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Association of Environmental Air Quality and Congenital Heart Defect Diagnosis

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One out of every 33 infants in the United States (U.S.) is born with a major birth defect. A birth defect occurs when the structure of one or more parts of the body forms abnormally during fetal development (Hoffman, Kaplan, & Liberthson, 2004). While nearly 120,000 babies are born each year with a birth defect in the U.S., nearly 40,000 infants having some variation of a congenital heart defect (CHD) (Center for Disease Control and Prevention [CDC], 2014; CDC, 2015). Among infants born with a CHD, one-quarter are diagnosed with a critical CHD requiring invasive cardiac intervention or surgical treatment within the first year of life for survival (Oster, Lee, Honein, Riehle-Colarusso, Shin, & Correa, 2013).

Despite the number of infants affected, over 80% of all CHD diagnoses have an unknown etiology (Botto & Correa, 2003; Snijder et al., 2012; Yu et al., 2014). Of the known causes, environmental risk factors, such as air pollution, are thought to play an important part (Hansen, Barnett, Jalaludin, & Morgan, 2009). The Environmental Protection Agency (EPA) identified six critical pollutants that are harmful not only the health of the population but also to the environment. These pollutants are monitored by the EPA based on established protocols and include particulate matter <2.5 ($PM_{2.5}$) and <10 (PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), lead (Pb), and carbon monoxide (CO). Within these commonly monitored air pollutants, $PM_{2.5}$ and O_3 have associations to one's health, especially in vulnerable populations and in certain exposure time periods (U.S. EPA, 2003; U.S. EPA, 2009; U.S. EPA, 2015).

The fetal heart develops between weeks two through eight of embryological development, also referred to as the critical exposure window. During this period, factors such as poor air quality may affect the infant's development and result in a CHD (Dadvand et al., 2011; Patel & Burns, 2013). Epidemiology research examining the relationship between CHD diagnosis and air pollutant exposure to the fetus during the critical exposure window is sparse. In a recent search of the literature, eight studies specific to $PM_{2.5}$ and 12 studies specific to O_3 have been found to be researching this relationship with a CHD diagnosis. The literature surrounding this topic has also measured different combinations of pollutant exposures and CHD outcomes. Thus the limited literature and inconsistent findings suggesting the need for further research. Therefore, the purpose of this research was to further explore the relationship between air quality and a CHD diagnosis. Specifically, we hypothesize increasing amounts of $PM_{2.5}$ and O_3 during the critical exposure window of fetal development will be associated with increased rates of critical CHD diagnosis.

To test our hypothesis, we conducted a case-control retrospective study. Inclusion criteria for cases included a nonrandom, purposive sample of infants that met the following criteria: born between January 1, 2014, to December 31, 2016; received care at an identified children's hospital with an established congenital heart program; were diagnosed with one of 13 critical CHDs identified; and were born to mothers with a Mississippi residence at the time of delivery. For each infant, the maternal residential address was mapped as a point on a spatial map using geographical information system (GIS) software. The air pollution values for calculated weeks one through twelve of gestation were obtained for each of the infants included in the study. Air pollution values were obtained from the Environmental Protection Agency's air quality monitors throughout the state of Mississippi. The air quality monitors and their associated values were also added as a layer on the map using GIS software.

Controls in the study were obtained from the Mississippi Department of Health Vital Statistics database. Inclusion criteria for the controls included infants born between January 1, 2014, to December 31, 2016, and were born in the state of Mississippi. Due to privacy concerns, infants in the control group were mapped based on the census block group in which their address was located. Because air pollution

values could not be obtained for an exact address, the pollutant value at the center of the block group was obtained from weeks one through twelve of gestation for each control.

A total of 199 cases and over 99700 controls met the inclusion criteria. Infants identified within a 50 kilometers boundary of an air quality monitoring station and that had a weekly value for each of the weeks one to twelve gestation were used for analysis. A total of 108 cases were included. Of the 42000 controls that were within the 50km boundary, 550 were randomly selected for the study to give a 1:5 case-control ratio.

The process of analyzing the data is currently underway conducting descriptive statistics, correlations, and regression models to determine if potential relationships can be identified. Because both of these variables are mapped spatially, we are also conducting spatial analytic statistics to identify if spatial patterns are found. The hope is that the knowledge gained from this study will not only add to the increasing literature that surrounds this topic but may also be useful evidence for healthcare by identifying potential risk factors for the fetal development of a CHD.

Title:
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Abstract Summary:
Congenital heart defects (CHD) impact nearly 40,000 infants annually with 80% of those diagnosed having an unknown etiology. Environmental risk factors potentially play an important part in CHD development. This research was conducted to further explore the relationship between air quality and a CHD diagnosis.

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I. Introduction

II. Background

A. Congenital Heart Defects

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