

Letter to the Editor

Synergistic effects of air pollution and psychosocial stressors on adolescent lung function

To the Editor:

A growing body of evidence has found that air pollutants, including traffic-related pollutants such as NO, NO₂, and NO_x, decrease lung function in children and adolescents.¹ It has been suggested that stress experienced in youth may modify the effects of air pollution on lung function, but data on stressors in adolescents are scarce. The Children's Health Study (CHS) conducted in Los Angeles, California, found that impairments in lung function due to traffic-related pollutants varied by parental stress levels measured earlier in the child's life.² However, a UK study of 11- to 13-year-olds found that perceived racism was not associated with lung function, and did not modify the effects of PM_{2.5} on lung function.³ Particularly in older children, it is important to capture their own perception of stressors, as peers or other social networks may alter perceived stress. Here, we explore whether self-reported psychosocial stress related to family, school, and neighborhood in adolescents' aged 10 to 17 years modifies estimated effects for the traffic-related air pollutants NO, NO₂, NO_x, and PM_{2.5} on lung function measured by spirometry.

A sample of 551 participants was drawn from the Los Angeles Family and Neighborhood Survey (L.A.FANS) wave 2, which has been previously described.⁴ Spirometry was assessed via Easy-One portable spirometers, which measured forced vital capacity (FVC), FEV₁, and forced expiratory mean flow between 25% and 75% of FVC (FEF₂₅₋₇₅). We created land use regression models relying on monitoring campaigns with passive Ogawa sampler badges for NO_x and NO₂ in 2 seasons of 2006-2007 to estimate spatially distinct annual average NO, NO_x, and NO₂ concentrations. In addition, PM_{2.5} concentrations were generated by kriging applied to available government monitoring data. Stress

was operationalized on the basis of self-reported dichotomized measures of family fighting, school safety, neighborhood safety, and paternal absence. We performed multiple linear regression for each lung function outcome (FVC, FEV₁, FEF₂₅₋₇₅) with single pollutant analyses (in separate models) for NO, NO₂, NO_x, and PM_{2.5} while adjusting for all covariates and individual psychosocial stressors. Robust standard errors were included to adjust for nonindependence due to the participation of siblings (112 sibling sets). In additional models we included Air pollutant × Stressor interaction terms, and subsequently stratified on presence/absence of stressors to assess the magnitude of effect measure modification. Additive interaction was confirmed when the interaction term had a *P* value of less than .10. In sensitivity analyses, we excluded those who reported ever having received a doctor's diagnosis of asthma and reported wheeze within the past 12 months (*n* = 37). A more complete description of the methods is available in the [Methods](#) section in this article's Online Repository at www.jacionline.org.

Descriptive and demographic characteristics of participants are presented in [Table E1](#) and air pollutant measurements are presented in [Table E2](#) in this article's Online Repository at www.jacionline.org.

All air pollutants were independently associated with FEV₁ in separate models when adjusted for absence of the father ([Table I](#)); paternal absence was not independently associated with any of the spirometry outcomes. When stratified by paternal presence/absence, air pollutants were associated with larger decrements of lung function in households where the father was absent compared with homes with a father present. This interaction was statistically significant for NO₂ and the lung function measures FEV₁ and FVC, as well as for NO and NO_x for the outcome measure FEF₂₅₋₇₅.

Similar but weaker interaction effects were observed when examining family fighting ([Table II](#)). All air pollutants were independently associated with FEV₁; however, family fighting

TABLE I. β (95% CI) for main effects of presence of father and select air pollutants, and modification of the effects of pollutants on lung function by presence/absence of father (*n* = 551)

