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Residential Proximity to Traffic and Female Pubertal Development

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Abstract

Background—Traffic-related air pollution (TRAP) has been linked with several adverse health outcomes, including preterm birth and low birth weight, which are both related to onset of puberty. No studies to date have investigated the association between TRAP and altered pubertal timing.

Objective—Determine the association between residential proximity to traffic, as a marker of long-term TRAP exposure, and age at pubertal onset in a longitudinal study of girls.

Methods—We analyzed data for 437 girls at the CYGNET study site of the Breast Cancer and Environment Research Program. TRAP exposure was assessed using several measures of residential proximity to traffic based on address at study entry. Using accelerated failure time models, we calculated time ratios (TRs) and their corresponding 95% confidence intervals (CIs) for specified traffic metrics and pubertal onset, defined as stage 2 or higher for breast or pubic hair development (respectively, B2+ and PH2+). Models were adjusted for race/ethnicity, household income, and cotinine levels.

Results—At baseline, 71% of girls lived within 150m of a major road. The median age of onset was 10.3 years for B2+ and 10.9 years for PH2+. Living within 150m downwind of a major road was associated with earlier onset of PH2+ (adjTR 0.96, 95% CI 0.93, 0.99). Girls in the highest quintile of either distance-weighted traffic density, annual average daily traffic, and/or traffic density also reached PH2+ earlier than girls in the lowest quintiles.

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Conclusions—In this first study to assess the association between residential proximity to traffic and pubertal onset we found girls with higher exposure reached one pubertal milestone several months earlier than low exposed girls, even after consideration of likely confounders. Results should be expanded in larger epidemiological studies, and with measured levels of air pollutants.

Keywords

Puberty; Breast development; Traffic-related air pollution; Cohort study

1. Introduction

Epidemiologic evidence suggests that the age of onset of pubertal maturation in girls has declined over the past thirty years (Biro et al., 2010; Biro et al., 2013; Herman-Giddens, 2007; Herman-Giddens et al., 1997). Markers of pubertal onset include breast development (thelarche), pubic hair development (pubarche), and later, menstruation (menarche). Early age at transition to these pubertal markers is associated with adverse psychosocial and health outcomes later in life, including obesity, depression, and breast cancer (Biro and Deardorff, 2013; Laitinen et al., 2001; Pierce and Leon, 2005). Specifically, early age at menarche is associated with a 30% increased risk for breast cancer (Okasha et al., 2003). Increases in childhood obesity may account for part of this decline in age of pubertal onset, though human and animal studies conclude that the trend cannot be explained by nutritional status alone (Biro et al., 2013; Himes et al., 2009; Wang, 2002).

Accumulating evidence suggests that exposure to environmental toxicants during critical windows of susceptibility may be associated with altered pubertal timing (Biro et al., 2012; Biro et al., 2009; Euling et al., 2008). Specifically, researchers have linked endocrine disrupting chemicals (EDCs) with altered pubertal maturation (Ozen and Darcan, 2011). Air pollution from traffic is composed of a diverse mixture of organic compounds, including particulate matter, which may have polycyclic aromatic hydrocarbons (PAHs) and oxidant metals adsorbed to the surface. PAHs are known to have estrogenic and endocrine disrupting properties and in animal studies have been found to interfere with both reproductive and pubertal development (Schug et al., 2011). Additionally, heavy metal exposure has previously been associated with delayed pubertal development in girls (Gollenberg et al., 2010; Selevan et al., 2003). Similarly, tobacco smoke contains thousands of chemicals, many found in air pollution, including PAHs, particulate matter, and metals. Several studies have examined prenatal maternal smoking in relation to age at menarche, with many, but not all, showing earlier onset, as reviewed in a recent meta-analysis (Yermachenko and Dvornyk, 2015). One study examined breast and pubic hair development in relation to prenatal tobacco smoke (PNS) exposure and also found evidence of earlier onset of pubertal transition (Maisonet et al., 2010). Only a handful of studies have examined childhood second-hand smoke exposure in relation to age at menarche, with a couple showing earlier onset, but results are inconsistent (Ferris et al., 2010; Reynolds et al., 2004; Shrestha et al., 2011; Windham et al., 2004).

