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# Psychosocial stressors and lung function in youth ages 10–17: an examination by stressor, age and gender

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## ABSTRACT

**Background** Research on the impact of psychosocial stressors on child and adolescent lung function is uncommon, and has primarily relied either on parents' own stress measures or parent-reported stressors the child experienced, which may be a poor proxy for perceived stress in older children and adolescents.

**Methods** We performed multivariate linear regression of spirometry measures (FVC, $FEV_1$ and $FEF_{25-75}$) and psychosocial stressors in 584 adolescents in the Los Angeles Family and Neighborhood Survey. We examined family conflict, unsafe neighborhood or school, and the absence of a father in models stratified by gender, adjusting for $PM_{2.5}$ and potential confounders.

**Results** We observed reductions in lung function in males related to the absence of a father in the house ($FEV_1$: −176.2 ml, 95% CI −322.7, −29.7) and family conflict ($FEV_1$: −156.2 ml, 95% CI −327.8, 15.5); associations were stronger in older males ages 15–17 years for each stressor (*P* for interaction of age and sex was 0.009 and 0.06, respectively).

**Conclusions** This research informs a very small literature on psychosocial stressors and lung function in adolescents. Our finding of differential vulnerability by age and gender warrants further exploration of adolescent psychosocial stressor response on lung function.

**Keywords** adolescents, epidemiology, social determinants

## Introduction

Psychosocial stressors have been associated with both the incidence and morbidity of asthma and wheeze in youth.[1–6] However, studies suggesting an association with decreases in lung function are rare.[7–9] Lung function, measured with spirometry, steadily increases throughout childhood and adolescence, plateauing in puberty/young adulthood. Decrements in the development of lung function in childhood and adolescence result in lower lung function by early adulthood, which is related to an increased risk of both all-cause mortality and ischemic heart disease mortality independent of smoking status.[10] Factors associated with inflammation, including asthma, smoking, respiratory infections and air pollution increase risk for impaired lung function.[7] A less investigated risk factor that is also hypothesized to operate through inflammatory mechanisms is stress. Perceived chronic stress may alter an individual's stress response through the differential expression of genes involved in the hypothalamus–pituitary–adrenal axis,[11,12] modulating immune function.[11] and heightening inflammatory activity.[13] Excessive pro-inflammatory responses that result in damage to the airway may result in lung structure and functional changes throughout development.[14]

Although a few studies have reported negative associations between adult negative mood, psychosocial stress and lung function,[14–16] the literature examining psychosocial stressors and lung function in children and adolescents is limited and

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inconsistent. Of three previous studies, one conducted in East Boston did not account for air pollution but found that the child's exposure to violence as measured by verbal aggression during interparental conflict was associated with a 5.5% decrease in percent predicted forced expiratory volume after 1 s ($FEV_1$), in 6- to 7-year-old girls only, while the child's exposure to community violence (as reported by the parent) was associated with a 3.4% lower $FEV_1$ in boys only.[7] A study of youth ages 11–13 years in the UK found no associations with either self-reported racism, particulate matter (PM) $PM_{2.5}$ or $PM_{10}$ air pollution for predicted lung function.[9] The third study in Southern California reported that maternal psychosocial stress did not independently predict lung function, but maternal psychosocial stress modified the impact of air pollution on lung function impairment in 11-year-old children. This last study relied solely on maternal stress assessed with a 4-question Perceived Stress Scale.[8] Measures of parental stressors or child stressors reported by the parents may not reflect the psychosocial stress that older children or adolescents perceive.[17]

Healthy growth and development of pulmonary function in childhood and adolescence is instrumental for respiratory health in adulthood. Investigating risk factors that may be modifiable in early life are critical in efforts to mitigate adult pulmonary diseases. To continue the early investigation into this hypothesized risk factor, we used cross-sectional data from the Los Angeles Family and Neighborhood Survey 2 (L.A.FANS-2) to explore whether 10- to 17-year-old adolescent's self-reported psychosocial stressors (family conflict, unsafe neighborhood or school) or the absence of a father in the home influenced lung function while adjusting for $PM_{2.5}$ air pollution exposure and potential confounders.