Lung function measure	Pollutant	Main effect of father absence coadjusted for pollutant	Main effect of pollutant coadjusted for father absence	Pollutant: Father present (<i>n</i> * = 355)	Pollutant: Father absent (<i>n</i> * = 178)	<i>P</i> value†
FEV ₁						
	NO ₂	-51.5 (-154.4 to 51.4)	-59.5 (-107.9 to -11.2)	-31.5 (-85.6 to 22.6)	-142.3 (-243.5 to -41.2)	.02
	NO _x	-52.4 (-155.5 to 50.6)	-75.9 (-134.6 to -17.2)	-55.8 (-124.3 to 12.7)	-126.4 (-237.2 to -15.7)	.10
	NO	-57.9 (-161.3 to 45.5)	-60.7 (-122.4 to 1.0)	-48.2 (-119.5 to 22.9)	-98.9 (-204.6 to 6.7)	.18
	PM _{2.5}	-56.2 (-158.9 to 46.5)	-38.1 (-61.4 to -14.8)	-34.7 (-61.4 to -7.9)	-60.7 (-125.4 to 4.0)	.44
FVC						
	NO ₂	-62.9 (-180.3 to 54.5)	-45.6 (-102.7 to 11.5)	-11.6 (-71.6 to 48.4)	-161.7 (-305.5 to -17.9)	.04
	NO _x	-63.4 (-181.5 to 54.6)	-59.0 (-129.7 to 11.6)	-42.7 (-122.4 to 36.9)	-106.9 (-262.5 to 48.8)	.30
	NO	-67.5 (-185.9 to 51.0)	-48.5 (-122.2 to 25.3)	-45.2 (-130.4 to 39.9)	-65.6 (-205.7 to 74.6)	.53
	PM _{2.5}	-65.3 (-182.8 to 52.2)	-32.3 (-59.9 to -4.7)	-24.4 (-52.1 to 3.3)	-80.4 (-167.1 to 6.4)	.29
FEF ₂₅₋₇₅						
	NO ₂	-79.9 (-254.5 to 94.6)	-85.8 (-191.2 to 19.5)	-47.8 (-170.7 to 75.1)	-164.1 (-344.5 to 16.3)	.13
	NO _x	-81.7 (-255.4 to 92.0)	-107.3 (-224.2 to 9.6)	-56.6 (-194.2 to 81.0)	-218.5 (-402.0 to -34.9)	.05
	NO	-91.0 (-264.6 to 82.5)	-75.2 (-188.8 to 38.4)	-29.0 (-158.2 to 100.2)	-195.3 (-370.6 to -20.1)	.06
	PM _{2.5}	-90.2 (-265.2 to 84.8)	-51.6 (-108.0 to 4.9)	-53.2 (-120.7 to 14.3)	-54.9 (-166.7 to 56.9)	.92

Models are adjusted for age, race/ethnicity, sex, federal poverty level, household smoking, height, height-squared, weight, weight-squared, and Sex × Age.

*Adjusted *n*.

†*P* for interaction of stressor (paternal presence/absence) and select pollutant.

TABLE II. β (95% CI) for main effects of presence of family fighting and select air pollutants, and modification of the effects of pollutants on lung function by presence/absence of family fighting (n = 551)

Pollutant	Main effect of family fighting coadjusted for pollutant	Main effect of pollutant coadjusted for family fighting	Pollutant: No family fighting (n* = 475)	Pollutant: Family fighting (n* = 56)	P value†
FEV₁					
NO ₂	-73.9 (-186.3 to 38.5)	-65.2 (-114.1 to -16.2)	-56.9 (-110.6 to -3.3)	-160.9 (-248.3 to -73.5)	.26
NO _x	-74.5 (-186.4 to 37.3)	-82.1 (-141.4 to -22.7)	-72.6 (-136.5 to -8.7)	-184.6 (-294.6 to -74.6)	.32
NO	-74.8 (-187.8 to 38.3)	-66.6 (-129.3 to -3.9)	-57.4 (-125.5 to 10.6)	-199.9 (-323.6 to -76.3)	.27
PM _{2.5}	-64.8 (-181.7 to 52.1)	-39.5 (-63.4 to -15.6)	-39.3 (-63.9 to -14.8)	-75.6 (-173.3 to 22.1)	.28
FVC					
NO ₂	-61.3 (-192.1 to 69.6)	-52.0 (-109.8 to 5.7)	-46.9 (-110.3 to 16.5)	-133.6 (-252.6 to -14.7)	.44
NO _x	-61.8 (-192.1 to 68.5)	-65.8 (-136.9 to 5.3)	-57.1 (-134.6 to 20.2)	-175.9 (-313.4 to -38.5)	.37
NO	-62.1 (-193.2 to 69.0)	-54.8 (-129.3 to 19.7)	-47.2 (-128.8 to 34.4)	-197.1 (-352.4 to -41.8)	.35
PM _{2.5}	-52.7 (-186.3 to 80.9)	-33.8 (-61.9 to -5.7)	-32.3 (-60.9 to -3.7)	-94.7 (-206.7 to 17.4)	.12
FEF₂₅₋₇₅					
NO ₂	-80.3 (-299.4 to 138.7)	-93.9 (-200.6 to 12.7)	-75.5 (-194.2 to 43.2)	-207.6 (-384.5 to -30.6)	.21
NO _x	-81.2 (-300.2 to 137.9)	-116.1 (-235.1 to 3.0)	-101.7 (-231.2 to 27.9)	-183.5 (-422.6 to 55.6)	.44
NO	-80.8 (-301.4 to 139.7)	-83.2 (-199.6 to 33.2)	-68.1 (-192.9 to 56.8)	-187.9 (-454.3 to 78.3)	.37
PM _{2.5}	-73.2 (-299.3 to 153.0)	-55.5 (-113.1 to 2.1)	-55.4 (-115.2 to 4.4)	-96.1 (-325.3 to 133.2)	.59