Both short and long-term traffic-related air pollution (TRAP) exposure has been associated with adverse health effects, particularly for vulnerable populations such as pregnant women

and children (McConnell et al., 2015; Olsson et al., 2015). Children are more vulnerable to the effects of air pollution because they breathe more air per unit of body weight than adults (Arcus-Arth and Blaisdell, 2007) and spend more time outdoors (Bateson and Schwartz, 2008). Studies have shown associations between TRAP during childhood with both immediate and long-term health outcomes, including obesity and asthma (Jerrett et al., 2014; McConnell et al., 2010). There is also compelling evidence for an association between air pollution exposure during pregnancy and several health outcomes in the offspring including low birth weight and small for gestational age (SGA) (Ghosh et al., 2012; Laurent et al., 2013; Stieb et al., 2016). Previous studies have found associations between low birth weight and SGA and early transition to several pubertal milestones including pubarche (Ibanez et al., 2011; Yermachenko and Dvornyk, 2014). No epidemiological studies to date have assessed the association between TRAP exposure and pubertal development, however, which is biologically plausible based on potential endocrine-disrupting activity or obesogenic effects.

To investigate the association between measures of traffic-related air pollution exposure and pubertal onset, we made use of rich existing longitudinal data from The Breast Cancer and the Environmental Research Program (BCERP). BCERP was developed with an aim of assessing prepubertal environmental exposures that may affect pubertal development and predispose a woman to breast cancer. Using data from BCERP, the current study aims to examine the association between exposure to traffic emissions, as measured by residential proximity to major roads and highways and various traffic metrics, and altered pubertal timing. Proximity to traffic metrics have been used in several other studies that have assessed the health effects of TRAP (Girguis et al., 2016; Green et al., 2009) and have been found to be correlated with measured levels of traffic related air pollutants (Gauderman et al., 2005; Spira-Cohen et al., 2010). Due to the ubiquitous nature of traffic-related air pollution, even small effects on age of puberty will have important public health implications.

2. Methods

2.1 Study Population

The study objective was investigated using data from the Cohort Study of Young Girls' Nutrition, Environment, and Transitions (CYGNET), which is part of the BCERP network (Hiatt et al., 2009). Kaiser Permanente Northern California (KPNC) recruited girls that were born in and current members of the KPNC Health Plan in the San Francisco Bay Area. Girls were eligible for participation in the study if they were between the ages of 6-8 and had no underlying endocrine-associated medical conditions. Informed consent was obtained from parent or guardian and assent was obtained from each child. The final enrolled sample consisted of 444 girls. Baseline clinical visits were conducted between June 2005 and August 2006 and girls were followed annually (until 2012 in this analysis) to measure onset and progression of pubertal maturation.

2.2 Data Collection

At each annual visit, girls' anthropometric measurements and Tanner staging for breast and pubic hair development were assessed (Biro et al., 2010). Tanner staging uses a five-stage scale for both breast and pubic hair development, with stage one (B1 and PH1) corresponding to a pre-pubertal state. Onset of pubertal development occurs when girls have reached breast stage 2 (B2) or pubic hair stage 2 (PH2) or above (2+). At baseline and each annual follow-up visit, breast and pubic hair stages were assessed by inspection and palpation by trained study staff (Biro et al., 2010). Height and weight were also measured annually, and for this analysis BMI at baseline (e.g. first exam visit) was calculated (kg/m^2). Centers for Disease Control and Prevention (CDC) growth charts were used to convert BMI values into age- and gender- specific BMI percentile levels. Categories of BMI were defined as girls below the age and sex-specific 50^{th} percentile, 50-85^{th} percentile, above the 85^{th} percentile (overweight) and above the 95^{th} percentile (obese).

At baseline and annually, the parent or guardian of each girl completed a questionnaire on medical history, product use and exposures, demographic variables, and residential history. Information was additionally collected on parental smoking status and number of smokers in the household. Cotinine, the primary metabolite of nicotine, was measured by a sensitive assay in urine collected at baseline and was categorized into quartiles. Parents or guardians identified girls as African American, White, Asian, or other, and ethnicity as Hispanic or non-Hispanic. Girl's race/ethnicity was classified into mutually exclusive categories in the following priority order: Black (regardless of ethnicity), Hispanic (including any race other than Black), non-Hispanic Asian or Pacific Islander, and non-Hispanic White or other race/ethnicity. Other potential covariates obtained from the baseline questionnaire included annual household income, education of the primary caregiver (the majority of whom were the mother), parental/guardian marital status, and maternal age at menarche.

