlotkowska R, Fletcher T. Parental Smoking and Lung Function in Children: an International Study. *Am J Respir Crit Care Med*. 2006
15. Limb SL, Brown KC, Wood RA, Wise RA, Eggleston PA, Tonascia J, Adkinson NF Jr. Irreversible lung function deficits in young adults with a history of childhood asthma. *J Allergy Clin Immunol*. 2005; 116:1213–1219. [PubMed: 16337448]
16. O'Connor GT, Sparrow D, Segal M, Weiss ST. Risk factors for ventilatory impairment among middle-aged and elderly men. The Normative Aging Study. *Chest*. 1993; 103:376–382. [PubMed: 8432122]
17. Wright R, Cohen S, Carey V, Weiss S, Gold D. Parental stress as a predictor of wheezing in infancy: a prospective birth-cohort study. *Am J Respir Crit Care Med*. 2002; 165:358–365. [PubMed: 11818321]
18. Wright RJ, Finn P, Contreras JP, Cohen S, Wright RO, Staudenmayer J, Wand M, Perkins D, Weiss ST, Gold DR. Chronic caregiver stress and IgE expression, allergen-induced proliferation, and cytokine profiles in a birth cohort predisposed to atopy. *J Allergy Clin Immunol*. 2004; 113:1051–1057. [PubMed: 15208584]
19. Wright R. Prenatal maternal stress and early caregiving experiences: implications for childhood asthma risk. *Paediatric and Perinatal Epidemiology*. in press.
20. Weiss ST, Tager IB, Speizer FE, Rosner B. Persistent wheeze. Its relation to respiratory illness, cigarette smoking, and level of pulmonary function in a population sample of children. *Am Rev Respir Dis*. 1980; 122:697–707. [PubMed: 7447154]
21. Cohen S, Frank E, Doyle WJ, Skoner DP, Rabin BS, Gwaltney JM Jr. Types of stressors that increase susceptibility to the common cold in healthy adults. *Health Psychol*. 1998; 17:214–223. [PubMed: 9619470]
22. Cohen S. Social status and susceptibility to respiratory infections. *Ann N Y Acad Sci*. 1999; 896:246–253. [PubMed: 10681901]
23. Shohaimi S, Welch A, Bingham S, Luben R, Day N, Wareham N, Khaw KT. Area deprivation predicts lung function independently of education and social class. *Eur Respir J*. 2004; 24:157–161. [PubMed: 15293619]