## Methods

### Sample and data source

#### Los Angeles Family and Neighborhood Survey 2

We used data from the L.A.FANS Wave 2 described previously.[18,19] Briefly, L.A.FANS was a population-based cohort conducted in two waves. The first wave (L.A.FANS-1) sampled 3090 households from 65 neighborhoods in Los Angeles County in 2000–01, selecting 3140 children ages 0–17 years. Households were sampled from three neighborhood strata: very poor, poor and not-poor with oversampling of very poor and poor census tracts and of households with children below age 18. In 2006–08, L.A.FANS-2 re-interviewed L.A.FANS-1 participants and added additional households within the sampled neighborhoods. Excluding children who participated in the Wave 1 survey who were older than 18 when the Wave 2 survey re-enrolled Wave 1 participants, there were 1091 children who were re-interviewed (64% response rate); also, 296 additional children from new families were added to the sample, for a total of 1387 children. In order to assess adolescents' self-reported psychosocial stressors, we included in our analyses only Wave 2 survey data for those between ages 10 and 17 years (here referred to as 'adolescent's' following World Health Organization nomenclature[20]) in our analyses and with a reproducible spirometry curve ($n = 584$).

The UCLA Office of the Human Research Protection Program and the RAND Corporation approved this research.

### Outcome, exposure and covariate definitions

#### Spirometry

Of the 1387 children in L.A.FANS-2, 1070 children aged 5–17 years participated in spirometry assessments via EasyOne™ portable spirometers, which measured forced vital capacity (FVC), forced expiratory volume after 1 s ($FEV_1$) and forced expiratory mean flow between 25 and 75% of FVC ($FEF_{25-75}$). Evaluation and acceptance criteria for spirometry curves have been previously detailed.[21] Briefly, an expert with specific experience evaluating spirometry data from children as part of the UC Berkeley Fresno Asthmatic Children's Environment Study reviewed all curves. To determine acceptability, all grading of spirometry curves was done based on the following criteria (some of which overlap with the 1994 ATS criteria[22]): (i) the back extrapolated volume must be $\leq$5% or 150 ml, whichever is greater; (ii) time to peak flow must be $\leq$120 ms; (iii) no abrupt end to test; (iv) FET must be $\geq$2 s; (v) time/volume curve must begin at origin (to ensure proper start of test); (vi) curve must show that subject exhaled using only one continuous blast of air and (vii) curve must show no leaks or negative flow throughout test (i.e. no inhalation). Only curves judged acceptable by the reviewer were included; curves judged acceptable by the EasyOne™portable spirometer without reviewer approval were not included in analyses.

Of the 775 children with at least one acceptable curve for analysis, 584 children were between 10 and 17 years of age.

#### Psychosocial stressors

*Adolescent-reported.* Adolescents self-reported whether 'people in my family fight a lot' (true versus sometimes true or not true), whether they 'feel safe in this neighborhood' (yes versus sometimes or no) and whether they 'feel safe at this school' (yes versus sometimes or no).

*Caregiver/family.* The individual who identified as head of household reported whether the father of each child lived in the house (yes/no).

### Air pollution estimates

Our air pollution estimates have been described previously in detail.[21] For this analysis, we adjusted models according to inter-quartile range units of $PM_{2.5}$, based upon its negative association with spirometry measures in the sample ($FEV_1$ males: −47.9 ml, 95% CI −92.3, −3.6; $FEV_1$ females: −56.9 ml, 95% CI −103.2, −10.6). $PM_{2.5}$ estimates were generated by Kriging available government monitoring data for the years 2002 and 2000, respectively. We used annual average measures of $PM_{2.5}$ at both residences and schools weighted for time spent in each location in the past 12 months.

### Child/household socio-demographic covariates

The following covariates were assessed in the survey and explored as covariates: the head of the household reported the adolescent's gender, age, race/ethnicity and nativity (US born versus non-US born), as well as household income used to calculate the household federal poverty level (FPL). Trained interviewers measured the adolescent's height and weight. Adolescents' were asked about their own smoking behavior, and whether they like to read, walk to activities or engage in sports. Finally, the mother/primary caregiver reported maternal/primary caregiver education, any smoking in the house and the adolescent's life time asthma history and wheeze within the past 12 months.

