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Longitudinal Analysis of Particulate Air Pollutants and Adolescent Delinquent Behavior in Southern California

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Abstract

Animal experiments and cross-sectional human studies have linked particulate matter (PM) with increased behavioral problems. We conducted a longitudinal study to examine whether the trajectories of delinquent behavior are affected by $PM_{2.5}$ (PM with aerodynamic diameter ≤2.5 μm) exposures before and during adolescence. We used the parent-reported Child Behavior Checklist at age 9–18 with repeated measures every ~2–3 years (up to 4 behavioral assessments) on 682 children from the Risk Factors for Antisocial Behavior Study conducted in a multi-ethnic cohort of twins born in 1990–1995. Based on prospectively-collected residential addresses and a spatiotemporal model of ambient air concentrations in Southern California, monthly $PM_{2.5}$ estimates were aggregated to represent long-term (1-, 2-, 3-year average) exposures preceding baseline and cumulative average exposure until the last assessment. Multilevel mixed-effects models were used to examine the association between $PM_{2.5}$ exposure and individual trajectories of delinquent behavior, adjusting for within-family/within-individual correlations and potential confounders. We also examined whether psychosocial factors modified this association. The results suggest that $PM_{2.5}$ exposure at baseline and cumulative exposure during follow-up was significantly associated ($p<0.05$) with increased delinquent behavior. The estimated effect sizes (per interquartile increase of $PM_{2.5}$ by 3.12–5.18 $\mu g/m^3$) were equivalent to the difference in delinquency scores between adolescents who are 3.5–4 years apart in age. The adverse effect was stronger in families with unfavorable parent-to-child relationships, increased parental stress or maternal depressive symptoms. Overall, these findings suggest long-term $PM_{2.5}$ exposure may increase delinquent behavior of urban-dwelling adolescents, with the resulting neurotoxic effect aggravated by psychosocial adversities.

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Keywords

adolescence; delinquency; ambient fine particles; environmental exposures; epidemiologic studies; longitudinal studies

Delinquency, defined as behaviors and attitudes that violate societal norms, values, and laws, is a strong predictor of future criminal and antisocial activities (Murray and Farrington 2010). Early delinquency predicts negative outcomes later in life, including academic underachievement, unemployment, mental disorders, substance use, and dysfunctional families (Murray and Farrington 2010). As youth incarceration costs state and local governments $8–12 billion annually (Petteruti 2011), delinquent behavior, if not appropriately intervened, may amount to a significant economic and social burden.

Adolescence is an important developmental period characterized by significant social, biological, and physiological changes. This phase of functional and structural change in the prefrontal lobe is a crucial time for shaping behavioral trajectories (Carroll et al. 2014), and is a vulnerable period during which developmental processes may be easily disrupted (Rauh et al. 2010). Delinquent behavior increases during this critical time and peaks during mid-adolescence (Murray and Farrington 2010), therefore, adolescents are an important population to target for early intervention strategies. Approximately 55–87% of the total variance of delinquency is attributable to environment (Burt 2009), but previous studies have mainly focused on social factors and overlooked the influence of physical environments. Rates of juvenile delinquency are highest in urban neighborhoods (Shaw and McKay 1942), highlighting the need to identify modifiable environmental factors in these areas.

Only few environmental neurotoxicants have been implicated as modifiable risk factors for delinquent behavior. Existing literature supports an association between prenatal secondhand smoke and externalizing problems, including delinquency (Tiesler and Heinrich 2014). Early-life exposure to lead increased delinquent behavior in pre-adolescent boys and predicted adjudicated delinquency in adolescents (Needleman et al. 1996). Over the last 15 years, both experimental animal models and epidemiologic studies have reported developmental neurotoxicity of ambient air pollutants (Block et al. 2012), especially particulate matter (PM). Air pollution is the most abundant source of environmentally induced inflammation and oxidative stress, and recent reports have revealed that small particles may enter the brain contributing to structural damage and neurodegeneration (Maher et al. 2016). Cross-sectional studies (Forns et al. 2015; Haynes et al. 2011; Perera et al. 2013; Peterson et al. 2015; Yorifuji et al. 2016) have examined the association of behavioral problems with particulate air pollutants. Although $PM_{2.5}$ (PM with aerodynamic diameter < 2.5-μm) exposure is common in urban areas, very little is known about its influence on delinquent behavior, especially over time. This longitudinal study aims to investigate whether the individual trajectories of delinquent behavior are affected by residential exposure to ambient air pollutants in urban-dwelling adolescents from Southern California, a region where outdoor $PM_{2.5}$ concentrations often exceed state and national annual standards (South Coast Air Quality Management District 2013). Our secondary aim was to explore whether social stressors may enhance a child's susceptibility to the putative

adverse behavioral effects of $PM_{2.5}$, as these possible stress-pollution interactions have become increasing recognized in the literature (Cooney 2011).

Methods

Study Design

Participants were drawn from the Risk Factors for Antisocial Behavior (RFAB) twin study (Baker et al. 2007). Families were recruited from Los Angeles and surrounding counties, representative of the multi-ethnic and socioeconomically-diverse population of the greater Los Angeles area (Baker et al. 2007). The initial cohort included over 780 monozygotic and dizygotic (same-sex and opposite sex) twin pairs and triplets born in 1990–1995 and aged 9–10 years at the RFAB inception in 2000. Study protocols were approved by the Institutional Review Board at the University of Southern California (USC). We obtained informed consent from parents and informed assent from all individual participants included in the study when the twins were under the age of 18 years. Once participants were 18 years of age or older, informed consent was obtained from both parents and the twins.