2.3 Exposure Assessment

Proximity to traffic metrics were used as markers of traffic-related air pollution exposure. Residential addresses were collected at study baseline (June 2005-August 2006) for all participants and were used to assign exposure levels. All traffic metrics were created using annualized traffic data from 2004. Thus, the assigned traffic metrics capture long-term averages of exposure for the year or so prior to baseline (or study enrollment). Residential addresses were geocoded using the California Environmental Health Tracking Program's (CEHTP) geocoding tool. The CEHTP geocoding tool provides 96-98% match rates and is used to standardize, verify, and geocode address data to latitude/longitude coordinates and other political boundaries. We were able to successfully geocode all but one address. Six of the addresses were PO boxes, thus were excluded from analyses. Therefore, the final sample size consisted of 437 participants.

Each geocoded address was linked to traffic exposure data using the CEHTP's Traffic Volume Linkage Tool. Supplementary Figure 1 provides example output from the linkage tool. This tool uses California Department of Transportation (CalTrans) Highway Performance Monitoring System (HPMS) data from 2004 to calculate average annual daily traffic (AADT), creating various traffic metrics (CA Env Health Tracking Program, 2013).

Only principal arterial interstates, principal arterial freeways and highways, minor arterials, and major and minor collectors were included in the HPMS database. The database does not include traffic flow for local residential streets. The AADT represents the average number of vehicles travelling in both directions on a road segment. Traffic metrics were calculated within a 150 m buffer zone around each geocoded address. We placed emphasis on the 150 m buffer since research shows that 80-90% of pollutants decay between 150 and 200 meters (English et al., 1999).

A variety of traffic metrics were used for the analysis (Table 1), including *a) distance to the nearest major road or highway*, measured in meters; calculated as the shortest distance from the residence to the middle of the nearest set of lanes of the major road or highway. Major road or highways include all the HPMS road types listed above, so do not include local residential streets. *b) Maximum AADT*, which represents the daily maximum traffic volume within the buffer, and *c) closest AADT*, defined as AADT on the closest major road within the buffer (b and c both measured in vehicles/day). *d)* The *distance-weighted traffic density* (DWTD); this metric is calculated with the assumption that 96% of traffic exhaust emissions disperse at 500 ft. (152.4 m) from the road according to a Gaussian distribution. For the DWTD metric, a Gaussian weight was applied based on inverse of distance from residence (Pearson et al., 2000). *e*) *Traffic density*, measured as the sum of all segment length-adjusted traffic volumes within buffer (vehicle-km/hr). When analyzing mean levels of traffic metrics (Table 2), a default AADT value of 400 and DWTD value of 50 was assigned to residential addresses with only small local roads within the buffer, for which no AADT or DWTD is provided (Wilhelm and Ritz, 2003), which form the comparison group for categorical analyses. Supplementary Table 1 provides descriptive statistics for the included traffic metrics.

2.4 Statistical Analyses

The mean and standard deviations of continuous traffic metrics were first examined by covariates of interest using analysis of variance (ANOVA) (with $p<0.05$ representing statistical significance). Traffic volume and density metrics were categorized into five categories, with the first level, or referent group, representing only local traffic exposure and the remainder divided into quartiles (hereinafter abbreviated as “quintiles”). Distance to nearest major roadway was categorized as <50 m, 50-100 m, 100-150 m, and only local traffic exposure (>150 m from a major road/highway). We also examined associations with traffic metrics taking wind direction into consideration. The majority of highways in the study area run north/south and winds in the study area are from the west. Thus for the current analysis, locations east of the freeway were classified as downwind and those west of the freeway were classified as upwind (Kim et al., 2008). Proximity to nearest roadway taking wind direction into consideration was categorized as local traffic (reference), within 150 m upwind, or within 150 m downwind of a major road.

