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Neighborhood food environment, dietary fatty acid biomarkers, and cardiac arrest risk

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ABSTRACT

We explored links between food environments, dietary intake biomarkers, and sudden cardiac arrest in a population-based longitudinal study using cases and controls accruing between 1990 and 2010 in King County, WA. Surprisingly, presence of more unhealthy food sources near home was associated with a lower 18:1 *trans*-fatty acid concentration (−0.05% per standard deviation higher count of unhealthy food sources, 95% Confidence Interval [CI]: 0.01, 0.09). However, presence of more unhealthy food sources was associated with higher odds of cardiac arrest (Odds Ratio [OR]: 2.29, 95% CI: 1.19, 4.41 per standard deviation in unhealthy food outlets). While unhealthy food outlets were associated with higher cardiac arrest risk, circulating 18:1 *trans* fats did not explain the association.

1. Introduction

Sudden cardiac arrest causes between 180,000 and 450,000 adult deaths in the United States each year, (Deo and Albert, 2012; Kong et al., 2011; Zheng et al., 2001) but both incidence and fatality vary by geographic locale. In particular, cardiac arrest incidence rates are higher in neighborhoods in lowest income quartiles (Reinier et al., 2011) while survival following cardiac arrest appears higher for residents of higher income neighborhoods (Sasson et al., 2011).

These disparities might result from disparities in neighborhood food environments. Several prior studies have shown density of unhealthy food outlets to be associated with incidence of harmful cardiovascular events such as stroke (Hamano et al., 2013) and cardiovascular mortality (Alter and Eny, 2005; Daniel et al., 2010). Some studies have suggested that the food environment shapes diet, though the evidence for this relationship remains inconclusive (Black et al., 2014; Caspi et al., 2012; Cummins et al., 2014; Mejia et al., 2015). Circulating *trans* fatty acids have been associated with higher risk of sudden cardiac death (Lemaitre et al., 2006a, 2006b, 2002), potentially as a result of fatty acid release and eicosanoid formation or vascular inflammation. (Lemaitre et al., 2016; Siddiqui et al., 2009) Thus, improving the food environment (Dannefer et al., 2012; Leggat et al., 2012) may decrease *trans* fat consumption and thereby either lower the risk of cardiac arrest, or decrease the severity of incident arrests (e.g. if a healthier diet lessens vascular inflammation (Esposito et al., 2004)), leading to better survival after arrest.

However, neighborhood research can be methodologically challenging.(Oakes, 2004) Neighborhood characteristics such as food environments are frequently correlated with other characteristics that are often also health determinants. By using dietary biomarkers to explore the relationship between food environment and diet in both cardiac arrest cases and community controls, we can test whether the food environment specifically, as opposed to some other neighborhood mechanism, is likely responsible for observed associations.

We examined associations between residential neighborhood food environment and 1) dietary biomarkers of fatty acid intake, 2) risk of cardiac arrest, and 3) survival after out of hospital cardiac arrest attended by paramedics and due to ventricular fibrillation.

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2. Methods

2.1. Setting

We used subject data from the Cardiac Arrest Blood Study – Repository (CABS-R). CABS-R, which has been described in more detail elsewhere (Johnson et al., 2012; Siscovick et al., 1995; Sotoodehnia et al., 2006), is a population-based registry that includes blood samples and interview data for out-of-hospital cardiac arrest incidents attended by paramedics within King County, Washington, as well as a smaller group of population-based controls. King County has about 2 million residents and is geographically large (approximately 6000 km^2) and varied, including both the city of Seattle and the Cascade Mountains. Rates of survival after cardiac arrest in King County are among the highest in the world (Lance and Becker, 2015).

The Human Subjects Review Committee at the University of Washington approved all data collection protocols, and the Human Subjects Review Committees at both the University of Washington and Columbia University Medical Center approved the geographic linkage and analyses for this study. The use of repository data from the CABS-R for the study analyses was authorized under a waiver of consent.

2.2. Subjects

Case ascertainment in CABS-R was based on paramedic reports, death certificates, medical examiner reports, and autopsy reports (Siscovick et al., 1995). Cardiac arrest cases were defined as a sudden, pulseless condition that arose in an otherwise apparently stable individual in the absence of a non-cardiac cause of arrest, such as traumatic injury or drug overdose. CABS-R case identification began in 1988 and is ongoing; for this analysis, we used subjects whose cardiac arrest occurred between 1990 and 2010. CABS-R staff classified subjects as having had definite, probable, or possible cardiac arrest after reviewing records of initial rhythm (ventricular fibrillation, pulseless electrical activity, or asystole), circumstances (e.g. witnessed or unwitnessed), and possible contribution of comorbidities to the event. To avoid misclassification, we excluded CABS-R cases with only possible cardiac arrest.