The present study used data collected with up to four behavioral assessments from childhood to adolescence (Baker et al. 2007). Our study base was defined as subjects with at least two assessments of delinquent behavior at age 9–18 (n = 1299). For eligibility, children had to provide valid residential addresses at the scheduled testing dates, and their co-twin or triplet siblings also had to participate. Effects of early-life exposure were not studied, because ambient $PM_{2.5}$ data were not collected until 1999. However, comparing the putative effects of long-term exposure estimated over an extended period before behavioral assessments started may provide valuable insights to understanding the contribution of more recent exposure (e.g., 1-year average) versus remote exposure (e.g., 3-year average) affecting the trajectories of delinquent behavior. Therefore, the analytic sample was limited to participants with complete data on $PM_{2.5}$ exposures (1-, 2-, and 3-year averages) before the baseline assessment. A total of 682 subjects (from 338 families) met these criteria (Figure 1). The restriction of exposure data was not extended beyond 3 years, as it would greatly reduce sample size and statistical power.

Behavioral Assessment

Delinquent behavior over the preceding 6 months was assessed with the parent-reported version of the widely used *Child Behavior Checklist (CBCL/6–18)*. The high reliability and validity of the CBCL has been reported elsewhere (Achenbach and Rescorla 2001). The *Delinquent (Rule-Breaking) Behavior* subscale consists of 13 items, including lying and cheating, feeling no guilt, truancy, stealing, vandalism, running away, fire setting, and substance use. Each item was scored on a 3-point scale (0: not true; 1: sometimes true; and 2: very true/often true), and a continuous raw score was created by summing across items. The CBCL assessment had a relatively high internal consistency (average Cronbach's Alpha: α = 0.71) in the RFAB cohort across 4 assessments (mean age ± SD: 9.22 ± 0.42; 11.32 ± 0.92; 14.49 ± 0.76; 16.70 ± 0.66).

Estimation of Ambient Air Pollution Exposure

Residential location data and geocoding—Residential addresses, prospectively collected through parent-reports at each wave, were sent to the USC Spatial Sciences Institute for geocoding, which followed standard procedures and returned high-quality data, with successful matching by exact parcel locations or specific street segments for 98.6% of RFAB families (see baseline geographic distribution presented in Figure S1). The remaining addresses were geocoded satisfactorily with Google Earth based on visual acceptance.

Spatiotemporal generalized additive modeling for $PM_{2.5}$—Daily $PM_{2.5}$ concentrations recorded at twenty-five monitors in our study area were acquired in 2000–2014. Cross-validated spatiotemporal generalized additive models (Wood 2006) were fit to monthly average concentrations in overlapping five-year (i.e., 2000–2005, 2004–2008, 2007–2011, 2010–2014) segments at each monitor (see Supplement for more details). These models (with an average model cross-validation R^2=0.71) were then applied to estimating $PM_{2.5}$ concentrations. From the resulting monthly exposure time-series in 2000–2014 at each geocoded home location, corresponding exposure estimates were aggregated to represent the average $PM_{2.5}$ 1-, 2-, and 3-years preceding baseline (i.e., the first valid CBCL assessment), as well as the cumulative exposure over follow-up.

CALINE freeway NO_x—We estimated near-roadway exposure to nitrogen oxides (NO_x), using the CALINE4 dispersion model (Benson 1992), which incorporates roadway geometry, traffic volume and emission rate by roadway link, and meteorological conditions. Average ambient NO_x concentrations from local (within 5-km) traffic were obtained during the testing year, and cumulative exposures over follow-up were also assigned (see Supplement).

Relevant Covariate Data

A directed acyclic graph (DAG) (Howards et al. 2012) was used to identify potential confounders (Figure S2) known to predict delinquency and likely influence where people lived (and thus their exposure to ambient air pollution), including age, gender, race/ethnicity, household socioeconomic status (SES), neighborhood socioeconomic (nSES) characteristics (defined by US census data), and self-perceived neighborhood quality. Potential confounding by other covariates, including freeway NO_x, traffic density (proxy for traffic noise), neighborhood greenspace, meteorological factors (relative humidity; temperature), urbanicity, prenatal secondhand smoke and maternal depressive symptoms (proxies for early-life and maternal risk factors) were also evaluated by DAG because they correlated with $PM_{2.5}$ or increased delinquent behavior. Also included were measures of parent-to-child affect (PCA), perceived parental stress and maternal depressive symptoms (by standardized/validated instruments; see Supplement) as potential moderators.

Statistical Analysis

Three-level mixed effects models (Diggle et al. 2002) with the restricted maximum likelihood and an unstructured covariance structure were constructed by regressing continuous raw delinquency scores on air pollution exposures (1-, 2-, and 3-years prior to baseline; cumulative exposure over follow-up), while accounting for within-family (random

intercept and slope [age]) and within-individual (random intercept) correlations and multiple potential confounders, including age as a time-varying covariate (see Supplement). Separate analyses were conducted to investigate the independent effects of air pollution at each exposure temporal scale. Further analyses were also conducted to investigate whether air pollution exposures might affect the slope of delinquent behavior over time (the age slope). We ran sensitivity analyses to evaluate other potential confounding and conducted exploratory analyses on possible effect modification (see Supplement). All analyses were performed using SAS (version 9.4) and figures were created using R software (version 2.15.2).

Results

Descriptive statistics of our main exposure and outcome of interest are presented in Table S1. Our study sample (n = 682) and the excluded subjects (n = 617) had similar delinquent behavior scores and did not significantly differ by gender, ethnicity, household SES, nSES, neighborhood quality, or freeway NO_x (Table S2). Subjects included in our analyses were slightly older ($p < 0.0001$), with more entering the cohort at Wave 3 (2006–2010) when $PM_{2.5}$ exposure was lower ($p < 0.0001$).