Potential confounders were identified *a priori* due to past associations with traffic-related air pollution exposure and pubertal onset. These covariates include: race/ethnicity, secondhand smoke exposure represented by cotinine level, and socioeconomic status (represented by household income). As BMI may be on the pathway to pubertal onset, mediation analyses

were conducted with BMI at baseline and a dichotomous measure of roadway using the Baron and Kenny method (Baron and Kenny, 1986). For the dichotomous measure of roadway proximity exposure, age at pubertal onset of girls within 150 m of a major road/highway were compared to girls living >150 m of a major road/highway (reference group).

We modeled exposure to traffic emissions and onset of puberty, assessing age at breast and pubic hair stage 2 or higher (B2+ and PH2+ respectively), throughout the approximate seven years of follow up. Weibull accelerated failure time (AFT) models were used to obtain time ratios (TR) and their 95% confidence intervals (CI) for the relation between each traffic metric and age at entry into B2+ and PH2+. A TR of >1.0 indicates a longer time to pubertal onset, while a TR of <1.0 generally indicates earlier age at pubertal development compared to the reference group. With typical median ages of B2+ and PH2+ between nine and 10 years old, small TRs can reflect a relatively large difference in age (e.g., 9.5y/10y = 0.95, representing a six-month lag or 5% earlier onset). We used model results to calculate the median age of onset of B2+ and PH2+ for the reference level of each traffic metric, along with differences (in months) in median age for each exposure level, compared to the referent.

Accelerated failure time models were used to account for interval censoring, since girls entered pubertal development at an unknown time between study visits (SAS Proc Lifereg, SAS v. 9.3). For girls who reached Tanner stage 2+ during observed follow-up visits, the interval was defined as the period from the last exam visit consistently at stage 1 to the first visit where the girl was observed to be consistently at stage 2 or greater (e.g. the girl did not return to B1 in a subsequent visit). AFT models also account for left and right censoring, e.g. of girls who had already entered puberty at enrollment and others that had not entered by the last annual visit included in this analysis, respectively. Because girls may have moved between baseline and onset of puberty, we conducted sensitivity analyses including only the girls who remained at the same address throughout the follow-up period (N=288) and results were compared with the full sample. All data were analyzed using SAS version 9.3 (SAS Institute, Cary, NC, USA).

3. Results

Of the 437 participants included, 311 (71%) lived within 150 m of a major road/highway at baseline. Table 2 shows variation in the mean traffic metric levels by covariates. Mean maximum AADT and DWTD levels varied significantly by household income, with girls in the lowest income category experiencing the highest levels of daily traffic exposure. African American girls and those who were either overweight or obese tended to live in closer proximity to a major road/highway, though mean distance levels did not vary significantly by any of the covariates of interest. Additionally, DWTD levels varied significantly by cotinine levels in girls, with those in the highest cotinine quartile experiencing the highest traffic volume exposure (not adjusted for other factors). The median age of onset was 10.3 years for B2+ and 10.9 years for PH2+. Modeled median age of onset of B2+ was 9.8, 10.1, 10.4, and 10.6 years for black, Asian, Hispanic, and non-Hispanic white participants, respectively (data not shown).

There was strong correlation between the DWTD, AADT, and traffic density metric groupings within the girls, but lower and inverse correlations with distance (Supplementary Table 2). The percentage of girls that stayed in the same exposure category across these traffic metrics was 57%. Interestingly, only 28 girls (6.3%) were in the highest exposure quintile for all four traffic volume metrics, while the same 126 girls always remained in the unexposed (local traffic) group, by definition.

Table 3 presents the adjusted associations between traffic metrics (closest AADT, maximum AADT, distance weighted traffic density, and traffic density) and onset of breast and pubic hair development, which were fairly similar to the unadjusted effect estimates. Generally, residential proximity to higher levels of traffic volume was associated with earlier transition to PH2+, but not in a monotonic trend pattern. Girls in the highest AADT, DWTD, and traffic density quintiles reached PH2+ earlier than girls in the lowest quintiles (adjusted TR 0.97, 95% CI: 0.93, 1.01, representing 3.8 months earlier than the referent group). Girls in other categories (above the reference) also had earlier onset, with some of these categories showing stronger statistical significance than the highest level. Specifically, the strongest association was for the fourth category of distance-weighted traffic density quintile with an adjusted TR of 0.93 (95% CI: 0.90, 0.97, representing 9 months earlier than the referent group).

