Environmental Research 121 (2013) 31–38

Contents lists available at SciVerse ScienceDirect

Environmental Research

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

Exposure to traffic emissions: Associations with biomarkers of antioxidant status and oxidative damage

Yanli Li [a,*], Jing Nie [a], Jan Beyea [b], Carole B. Rudra [c], Richard W. Browne [d], Matthew R. Bonner [a], Lina Mu [a], Maurizio Trevisan [e], Jo L. Freudenheim [a,*]

[a] Department of Social and Preventive Medicine, University at Buffalo, Buffalo, NY, USA
[b] Consulting in the Public Interest, Lambertville, NJ, USA
[c] Independent Health, Buffalo, NY, USA
[d] Department of Biotechnical and Clinical Laboratory Sciences, University at Buffalo, Buffalo, NY, USA
[e] Sophie Davis School of Biomedical Education, City College of New York, NY, USA

ARTICLE INFO

Article history:
Received 16 March 2012
Received in revised form
17 July 2012
Accepted 4 October 2012
Available online 8 November 2012

Keywords:
Traffic emissions
Polycyclic aromatic hydrocarbon
Oxidative stress

ABSTRACT

Background: Oxidative stress has been implicated as a possible mechanism for adverse health effects associated with traffic emissions. We examined the association of an estimate of traffic emissions with blood biomarkers of antioxidant capacity (glutathione, glutathione peroxidase, trolox-equivalent antioxidant capacity) and oxidative damage (thiobarbituric acid-reactive substances (TBARS)) among 1810 healthy women, randomly selected from Erie and Niagara Counties in Western New York.
Methods: A geographic traffic emission and meteorological dispersion model was used to estimate annual polycyclic aromatic hydrocarbon (PAH) exposure from traffic emissions for each woman based on her residence at the time of study. Associations of traffic-related PAH exposure with measures of oxidative stress and antioxidant capacity were examined in multiple regression analyses with adjustment for potential confounders.
Results: Higher traffic-related PAH exposure was associated with decreased glutathione and increased glutathione peroxidase. Stronger associations between traffic-related PAH exposure and levels of glutathione and glutathione peroxidase were suggested among nonsmoking women without second-hand smoke exposure, especially among premenopausal nonsmoking women. Associations were also stronger for measurements made in warmer months.
Conclusions: These findings suggest that PAHs or other components of traffic emissions may impact anti-oxidative capacity among healthy women, particularly premenopausal non-smokers without secondhand smoke exposure.

1. Introduction

Research in recent decades consistently indicates that outdoor air pollution adversely affects health and that air pollution stemming from transportation is an important contributor (World Health Organization, 2005). There is evidence that exposure to traffic emissions increases risk of asthma and other respiratory diseases as well as of cardiovascular diseases, lung cancer, breast cancer and childhood leukemia (Amigou et al., 2011; Crouse et al., 2010; Ito et al., 2010; Liao et al., 2011; Nie et al., 2007; Patel et al., 2010).

The underlying mechanisms behind traffic emission-related health effects are not well understood; it has been suggested that oxidative stress might be one possible mechanism (Laumbach and Kipen, 2010). Oxidative stress, defined as an imbalance between free radical production and protective radical scavenging antioxidants, can lead to damage of biologic macromolecules and dysregulation of normal metabolism and physiology (Terada, 2006). There is evidence that oxidative stress is important in the etiology of cardiovascular diseases, lung diseases and cancer (Olinski et al., 2002; Spector, 2000).

Traffic emissions contain a multitude of air contaminants with pro-oxidant properties. These include heavy metals, volatile organic compounds and polycyclic aromatic hydrocarbons (PAHs). PAHs, a class of organic compounds containing only carbon and hydrogen, and comprised of two or more fused aromatic rings (IARC, 1998, 2010) have been reported to increase production of free radicals and to possess carcinogenic, mutagenic and endocrine disrupting properties (Brunekreef et al., 2009; Kim and Lee, 1997; Kiruthiga et al., 2010). Motor vehicle exhaust is a major source of PAH exposure in urban areas (Zhang et al., 2006).

Several human studies have been conducted to examine PAH exposure and oxidative stress biomarkers in occupations with high PAH exposure and among children (Bae et al., 2010; Jeng et al., 2010;

* Corresponding authors. Fax: +1 716 829 2979.
E-mail addresses: yanlili@buffalo.edu (Y. Li), jfreuden@buffalo.edu (J.L. Freudenheim).