Subjects who were enrolled earlier, girls, racial/ethnic minority groups (e.g., non-Hispanic whites; African American), from households of lower SES, living in urban areas, neighborhoods with unfavorable nSES characteristics or perceiving poorer neighborhood quality, had higher levels of ambient $PM_{2.5}$ exposures, as compared to their counterparts (Table 1). Higher $PM_{2.5}$ estimates were also observed in locations with elevated NO_x from freeway, limited greenspace, and higher temperatures and relative humidity. Parents with higher $PM_{2.5}$ exposures perceived the highest level of stress, but tended to have more favorable PCA, except for the subscale of parent-reported negative PCA. Mothers of twins with higher $PM_{2.5}$ exposures reported more depressive symptoms.

Delinquent behavior increased during adolescence. According to the intra-class correlation, 42% of the variability in delinquency was attributable to between-family differences, with the remaining 58% from within-family differences. More delinquent behavior was found in boys, African Americans, lower SES household, families perceiving poorer neighborhood quality or those living with unfavorable nSES or limited greenspace, as compared to their counterparts (Table 2). Delinquency increased with more unfavorable PCA, higher levels of parental stress, and maternal depressive symptoms.

In Table 3, we present the results of the multilevel mixed-effects models, with the regression coefficient β (95% confidence interval [CI]) expressed as the difference in delinquency scores per one interquartile range (IQR) increase of $PM_{2.5}$ (IQRs: 4.77-, 4.93-, 5.18-, and 3.12-$\mu g/m^3$ for average 1-, 2-, 3-year and cumulative monthly average, respectively). In the base model of age-dependent trajectory accounting for within-family/individual correlations, $PM_{2.5}$ was associated with increased delinquent behavior (all Ps < 0.05; Table 3). In a separate set of analyses examining interaction between $PM_{2.5}$ and age, $PM_{2.5}$ did not affect the age-related rate of change in delinquency (Table S3). Adjustment for gender, race, household SES, nSES, and perceived neighborhood quality only modestly decreased the

effect estimates and the adverse effects remained significant (Table 3). This jointly positive confounding was primarily caused by nSES characteristics, but other covariates (family SES; race/ethnicity; neighborhood quality) also had partial contributions (Tables 1 & 2). There was a consistent pattern of increased delinquency associated with $PM_{2.5}$, and the estimates of adverse effects were equivalent to the difference in delinquency scores between subjects 3.5–4 years apart in age (Table S4).

Sensitivity analyses (Table 3) showed no substantial changes to the observed adverse $PM_{2.5}$ effects for 1-year average after further accounting for freeway NO_x, traffic densities, neighborhood greenspace, ambient temperature, urbanicity, prenatal secondhand smoke, or maternal depression. Although a few estimates for 2-year, 3-year, and cumulative monthly average became less precise and did not reach statistical significance, the results were fairly consistent as indicated by the largely overlapping confidence intervals. When dichotomized age was used in the adjusted analyses, the $PM_{2.5}$ effects were fairly robust (Table 3: Model VIII), and interaction between $PM_{2.5}$ and age remained non-significant (Table S2).

Delinquency increased with high levels of freeway NO_x in the base model (Table S5). However, adjustment of potential confounders diminished the effect estimates, which became statistically non-significant.

The associations of increased delinquency with $PM_{2.5}$ did not vary substantially by either household SES or nSES, but were slightly stronger among boys and in families perceiving better neighborhood quality (Figure 2; Table S6). Children with lower levels of self-reported positive PCA, denoting a poorer parent-to-child relationship as compared to their counterpart, had much stronger (3- to 5-fold) adverse $PM_{2.5}$ effects, and most of these differences were statistically significant ($p < 0.05$; Table S7). Interestingly, the corresponding interactions were not evident for parent-related positive PCA. The adverse $PM_{2.5}$ effect was slightly greater in families with high (versus low) levels of negative PCA (with a worse parent-to-child relationship assessed by either children or parents), and also strengthened by parental stress or maternal depressive symptoms.

Discussion

In this longitudinal study on urban-dwelling children and adolescents (ages 9–18), we found parent-reported frequency of delinquent behavior increased with residential exposure to ambient $PM_{2.5}$ estimated at baseline (1-, 2-, and 3-year averages) and during follow-up. The adverse $PM_{2.5}$ effect estimates per one IQR increase were equivalent to the difference in delinquency scores between adolescents who are 3.5–4 years apart in age. These associations could not be explained by individual or household sociodemographic factors, neighborhood socioeconomic characteristics, or perceived neighborhood quality, and remained largely consistent, after statistical adjustment for freeway NO_x, traffic density, neighborhood greenspace, meteorological factors, urbanicity, prenatal secondhand smoke, or maternal depression. The observed neurotoxic $PM_{2.5}$ effects on delinquent behavior were strengthened by unfavorable parent-child relationships and parental psychosocial distress. Major strengths of this study include the well-characterized sample, the longitudinal study

design, the extended follow-up from late childhood through adolescence, and the valid measures on psychosocial risk factors, allowing us to explore social-chemical interactions.