When using distance to nearest major road/highway as the exposure metric (Table 4), associations with earlier onset of pubic hair development were seen for closer proximity. However, this again was not in a monotonic trend, but rather a threshold for any category more than local traffic, with those living within 50-100 m of a major road or highway developing the earliest (TR: 0.95, 95% CI: 0.91, 0.99, or 6.4 months earlier compared to the referent group). Those living within 150 m downwind of a major road or highway also reached PH2+ earlier than those living further away, with a TR of 0.96 (95% CI: 0.93, 0.99), but these were very similar to upwind effect estimates. Results were similar for girls who remained at the same address throughout the study period, which should reduce potential misclassification (data not shown). There were no consistent significant associations between any of the traffic volume, density, or distance metrics and onset of breast development.

Although BMI (categorized as overweight/obese vs. normal) was associated with both breast (TR: 0.96, 95% CI: 0.94, 0.99) and pubic hair development (TR: 0.95, 95% CI: 0.92, 0.97), it was not associated with the traffic metrics. Further, there was no evidence from our analyses that BMI mediated the association between residential proximity to traffic and the pubertal outcomes (data not shown).

4. Discussion

We investigated the association between residential proximity to traffic, as a marker for traffic-related air pollution, and age of pubertal onset in a longitudinal study of girls, the first study to do so to our knowledge. We found that girls with potentially higher exposure reached one pubertal milestone several months earlier than low exposed girls. Specifically, we found that girls living within 150 m of a major road or highway reached pubarche earlier,

which was consistent with findings for the highest distance weighted traffic density, annual average daily traffic, and traffic density quintiles. In contrast, we did not find significant associations of any of the traffic metrics with timing of breast development.

This particular exposure is of interest because chemicals in traffic-related air pollution may have endocrine disrupting properties that could accelerate or delay pubertal development in girls. Specifically, PAHs are known to have estrogenic and endocrine disrupting properties and in animal studies have been found to interfere with both reproductive and pubertal development (Schug et al., 2011). Findings from previous analyses on early life tobacco exposure and pubertal development have shown tobacco exposure, which contains some of same type of chemicals, to be associated with earlier pubertal maturation (Maisonet et al., 2010; Windham et al., 2004). Results from this cohort also indicate earlier onset of pubarche with greater secondhand and prenatal smoke exposures (Windham et al., 2014). Further, studies have shown associations between in utero exposure to air pollution and both low birth weight and SGA, which may also be contributing factors to early puberty in girls (Stieb et al., 2016; Wilhelm et al., 2012). Studies have found low birth weight and SGA to be risk factors for premature pubarche, which may be mediated through insulin resistance (Ibanez et al., 2011; Ibanez et al., 2001; Yermachenko and Dvornyk, 2014).

Previous studies have also found exposure to endocrine disrupting chemicals (EDCs) in general to be associated with both early and late pubertal development, often measured as menarche, not the earlier markers we examined. Specifically, epidemiologic studies have suggested that exposure to EDCs such as phthalates, dichlorodiphenyldichloroethylene (DDE), and polybrominated biphenyls (PBB) may be associated with earlier menarche (Ozen and Darcan, 2011). However, other studies have found opposite associations, e.g. exposure to EDCs such as lead, phenols, and PCBs was associated with delayed pubertal development in girls, including in the same cohort (Selevan et al., 2003; Windham et al., 2015; Wolff et al., 2015).

Previous epidemiological studies have found associations between early life air pollution exposure and childhood obesity (Fleisch et al., 2016; Jerrett et al., 2014; McConnell et al., 2016; McConnell et al., 2015). There is as well an established association between overweight/obesity in girls and early onset of pubertal development (Biro et al., 2006). Specifically, previous analyses from BCERP found that girls with a higher BMI reached breast maturation earlier (Biro et al., 2013). Thus, it is plausible that BMI could potentially be a mediator on the pathway between early life TRAP exposure and pubertal development. Air pollution from traffic sources could be a potential obesogen and exposure to high levels of air pollution during early life could therefore lead to overweight/obesity in some girls, which could contribute to early pubertal development. In the current study, however, we did not find a strong association between BMI and measures of traffic exposure, nor evidence for mediation of the proximity to traffic-puberty association by overweight/obesity status.