24. Jackson B, Kubzansky LD, Cohen S, Weiss S, Wright RJ. A matter of life and breath: childhood socioeconomic status is related to young adult pulmonary function in the CARDIA study. *Int J Epidemiol.* 2004; 33:271–278. [PubMed: 15082626]
25. Lawlor DA, Ebrahim S, Davey Smith G. Association between self-reported childhood socioeconomic position and adult lung function: findings from the British Women's Heart and Health Study. *Thorax.* 2004; 59:199–203. [PubMed: 14985552]
26. Wright RJ. Health effects of socially toxic neighborhoods: the violence and urban asthma paradigm. *Clin Chest Med.* 2006; 27:413–421. [PubMed: 16880051]
27. Christopher M. A broader view of trauma: a biopsychosocial-evolutionary view of the role of the traumatic stress response in the emergence of pathology and/or growth. *Clin Psychol Rev.* 2004; 24:75–98. [PubMed: 14992807]
28. Cohen, S.; Kessler, R.; Underwood Gordon, L. Measuring stress: a guide for health and social scientists. New York: Oxford University Press; 1995.
29. Richters JE, Martinez P. The NIMH community violence project: I. Children as victims of and witnesses to violence. *Psychiatry.* 1993; 56:7–21. [PubMed: 8488215]
30. Cicchetti D, Rogosch FA. The impact of child maltreatment and psychopathology on neuroendocrine functioning. *Dev Psychopathol.* 2001; 13:783–804. [PubMed: 11771908]
31. Gunnar MR, Donzella B. Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology.* 2002; 27:199–220. [PubMed: 11750779]
32. Kliewer W. Violence exposure and cortisol responses in urban youth. *Int J Behav Med.* 2006; 13:109–120. [PubMed: 16712428]
33. Osofsky J, Wewers S, Hann D, Fick A. Chronic Community Violence: What is happening to our children? *Psychiatry.* 1993; 56:36–45. [PubMed: 8488211]
34. Wright RJ, Wright RO, Isaac NE. Response to battered mothers in the pediatric emergency department: a call for an interdisciplinary approach to family violence. *Pediatrics.* 1997; 99:186–192. [PubMed: 9024444]
35. Thomson CC, Roberts K, Curran A, Ryan L, Wright RJ. Caretaker-child concordance for child's exposure to violence in a preadolescent inner-city population. *Arch Pediatr Adolesc Med.* 2002; 156:818–823. [PubMed: 12144374]
36. Straus M, Gelles R. Physical Violence in American Families: risk factors and adaptations to violence in 8145 families. New Brunswick, NJ: Transaction Publishers. 1990
37. Connelly CD, Newton RR, Aarons GA. A psychometric examination of English and Spanish versions of the Revised Conflict Tactics Scales. *J Interpers Violence.* 2005; 20:1560–1579. [PubMed: 16246917]
38. Straus M. The revised Conflict Tactics Scales (CTS2): Development and preliminary psychometric data. *Journal of Family Issues.* 1996; 17.
39. Standardization of Spirometry, 1994 Update. American Thoracic Society. *Am J Respir Crit Care Med.* 1995; 152:1107–1136. [PubMed: 7663792]
40. Eigen H, Bieler H, Grant D, Christoph K, Terrill D, Heilman DK, Ambrosius WT, Tepper RS. Spirometric pulmonary function in healthy preschool children. *Am J Respir Crit Care Med.* 2001; 163:619–623. [PubMed: 11254514]
41. Ferris BG. Epidemiology Standardization Project (American Thoracic Society). *Am Rev Respir Dis.* 1978; 118:1–120. [PubMed: 742764]
42. Kindlon D, Wright B, Raudenbush S, Earls F. The measurement of Children's exposure to violence: a rasch analysis. *International Journal of Methods in Psychiatric Research.* 1996; 6:187–194.
43. Franco Suglia, S. The Influence of the Social and Physical Environment on Child Health. Boston, MA: Harvard School of Public Health; 2006.
44. Horton NJ, Laird NM, Murphy JM, Monson RR, Sobol AM, Leighton AH. Multiple informants: mortality associated with psychiatric disorders in the Stirling County Study. *Am J Epidemiol.* 2001; 154:649–656. [PubMed: 11581099]
45. Howard DE, Cross SI, Li X, Huang W. Parent-youth concordance regarding violence exposure: relationship to youth psychosocial functioning. *J Adolesc Health.* 1999; 25:396–406. [PubMed: 10608579]