Our novel findings may have important public health and clinical implications. In an effort to estimate the societal impact of these results, we used the individual-level effects reported here to calculate the projected population-level effects following procedures presented in Weiss (Weiss 1988). An estimated 3.5-million adolescents reside in California (Office of Women's Health 2009), with approximately 95% within urban areas (United States Census Bureau 2012). In our full sample, the average (±SD) delinquency score across all waves was 1.34 (± 2.06), almost identical to previous estimates for U.S. children and adolescents (Crijnen et al. 1999). A raw score at 2-SD above the mean (Achenbach and Rescorla 2001) represents a clinically significant level of CBCL-defined delinquent behavior. If the population of urban-dwelling adolescents in California (N=3.3 million) assumes the same empirical distributions (mean ± SD), 75,900 would present with delinquent behavior above the clinical range. In our study, an inter-quartile increase of $PM_{2.5}$ was associated with a 0.26- to 0.32- point increase in delinquency scores. Given this effect size (~0.3-points), long-term $PM_{2.5}$ exposure could presumably shift the distribution (i.e., population average) from 1.34 to 1.64. Assuming the same SD and clinically significant cut-off applies to this hypothetical scenario, this would result in an additional 16,170 cases (total cases = 92,070; a 22.4% increase) of clinically significant delinquency among California adolescents alone. Delinquency at the clinical level at age 12–16 years is a strong predictor of adult externalizing psychopathology (Hofstra et al. 2000) and future criminal conviction (Murray and Farrington 2010). It is noteworthy that both ambient $PM_{2.5}$ concentrations (South Coast Air Quality Management District 2013) and crime rates (Harris 2014) have been decreasing in Southern California. Therefore, it will be of great interest to conduct quasi-experimental studies (Dominici et al. 2014) investigating whether the declining $PM_{2.5}$ due to environmental regulations might have contributed to the reduced crime rates.

Our study addresses a critical knowledge gap in the emerging area of environmental neurosciences in studying the neurobehavioral toxicity of ambient air pollutants. While several reports have linked air pollution with cognitive deficits and internalizing behavior (Block et al. 2012), the literature is scant on externalizing psychopathology. An ecological study in Ohio reported an association of adolescents' delinquent behavior with exposure to ambient $PM_{2.5}$ and PM_{10} (Haynes et al. 2011). In a cross-sectional study of 7 to 11 year olds in Barcelona (Forns et al. 2015), parent-reported conduct problems were associated with outdoor elemental carbon (EC; highly correlated with traffic-related $PM_{2.5}$), but not with outdoor NO_2. In a Cincinnati birth cohort, parent-reported conduct problems at age 7 increased with early-life EC exposure, but the adjusted association was statistically non-significant (Newman et al. 2013). Findings from these previous studies suffered from their respective methodological weaknesses (e.g., ecological design; lack of temporality in cross-sectional analyses). In our individual-level longitudinal study, residing in places with higher ambient $PM_{2.5}$ before baseline increased delinquent behavior and such adverse effects continued during adolescence, and the observed associations remained after multiple statistical adjustments. Our study is the first to investigate whether $PM_{2.5}$ adversely affects the trajectory of delinquent behavior in children. Although delinquent behavior became more frequent as the participants grew into adolescence, our analyses suggest no evidence

for adverse $PM_{2.5}$ effects interacting with age. If these results are substantiated in studies using similar methodological approaches, criminologists and behavioral scientists studying the effects of community and neighborhood on violent and criminal behavior may need to address the potential confounding by air pollution, as it is highly correlated with many of these factors, including poverty, neighborhood crime and violence, and neighborhood quality.

Growing experimental data have provided strong neurobiological evidence supporting adverse effects of airborne particles on increased delinquent behavior. Impulsivity and emotion dysregulation are early behavioral risk factors for externalizing psychopathology (Beauchaine 2012). Behavioral phenotypes consistent with impulsivity (preference for immediate reward; fixed interval schedule-controlled performance) were increased in mice with early-life exposures to concentrated ambient ultrafine particles (Allen et al. 2014). Such neurobehavioral effects may be mediated by particle-induced alteration of the mesocorticolimbic dopamine system (Allen et al. 2014) associated with impulsive-antisocial behavior of individuals with psychopathic personality traits. Increased depressive-like responses were also observed in mice exposed to concentrated ambient $PM_{2.5}$ (Fonken et al. 2011) or nanoparticles from urban traffics (Davis et al. 2013). Very few human studies attempted to elucidate the neurobehavioral pathways linking air pollution exposure with externalizing psychopathology, and the reported results were mixed. In an urban Boston birth cohort, prenatal $PM_{2.5}$ exposure was not associated with impulsivity (commission errors of the Conner's Continuous Performance Test) at age 7 (Chiu et al. 2016). Interestingly, using the same neuropsychological tool for another urban Boston birth cohort, investigators found that traffic-related black carbon was associated with increased impulsivity ascertained at 7–14 years of age, with more prominent adverse effects in boys (Chiu et al. 2013). One cross-sectional study in Japan found that early-life exposure to suspended PM increased impulsivity-related behaviors at age 5.5 years (Yorifuji et al. 2016). In a minority urban cohort in NYC where maternal DNA adducts with polycyclic aromatic hydrocarbon (PAH) was used to measure prenatal exposure to air pollution mixture, an adverse effect on emotional self-regulation of children before age 11 was suggested, but did not adjust for secondhand smoke (Margolis et al. 2016). In a small neuroimaging subsample (n=40) selected from the same cohort, prenatal PAH exposure determined in $PM_{2.5}$ air samples did not predict externalizing symptoms (including conduct problems) at age 7–9 (Peterson et al. 2015).