Models are adjusted for age, race/ethnicity, sex, federal poverty level, house smoke, height, height-squared, weight, weight-squared, and Sex $\times$ Age.

*Adjusted n.

†P for interaction of stressor (family fighting yes/no) and select pollutant.

was not. Although the trends were stronger for air pollutant and spirometry measures in those who self-reported family fighting compared with those who did not, none of the interaction terms reached statistical significance.

Results for self-reported neighborhood safety, school safety, and sensitivity analyses are presented in the [Results](#) section in this article's Online Repository at www.jacionline.org.

Our findings of modification of air pollutant effects on lung function by select psychosocial stressors corroborate findings from the CHS² for parental stress measures. L.A.FANS did not use validated stress measures; therefore, we chose psychosocial stressors that pertain to the adolescent's physical surroundings or family functioning that have previously been associated either with a stress response or with reduced lung function (for detailed discussion of selected stressors, see the [Discussion](#) section in this article's Online Repository at www.jacionline.org). Psychosocial stressors, which often cluster in economically deprived neighborhoods, may explain some of the adverse effects on respiratory health observed with measures of socioeconomic status.⁵ The biologic underpinnings for synergisms between air pollution and stress on lung function may be found in the immune response and inflammatory reactions.^{6,7} Many air pollutants consist of free radicals, which in the lung tissue result in oxidative stress that generates an inflammatory response, releasing additional free radicals that ultimately damage lung tissue.⁶ Psychosocial stressors, acting through the hypothalamic-pituitary-adrenal axis modifications, also heighten inflammatory activity and modulate immune function,⁸ potentially increasing susceptibility to environmental insults. This pathway may contribute to some of the differential pulmonary vulnerability to air pollutants observed in those with higher levels of psychosocial stress.²

Briefly, strengths of our study include the use of adolescent self-reported psychosocial stressors. In addition, our spirometry estimates were sensitive to the effects of air pollution and both measures were similar to estimates obtained for air pollutants and pulmonary function from the CHS.⁹ However, validated or

more psychometrically sound instruments would have been preferential to the stress measures that we used. Although we chose psychosocial stressors based on 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. Our findings of paternal absence are difficult to interpret, as the adolescents were not further queried about their own feelings about the familial composition or related stress. In addition, our sample size did not allow us to analyze pulmonary function as a change from predicted value from a standard population, nor was our sample size large enough to calculate our own standard reference. Thus, we have reported absolute changes in pulmonary function values.

Healthy growth and development of pulmonary function in childhood and adolescence is instrumental for respiratory health in adulthood. Our findings contribute modest evidence to the hypothesis that psychosocial stress modifies the effects of air pollutants on lung function, and we hope they may inspire researchers to measure stress when conducting research on respiratory health.

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METHODS

Sample

Los Angeles Family and Neighborhood Survey 2. Participants were drawn from the Los Angeles Family and Neighborhood Survey (L.A.FANS) wave 2. The first wave (L.A.FANS-1) sampled 3090 households from 65 neighborhoods in Los Angeles County in 2000-2001, interviewing 3140 children aged 0 to 17 years with oversampling of very poor and poor households. The cohort included 55% Hispanic, 26% white, 10% black, and 7% Asian participants. In 2006-2008, L.A.FANS-2 reinterviewed L.A.FANS-1 participants and added additional households from within the same neighborhoods. A total of 1091 children were reinterviewed (64% response rate) and 296 new children were added to the sample, for a total of 1387 children. This second wave also added spirometry data collection to its protocol. To minimize lung function measurement error issues, likely to be larger in younger children, and to allow us to assess adolescents' self-reported psychosocial stress, we restricted our analyses to individuals aged 10 to 17 years (here referred to as "adolescents" following the World Health Organization nomenclature^{E1}) with a reproducible spirometry curve ($n = 551$).