### Statistical analyses

FVC, $FEV_1$ and $FEF_{25-75}$ were stratified by gender and confirmed to have normal distributions. Multiple linear regression was performed, separately for each stressor (family conflict, unsafe neighborhood, unsafe school and the absence of father in the house) and each lung function measure (FVC, $FEV_1$, $FEF_{25-75}$), unadjusted and adjusted for potential confounders, including $PM_{2.5}$, child's age, FPL, the presence of smokers in the household, child's race/ethnicity, maternal education, height, height-square, weight, weight-square, and in models that included both genders, gender and gender*age. Covariates were retained in models based upon previous literature or a change in estimate of the association of interest by >10%. Few adolescents ($n = 10$) reported smoking, and inclusion in the models did not change effect estimates; we thus did not include it in the final models. However, the presence of smokers in the house was retained. Neither nativity of the child nor level of sports engagement changed estimates and were also not included in the final adjusted models. Robust standard errors were included to adjust for non-independence of sibling data (112 sibling sets) and neighborhood factors. Models were stratified by age group (10–14 and 15–17 years) and by gender to examine potential effect measure modification. Finally, models were created with a stressor*gender term to assess statistical interaction.

Given the previous literature linking both air pollution and psychosocial stressors to childhood asthma,[2,3,23,24] reduced lung function may be on the causal path to asthma, and thus we did not include previous asthma in the models. In sensitivity analyses, we excluded those who had a doctor's diagnosis of asthma with wheeze in the past 12 months ($n = 42$). Information regarding timing of last use of asthma medications (if any) prior to spirometry was not collected.

## Results

The mean ambient $PM_{2.5}$ concentration was 8.5 $\mu m/m^3$ (inter-quartile range 2.3 $\mu m/m^3$). Spirometry estimates [mean (standard deviation)] were as follows: FVC (ml) boys: 3559.9 (1112.4), girls: 3091.8 (689.4); $FEV_1$ (ml) boys: 2967.3 (953.1), girls: 2625.6 (610.6); $FEF_{25-75}$ (ml/s) boys: 3139.9 (1272.1), girls: 3020.7 (987.6).

The majority of adolescents sampled were US-born Hispanic, with over 50% living at or below 200% of the FPL. While only 10 children reported smoking, 22.3% lived in a household with a smoker. There was little difference by gender for perceived school safety, family conflict or the absence of a father in the household. Boys were more likely to feel that their neighborhood was safe (Table 1).

When stratified by gender (Table 2), a negative association was suggested between adjusted estimates for family conflict and both FVC and $FEV_1$ in males, but confidence intervals included the null value. Similarly, $FEV_1$ and $FEF_{25-75}$ were lower in males when a father was absent, while $FEF_{25-75}$ values were higher in females for this stressor. There were no other associations between selected psychosocial stressors and lung function observed in females or males.

When gender*stressor interaction terms were included into adjusted models, interactions terms were statistically significant for gender and the absence of the father ($P = 0.009$), and very close to statistically significant for gender and family conflict (0.060). No other stressors had statistically significant gender*stressor interaction terms. When stratified by gender and age (Fig. 1), both of these psychosocial stressors had the largest coefficients for reduction of $FEV_1$ in males 15–17 years of age.

Finally, when asthmatics with current wheeze were excluded, point estimates for the stressors remained within 10% of the original models' estimates; i.e. the results generally did not change when excluding asthmatics (data not shown).