Increasing data suggest that psychological stress may enhance a child's vulnerability to certain chemical exposures (Cooney 2011), denoting the significance of studying interactions of social-chemical stressors. In our study, the $PM_{2.5}$-delinquency association was several-fold stronger in adolescents with psychosocial risk factors, including low-level positive PCA (self-reported), high-level negative PCA (self- and parent-reported), parental stress, and increased maternal depressive symptoms. The neurobiological mechanisms underlying the suggested psychosocial moderation of adverse neurobehavioral effects of airborne particles are unclear. Children who endure ongoing parental neglect, harsh parenting, physical or emotional abuse, poverty, or neighborhood crime and violence are in a prolonged state of toxic stress (McEwen and Tucker 2011). Growing evidence suggests chronic stress may act upon one or more of the same critical physiological pathways as air

pollutants, including oxidative stress, inflammation, and autonomic disruption (Cooney 2011). One experimental model demonstrated that maternal stress during late gestation increased the offspring's susceptibility to the deleterious effects of prenatal diesel exhaust particles exposure (Bolton et al. 2013). The researchers further posited that the synergism of air pollutants and psychosocial factors might involve their joint actions on innate immune recognition genes and downstream neuroinflammatory cascades within the developing brain. Further studies are needed to substantiate these novel hypotheses.

Our study had limitations. First, we were unable to explore early-life exposure effects because ambient $PM_{2.5}$ data were not collected until 1999 when subjects were already 9–10 years old. Prenatal and early-childhood are critical exposure periods, which may impart much stronger neurotoxic effects, perhaps due to the higher levels of $PM_{2.5}$ prior to 1999, so our reported association may have underestimated the true adverse $PM_{2.5}$ effects on delinquency. Additionally, although our analyses did not show an interaction between $PM_{2.5}$ and age, we cannot rule out the possibility of exposure effect on early-life trajectories before age 9. Second, we used the parent-reported CBCL to assess delinquent behavior, and parents may not be aware of their children's behaviors outside of the home. Although the Youth Self Report of CBCL was available, it is not validated for ages ≤10 and was only administered after the ages 14–15, making it unsuitable for our longitudinal analyses over ages 9–18. Third, while $PM_{2.5}$ exposures based on spatiotemporal interpolation were well cross-validated, non-differential measurement errors in such estimates were unavoidable and likely attenuated the observed associations. Fourth, we did not study $PM_{2.5}$ constituencies such as metals or organic species (e.g., EC). However, the correlation between EC and $PM_{2.5}$ was very high (R^2=0.93) in Southern California communities (Gauderman et al. 2002). Fifth, we only included subjects with complete data, potentially limiting the generalizability of our findings. However, our analyses accounted for all the major differences between our analytic sample and those excluded, and the observed associations remained in various sensitivity analyses. Sixth, we could not completely rule out the possibility of confounding by urban noise exposure. Although noise exposure has well-established adverse effects on memory functions and learning (Ferguson et al. 2013), our literature review suggests no clear evidence of a relationship between noise and externalizing behaviors (especially delinquency), and the observed $PM_{2.5}$-effect remained statistically significant in our sensitivity analyses adjusting for traffic density. Lastly, we could not completely rule out the possibility of unmeasured or residual confounding by other environmental determinants of delinquent behavior. However, we conducted several sensitivity analyses (Table 3) and the $PM_{2.5}$-delinquency associations persisted after statistical adjustment of multiple spatial covariates correlated with the exposure.

Conclusions

This first longitudinal study provides supporting evidence that ambient $PM_{2.5}$ may increase delinquent behavior before and during adolescence amongst urban-dwelling 9–18 year olds. These adverse effects may be aggravated by unfavorable parent-child relationships and parental psychosocial distress. Future studies are needed to investigate whether $PM_{2.5}$ may affect early-life trajectories of externalizing behaviors. If the adverse neurobehavioral effects

of $PM_{2.5}$ are substantiated, the resulting knowledge may shed new light on interventions for adolescent delinquency and offer additional impetus to strengthen regulatory standards.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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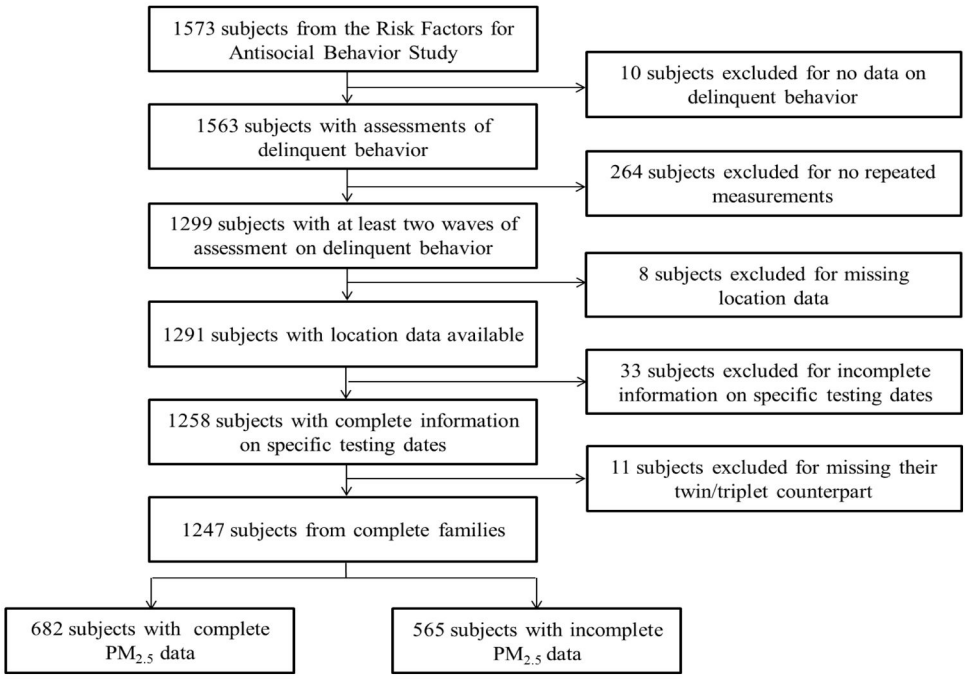

Figure 1.
Study flow chart indicating selection of study participants for current analyses.

