Environment International 73 (2014) 216–223

Contents lists available at ScienceDirect

Environment International

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

Racial differences in levels of serum lipids and effects of exposure to persistent organic pollutants on lipid levels in residents of Anniston, Alabama

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ARTICLE INFO

Article history:
Received 27 March 2014
Accepted 24 July 2014
Available online xxxx

Keywords:
PCBs
Pesticides
Cardiovascular disease
Cholesterol
Triglycerides
HDL

ABSTRACT

Serum lipid levels are major risk factors for cardiovascular disease. In addition to diet, exercise, genetics, age and race, serum concentrations of persistent organic pollutants (POPs) influence concentrations of serum lipids. We investigated associations between fasting concentrations of 35 polychlorinated biphenyl (PCB) congeners and nine organochlorine pesticides in relation to total serum lipids, total cholesterol, low-density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol and triglycerides in 525 Caucasian and African American residents of Anniston, Alabama, who were not on any lipid-lowering medication. In Model 1, data were adjusted for age, age quadratic, gender, BMI, alcohol consumption, smoking and exercise, while in Model 2, additional adjustment was done for other POPs. As compared to Caucasians, African Americans had lower levels of total lipids and triglycerides with higher concentrations of HDL cholesterol, but higher concentrations of PCBs and pesticides. Total pesticides were more strongly associated with elevations in serum lipids than were total PCBs, and the associations were stronger in African Americans. Total DDTs were not associated with serum lipids after adjustment for other POPs in either racial group, while the strongest positive associations were seen for hexachlorobenzene (HCB) in both racial groups. Racial differences in lipid profiles, concentrations of POPs and associations between POP concentrations and serum lipids are relevant to racial differences in rates of cardiovascular disease.

1. Introduction

Concentrations of serum lipids are known to be one of the major risk factors for cardiovascular disease, along with hypertension, smoking and diabetes (Berry et al., 2012; Prospective Studies Collaboration et al., 2007). Elevated concentrations of total cholesterol, triglycerides and especially low-density-lipoprotein (LDL) cholesterol are major risk factors for cardiovascular disease (Chapman et al., 2011), as is low high-density-lipoprotein (HDL) cholesterol (Emerging Risk Factors Collaboration et al., 2009). However, there are known racial differences in "normal" levels of the lipid components, with African Americans having lower total/HDL cholesterol ratios and triglyceride concentrations, as well as higher HDL cholesterol levels, while Hispanics have lower LDL cholesterol, total cholesterol and HDL cholesterol as compared to Caucasians (Rodriguez et al., 2002). Willey et al. (2011) reported that levels of HDL were inversely predictive of risk of a myocardial infarction in African-Americans and Caucasians but not in Hispanics. Chang et al. (2011) have reported significant racial differences in single-nucleotide polymorphisms among the major racial groups in the Third National Health and Nutrition Examination Survey (NHANES), and Dumitrescu et al. (2011) have found racial differences in common genetic variants associated with HDL, LDL and triglycerides, which is consistent with the hypothesis that the differences reported above are at least in part due to genetic variability. However, in spite of having on average higher concentrations of HDL cholesterol, African Americans have a higher mortality from coronary heart disease as compared to either Caucasians or Hispanics (Lloyd-Jones et al., 2010; Roger et al., 2012).

Lifestyle factors, including diet (Hu and Willett, 2002), tobacco (Benowitz, 2003), binge alcohol use (Graff-Iversen et al., 2012) and frequency of exercise (Wisløff et al., 2005) are important in relation to serum levels of the various lipid components. There are significant racial

Abbreviations: AA, African Americans; ATSDR, Agency for Toxic Substances and Disease Registry; BMI, body mass index; CA, Caucasian Americans; DDE, dichlorodiphenydichloroethylene; DDT, dichlorodiphenyltrichloroethane; HCB, hexachlorobenzene; HCCH, hexachlorocyclohexane; HDL, high density lipoprotein cholesterol; LDL, low density lipoprotein cholesterol; PCBs, Polychlorinated biphenyls; POPs, persistent organic pollutants; ppb, parts per billion (ng/g); ww, wet weight.

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http://dx.doi.org/10.1016/j.envint.2014.07.022
0160-4120/

and geographical differences in dietary intakes in black and white women (Newby et al., 2012). Smoking is more common, but heavy alcohol consumption is less common in African Americans than Caucasians in at least some studies (White et al., 2012).

The role of exposure to environmental contaminants and cardiovascular disease is often not acknowledged in large population studies, but is important. Exposure to lead (Glenn et al., 2003) and polychlorinated biphenyls (PCBs) (Goncharov et al., 2010) increases risk of hypertension, while exposure to arsenic (Chang et al., 2004) and methylmercury (Guallar et al., 2002) increases risk of coronary artery disease. Several authors have reported elevations in triglycerides (Baker et al., 1980; Chase et al., 1982) and total cholesterol (Stehr-Green et al., 1986; Takamatsu et al., 1984; Tokunaga and Kataoka, 2003) in PCB-exposed populations. Persons with occupational PCB exposures were found to have elevations in rates of cardiovascular deaths (Gustavsson and Hogstedt, 1997). In a Native American population, higher PCB levels were associated with elevations of both triglycerides and total cholesterol, as well as with self-reported hypertension and cardiovascular disease (Goncharov et al., 2008).