46. Kuo M, Mohler B, Raudenbush SL, Earls FJ. Assessing exposure to violence using multiple informants: application of hierarchical linear model. *J Child Psychol Psychiatry*. 2000; 41:1049–1056. [PubMed: 11099121]
47. Brennan R, Molnar B, Earls F. Refining the Measurement of Exposure to Violence (ETV) in Urban Youth. *J Community Psychol*. 2007 In press.
48. Selner-O'Hagan MB, Kindlon DJ, Buka SL, Raudenbush SW, Earls FJ. Assessing exposure to violence in urban youth. *J Child Psychol Psychiatry*. 1998; 39:215–224. [PubMed: 9669234]
49. Richters JE, Saltzman W. Survey of Children's Exposure to Community Violence: Parent report. 1990
50. Olsen J. What characterises a useful concept of causation in epidemiology? *J Epidemiol Community Health*. 2003; 57:86–88. [PubMed: 12540681]
51. Gilliland FD, Berhane K, McConnell R, Gauderman WJ, Vora H, Rappaport EB, Avol E, Peters JM. Maternal smoking during pregnancy, environmental tobacco smoke exposure and childhood lung function. *Thorax*. 2000; 55:271–276. [PubMed: 10722765]
52. Cunningham J, Dockery DW, Speizer FE. Maternal smoking during pregnancy as a predictor of lung function in children. *Am J Epidemiol*. 1994; 139:1139–1152. [PubMed: 8209873]
53. McEwen BS. Protection and damage from acute and chronic stress: allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Ann N Y Acad Sci*. 2004; 1032:1–7. [PubMed: 15677391]
54. Carrion VG, Weems CF, Ray RD, Glaser B, Hessel D, Reiss AL. Diurnal salivary cortisol in pediatric posttraumatic stress disorder. *Biol Psychiatry*. 2002; 51:575–582. [PubMed: 11950459]
55. Ford JG, Li Y, O'Sullivan MM, Demopoulos R, Garte S, Taioli E, Brandt-Rauf PW. Glutathione S-transferase M1 polymorphism and lung cancer risk in African-Americans. *Carcinogenesis*. 2000; 21:1971–1975. [PubMed: 11062156]
56. Spiteri MA, Bianco A, Strange RC, Fryer AA. Polymorphisms at the glutathione S-transferase, GSTP1 locus: a novel mechanism for susceptibility and development of atopic airway inflammation. *Allergy*. 2000; 55 Suppl 61:15–20. [PubMed: 10919500]
57. Adachi S, Kawamura K, Takemoto K. Oxidative damage of nuclear DNA in liver of rats exposed to psychological stress. *Cancer Res*. 1993; 53:4153–4155. [PubMed: 8364908]
58. Kosugi H, Enomoto H, Ishizuka Y, Kikugawa K. Variations in the level of urinary thiobarbituric acid reactant in healthy humans under different physiological conditions. *Biol Pharm Bull*. 1994; 17:1645–1650. [PubMed: 7735211]
59. DeWit DJ, Chandler-Coutts M, Offord DR, King G, McDougall J, Specht J, Stewart S. Gender differences in the effects of family adversity on the risk of onset of DSM-III-R social phobia. *J Anxiety Disord*. 2005; 19:479–502. [PubMed: 15749569]
60. Reese LE, Vera EM, Thompson K, Reyes R. A qualitative investigation of perceptions of violence risk factors in low-income African American children. *J Clin Child Psychol*. 2001; 30:161–171. [PubMed: 11393917]
61. Rasmussen A, Aber MS, Bhana A. Adolescent coping and neighborhood violence: perceptions, exposure, and urban youths' efforts to deal with danger. *Am J Community Psychol*. 2004; 33:61–75. [PubMed: 15055755]
62. Edwards A, Shipman K, Brown A. The socialization of emotional understanding: a comparison of neglectful and nonneglectful mothers and their children. *Child Maltreat*. 2005; 10:293–304. [PubMed: 15983112]
63. Shipman K, Edwards A, Brown A, Swisher L, Jennings E. Managing emotion in a maltreating context: a pilot study examining child neglect. *Child Abuse Negl*. 2005; 29:1015–1029. [PubMed: 16159666]
64. Chaplin TM, Cole PM, Zahn-Waxler C. Parental socialization of emotion expression: gender differences and relations to child adjustment. *Emotion*. 2005; 5:80–88. [PubMed: 15755221]
65. Roy A. Childhood trauma and neuroticism as an adult: possible implication for the development of the common psychiatric disorders and suicidal behaviour. *Psychol Med*. 2002; 32:1471–1474. [PubMed: 12455946]
66. Bunce SC, Larsen RJ, Peterson C. Life after trauma: personality and daily life experiences of traumatized people. *J Pers*. 1995; 63:165–188. [PubMed: 7782991]