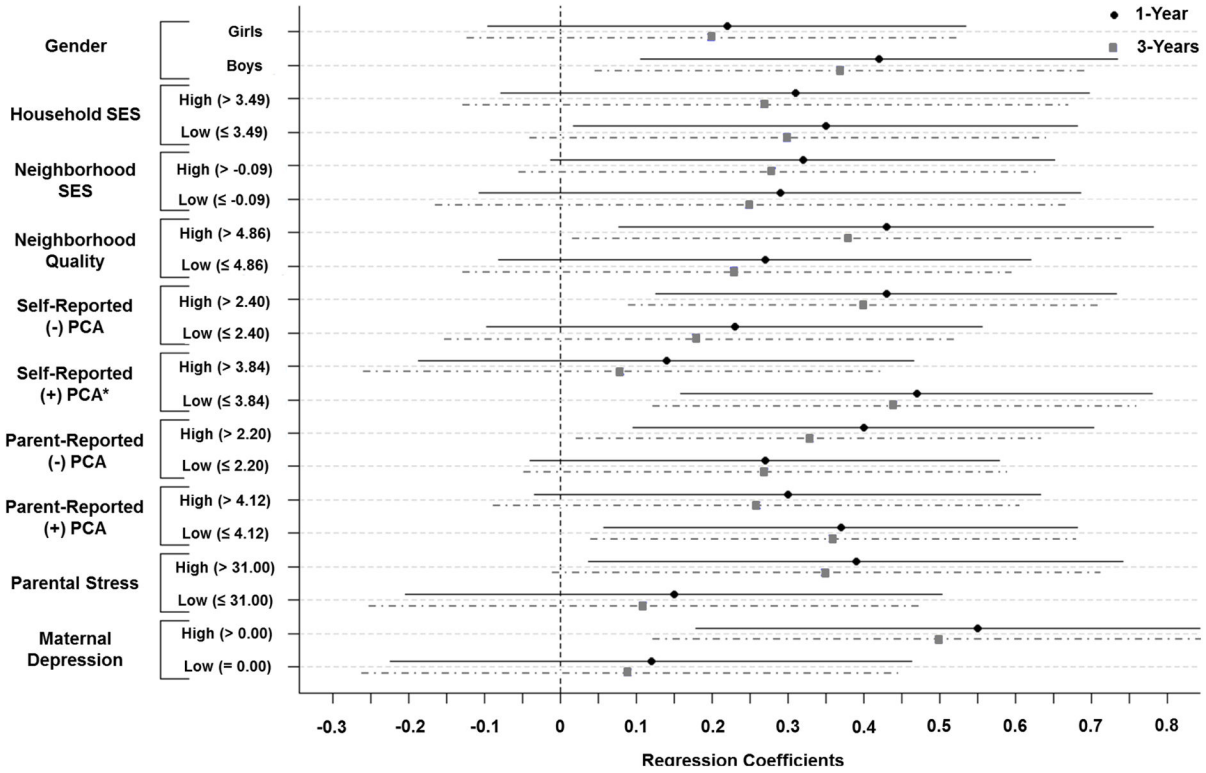

Figure 2.

Modification of 1- and 3-year $PM_{2.5}$ ($\mu g/m^3$) effects[a] on delinquent behavior by sociodemographic, neighborhood, and psychosocial factors[b].

Abbreviations: PCA, parent-to-child affect; $PM_{2.5}$, particulate matter with aerodynamic diameter < 2.5 μm; SES, socioeconomic status.

[a]Symbols (bars) represent regression coefficients (95% confidence intervals) given by multilevel mixed-effects models with statistical adjustment for within-family and within-individual correlations, age, gender, race/ethnicity, household socioeconomic status, neighborhood socioeconomic characteristics, neighborhood quality, and the corresponding effect modifier (dichotomized at the median). Black lines/symbols denote 1-year $PM_{2.5}$ and blue lines/symbols denote 3-year $PM_{2.5}$.

[b]SES: higher scores corresponding to higher SES levels; neighborhood quality: higher score for a more positive perception of neighborhood quality; PCA: higher positive scores represent better relationships and higher negative scores represent worse relationships).

*$P_{interaction} \leq 0.05$.

Table 1

Population Characteristics at Baseline in Relation to $PM_{2.5}$ Exposure 1-year Prior to Baseline (n=682)[a]