**Table 1** Descriptive and demographic data for participants ages 10–17 years in L.A.FANS-2, a neighborhood and household survey in Los Angeles, CA, 2006–08 ($n = 584$)

| | *TOTAL (n = 584)* n, % | *BOYS (n = 316)* n, % | *GIRLS (n = 268)* n, % |
|---|---|---|---|
| Psychosocial stressors | | | |
| Neighborhood feels safe | 367 (62.8) | 215 (68.0) | 152 (56.7) |
| School feels safe | 443 (75.8) | 244 (77.2) | 199 (74.3) |
| Family does not fight | 519 (88.9) | 280 (88.6) | 239 (89.2) |
| Dad lives in the house | 383 (65.6) | 202 (63.9) | 181 (67.5) |
| Child covariates | | | |
| Age mean (SD) | 13.5 (2.3) | 13.4 (2.3) | 13.6 (2.2) |
| 10–14 years | 358 (61.3) | 196 (62.0) | 162 (60.5) |
| 15–17 years | 226 (38.7) | 120 (38.0) | 106 (39.5) |
| Height (cm) mean (SD) | 158.8 (11.8) | 161.1 (13.4) | 156.2 (8.9) |
| Weight (kg) mean (SD) | 61.2 (19.3) | 63.7 (20.8) | 58.3 (16.9) |
| Race | | | |
| Non-Hispanic White | 84 (14.4) | 44 (13.9) | 40 (14.9) |
| Hispanic | 392 (67.1) | 203 (64.2) | 189 (70.5) |
| Black | 32 (5.5) | 19 (6.0) | 13 (4.9) |
| Asian/Pacific Islander | 28 (4.8) | 19 (6.0) | 9 (3.4) |
| Other (multiple races) | 48 (8.2) | 31 (9.8) | 17 (6.3) |
| Child's nativity | | | |
| US-born | 532 (91.1) | 289 (91.5) | 243 (90.7) |
| Child smokes | 10 (1.7) | 8 (2.5) | 2 (0.8) |
| Asthma diagnosis with wheeze (12 months) | 42 (7.2) | 29 (9.2) | 13 (4.9) |
| Moderate (4+/week) sports | 284 (48.6) | 174 (55.1) | 110 (41.0) |
| Vigorous (4+/week) sports | 272 (46.6) | 170 (53.8) | 102 (38.1) |
| Family/household covariates | | | |
| Maternal Education | | | |
| <8th grade | 137 (23.5) | 75 (23.7) | 62 (23.1) |
| 9–12th grade | 206 (35.3) | 101 (32.0) | 105 (39.2) |
| Vocational school | 33 (5.7) | 14 (4.4) | 19 (7.1) |
| AA/some college | 126 (21.6) | 78 (24.7) | 48 (17.9) |
| College+ | 78 (13.4) | 45 (14.2) | 33 (12.3) |
| Federal poverty level | | | |
| <100% | 170 (29.1) | 94 (29.7) | 76 (28.4) |
| 101–200% | 172 (29.5) | 93 (29.4) | 79 (29.5) |
| 201–300% | 94 (16.1) | 49 (15.5) | 45 (16.8) |
| 301%+ | 148 (25.3) | 80 (25.3) | 68 (25.4) |

**Table 2** Adjusted[a] Beta, 95% confidence interval estimates for psychosocial stressors and lung function by gender in adolescents 10–17 in L.A.FANS ($n = 584$[b])

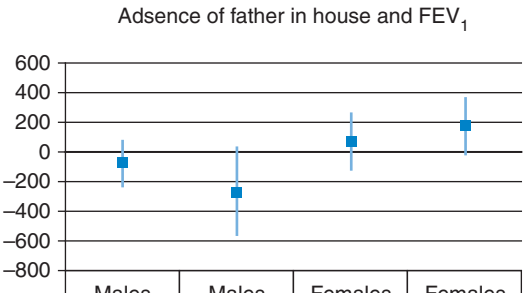
| | *FVC (ml)* | | *$FEV_1$ (ml)* | | *$FEF_{25-75}$ (ml/s)* | |
|---|---|---|---|---|---|---|
| | *Males* | *Females* | *Males* | *Females* | *Males* | *Females* |
| Family conflict[c] | −174.1 (−362.5, 14.3) | 90.7 (−89.7, 271.1) | −156.2 (−327.8, 15.5) | 41.5 (−101.4, 184.4) | −68.4 (−400.1, 263.4) | −74.6 (−369.2, 220.0) |
| Unsafe school[d] | −56.6 (−230.7, 117.5) | 3.0 (−134.2, 140.3) | −85.2 (−229.5, 59.1) | −50.0 (−184.1, 84.1) | −138.4 (−416.6, 139.9) | −119.1 (−384.4, 146.2) |
| Unsafe neighborhood[e] | 68.8 (−87.0, 224.8) | 13.4 (−114.1, 140.9) | 13.0 (−121.3, 147.4) | 29.3 (−89.5, 148.1) | −89.4 (−328.3, 149.5) | 143.1 (−79.5, 365.8) |
| Absence of father[f] | −156.9 (−329.9, 16.0) | 35.5 (−107.5, 178.5) | −176.2 (−322.7, −29.7) | 76.7 (−52.8, 206.2) | −371.8 (−607.8, −135.8) | 253.0 (12.3, 493.8) |