67. Jackson B, Kubzansky LD, Cohen S, Jacobs DR Jr, Wright RJ. Does harboring hostility hurt? Associations between hostility and pulmonary function in the Coronary Artery Risk Development in (Young) Adults (CARDIA) study. *Health Psychol.* 2007; 26:333–340. [PubMed: 17500620]
68. Kubzansky LD, Wright RJ, Cohen S, Weiss S, Rosner B, Sparrow D. Breathing easy: a prospective study of optimism and pulmonary function in the Normative Aging Study. *Ann Behav Med.* 2002; 24:345–353. [PubMed: 12434946]
69. Sarid O, Anson O, Yaari A, Margalith M. Academic stress, immunological reaction, and academic performance among students of nursing and physiotherapy. *Res Nurs Health.* 2004; 27:370–377. [PubMed: 15362147]
70. Kumari V, Corr PJ. Menstrual cycle, arousal-induction, and intelligence test performance. *Psychol Rep.* 1996; 78:51–58. [PubMed: 8839295]
71. Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J. The effect of air pollution on lung development from 10 to 18 years of age. *N Engl J Med.* 2004; 351:1057–1067. [PubMed: 15356303]
72. Rauh VA, Chew GR, Garfinkel RS. Deteriorated housing contributes to high cockroach allergen levels in inner-city households. *Environ Health Perspect.* 2002; 110 Suppl 2:323–327. [PubMed: 11929744]
73. Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, Kahn RL, Syme SL. Socioeconomic status and health. The challenge of the gradient. *Am Psychol.* 1994; 49:15–24. [PubMed: 8122813]
74. von Mutius E. Childhood experiences take away your breath as a young adult. *Am J Respir Crit Care Med.* 2002; 165:1467–1468. [PubMed: 12045117]

Figure 1.
Girls percent predicted (95% confidence interval) lung function measures: Adjusted linear regression models*

Figure 2.
Boys percent predicted (95% confidence interval) lung function measures: Adjusted linear regression models*

Table 1

Mean and standard error of height adjusted lung function outcomes by demographic and exposure characteristics; stratified by gender

Males		FEV₁	FVC	FEF_{25-75%}
Demographics	N (%)	Mean (SE)	Mean (SE)	Mean (SE)
Child's Age at PFT				
Less than 6	107 (68.6)	1.30 (.02)	1.41 (.02)	1.80 (.04)
Six or older	49 (31.4)	1.33 (.02)	1.43 (.03)	1.92 (.06)
Child's Race				
Non-Hispanic [†]	76 (48.7)	1.29 (.02)	1.39(.02)	1.82 (.05)
Hispanic	80 (51.3)	1.33 (.02)	1.42 (.02)	1.85 (.05)
Maternal Education Level				
Some College	32 (20.5)	1.33 (.03)	1.43 (.03)	1.90 (.07)
High School Grad/ Tech	63 (40.4)	1.30 (.02)	1.41 (.02)	1.78 (.05)
Less High School/ No Grad	61 (39.1)	1.32 (.02)	1.42 (.03)	1.86 (.05)
Medical History				
Birthweight				
Greater than or equal to 2500g	142 (91.0)	1.32 (.01)	1.43 (.02)	1.85 (.03)
Less than 2500 g	14 (9.0)	1.26 (.05)	1.35 (.05)	1.70 (.11)
Physician Diagnosed Asthma				
No	124 (79.5)	1.32 (.02)	1.42 (.02)	1.87 (.04)
Yes	32 (20.5)	1.30 (.03)	1.44 (.04)	1.72 (.07)
Allergies				
No	135 (86.5)	1.31 (.01)	1.41 (.02)	1.83 (.03)
Yes	21 (13.5)	1.32 (.04)	1.45 (.04)	1.86 (.09)
Lower Respiratory Infections				
None	37 (25.7)	1.35 (.03)*	1.44 (.03)*	1.97 (.07)*
One to Two	52 (36.1)	1.34 (.02)	1.45 (.03)	1.85 (.06)
Three or more	55 (38.2)	1.26 (.02)	1.37 (.02)	1.75 (.05)
Violence Exposures				
CTS- Verbal Aggression[‡]				
1st Tertile	54 (34.6)	1.31 (.02)	1.43 (.03)	1.81 (.06)
2nd Tertile	56 (35.9)	1.33 (.02)	1.43 (.03)	1.85 (.05)
3rd Tertile	46 (29.5)	1.30 (.02)	1.38 (.03)	1.84 (.06)
ETV Score[‡]				
1st Tertile	69 (44.2)	1.34 (.02)	1.46 (.02)*	1.80 (.05)
2nd Tertile	34 (21.8)	1.32 (.03)	1.41 (.03)	1.85 (.07)
3rd Tertile	53 (34.0)	1.28 (.02)	1.37 (.03)	1.87 (.06)