	Quartile of $PM_{2.5}$ (ug/m^3)				
Population Characteristics[b]	**7.3–14.9 Median = 13.2 (*n* = 171)**	**14.9–18.3 Median = 16.1 (*n* = 170)**	**18.3–20.5 Median = 19.4 (*n* = 170)**	**20.5–23.2 Median = 20.9 (*n* = 171)**	
Categorical variables	***n* (%)**	***n* (%)**	***n* (%)**	***n* (%)**	**p-value[c]**
Gender					<0.01
Boys	101 (31.0)	79 (24.2)	76 (23.3)	70 (21.5)	
Girls	70 (19.7)	91 (25.6)	94 (26.4)	101 (28.4)	
Ethnicity					<0.01
White	84 (37.3)	67 (29.8)	49 (21.8)	25 (11.1)	
Hispanic	32 (14.8)	40 (18.5)	52 (24.1)	92 (42.6)	
African American	10 (11.6)	18 (20.9)	28 (32.6)	30 (34.9)	
Mixed	41 (32.8)	31 (24.8)	31 (24.8)	22 (17.6)	
Other or missing	4 (13.3)	14 (46.7)	10 (33.3)	2 (6.7)	
Prenatal SHS exposure					<0.01
No	147 (27.8)	136 (25.7)	119 (22.5)	127 (24.0)	
Yes	16 (13.8)	26 (22.4)	40 (34.5)	34 (29.3)	
Urbanicity					<0.01
Non-urban areas	10 (100%)	0 (0%)	0 (0%)	0 (0%)	
Urban areas	161 (24.0%)	170 (25.3%)	170 (25.3%)	171 (25.5%)	
Continuous variables	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**p-value[d]**
Age, y	13.9 (1.9)	12.1 (2.4)	9.5 (0.7)	9.6 (0.6)	<0.01
Household SES	43.2 (8.7)	44.6 (11.3)	43.2 (13.3)	39.3 (12.6)	<0.01
Neighborhood SES	0.4 (0.9)	0.3 (1.0)	0.1 (0.9)	−0.5 (0.7)	<0.01
Neighborhood quality	51.4 (8.9)	51.1 (8.4)	50.5 (9.4)	45.1 (13.5)	<0.01
Freeway NO_x, ppb	11.1 (15.0)	16.9 (21.6)	21.4 (18.3)	23.6 (16.0)	<0.01
Traffic density	73.8 (153.5)	73.7 (124.7)	82.5 (124.7)	88.4 (146.2)	0.69
Neighborhood greenspace	0.3 (0.1)	0.3 (0.1)	0.3 (0.1)	0.3 (0.1)	<0.01
Temperature, °C	17.5 (0.9)	17.5 (0.8)	17.4 (0.7)	17.9 (0.6)	<0.01
Relative humidity, %	57.0 (9.3)	59.7 (8.1)	66.6 (7.1)	64.3 (4.9)	<0.01

Population Characteristics[b]	Quartile of $PM_{2.5}$ (ug/m^3) 7.3–14.9 Median = 13.2 (*n* = 171)	14.9–18.3 Median = 16.1 (*n* = 170)	18.3–20.5 Median = 19.4 (*n* = 170)	20.5–23.2 Median = 20.9 (*n* = 171)	
Self-reported (−) PCA	2.5 (0.7)	2.4 (0.7)	2.2 (0.8)	2.2 (0.7)	<0.01
Self-reported (+) PCA	3.6 (0.6)	3.7 (0.6)	3.9 (0.6)	3.9 (0.6)	<0.01
Parent-reported (−) PCA	2.3 (0.6)	2.4 (0.6)	2.3 (0.6)	2.3 (0.5)	0.22
Parent-reported (+) PCA	4.0 (0.4)	4.0 (0.4)	4.2 (0.4)	4.2 (0.4)	<0.01
Parental stress	30.0 (8.0)	29.7 (8.3)	32.6 (9.9)	34.2 (8.3)	<0.01
Maternal depression	0.19 (0.33)	0.18 (0.29)	0.35 (0.54)	0.37 (0.48)	<0.01

Abbreviations: NO_X, nitrogen oxides; PCA, parent-to-child affect; $PM_{2.5}$, particulate matter with aerodynamic diameter < 2.5 μm; SES, socioeconomic status; SHS, secondhand smoke.

[a] Total number of subjects decreases slightly due to missing values.

[b] SES: high scores corresponding to higher SES levels; neighborhood quality: higher score for a more positive perception of neighborhood quality; neighborhood greenspace: measured by Normalized Difference Vegetation Index, with higher scores representing more vegetation; PCA: higher positive scores represent better relationships and higher negative scores represent worse relationships.

[c] P-value for Pearson χ^2 test comparing the distribution of $PM_{2.5}$ across population characteristics.

[d] P-value for ANOVA test comparing means of population characteristics for quartile of $PM_{2.5}$.

Table 2

Population Characteristics in Relation to Levels of Delinquent Behavior Scores at Baseline (n=682[a])