Population-based controls available for this analysis were recruited by random-digit dialing within King County as part of the Diet and Primary Cardiac Arrest Study (Siscovick et al., 1995). Controls were matched to a subset of CABS-R cases without diagnosed heart disease before their cardiac arrest, based on sex, age (+/- 7 years), and calendar year. Subjects were not matched on neighborhood geography or neighborhood features. The control response rate was 64%.

Paramedics collected blood samples from cases, usually within 45 min of cardiac arrest. CABS-R staff collected blood samples from controls during a home interview. Case and control samples were assessed using gas chromatography to measure red blood cell membrane fatty acids. We used two measures of *trans* fats, each compiled as a percentage of total circulating fatty acids. First, we summed concentrations of five *trans* isomers of oleic acid (12 *trans*-18:1, 11 *trans*-18:1, 10 *trans*-18:1, 9 *trans*-18:1, and mix of 6–8 *trans*-18:1). Oleic acids have previously been associated with the consumption of foods commonly sold by unhealthy food outlets, due to outlets' use of partially hydrogenated vegetable oil (Micha et al., 2010; Tyburczy et al., 2012). We also used chromatography measures of linoleic acids (*trans*-18:2 fatty acids), which have previously been more strongly associated with sudden cardiac arrest (Lemaitre et al., 2006b, 2002). Linoleic acids are present in small amounts in most cooking oils and are less likely to be affected by neighborhood food environments. Because uncertainty remains about each isomer's impact on cardiac arrest risk and association with food environment, we analyzed both. Lab procedures and validation against a dietary intake questionnaire for these data have been described previously (Lemaitre et al., 2002, 2014) Biomarker data were available for 416 controls and 1570 cases.

2.3. Retail and socio-economic environment

We used the National Establishment Time-Series (NETS) database, which contains yearly registration data for businesses, including company name, street address, and Standard Industry Classification (SIC) code (Kaufman et al., 2015; Walls, 2009) to build a spatio-temporal food environment measure for each US Census tract (geographical units typically containing about 4000 residents (Bureau, 2012)) in King County for each year from 1990 to 2010. NETS was developed from Dun & Bradstreet's business registry, which has previously been validated for neighborhood food environment research (Liese et al., 2010).

After cleaning and processing the data as described elsewhere (Kaufman et al., 2015), our procedure for estimating food outlet density within each tract for each year comprised: 1) geocoding each businesses' reported address, 2) using SIC codes and business names to classify each retail establishment into potentially health relevant categories informed by previous literature, of which healthy food sources (e.g. supermarkets, fish markets) and unhealthy food sources (e.g. fast food restaurants, bakeries) are of central interest for this analysis, 3) using ArcGIS to identify the year 2000 census tract in which each business was located, and 4) tabulating by census tract identifier to estimate the number of each type of business within each census tract. This food environment measure, though imperfect, is consistent with prior research.(McInerney et al., 2016; Mercille et al., 2016) Complete details on the classification of food outlets into healthy and unhealthy are given in the online appendix. Notably, we classified supermarkets as healthy, as is conventional when measuring food environment from administrative data (Chaparro et al., 2017; Cobb et al., 2015; McInerney et al., 2016), despite many supermarkets selling unhealthy foods.

Next, we used ArcGIS to identify the year 2000 census tract for each subject's reported residential address. We considered the count of each type of food source present within the year 2000 tract's boundaries in the index year to represent the food environment context. Our analyses operationalized the food environment in three ways: 1) As a count of healthy food outlets in the tract, 2) as a count of unhealthy food outlets in the tract, and 3) as the proportion of total food outlets classified as healthy.

2.4. Primary analyses

We theorized that a relationship between the food environment and cardiac arrest might be mediated by the previously established relationship between dietary and circulating fatty acids and cardiac outcomes (Leaf et al., 2003; Lemaitre et al., 2006b,2002,2014; Siscovick et al., 1995). For analysis 1, we fit linear regression models predicting each participant's biomarker concentration from the food environment in the home census tract in the year of SCA event or control recruitment, adjusting for year, age and sex. We present this analysis stratified by case/control status because of the risk of "collider bias" in cases (Hernán et al., 2004). (The risk of bias is this: If higher levels of fatty acids result in cardiac arrest, and if neighborhood factors also associated with more food outlets (e.g. air pollution) also cause cardiac arrest, then an analysis oversampling cases as compared to the general population will display an artificially inflated association between food outlets and fatty acids. This bias will not be present in the controls because their inclusion in the study was not conditional on a cardiac arrest incident.)

Analysis 2 focused on risk of cardiac arrest. From the registry, we identified population-based controls and the cases that matched the controls' selection criteria, which comprised being 79 or younger, lacking a history of diagnosed heart disease and having an index event prior to 2007. Because case ascertainment was less complete outside Seattle city limits during the early years of CABS-R, we further limited this analysis to the 475 cases and 215 controls who met the above criteria and also lived within Seattle city limits. Among the selected