Males		FEV ₁	FVC	FEF _{25-75%}
Demographics	N (%)	Mean (SE)	Mean (SE)	Mean (SE)
CTS- Composite Physical violence^{††}				
1st Tertile	85 (54.8)	1.31 (.02)	1.42 (.02)	1.81 (.04)
2nd Tertile	22 (14.2)	1.28 (.04)	1.38 (.04)	1.74 (.09)
3rd Tertile	48 (31.0)	1.34 (.02)	1.44 (.03)	1.94 (.06)
Environmental Exposures				
Tobacco Exposure				
Non Smoker	47 (30.1)	1.32 (.02)	1.44 (.03)*	1.82 (.06)
UTS and SHS [§]	33 (21.2)	1.27 (.03)	1.37 (.03)	1.73 (.07)
Early SHS ^{§§}	23 (14.7)	1.38 (.03)	1.51 (.04)	1.87 (.08)
Late SHS ^{§§§}	53 (34.0)	1.31 (.02)	1.40 (.03)	1.91 (.06)
Females		FEV ₁	FVC	FEF _{25-75%}
Demographics	N (%)	Mean (SE)	Mean (SE)	Mean (SE)
Child's Age at PFT				
Less than 6	102 (65.0)	1.24 (.01)	1.35 (.02)	1.77 (.04)
Six or older	55 (35.0)	1.27 (.02)	1.36 (.02)	1.89 (.05)
Child's Race				
Non-Hispanic [†]	77 (49.0)	1.22 (.02)*	1.33 (.02)	1.72 (.04)*
Hispanic	80 (51.0)	1.28 (.02)	1.38 (.02)	1.90 (.04)
Maternal Education Level				
Some College	30 (19.1)	1.20 (.03)	1.30 (.03)	1.73 (.07)
High School Grad/ Tech	65 (41.4)	1.27 (.02)	1.37 (.02)	1.84 (.05)
Less High School/ No Grad	62 (39.5)	1.25 (.02)	1.36 (.02)	1.83 (.05)
Medical History				
Birthweight				
Greater than or equal to 2500g	145 (92.4)	1.25 (.01)	1.36 (.01)	1.81 (.03)
Less than 2500 g	12 (7.6)	1.25 (.04)	1.33 (.05)	1.80 (.11)
Physician Diagnosed Asthma				
No	132 (84.1)	1.25 (.01)	1.36 (.01)	1.82 (.03)
Yes	25 (15.9)	1.23 (.03)	1.31 (.03)	1.80 (.08)
Allergies				
No	143 (91.1)	1.25 (.01)	1.36 (.01)	1.81 (.03)
Yes	14 (8.9)	1.22 (.04)	1.31 (.05)	1.79 (.10)
Lower Respiratory Infections				
None	44 (29.9)	1.28 (.02)	1.38 (.02)	1.83 (.06)
One to Two	62 (42.2)	1.22 (.02)	1.32 (.02)	1.80 (.05)