Population Characteristics[b]	No Delinquency Score = 0.0 (*n* = 322)	Few Delinquencies Score = 1.0 (*n* = 161)	More Delinquencies Score ≥ 2.0 (*n* = 199)	
Categorical variables	***n* (%)**	***n* (%)**	***n* (%)**	**p-value[c]**
Gender				<0.01
Boys	130 (39.9)	81 (24.9)	115 (35.3)	
Girls	192 (53.9)	80 (22.5)	84 (23.6)	
Ethnicity				0.01
White	122 (54.2)	48 (21.3)	55 (24.4)	
Hispanic	89 (41.2)	63 (29.2)	64 (29.6)	
African American	39 (45.4)	16 (18.6)	31 (36.1)	
Mixed	60 (48.0)	22 (17.6)	43 (34.4)	
Other or missing	12 (40.0)	12 (40.0)	6 (20.0)	
Prenatal SHS exposure				0.08
No	255 (48.2)	129 (24.4)	145 (27.4)	
Yes	48 (41.4)	24 (20.7)	44 (37.9)	
Urbanicity				0.08
Non-urban areas	8 (80.0%)	0 (0%)	2 (20.0%)	
Urban areas	314 (46.7%)	161 (24.0%)	197 (29.3%)	
Continuous variables	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**p-value[d]**
Age, y	11.4 (2.5)	11.1 (2.3)	11.2 (2.5)	0.31
Household SES	44.7 (11.3)	41.1 (13.0)	40.3 (10.8)	<0.01
Neighborhood SES	0.2 (0.9)	0.1 (0.9)	−0.1 (0.9)	<0.01
Neighborhood quality	51.0 (9.5)	48.2 (11.5)	48.2 (11.2)	<0.01
Freeway NO_x, ppb	17.6 (17.8)	17.9 (18.2)	19.5 (19.7)	0.53
Traffic density	71.6 (115.9)	79.0 (133.5)	92.2 (169.8)	0.26
Neighborhood greenspace	0.3 (0.1)	0.3 (0.1)	0.3 (0.1)	0.02
Temperature, °C	17.6 (0.7)	17.7 (0.7)	17.6 (0.9)	0.54
Relative humidity, %	62.3 (8.1)	61.5 (9.0)	61.6 (8.4)	0.54
Self-reported (−) PCA	2.3 (0.7)	2.2 (0.7)	2.5 (0.7)	<0.01
Self-reported (+) PCA	3.8 (0.6)	3.8 (0.6)	3.7 (0.6)	0.02
Parent-reported (−) PCA	2.2 (0.6)	2.4 (0.6)	2.5 (0.6)	<0.01
Parent-reported (+) PCA	4.2 (0.4)	4.1 (0.4)	4.0 (0.4)	0.01
Parental stress	29.8 (8.7)	31.6 (7.9)	34.6 (9.0)	<0.01
Maternal depression	0.24 (0.42)	0.26 (0.42)	0.36 (0.47)	<0.01

Abbreviations: NO_X, nitrogen oxides; PCA, parent-to-child affect; SES, socioeconomic status; SHS, secondhand smoke.

[a] Total number of subjects decreases slightly due to missing values.

[b] SES: higher scores corresponding to higher SES levels; neighborhood quality: higher score for a more positive perception of neighborhood quality; neighborhood greenspace: measured by Normalized Difference Vegetation Index, with higher scores representing more vegetation; PCA: higher positive scores represent better relationships and higher negative scores represent worse relationships.

[c] P-value for Pearson χ^2 test comparing the distribution of delinquent behavior across population characteristics.

[d] P-value for ANOVA test comparing means of population characteristics for level of behavior.

Table 3

Associations[a] Between $PM_{2.5}$ Exposure and Repeated Measures of Delinquent Behavior

Models[b]	$PM_{2.5}$ 1-year prior to baseline β (95% CI)	$PM_{2.5}$ 2-years prior to baseline β (95% CI)	$PM_{2.5}$ 3-years prior to baseline β (95% CI)	$PM_{2.5}$ average over follow-up β (95% CI)
Base[c]	0.36* (0.12, 0.60)	0.32* (0.08, 0.56)	0.33* (0.08, 0.58)	0.30* (0.09, 0.52)
Fully Adjusted[d]	0.32* (0.06, 0.59)	0.28* (0.02, 0.54)	0.28* (0.01, 0.56)	0.26* (0.02, 0.51)
Sensitivity Analyses				
I[e]	0.30* (0.03, 0.57)	0.25 (−0.02, 0.52)	0.25 (−0.02, 0.53)	0.25* (0.00, 0.50)
II[f]	0.32* (0.06, 0.59)	0.28* (0.02, 0.54)	0.28* (0.01, 0.56)	0.26* (0.02, 0.51)
III[g]	0.34* (0.07, 0.62)	0.30* (0.03, 0.56)	0.30* (0.02, 0.58)	0.29* (0.04, 0.54)
IV[h]	0.33* (0.06, 0.59)	0.28* (0.02, 0.54)	0.29* (0.01, 0.56)	0.25 (−0.00, 0.50)
V[i]	0.32* (0.05, 0.59)	0.27* (0.01, 0.54)	0.28* (0.00, 0. 56)	0.26* (0.01, 0.51)
VI[j]	0.30* (0.03, 0.58)	0.25 (−0.02, 0.52)	0.26 (−0.02, 0.54)	0.25 (−0.00, 0.51)
VII[k]	0.33* (0.05, 0.60)	0.27* (0.00, 0.54)	0.28* (0.00, 0.57)	0.26* (0.01, 0.51)
VIII[l]	0.33* (0.06, 0.61)	0.28* (0.01, 0.56)	0.28 (−0.00, 0.57)	0.27* (0.02, 0.52)

Abbreviations: CI, confidence interval; $PM_{2.5}$, particulate matter with aerodynamic diameter < 2.5 μm.

[a] The results of the multilevel mixed-effects models with the regression coefficient (95% CI) representing the difference in parent-reported delinquent behavior scores across the interquartile range of $PM_{2.5}$ (4.77-, 4.93-, 5.18-, and 3.12-μg/m^3 for average 1-, 2-, 3-year and cumulative monthly average, respectively).

[b] n = 682 included in analyses except for sensitivity analyses VI (n=645) and VII (n=656)

[c] Accounting for within-family (random intercept and slope [age])/within-individual (random intercept) correlations in the model of age-dependent trajectory.

[d] Base + gender, ethnicity, household socioeconomic status, neighborhood socioeconomic characteristics, and neighborhood quality.

[e] Adjusted + freeway NO_X.

[f] Adjusted + traffic density in 300m area.

[g] Adjusted + neighborhood greenspace in 250m buffer.

[h] Adjusted + ambient temperature.

[i] Adjusted + urbanicity.

[j] Adjusted + prenatal secondhand smoke exposure.

[k] Adjusted + maternal depression.

[l] Adjusted model using age as dichotomized variable (age ≥ 13 years vs. age < 12 years).

* $p<0.05$.