0013-9351/$ - see front matter © 2012 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.envres.2012.10.003

Rossner et al., 2008b; Singh et al., 2008; Wei et al., 2010; Wu et al., 2003). However, few studies have been conducted to specifically examine the health effects of traffic emissions on oxidative stress status among the general population.

In the present study, we assessed cross-sectional associations of an estimate of PAH exposure from traffic emissions with blood biomarkers of antioxidant capacity (glutathione, glutathione peroxidase, trolox-equivalent antioxidant capacity) and oxidative damage to lipids (thiobarbituric acid-reactive substances (TBARS)) among 1810 healthy women, randomly selected from Erie and Niagara Counties in Western New York. We hypothesized that higher PAH exposure from traffic emissions would adversely affect antioxidant capacity and increase oxidative stress. Since tobacco smoke is another major source of PAHs, we further hypothesized that the associations of traffic-related PAH exposure with oxidation biomarkers might be more easily detected among never-smokers without secondhand smoke exposure. In addition, we hypothesized that pre- and post-menopausal women might differ in their responses to PAH exposure because of the estrogen-mimicking properties of some PAHs (Clemons et al., 1998).

2. Methods

2.1. Study population

We randomly selected 2115 healthy women from the general population of Erie and Niagara counties in Western New York between 1996 and 2001. These women were originally selected as controls for a series of case-control studies including the Western New York Exposures and Breast Cancer study, described in detail elsewhere (Bonner et al., 2005; Nie et al., 2007). Briefly, eligible control participants were English-speaking, aged 35–79 years, who were current residents of Erie or Niagara counties, with no previous history of malignancy other than non-melanoma skin cancer. Women under age 65 years were selected from driver's license rolls; those aged 65 years or older were selected from the rolls of the Health Care Financing Administration. In the original case-control study, the response rate for controls was 63% among those determined to be eligible. All participants provided written informed consent, and the study protocol was approved by the Institutional Review Board of the University at Buffalo.

For these analyses, we excluded individuals who had either missing measurement of blood oxidation biomarkers ($n=287$) or were missing the estimate of PAHs from traffic emissions ($n=30$), resulting in data from 1810 women for the current analyses. The main reasons for missing blood biomarkers were lack of a blood sample or that the quantity of the sample collected was insufficient for assay. Missing traffic emissions estimates were the result either of lack of information about the participant's residence at the time of study enrollment or inability to geocode the address. In comparisons of those excluded from these analyses to those included, body mass index and smoking status were not different. However, excluded women tended to be nonwhite (16.38% versus 8.73%), older in age (mean age 62.72 versus 57.08 years) and less educated (12.75 versus 13.39 years of education) compared to those included.

2.2. Data collection

Information on demographics, reproductive history, smoking and secondhand smoke exposure, diet, alcohol consumption, non-steroidal anti-inflammatory drugs use, and residential history was collected through in-person interviews. Women were classified as current smokers if they had smoked more than 100 cigarettes in their lifetime and reported smoking at the time of interview. They were classified as former smokers if they had smoked more than 100 cigarettes in their lifetime but were not smokers at the time of interview. The most recent information on alcohol drinking, non-steroidal anti-inflammatory drugs use and vitamin supplement use collected in this study was for the period 12–24 months prior to the interview. Recent alcohol nondrinkers were defined as those who had had less than one drink per month during the 12 to 24 months prior to interview. Those who had never had at least 12 drinks of alcohol in any 12-month period were classified as lifetime nondrinkers. Recent non-steroidal anti-inflammatory drugs use, including aspirin and ibuprofen use, 12–24 months prior to the interview was categorized as non-users (0 days/month), infrequent users (≤ 14 days/month), and regular users (>14 days/month). Current height and weight at the time of interview were measured by trained interviewers according to a standardized protocol. Body mass index was calculated by dividing weight (in kilograms) by height (in meters) squared. Daily vitamin C and vitamin E intakes 12 to 24 months prior to interview were calculated by combining dietary and supplemental use. Dietary intakes were assessed through a modified version of the Health Habits and History food frequency questionnaire. Dietary vitamin C and vitamin E intakes were calculated from the food frequency questionnaire using the DietSys (version 3.7) nutrient analysis software utilizing food composition data from the United States Department of Agriculture (Block et al., 1986). Vitamin C and vitamin E supplement intakes were queried in the questionnaire. Those who reported no secondhand smoke exposure at home or at work in the past 10 years were defined as being without secondhand smoke exposure. For assessment of occupational exposure, women were dichotomized based on their likelihood of being exposed to PAHs in the working environment. Included in the high exposure category were women who had ever had worked as coke oven workers, auto mechanics, professional drivers, or cooks. Season at time of interview was classified as follows: spring (March to May), summer (June to August), autumn (September to November), and winter (December to next February). In addition to this classification of season, we also divided the year into two periods: a warm season (April to October) and cold season (November to March), based on usual temperatures in the study area and the likelihood of opening windows in the residence.