In the current study, we found associations between residential proximity to traffic and earlier onset of pubarche. Exposed girls developed on average 2-9 months earlier than unexposed girls, which is comparable to other predictors of pubertal timing including BMI. Premature pubarche is associated with several later life health outcomes. Specifically,

studies have found premature pubarche to be a risk factor for later development of PCOS (Ibanez et al., 2007; Ibanez et al., 2001). In addition, metabolic syndrome is more commonly seen in young women with both premature pubarche and obesity (Ibanez et al., 2009). We did not observe associations between proximity to traffic and onset of breast development. Pubarche and thelarche are controlled by different mechanisms, with the former being triggered from activation of the adrenals and the latter from the ovaries. As a result, EDC exposures can have different effects depending on their mode of action, e.g. estrogenic, androgenic or anti-androgenic. It is additionally possible that misclassification of breast development could have biased results toward the null for onset of breast development.

The current study has several strengths and limitations. This first study to address the question of whether early life proximity to traffic is associated with pubertal development is based on prospective data from a multiethnic cohort with clinical Tanner staging measured at annual follow-up visits by trained study staff (vs. retrospective, or self-report as in previous studies of EDCs). Further, results were consistent across metrics and adjusted for some confounders, including exposure to tobacco smoke.

The study is not without potential limitations, which should be considered. Studies, such as ours, that use traffic proximity as a marker of traffic-related air pollution, rather than actual measures of pollutants, may be subject to misclassification. By using these traffic metrics to characterize exposure, we are assuming that closer proximity equates to greater exposure to the pollutants from traffic-related air pollution, which has been substantiated by prior studies (Spira-Cohen et al., 2010). Further, all traffic metrics were created using annualized traffic data from 2004 and the address at baseline of study was used for linking participants to traffic metrics. Thus, the assigned traffic metrics capture long-term averages of exposure for the year or so prior to baseline. It may be, however, that a more appropriate window of exposure for pubertal development is during the *in utero* period or first year of life. Alternatively, exposure just prior to pubertal onset may have effects, which we attempted to examine in models that included only girls with the same address throughout the follow-up period, but did not see differences in results. We additionally did not account for changes in traffic patterns in the same neighborhood over time.

We did not observe monotonic trends with the traffic metrics and pubarche, which could reflect misclassification of exposure or be due to uncontrolled confounding or small number of participants in each exposure category. We chose residents >150m from of a major road or highway to serve as our reference group for all analyses. It's possible that those living further from major roads could differ with regards to the presence of measured or unmeasured factors. We did account for some of those factors, such as measures of SES and smoke exposure, but some were unmeasured such as other environmental stressors and neighborhood characteristics.

5. Conclusions

The current study takes advantage of a diverse population with longitudinal clinical outcome data measured annually to examine the hypothesis that residential proximity to traffic is associated with altered pubertal timing in girls. We found girls with potentially higher

exposure reached one pubertal milestone several months earlier than low exposed girls. Future studies should examine the question using the entire BCERP cohort or other carefully designed epidemiological studies that can examine multiple exposure windows or measured levels of air pollutants such as NOx and $PM_{2.5}$.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- We assessed associations of traffic-related air pollution and altered puberty
- Proximity to traffic was used as a marker of traffic-related air pollution exposure
- Higher traffic exposure was associated with earlier onset of one pubertal milestone
- Results should be expanded in larger studies and with measured levels of pollutants

Table 1

Description of primary traffic metrics of interest

Traffic metric	Measurement	Description
Distance to nearest major road/highway	Meters	Distance to nearest major road or highway (does not include local roads).
Max annual average daily traffic (AADT) - traffic volume	Vehicles/day	Daily maximum traffic volume within 150 m buffer.
Closest AADT - traffic volume	Vehicles/day	Traffic volume of closest major road or highway within 150 m buffer.
Distance-weighted traffic density (DWTD)	Vehicles/day	Road segment with highest AADT within 150 m buffer, with application of Gaussian weight based on distance from residence.
Traffic density	Vehicle-km/hr	Sum of all length-adjusted traffic volumes within 150 m buffer.

