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

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Capsule summary

Research on synergism of stress and air pollution on adolescent lung function is uncommon. Our study found statistically significant synergism between select psychosocial stressors and air pollution on the lung function of adolescents.

Keywords

Air pollutants; lung function; spirometry; adolescents; psychosocial stressors; synergism

To the editor

A growing body of evidence has found that air pollutants, including traffic-related pollutants such as NO, NO_2 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 is scarce. The Children's Health Study (CHS) conducted in Los Angeles, California found 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 11–13 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 10–17

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years modifies estimated effects for the traffic-related air pollutants NO, NO_2, NO_X, and $PM_{2.5}$ on lung function measured by spirometry.

A sample of 551 participants were drawn from the Los Angeles Family and Neighborhood Survey (L.A.FANS) wave 2, which has been previously described.[4] Spirometry was assessed via EasyOne™ portable spirometers, which measured forced vital capacity (FVC), forced expiratory volume after 1 second (FEV_1), and forced expiratory mean flow between 25% and 75% of FVC ($FEF_{25–75}$). We created land use regression models relying on monitoring campaigns with passive Ogawa sampler badges for NO_X and NO_2 in two seasons of 2006/7 to estimate spatially distinct annual average NO, NO_X and NO_2 concentrations. In addition, $PM_{2.5}$ concentrations were generated by kriging applied to available government monitoring data. Stress was operationalized based on 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, FEV, $FEF_{25–75}$) with single pollutant analyses (in separate models) for NO, NO_2, NO_X, and $PM_{2.5}$ while adjusting for all covariates and individual psychosocial stressors. Robust standard errors were included to adjust for non-independence 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 $p<0.10$. 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 available in Table E1, and air pollutant measurements in Table E2 in this article's Online Repository at www.jacionline.org.

All air pollutants were independently associated with FEV_1 in separate models when adjusted for absence of the father (Table 1); 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 to homes with a father present. This interaction was statistically significant for NO_2 and the lung function measures FEV_1 and FVC, as well as for NO and NO_X for the outcome measure $FEF_{25–75}$.

Similar but weaker interaction effects were observed when examining family fighting (Table 2). All air pollutants were independently associated with FEV_1; however, family fighting was not. While 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 CHS[2] 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 either previously been associated with a stress response or with reduced lung function (for detailed discussion of selected stressors, see 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 (SES).[6] The biologic underpinnings for synergisms between air pollution and stress on lung function may be found in the immune response and inflammatory reactions.[7,8] 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.[7] Psychosocial stressors, acting through HPA axis modifications, also heighten inflammatory activity and modulate immune function,[9] 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. Additionally, 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.[5] However, validated or more psychometrically sound instruments would have been preferential to the stress measures that we employed. Although 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. Our findings of paternal absence are difficult to interpret, as the adolescent was not further queried about their own feelings about the familial composition or related stress. Additionally, our sample size did not allow for 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.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Table 1

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

	Pollutant	Main effect of father absence co-adjusted for pollutant	Main effect of pollutant co-adjusted for father absence	Pollutant: Father present (n^1=355)	Pollutant: Father absent (n^1=178)	p*
FEV_1						
	NO_2	−51.5 (−154.4, 51.4)	−59.5 (−107.9, −11.2)	−31.5 (−85.6, 22.6)	−142.3 (−243.5, −41.2)	0.02
	NO_X	−52.4 (−155.5, 50.6)	−75.9 (−134.6, −17.2)	−55.8 (−124.3, 12.7)	−126.4 (−237.2, −15.7)	0.10
	NO	−57.9 (−161.3, 45.5)	−60.7 (−122.4, 1.0)	−48.2 (−119.5, 22.9)	−98.9 (−204.6, 6.7)	0.18
	$PM_{2.5}$	−56.2 (−158.9, 46.5)	−38.1 (−61.4, −14.8)	−34.7 (−61.4, −7.9)	−60.7 (−125.4, 4.0)	0.44
FVC						
	NO_2	−62.9 (−180.3, 54.5)	−45.6 (−102.7, 11.5)	−11.6 (−71.6, 48.4)	−161.7 (−305.5, −17.9)	0.04
	NO_X	−63.4 (−181.5, 54.6)	−59.0 (−129.7, 11.6)	−42.7 (−122.4, 36.9)	−106.9 (−262.5, 48.8)	0.30
	NO	−67.5 (−185.9, 51.0)	−48.5 (−122.2, 25.3)	−45.2 (−130.4, 39.9)	−65.6 (−205.7, 74.6)	0.53
	$PM_{2.5}$	−65.3 (−182.8, 52.2)	−32.3 (−59.9, −4.7)	−24.4 (−52.1, 3.3)	−80.4 (−167.1, 6.4)	0.29
$FEF_{25–75}$						
	NO_2	−79.9 (−254.5, 94.6)	−85.8 (−191.2, 19.5)	−47.8 (−170.7, 75.1)	−164.1 (−344.5, 16.3)	0.13
	NO_X	−81.7 (−255.4, 92.0)	−107.3 (−224.2, 9.6)	−56.6 (−194.2, 81.0)	−218.5 (−402.0, −34.9)	0.05
	NO	−91.0 (−264.6, 82.5)	−75.2 (−188.8, 38.4)	−29.0 (−158.2, 100.2)	−195.3 (−370.6, −20.1)	0.06
	$PM_{2.5}$	−90.2 (−265.2, 84.8)	−51.6 (−108.0, 4.9)	−53.2 (−120.7, 14.3)	−54.9 (−166.7, 56.9)	0.92