2.3. Traffic-related PAH exposure assessment

Residential addresses of the participants at the time of interview were geocoded using ArcView 3.2 (ESRI, Inc., Redlands, CA), with GDT/Dynamap 2000 (GDT, Inc., Lebanon, NH) as the reference theme. ZP4 (Semaphore Co., Aptos, CA) software was used to correct and update the zip code for each address before the geocoding process. A validation study showed very good positional accuracy of the geocoded addresses (Bonner et al., 2003).

A geographic traffic emissions and meteorological dispersion model was applied to estimate PAH exposure from traffic emissions in the calendar year of blood collection based on each participant's residence. In this model, benzo[a]-pyrene, the most commonly used marker of PAHs exposure, was used as a surrogate for total PAH exposure from traffic emissions. The geographic traffic exposure model, originally developed for the Long Island Breast Cancer Study Project, was modified for the Western New York region with region-specific meteorological and traffic data (Nie et al., 2007). Meteorological data were obtained from the National Climatic Data Center and traffic count data were obtained from the Greater Buffalo-Niagara Regional Transportation Council (Appendix Table A1). Briefly, annual average traffic density, distance from road, wind speed, direction and other meteorological conditions, as well as tailpipe emission including cruise emissions and excess emissions at intersections and during engine warm-up were modeled to estimate traffic exposures for each residence of the study participants. Cold start emissions were not explicitly included in the final model, because we had previously found them to contribute only negligibly to a regression model for soil PAH data, the preferred environmental dataset (Beyea et al., 2006). We did include intersection data in the model. Cold start emissions, highest at intersections, would contribute to this term. The model produced relative rather than absolute estimates of PAH exposure, because the former are less sensitive to uncertainties in model parameters (Nie et al., 2007).

This estimate of exposure to traffic emissions was validated and calibrated in a subset of the Long Island Breast Cancer Study participants (Beyea et al., 2006, 2005). The model was shown to predict successfully measured PAH, including soil benzo[a]pyrene concentrations, PAH-DNA adducts in study participants' blood and carbon monoxide level at a U.S. Environmental Protection Agency monitoring station (Beyea et al., 2006, 2005). To examine if the model were valid in our study region and also to further calibrate the model parameters, we performed an additional validation study using measured historical data on benzo[a]pyrene in air and carbon monoxide concentrations from the Erie and Niagara areas. The correlation coefficient between historical measured and predicted benzo[a]pyrene was 0.54 and 0.43 for the Pearson and Spearman correlations, respectively (Appendix Fig. A1). The model, adapted to predict carbon monoxide concentrations at four U.S. Environmental Protection Agency monitoring stations, was also able to reproduce the patterns in hourly carbon monoxide concentrations reasonably well (Appendix Fig. A2 to Fig. A5). Annual average values predicted for the four stations followed the trend in measurement between stations (Appendix Fig. A6).

2.4. Laboratory analysis of oxidation biomarkers

Fasting blood samples were collected in the morning of the interview and were processed within 30 min for glutathione measurement and within 2 h for the other oxidative stress biomarkers. Samples were stored at $-80\ ^{\circ}C$ until assayed (Trevisan et al., 2001).

Total erythrocyte glutathione (mg/dl of packed red blood cells) was measured in EDTA whole blood using the method of Browne and Armstrong (1998). Plasma glutathione peroxidase activity (IU/l) was measured using a Cobas Mira automated chemistry analyzer (Pippenger et al., 1998). Trolox-equivalent antioxidant capacity was measured using EDTA plasma and is expressed as a percent inhibition of the radical-generating reaction relative to the vitamin E analogue