Table 2

Distribution of traffic metrics (mean ± SD) by covariates

Characteristic	N (%)	Distance to nearest major road[a] (m)	Max AADT - traffic volume[b] (vehicles/day)	Distance-weighted traffic density (vehicles/day)
Child age at baseline (yrs)				
6-6.99	87 (19.9)	127.5 ± 113.4	21,070 ± 42,150	7,681 ± 23,248
7-7.99	330 (75.5)	141.2 ± 189.7	15,992 ± 33,137	6,158 ± 13,052
≥ 8.0	20 (4.5)	124.3 ± 77.2	24,749 ± 53,930	4,732 ± 6,790
Race/ethnicity				
African American	97 (22.2)	102.0 ± 87.1	22,101 ± 40,855	7,608 ± 16,047
Hispanic	106 (24.3)	140.0 ± 181.3	18,788 ± 39,931	7,153 ± 20,841
Asian	52 (11.9)	147.1 ± 272.9	15,693 ± 34,330	4,583 ± 7,528
Non-Hispanic White	182 (41.6)	152.7 ± 165.0	14,582 ± 31,579	5,828 ± 12,869
BMI age-specific percentile at baseline				
< 50th	147 (33.6)	159.8 ± 224.6	18,116 ± 41,016	6,927 ± 18,282
50-85th	158 (36.2)	129.2 ± 146.0	14,973 ± 29,603	5,042 ± 7,259
85-95th	65 (14.9)	122.5 ± 101.9	14,890 ± 26,828	4,936 ± 5,797
≥ 95th	67 (15.3)	124.2 ± 155.2	24,012 ± 45,825	9,841 ± 25,566
Family income				
<$50,000	93 (21.6)	114.3 ± 137.4	27,781 ± 51,241	10,426 ± 23,261
≥$50,000-100,000	155 (36.0)	145.8 ± 189.6	17,114 ± 35,080	5,415 ± 12,568
≥$100,000	182 (42.3)	145.7 ± 177.1	11,396 ± 19,953*	5,009 ± 11,943*
Missing	7			
Parental or guardian education				
High school	81 (18.6)	148.8 ± 217.1	21,155 ± 45,431	9,429 ± 24,810
Some college	136 (31.2)	118.6 ± 96.6	21,304 ± 42,086	7,825 ± 17,679
≥ BS	219 (50.2)	146.0 ± 191.1	13,652 ± 27,220	4,411 ± 6,867*
Missing	1			
Smoker in home				
Yes	80 (18.8)	144.4 ± 203.8	20,749 ± 39,126	7,812 ± 23,513
No	345 (81.2)	131.2 ± 154.3	17,056 ± 36,092	6,223 ± 13,140
Missing	12			
Cotinine				
1st quartile (<.12)	111 (28.1)	126.1 ± 108.6	12,329 ± 21,395	5,602 ± 13,470
2nd quartile (.12-<.22)	93 (23.5)	130.6 ± 139.8	17,685 ± 32,730	5,388 ± 6,296
3rd quartile (.22-<.50)	93 (23.5)	157.0 ± 225.4	16,114 ± 35,138	4,459 ± 8,474
4th quartile (≥.50)	98 (24.8)	107.0 ± 93.2	24,855 ± 51,139	10,580 ± 26,528*
Missing	42			

[a] Includes major roads and highways, not local residential streets

[b] AADT = Average annual daily traffic. The daily number of vehicles within 150 m of residence.

* ANOVA $p < 0.05$

Table 3

Adjusted[a] associations of traffic metrics and pubertal development (time ratios with 95% confidence intervals and change in median age of onset)

