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DESCRIPTION DETAILS RESULTS HISTORY SUBPROJECTS SIMILAR PROJECTS NEARBY PROJECTS BETA LINKS NEWS AND MORE

Project Number: 5F31ES025096-02

Title: AIR POLLUTION AND HIGH DENSITY LIPOPROTEIN STRUCTURE AND FUNCTION

Contact PI / Project Leader: [BELL, GRIFFITH](#)

Awardee Organization: UNIVERSITY OF WASHINGTON

Abstract Text:

DESCRIPTION (provided by applicant): Cardiovascular disease is most common cause of morbidity and mortality worldwide. Evidence suggests that air pollution increases the risk of CVD, and better understanding of the biological causes of this relationship could have substantial benefits. Unlike smoking and some dietary risk factors, individuals (particularly children) are largely unable to choose whether or not to be exposed to air pollution in their daily life, making air pollution a crucial subject of public health research for policy makers. Improved characterization of the biological pathway between air pollution and CVD could improve health by allowing for more directed air quality management policies, and better identification of at risk groups, reducing the need for interventions with harmful side effects. Several laboratory studies report that particulate matter (PM) reacts with oxidized phospholipids in human endothelial cells, resulting in expression of pro-inflammatory, pro-oxidant, unfolded protein response pathways related to vascular inflammation, and the creation of dysfunctional high-density lipoproteins (HDL). This PM-induced dysfunctional HDL loses its cardio-protective qualities and may even become atherogenic. Our proposal will further characterize this potential CVD pathway, and explore whether exposure to a suite of traffic-related air pollutants (particulate matter <2.5µm, nitrogen dioxide, oxides of nitrogen, and black carbon) is associated with changes in HDL in humans. To broadly examine this topic, we will examine exposure to traffic-related air pollutants (TRAP) in two different settings, and examine a suite of advanced HDL measurements. First, we will examine the cross-sectional relationship between air pollution exposure (estimated using a validated spatiotemporal land-use regression model based on EPA and cohort-specific monitoring) and HDL particle number (HDL-P, a measure of HDL particle number more strongly associated with CVD events than HDL cholesterol) in the Multi-Ethnic Study of Atherosclerosis Air Pollution study (MESA Air), a multiethnic cohort of men and women. Second, we will examine whether the relationship between air pollution and progression of subclinical atherosclerosis (as measured by coronary artery calcium) is mediated by HDL-P, employing traditional and causal mediation analysis frameworks to assess direct and indirect effects. Third, we will use mixed models to examine whether controlled exposure to air pollution to diesel exhaust from a human exposure trial from the DISCOVER center effects functional and proteomic qualities of HDL. In this study, we examine the effect of a short-term exposure to diesel exhaust on HDL anti-oxidant index (HOI), as well as the HDL proteome, both measures of HDL structure and function. Understanding the relationship between TRAP and HDL structure and function will help to elucidate the underlying biological mechanisms by which TRAP affects CVD.

Public Health Relevance Statement:

PUBLIC HEALTH RELEVANCE: Identification of biological pathways through which air pollution affects cardiovascular disease would be expected to have significant public health benefits. High density lipoproteins, which have recently come under renewed interest due to their complex relationship with CVD and linkage to a remarkable number of cardio-protective and endocrine-like systems, have strong potential to explain the relationship between air pollution and cardiovascular disease. Further exploration of this pathway may lead to important insights into at risk populations, possible pharmaceutical or policy interventions, and reduction in the use of drugs with deleterious side effects, greatly improving the well-being of the population at large.

Project Terms:

Adverse effects; Affect; Air; Air Pollutants; Air Pollution; ambient air pollution; Antioxidants; Atherosclerosis; base; Biological; Blood; Calcium; Carbon Black; Cardiovascular Diseases; cardiovascular disorder risk; cardiovascular risk factor; Cessation of life; Characteristics; Child; Cholesterol; cholesterol transporters; cohort; Complex; Coronary artery; Data; Diesel Exhaust; Diet; Disease Pathway; Double-Blind Method; Drug usage; Early identification; Endocrine; Endothelial Cells; ethnic diversity; Event; experience; exposed human population; Exposure to; Genetic study; Health; Health Benefit; High Density Lipoprotein Cholesterol; High Density Lipoproteins; high risk; Hour; Human; improved; indexing; Individual; Inflammatory; insight; interest; Intervention; Laboratory Study; land use; Lead; Life; Link; Long-Term Effects; Measurement; Measures; Mediating; Mediation; men; Methods; Modeling; Monitor; Morbidity - disease rate; mortality; Nitrogen Dioxide; Nitrogen Oxides; NMR Spectroscopy; novel; Observational Study; Outcome; Participant; particle; Particulate Matter; Pathway interactions; Personal Satisfaction; Pharmacologic Substance; Phospholipids; Policies; Policy Maker; Population; Populations at Risk; Proteins; Proteome; Proteomics; Public Health; public health research; Randomized; Reactive Oxygen Species; Reporting; Research; Research Design; research study; response; Risk; Risk Factors; Risk Marker; Shotguns; Smoking; Source; spatiotemporal; Structure; System; Testing; Time; trafficking; Translating; vascular inflammation; Woman

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