Models are adjusted for age, race/ethnicity, sex, FPL, household smoking, height, height squared, weight, weight squared, sex*age

^{1}adjusted n

*p for interaction of stressor (paternal presence/absence) and select pollutant

Table 2

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

	Pollutant	Main effect of family fighting co-adjusted for pollutant	Main effect of pollutant co-adjusted for family fighting	Pollutant: No family fighting (n^1=475)	Pollutant: Family fighting (n^1=56)	p*
FEV_1						
	NO_2	−73.9 (−186.3, 38.5)	−65.2 (−114.1, −16.2)	−56.9 (−110.6, −3.3)	−160.9 (−248.3, −73.5)	0.26
	NO_X	−74.5 (−186.4, 37.3)	−82.1 (−141.4, −22.7)	−72.6 (−136.5, −8.7)	−184.6 (−294.6, −74.6)	0.32
	NO	−74.8 (−187.8, 38.3)	−66.6 (−129.3, −3.9)	−57.4 (−125.5, 10.6)	−199.9 (−323.6, −76.3)	0.27
	$PM_{2.5}$	−64.8 (−181.7, 52.1)	−39.5 (−63.4, −15.6)	−39.3 (−63.9, −14.8)	−75.6 (−173.3, 22.1)	0.28
FVC						
	NO_2	−61.3 (−192.1, 69.6)	−52.0 (−109.8, 5.7)	−46.9 (−110.3, 16.5)	−133.6 (−252.6, −14.7)	0.44
	NO_X	−61.8 (−192.1, 68.5)	−65.8 (−136.9, 5.3)	−57.1 (−134.6, 20.2)	−175.9 (−313.4, −38.5)	0.37
	NO	−62.1 (−193.2, 69.0)	−54.8 (−129.3, 19.7)	−47.2 (−128.8, 34.4)	−197.1 (−352.4, −41.8)	0.35
	$PM_{2.5}$	−52.7 (−186.3, 80.9)	−33.8 (−61.9, −5.7)	−32.3 (−60.9, −3.7)	−94.7 (−206.7, 17.4)	0.12
$FEF_{25–75}$						
	NO_2	−80.3 (−299.4, 138.7)	−93.9 (−200.6, 12.7)	−75.5 (−194.2, 43.2)	−207.6 (−384.5, −30.6)	0.21
	NO_X	−81.2 (−300.2, 137.9)	−116.1 (−235.1, 3.0)	−101.7 (−231.2, 27.9)	−183.5 (−422.6, 55.6)	0.44
	NO	−80.8 (−301.4, 139.7)	−83.2 (−199.6, 33.2)	−68.1 (−192.9, 56.8)	−187.9 (−454.3, 78.3)	0.37
	$PM_{2.5}$	−73.2 (−299.3, 153.0)	−55.5 (−113.1, 2.1)	−55.4 (−115.2, 4.4)	−96.1 (−325.3, 133.2)	0.59

Models are adjusted for age, race/ethnicity, sex, FPL, house smoke, height, height squared, weight, weight squared, sex*age

^{1}adjusted n

*p for interaction of stressor (family fighting yes/no) and select pollutant