		Breast onset		Pubic hair onset	
Traffic metric	**N (%)**	**TR (95% CI)**	**Median age of onset**	**TR (95% CI)**	**Median age of onset**
Closest AADT - traffic volume (vehicles/ day)					
Local traffic	110 (28.2)	1.00	10.0 years	1.00	10.7 years
<4,400	69 (17.7)	1.01 (0.97, 1.05)	1.2 months	0.97 (0.93, 1.01)	−3.8 months
4,400-9,400	72 (18.5)	0.98 (0.95, 1.02)	−2.4 months	0.96 (0.92, 1.00)	−5.1 months
9,400-17,300	68 (17.4)	0.99 (0.95, 1.03)	−1.2 months	0.95 (0.91, 0.99)	−6.4 months
>17,300	71 (18.2)	1.01 (0.98, 1.05)	1.2 months	0.97 (0.93, 1.01)	−3.8 months
Maximum AADT - traffic volume (vehicles/ day)					
Local traffic	110 (28.2)	1.00	10.0 years	1.00	10.7 years
<5,870	69 (17.7)	1.03 (0.99, 1.06)	3.6 months	0.96 (0.93, 1.01)	−5.1 months
5,870-13,771	68 (17.4)	0.99 (0.95, 1.02)	−1.2 months	0.95 (0.91, 0.98)	−6.4 months
13,771-23,600	72 (18.5)	0.97 (0.93, 1.00)	−3.6 months	0.96 (0.93, 1.00)	−5.1 months
>23,600	71 (18.2)	1.01 (0.97, 1.04)	1.2 months	0.97 (0.93, 1.01)	−3.8 months
Distance-weighted traffic density (vehicles/day)					
Local traffic	110 (28.2)	1.00	10.0 years	1.00	10.7 years
<1,815.8	72 (18.5)	1.02 (0.98, 1.05)	2.4 months	0.96 (0.92, 1.00)	−5.1 months
1,815.8-4,538.7	67 (17.2)	0.99 (0.95, 1.02)	−1.2 months	0.98 (0.94, 1.03)	−2.6 months
4,538.7-9,857.5	72 (18.5)	0.99 (0.96, 1.03)	−1.2 months	0.93 (0.90, 0.97)	−9.0 months
>9,857.5	69 (17.7)	1.00 (0.96, 1.04)	0 months	0.97 (0.93, 1.01)	−3.8 months
Traffic Density (vehicle-km/hr)					
Local traffic	110 (28.2)	1.00	10.0 years	1.00	10.7 years
<67.3	71 (18.2)	1.02 (0.99, 1.06)	2.4 months	0.97 (0.93, 1.01)	−3.8 months
67.3-157.9	68 (17.4)	0.98 (0.94, 1.01)	−2.4 months	0.95 (0.91, 0.99)	−6.4 months
157.9-347.1	72 (18.5)	0.98 (0.94, 1.01)	−2.4 months	0.96 (0.92, 1.00)	−5.1 months
>347.1	69 (17.7)	1.01 (0.98, 1.05)	1.2 months	0.97 (0.93, 1.01)	−3.8 months

[a]Models are adjusted for race/ethnicity, household income, and girls' cotinine level. Total N for adjusted models is 390, 47 girls were excluded for missing data on income and cotinine.

Table 4

Adjusted[a] associations of distance metrics and pubertal development

		Breast onset		Pubic hair onset	
Traffic metric	**N (%)**	**TR (95% CI)**	**Median age of onset**	**TR (95% CI)**	**Median age of onset**
Distance to nearest major road (m)					
Local traffic (>150 m)	110 (28.2)	1.00	10.1 years	1.00	10.7 years
>100 to ≤ 150 m	83 (21.3)	1.02 (0.98, 1.05)	2.4 months	0.96 (0.92, 0.99)	−5.1 months
>50 to ≤ 100 m	92 (23.6)	0.99 (0.96, 1.02)	−1.2 months	0.95 (0.91, 0.99)	−6.4 months
≤50 m	105 (26.9)	0.99 (0.96, 1.02)	−1.2 months	0.97 (0.94, 1.01)	−3.8 months
Distance to road/wind [b]					
Local traffic (>150 m)	110 (28.2)	1.00	10.1 years	1.00	10.7 years
≤150 m upwind	142 (36.4)	1.01 (0.98, 1.04)	1.2 months	0.96 (0.93, 1.00)	−5.1 months
≤150 m downwind	138 (35.4)	0.99 (0.97, 1.02)	−1.2 months	0.96 (0.93, 0.99)	−5.1 months

[a]Adjusted models are adjusted for race/ethnicity, household income, and girls' cotinine level.

[b]Highways in the area run north/south, prevailing winds in the study area are from the west. Locations east of the freeways are classified as downwind and those west of the freeway are classified as upwind.