[a]Adjusted for: $PM_{2.5}$, age, FPL, current house smoke, race, maternal education, height (cm), height-square, weight (kg) weight-square.
[b]Adjusted *n*'s: males $n = 281$, female $n = 245$.
[c]Reference = no/sometimes family fighting.
[d]Reference = safe all of the time.
[e]Reference = safe all of the time.
[f]Reference = father present in the house.

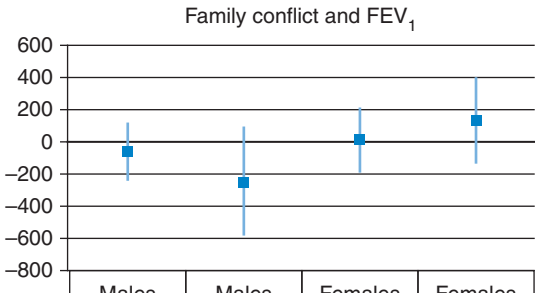
## Discussion

### Main findings of this study

Exploring a range of self-reported psychosocial stressors in adolescents' ages 10–17 years in Los Angeles, we found that the absence of a father or family conflict was associated with reduced lung function in males, particularly older males. We




| | Males 10–14 | Males 15–17 | Females 10–14 | Females 15–17 |
|---|---|---|---|---|
| Upper CI | 83.1 | 31.8 | 258.6 | 365.3 |
| Lower CI | –226 | –570.2 | –121.2 | –16 |
| Point estimate | –71.5 | –269.2 | 68.7 | 174.6 |




| | Males 10–14 | Males 15–17 | Females 10–14 | Females 15–17 |
|---|---|---|---|---|
| Upper CI | 121.6 | 84.3 | 214.2 | 393.2 |
| Lower CI | –243.8 | –592.5 | –187.6 | –133.7 |
| Point estimate | –61.1 | –254.1 | 13.2 | 129.8 |

**Fig. 1** Select psychosocial stressors and $FEV_1$ estimates stratified by gender and age group. *P* for interaction (**a**) 0.009, (**b**) 0.06.

did not find any evidence of an association between psychosocial stressors and reduced lung function in females.

In the absence of validated measures of perceived stress, our aim was to examine the association between lung function and stress by examining a range of potential psychosocial stressors pertaining to place and family that may better characterize an adolescent's stress than the parental measures previously examined. Previous research has relied primarily upon parental reports of their own stress[8] or their perception of the stress in the offspring,[7] both of which may misclassify the stress that a child is internalizing and attenuate results. Particularly as a child ages, it becomes more and more important and possible to capture their own perception, as peer networks may alter perceived stress and social positions, and their family roles might change considerably.

## What is already know on this topic

According to stress theory,[25] external challenging circumstances are stressors, while stress is a biological response resulting from to the inability of the individual to achieve a homeostasis when facing stressors. The progression from stressor to an internal stress response varies by individual and their resources and coping skills.[25] Here, in the absence of validated stress measures, we chose psychosocial stressors that pertain to the adolescent's physical surroundings or family functioning that have either previously been associated with a stress response or with reduced lung function.[26–31]

We examined school and neighborhood safety based on both their face validity (e.g. do you feel safe in your school/neighborhood: yes, no, sometimes) as well as based on previous studies that found that a child's perceived neighborhood safety[26] and school safety[27] are associated with psychological distress and psychological trauma symptoms. However, since few participants reported not feeling safe at all in their school or neighborhood, we combined the categories 'not at all' and 'sometimes' not feeling safe (reference: always feeling safe), which may have resulted in a measures of lower stress than previously assessed.