Outcome, exposure, and covariate definitions

Spirometry. Of the 1387 children in L.A.FANS-2, 1070 children aged 5 to 17 years participated in spirometry assessments. Evaluation and acceptance criteria for spirometry curves have been previously detailed.^{E2} 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 on the basis of the following criteria (some of which overlap with the 1994 American Thoracic Society criteria^{E3}): (1) The back extrapolated volume must be 5% or less or 150 mL, whichever is greater; (2) Time to peak flow must be 120 milliseconds or less; (3) No abrupt end to test; (4) Forced expiratory time must be 2 seconds or more; (5) Time/volume curve must begin at origin (to ensure proper start of test); (6) Curve must show that subject exhaled using only a continuous blast of air; and (7) Curve must show no leaks or negative flow throughout test (ie, no inhalation). Only those curves that were 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 1 acceptable curve for analysis, 551 children were aged between 10 and 17 years.

Air pollution estimates. Our air pollution measures have been described previously in detail.^{E2} We monitored pollution using 186 passive Ogawa sampler badges for NO_x and NO₂ in 2 seasons of 2006-2007 in neighborhoods in which L.A.FANS participants resided. This neighborhood-level NO_x and NO₂ measurement data were then used to develop a land use-based regression model for the Los Angeles Basin, building land use-based regression prediction surfaces^{E4,E5} for NO, NO₂, and NO_x. PM_{2.5} estimates were generated by kriging available government monitoring data from 23 state and local district monitoring stations in the Los Angeles Basin for the year 2000. Participants in L.A.FANS-2 provided residential history information for the 6 years between survey waves, allowing us to create annual average measures weighted for time spent at each location for the past 5+ years. In a previous analysis, we showed that exposure measures for the 5-year, 2-year, 12-month, and current home-only models were very similar.^{E2} Thus, for the current analyses, we extracted from our models annual average measures of each pollutant, weighted for time spent at both residences and schools in the past 12 months.

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

Adolescent/household sociodemographic covariates. Data on the following covariates were collected regarding the adolescents: sex, age, height and weight, race/ethnicity, nativity, self-reported smoking, asthma

history, and wheeze within the past 12 months. Data on maternal education, household income (which was used to categorize federal poverty level), and any reported smoking in the house were also collected in the survey.

Statistical analyses

Potential confounders were selected and included in all models on the basis of the previous literature and by assessing changes in estimates for air pollutants greater than 10%. These covariates included adolescent's age, federal poverty level, smokers in the household, adolescent's race/ethnicity, height, height-squared, weight, weight-squared, sex, and Sex $\times$ Age. Few adolescents ($n = 10$) reported smoking. Inclusion of this variable in the models did not change estimates for the air pollutants and thus was not retained in final models. Analyses were conducted in SAS 9.3 (Cary, NC).

RESULTS

Neither self-reported neighborhood safety nor school safety was associated with spirometry measures, although all coadjusted air pollutant estimates were independently associated with the spirometry measures. We did not observe statistical interaction of these psychosocial stressors with any of the air pollutants (data not shown).

In sensitivity analyses, removing those with an asthma diagnosis who experienced wheeze in the past 12 months ($n = 37$) resulted in slight attenuation but did not change air pollutant estimates or interactions by more than 10% (data not shown).

DISCUSSION

Using information on family, neighborhood, and school-related psychosocial stressors collected in L.A.FANS, we were able to assess whether these measures modified the associations between select air pollutants and spirometry outcomes in adolescents aged 10 to 17 years. We found that paternal absence, while not independently associated with lung function measures, modified the association of NO₂ and both FEV₁ ($P = .02$) and FVC ($P = .04$). Although associations were modest, the deficits of FEV₁ associated with NO₂ were approximately 2% of the median FEV₁ values in the sample (2814 mL), while deficits were 5% accounting for the interaction with paternal absence. Although not statistically significant, NO, NO_x, and PM_{2.5} followed the same pattern, with stronger negative associations observed between the air pollutants and spirometry measures in adolescents from households without a father than in households with a father present. Similar trends were observed when we compared adolescents' self-reported family fighting with those who reported no family fighting, although interaction terms did not reach statistical significance. Neither self-reported school safety nor neighborhood safety modified associations of air pollutant and spirometry measures of lung function.