PCBs were manufactured in Anniston, Alabama, from 1929 until 1971. In 2003 ATSDR funded a consortium of universities to determine exposure of nearby residents and the resulting health impacts. The serum PCB levels in 758 Anniston residents ages 19–93 years ranged from 0.1 to 170.4 ng/g (ppb) with a median of 3.2 ng/g (Goncharov et al., 2010). These concentrations can be compared to the statement by ATSDR (2000) that the average PCB concentration in persons that are not unusually exposed is between 0.9 and 1.5 ng/g.

We have previously reported evidence that serum concentrations of various PCB congeners and chlorinated pesticides are associated with the alteration of the profile of serum lipid levels in Anniston residents in a study where we controlled for race (Aminov et al., 2013). The goal of the present study is to explore racial differences both among serum lipid components in a population that has nearly identical numbers of African Americans and Caucasians, and to determine how total PCBs, PCB congener groups, total pesticides and concentrations of individual pesticides or pesticide groups may alter these serum lipid associations.

2. Materials and method

Previous reports have described the study population, sampling methods, data collection techniques and laboratory analyses (Aminov et al., 2013; Goncharov et al., 2010, 2011), and so these methods will be presented only briefly here.

2.1. Study site and population

A pool of 3320 eligible addresses was randomly selected from a commercial list of all residential properties in Anniston with intentional oversampling (two thirds of all eligible) of residences in West Anniston, which is the area nearest the former PCB manufacturing facility, resulting in a stratified sample that facilitated enrollment of individuals with residences closer to the plant. An adult member of each household was invited to enroll in the study. Interviews were conducted by trained interviewers with 1110 persons (ages 18 years and older) who were enrolled in the study. The objective of the interview was to obtain information about each study participants demographics, lifestyle, and health-related variables.

A total of 774 persons provided fasting blood samples for analysis of serum PCBs and pesticides, serum lipids and fasting glucose. PCB results were missing for eight subjects failing quality control/assurance procedures, there was no medication information for one subject, no race/ethnicity information for one subject, and height and weight parameters were not available for two participants, and so these subjects were excluded. To exclude a possible influence of confounding by lipid-lowering medication, for our study we used 575 study participants who reported that they are not taking any lipid-lowering medication.

2.2. Laboratory analyses

The Centers for Disease Control and Prevention's National Center for Environmental Health laboratory performed analyses of levels of 35 PCB congeners (PCBs 28, 44, 49, 52, 66, 74, 87, 99, 101, 105, 110, 118, 128, 146, 149, 151, 153, 156, 157, 138–158, 167, 170, 172, 177, 178, 180, 183, 187, 189, 194, 195, 199, 196–203, 206, 209) and 9 organochlorine pesticides [hexachlorobenzene (HCB), β-hexachlorocyclohexane (β-HCCH), γ-HCCH, oxychlordane, trans-nonachchlor, dichlorodiphenyltrichloroethanes (p,p′-DDE, p,p′-DDT, o,p′-DDT, the sum of which will be called “total DDTs”) and mirex] in serum, measured by high resolution gas chromatography/isotope dilution mass spectrometry (Sjödin et al., 2004).

The Clinical Chemistry Laboratory of the Jacksonville, Alabama Medical Center measured serum levels of different lipid fractions (triglycerides, total cholesterol, LDL cholesterol and HDL cholesterol). The formula proposed by Bernert et al. (2007) was applied to calculate total lipid levels.

2.3. Statistical analysis

All statistical analyses were conducted using SAS System 9.1.3 package (SAS Institute, Inc., Cary, NC). Descriptive statistics were calculated for outcome variables, demographic characteristics, and exposure variables. The Cochran–Mantel–Haenszel test was used to compare demographic and behavioral characteristics of the two racial groups. Student's *t*-test was used in order to assess racial differences in demographic and serum characteristics. Normality of covariates was assessed graphically and by the Kolmogorov–Smirnov test. Since serum POP concentrations were highly skewed, they were natural log transformed to achieve approximately normal distribution before including them into regression models. Serum lipid levels were less skewed but were also natural log transformed. Multiple linear regression modeling was used to evaluate relationships between five outcome variables (serum concentrations of total lipids, total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides) and exposure variables (serum levels of total PCBs and total pesticides as well as various PCB and pesticide groups) in each racial group. All regression models were adjusted for confounders defined by selected demographic and behavioral characteristics of the study population.

As previously described (Aminov et al., 2013), we used two models. In Model 1 adjustment was made only for age, age quadratic, gender, BMI, smoking, alcohol consumption and exercise. In Model 2 adjustment was made, in addition to all those variables in Model 1, for concentrations of all other POPs than the one under study. While theoretically preferable because it can assist in distinguishing actions of different contaminants that are all found in serum lipids, use of Model 2 can lead to results that are statistically significant but biologically implausible, especially if the apparent association changes sign (see Additional file 1 in Aminov et al., 2013). Thus results using Model 2 must be interpreted with caution.

Predictive models were validated by evaluating R-squared statistics. All possible interaction terms initially were in the models, including “race × PCBs”, “race x pesticides”, “age × PCBs” and “age × pesticides”, but in final models they were removed since none of the interaction terms had significant estimates or significantly contributed to the models. To address the issue of multicollinearity between exposure variables, we have assessed the correlation between groups of POPs. The correlation between groups of PCBs organized by number of *ortho*-substituted chlorines and the various pesticides was assessed by calculation of Spearman's correlation coefficients by race. The exposure variables (concentrations of total PCBs, total pesticides, groups of PCBs, and pesticides) and hypotheses tested have been defined a priori,