We selected self-reported 'family conflict' based upon findings associating interparental conflict with increased cortisol levels in children,[28,29] as well as findings that 6- to 7-year-old girls had reduced $FEV_1$ and FVC when mothers reported high levels of interparental conflict.[7] There was a weak negative association between FVC and $FEV_1$ and family conflict among males, but confidence intervals were wide. Previous research on interparental conflict found that the child's involvement in the family conflict (e.g. comforting the parent) as well as externalizing behaviors mediated cortisol response, which we were unable to assess. Additionally, in previous research the children were much younger (ages 5–7),[28,29] limiting the relevance of these findings for our adolescent sample.

A large body of evidence suggests that paternal absence has many negative consequences for children, including behavioral problems and psychological distress (review paper[30]). We found a negative association between $FEV_1$ and $FEF_{25-75}$ and paternal absence in males, and the association was stronger in older males 15–17, where a reduction of 269.2 ml in $FEV_1$ represents a 7% decrease from their mean $FEV_1$ (3767 ml). We are not able to explain the finding that older females with an absent father had increased $FEF_{25-75}$, but feel it might be driven by the large variability in the $FEF_{25-75}$ measure. A previous study found father-absent male adolescents had higher cortisol levels compared with father-present adolescents, but there was no difference in cortisol levels between father-present/absent adolescent females.[31] In LAFANS, we were not able to assess if a father-surrogate (stepfather, grandfather) was present in the house, which would be important in future

work to understand what 'father's absence' is truly measuring. The findings of an interaction between the absence of the father and gender are interesting and may warrant further examination.

### What this study adds

This study adds evidence to scarce literature on psychosocial stress in adolescents and reduced lung function. Our observations that effect estimates differ by gender are similar to findings by Suglia *et al.*, who reported that young boys, but not girls, had decrements in $FEV_1$ associated with exposure to community violence. Taken together, these findings are particularly thought provoking in light of recent work on gender differences and disadvantage. Researchers at the Institute for Policy Research at Northwestern demonstrated a gender gap in behavioral and educational outcomes that was more pronounced in families of low economic means. The authors found that the impact of family disadvantage, while not apparent at birth, was already pronounced in male offspring by kindergarten entry, resulting in sizable gender gaps in high school graduation and crime rates by ages 16–18 years.[32] Although the authors were agnostic to the pathways involved, they noted that low quality neighborhoods and schools were particularly detrimental to boys. As our study oversampled low-income families, our findings that the physiological effects of psychosocial stressors, as measured by lung function, may be greater among male adolescents offers modest evidence in support of this report.

### Limitations of this study

Limitations include the cross-sectional report of stressors and spirometry assessments, making it more difficult to discern temporality as well as the impact of duration for which the psychosocial stressor was experienced. Additionally, while we chose psychosocial stressors based upon empirical evidence of cortisol activity in other research, without such biomarkers, we do not know whether reported psychosocial stressors caused a stress response in the adolescent. The range of chosen stressors also necessitates multiple comparisons, lending to the possibility that some of our observed associations may have been due to chance. Future work should employ validated stress scales aimed at capturing adolescent perceived stress. Also, our sample overrepresented low-income adolescents, who may be at higher risk for adverse respiratory outcomes given their disproportionate exposure to other environmental hazards. It should therefore be recognized that our findings may have limited generalizability to different, less disadvantaged populations. Finally, the limited sample size of children ages 10–17 years with a reproducible spirometry curve for analysis did not permit explorations by race/ethnicity or nativity in our majority Hispanic sample. Non-analyzable curves were interviewer dependent and more often seen in younger children whom we did not included in the analysis. While the exclusion of unreliable measures further limited our statistical power, we expect the resulting bias to be non-differential in their impact on the reported estimate. This project also has a number of strengths. Lung function estimates for adolescents in our sample were sensitive to air pollutants [$PM_{2.5}$, $NO_2$ (data not shown)] as observed in many other studies,[33,34] giving us confidence that the spirometry estimates were robust and appropriate also for examination with psychosocial stressors. Additionally, our mean $FEV_1$ values by age and gender were similar to a large six-city cohort study conducted in the 1970–80s[35] which measured lung function growth in over 11 000 children and adolescents (data not shown).