Males		FEV ₁	FVC	FEF _{25-75%}
Demographics	N (%)	Mean (SE)	Mean (SE)	Mean (SE)
Three or more	41 (27.9)	1.23 (.02)	1.34 (.03)	1.79 (.06)
Violence Exposures				
CTS- Verbal Aggression[†]				
1st Tertile	58 (36.9)	1.29 (.02) *	1.40 (.02) *	1.85 (.05)
2nd Tertile	37 (23.6)	1.26 (.02)	1.35 (.03)	1.83 (.06)
3rd Tertile	62 (39.5)	1.21 (.02)	1.31 (.02)	1.77 (.05)
ETV Score[†]				
1st Tertile	74 (47.1)	1.26 (.02)	1.37 (.02)	1.83 (.05)
2nd Tertile	27 (17.2)	1.21 (.03)	1.32 (.03)	1.74 (.08)
3rd Tertile	56 (35.7)	1.26 (.02)	1.35 (.02)	1.83 (.05)
CTS- Composite Physical violence^{††}				
1st Tertile	77 (49.7)	1.26 (.02)	1.37 (.02)	1.82(.04)
2nd Tertile	25 (16.1)	1.25 (.03)	1.35 (.03)	1.83 (.08)
3rd Tertile	53 (34.2)	1.23 (.02)	1.34 (.02)	1.79 (.05)
Environmental Exposures				
Tobacco Exposure				
Non Smoker	48 (30.6)	1.31 (.02) *	1.41 (.02) *	1.91 (.05) *
UTS and SHS [§]	33 (21.0)	1.21 (.03)	1.32 (.03)	1.62 (.07)
Early SHS ^{§§}	18 (11.5)	1.21 (.03)	1.34 (.04)	1.68 (.09)
Late SHS ^{§§§}	58 (36.9)	1.24 (.02)	1.33 (.02)	1.88 (.05)

* p-value < 0.05 based on chi-squared analysis

SE indicates Standard Error

[†] 143 identified as White, 3 as Black, and 7 as other race/ethnicity

[†] Tertiles do not break evenly due to presence of ties

^{††} Three children are missing physical violence information

[§] Includes those exposed to in utero tobacco smoke (UTS) and postnatal second hand smoke (SHS)

^{§§} Includes those exposed to SHS from birth up to age 25 months only

^{§§§} Includes those exposed to SHS either from birth up to the time of lung function testing or those exposed to SHS from 26 months of age up to the time of lung function testing

SE indicates Standard Error

Table 2

Percent predicted (95% confidence interval) lung function measures stratified by gender: Adjusted linear regression models*

	FEV ₁		FVC		FEF _{25-75%}	
	Girls	Boys	Girls	Boys	Girls	Boys
Verbal Aggression (per SD)	-2.4 (-4.0, -0.7) [†]	-0.7 (-2.8, 1.5)	-2.1 (-3.9, -0.3) [†]	-1.8 (-4.1, 0.6)	-1.4 (-4.6, 1.9)	0.7 (-3.1, 4.5)
ETV Scores (per SD)	0.3 (-1.4, 2.1)	-1.5 (-3.7, 0.7)	0.4 (-1.5, 2.2)	-2.0 (-4.3, 0.4)	0.8 (-2.5, 4.1)	1.7 (-2.2, 5.6)
Physical Aggression (per SD)	-0.4 (-2.0, 1.3)	1.3 (-1.0, 3.6)	0.1 (-1.6, 1.9)	0.8 (-1.8, 3.3)	-1.7 (-4.8, 1.4)	2.9 (-1.2, 7.0)

* Each line is a separate multivariate model adjusted for race/ethnicity, birthweight, age, tobacco smoke exposure and maternal education.

[†] p-value < .05

UTS=In utero tobacco exposure; ETV=Exposure to violence; SHS=Second hand smoke