We were compelled by the findings of synergism between air pollutants and stress in the CHS,^{E6} but relying on early static measures of parental stress only may not appropriately represent and thus potentially misclassify the stress an adolescent is experiencing. The stress burden in a household likely changes with time and circumstance, and although parental measures of stress may influence the perceived stress of a 5-year-old child, to our knowledge the role of timing of stress during lung development in relation to decrements in lung function has not been established. As children age into adolescence, their own coping skills, peer networks, and autonomy increase. This

highlights the importance of considering adolescents' stress burden on their pulmonary function from their point of view in addition to the parents'. Without having validated stress measures available in L.A.FANS, we used measures that had face validity or previously reported associations with cortisol or lung function, as follows.

We examined school and neighborhood safety on the basis of both their face validity (eg, do you feel safe in your school/neighborhood: yes, no, sometimes) and previous studies that found that a child's perceived neighborhood safety^{E7} and school safety^{E8} are associated with psychological distress. However, because 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 measure representing much lower stress than has previously been assessed.

We selected self-reported "family conflict" on the basis of findings associating interparental conflict with increased cortisol levels in children,^{E9,E10} as well as findings that 6- to 7-year-old girls had reduced FEV₁ and FVC when mothers reported high levels of interparental conflict.^{E11} Previous research on interparental conflict found that the child's involvement in the family conflict (eg, comforting the parent) as well as externalizing behaviors mediated cortisol response. However, we acknowledge that in this previous research the children were much younger (ages 5-7 years),^{E9,E10} potentially limiting the relevance of the 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 article^{E12}). A previous study found that 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.^{E13} In L.A.FANS, we were

not able to assess whether a father-surrogate (stepfather, grandfather) was present in the house, which would be helpful to assess in future work to understand what "father's absence" is measuring.

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TABLE E1. Descriptive and demographic data for participants aged 10 to 17 years in L.A.FANS-2, a neighborhood and household survey in Los Angeles, California, 2006-2008 (n = 551)

Descriptive and demographic data	Mean $\pm$ SD or n (%)
Spirometry	
FVC (mL)	3353.1 $\pm$ 961.2
FEV ₁ (mL)	2814.0 $\pm$ 823.6
FEF ₂₅₋₇₅ (mL/s)	3114.2 $\pm$ 1153.2
Psychosocial stressors	
Neighborhood feels safe	349 (63.4)
Missing	2 (0.4)
School feels safe	423 (76.8)
Missing	5 (0.9)
Family does not fight	490 (88.9)
Missing	2 (0.4)
Dad lives in the house	367 (66.6)
Child covariates	
Age (y)	13.4 $\pm$ 2.3
Height (cm)	158.9 $\pm$ 11.7
Weight (kg)	61.4 $\pm$ 19.3
Race	
Hispanic	369 (66.8)
Non-Hispanic white	80 (14.5)
Black	27 (4.9)
Asian/Pacific Islander	28 (5.1)
Other (multiple races)	47 (8.5)
Child's nativity	
US-born	505 (91.7)
Child smokes	10 (1.8)
Missing	4 (0.7)
Asthma diagnosis with wheeze (12 mo)	37 (6.7)
Family/household covariates	
Maternal education	
$\leq$ 8th grade	133 (24.1)
9-12th grade	191 (34.7)
Vocational school	32 (5.8)
AA/some college	116 (21.1)
College+	76 (13.8)
Missing	3 (0.5)
Federal poverty level	
<100%	158 (28.7)
101% to 200%	161 (29.2)
201% to 300%	93 (16.9)
301%+	139 (25.2)
Current smoker in house	119 (21.6)

TABLE E2. Pollutant median values and ranges for L.A.FANS adolescent respondents with spirometry measures (n = 551)

Pollutant	Median, range	IQR
NO ₂ (ppb)	23.7 (6.2-34.6)	5.3
NO _x (ppb)	47.7 (11.8-90.1)	16.5
NO (ppb)	23.5 (3.5-56.5)	10.4
PM _{2.5} μm/m ³	21.7 (8.5-23.6)	1.8

IQR, Interquartile range; *ppb*, parts per billion.