By studying self-reported potential psychosocial stressors in a sample over-representing poor families, we attempted to elucidate the complex relationship of psychosocial stressors and respiratory health in adolescents. Our results vary by gender and age, and suggest that the lung function in older adolescent males may be negatively affected by the absence of a father or familial conflict, warranting further investigation into interactions with gender when assessing the influence on lung function in adolescents. Should these findings be replicated, they suggest that we may consider targeting public health resources towards underprivileged adolescent males when seeking to mitigate the physiological impacts of stress among adolescents.

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## References

1 Camacho-Rivera M, Kawachi I, Bennett GG *et al.* Perceptions of neighborhood safety and asthma among children and adolescents in Los Angeles: a multilevel analysis. *PLoS ONE* 2014;**9(1)**:e87524.

2 Shankardass K, McConnell R, Jerrett M *et al*. Parental stress increases the effect of traffic-related air pollution on childhood asthma incidence. *Proc Natl Acad Sci USA* 2009;**106(30)**:12406–11.

3 Turyk ME, Hernandez E, Wright RJ *et al*. Stressful life events and asthma in adolescents. *Pediatr Allergy Immunol* 2008;**19(3)**:255–63.

4 Wolf JM, Miller GE, Chen E. Parent psychological states predict changes in inflammatory markers in children with asthma and healthy children. *Brain Behav Immun* 2008;**22(4)**:433–41.

5 Wright RJ, Cohen S, Carey V *et al*. Parental stress as a predictor of wheezing in infancy: a prospective birth-cohort study. *Am J Respir Crit Care Med* 2002;**165**:358–65.

6 Milam J, McConnell R, Yao L *et al*. Parental stress and childhood wheeze in a prospective cohort study. *J Asthma* 2008;**45(4)**: 319–23.

7 Suglia SF, Ryan L, Laden F *et al*. Violence exposure, a chronic psychosocial stressor, and childhood lung function. *Psychosom Med* 2008;**70(2)**:160–9.

8 Islam T, Urman R, Gauderman WJ *et al*. Parental stress increases the detrimental effect of traffic exposure on children's lung function. *Am J Respir Crit Care Med* 2011;**184(7)**:822–7.

9 Astell-Burt T, Maynard MJ, Lenguerrand E *et al*. Effect of air pollution and racism on ethnic differences in respiratory health among adolescents living in an urban environment. *Health Place* 2013;**23(100)**:171–8.

10 Schünemann HJ, Dorn J, Grant BJ *et al*. Pulmonary function is a long-term predictor of mortality in the general population: 29-year follow-up of the Buffalo Health Study. *Chest* 2000;**118(3)**: 656–64.

11 Wright RJ. Epidemiology of stress and asthma: from constricting communities and fragile families to epigenetics. *Immunol Allergy Clin North Am* 2011;**31(1)**:19–39.

12 Provençal N, Binder E. The effects of early life stress on the epigenome: from the womb to adulthood and even before. *Exp Neurol* 2014;**268**:1–10.

13 Hänsel A, Hong S, Cámara RJ *et al*. Inflammation as a psychophysiological biomarker in chronic psychosocial stress. *Neurosci Biobehav Rev* 2010;**35**:115–21.

14 Wright RJ. Perinatal stress and early life programming of lung structure and function Rosalind. *Child Global J Child Res* 2010;**84(1)**:46–56.

