Environment International 94 (2016) 635–641

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Residential proximity to traffic and female pubertal development

Laura A. McGuinn [a,*], Robert W. Voss [b], Cecile A. Laurent [c], Louise C. Greenspan [d], Lawrence H. Kushi [c], Gayle C. Windham [b]

[a] Department of Epidemiology, University of North Carolina, Chapel Hill, North Carolina, USA
[b] Division of Environmental and Occupational Disease Control, California Dept. of Public Health, Richmond, CA, USA
[c] Division of Research, Kaiser Permanente Northern California, Oakland, CA, USA
[d] Department of Pediatrics, Kaiser Permanente San Francisco, San Francisco, CA, USA

ARTICLE INFO

Article history:
Received 18 February 2016
Received in revised form 26 June 2016
Accepted 26 June 2016
Available online 1 July 2016

Keywords:
Puberty
Breast development
Traffic-related air pollution
Cohort study

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 150 m of a major road. The median age of onset was 10.3 years for B2+ and 10.9 years for PH2+. Living within 150 m downwind of a major road was associated with earlier onset of PH2+ (TR 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.
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.

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).

* Corresponding author at: Department of Epidemiology, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, CB # 7435, Chapel Hill, North Carolina 27599, USA.
E-mail address: lmcguinn@live.unc.edu (L.A. McGuinn).

http://dx.doi.org/10.1016/j.envint.2016.06.031
0160-4120/

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 50th percentile, 50–85th percentile, above the 85th percentile (overweight) and above the 95th 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 Fig. 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