15 Kubzansky LD, Wright R, Cohen S *et al*. Breathing easy: a prospective study of optimism and pulmonary function in the Normative Aging Study. *Ann Behav Med* 2002;**24(4)**:345–53.

16 Kubzansky LD, Sparrow D, Jackson B *et al*. Angry breathing: a prospective study of hostility and lung function in the Normative Aging Study. *Thorax* 2006;**61(10)**:863–8.

17 Lippman L, Anderson Moore K, Guzman L *et al*. Cognitive Interviews: Designing Survey Questions for Adolescents. In: *Flourishing Children. Defining and Testing Indicators of Positive Development*. 1st edn. Netherlands: Springer, 2014,28–9.

18 Sastry N, Ghosh-Dastidar B, Adams J *et al*. The design of a multilevel survey of children, families, and communities: The Los Angeles Family and Neighborhood Survey. *Soc Sci Res* 2006;**35(4)**:1000–24.

19 Peterson CE, Pebley AR, Sastry N *et al*. *The Los Angeles Family and Neighborhood Survey, Wave 2: User's Guide and Codebook*. Santa Monica, CA: RAND Corporation, 2012.

20 World Health Organization adolescent health. http://www.who.int/topics/adolescent_health/en/ (8 May 2015, date last accessed).

21 Ritz BR, Ghosh JKC, Tuner MW *et al*. Traffic-Related Air Pollution and Asthma in Economically Disadvantaged and High Traffic Density Neighborhoods in Los Angeles County, California. 2009:1–113.

22 Crapo R, Hankinson J, Irvin C *et al*. Standardization of spirometry. *Am J Respir Crit Care Med* 1995;**152(6)**:1107–36.

23 McConnell R, Islam T, Shankardass K *et al*. Childhood incident asthma and traffic-related air pollution at home and school. *Environ Health Perspect* 2010;**118(7)**:1021–6.

24 Wright RJ, Rodriguez M, Cohen S. Review of psychosocial stress and asthma: an integrated biopsychosocial approach. *Thorax* 1998; **53**:1066–74.

25 Aneshensel CS. Social stress: theory and research. *Ann Rev Sociol* 1992;**18**:15–38.

26 Booth J, Ayers SL, Marsiglia FF. Perceived neighborhood safety and psychologival distress: exploring protective factors. *J Sociol Soc Welfare* 2012;**39(4)**:137–56.

27 Flannery DJ, Wester KL, Singer MI. Impact of exposure to violence in school on child and adolescent mental health and behavior. *J Community Psychol* 2004;**32**:559–73.

28 Davies PT, Sturge-apple ML, Cicchetti D *et al*. The role of child adrenocortical functioning in pathways between interparental conflict and child maladjustment. *Dev Psychol* 2007;**43(4)**:918–30.

29 Davies PT, Sturge-Apple ML, Cicchetti D *et al*. Adrenocortical underpinnings of children's psychological reactivity to interparental conflict Patrick. *Child Dev* 2008;**79(6)**:1693–706.

30 McLanahan S, Tach L, Schneider D. The causal effects of father absence. *Ann Rev Sociol* 2013;**399(June)**:399–427.

31 Flinn MV, Quinlan RJ, Decker SA *et al*. Male-female differences in effects of parental absence on glucocorticoid stress response. *Hum Nat* 1996;**7(2)**:125–62.

32 Autor D, Figlio D, Roth J *et al*. Family Disadvantage and the Gender Gap in Behavioral and Educational Outcomes. Evanston, 2015. 1–68. Report No.: WP-15-16.

33 Chen Z, Salam MT, Eckel SP *et al*. Chronic effects of air pollution on respiratory health in Southern California children: findings from the Southern California Children's Health Study. *J Thorac Dis* 2015;**7(17)**:46–58.

34 Anderson JO, Thundiyil JG, Stolbach A. Clearing the air: a review of the effects of particulate matter air pollution on human health. *J Med Toxicol* 2012;**8**:166–75.

35 Wang X, Dockery DW, Wypij D *et al*. Pulmonary function between 6 and 18 years of age. *Pediatr Pulmonol* 1993;**15(2)**:75–88.