The Social Context of Environmental Exposures: an Application to Swine CAFO Air Effluent and Pregnancy Outcomes in North Carolina

by

Melissa Ann Tosiano

Environment
Duke University

Date:_______________________
Approved:

____
Marie Lynn Miranda, Supervisor

____
Dean Urban

____
Jerome Reiter

____
Timothy Barzyk

Thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Environment in the Graduate School of Duke University

2012

ABSTRACT

The Social Context of Environmental Exposures: an Application to Swine CAFO Air Effluent and Pregnancy Outcomes in North Carolina

by

Melissa Ann Tosiano

Environment
Duke University

Date:_______________________
Approved:

____
Marie Lynn Miranda, Supervisor

____
Dean Urban

____
Jerome Reiter

____
Timothy Barzyk

An abstract of a thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Environment in the Graduate School of Duke University

2012

Abstract

Compared to full weight infants, low birth weight infants are at greater risk for short and long term health consequences. Maternal exposure to air pollution is associated with low birth weight, although these studies focused on urban communities and did not extend to rural sources of air pollution[1]. The aim of this study was to investigate the association of maternal exposure to swine CAFO air emissions with birth weight. Information on all North Carolina births from 2004-2008 was extracted from the NCDBR. Due to the scarcity of data concerning maternal exposure to swine CAFO air emissions, metrics of estimated exposure were generated by incorporating CAFO water release permitting and hog steady state live weight (SSLW) into a geographic information system (GIS). Binary exposure metrics were generated using a series of radii around the maternal residence 1, 3, 5km (yes or no). Interaction metrics were generated by multiplying the binary value by the sum of the SSLW within the buffer. An exponential decay metric assumed that 5% of the SSLW remained at the critical distances of 1, 3, or 5km.

Using ordinary least squares regression modeling, this study demonstrates a statistically, but not clinically significant association between exposure to swine CAFO emissions and birth weight. As the metric of estimated maternal exposure to swine CAFO air emissions moved from strictly binary to continuous (from binary to

interaction terms to exponential decay), the association between swine CAFO exposure and decreased birth weight was strengthened. Prior studies have found associations between swine CAFOs and poor health in proximate communities, but none have addressed birth weight [2-8]. The results of this research indicate rural sources of air pollution could potentially adversely affect birth outcomes.

Contents

Abstract iv

List of Tables viiii

List of Figures ix

Acknowledgements x

1. Introduction 1

1.1 Background and Motivation 1

1.1.1Consequences of Adverse Pregnancy Outcomes……………………………….2

1.1.2 Ethnic Disparities in Adverse Pregnancy Outcomes…………………………..3

1.1.3 Environmental Disparities Between Ethnic Groups May Contribute to Ethnic Disparities in Birth Outcomes……………………………………………….5

1.2 Swine CAFOs and Human Health 6

1.2.1Trends in North Carolina Swine Production……………………………………6

1.2.2 Air Emissions from Industrial Swine Production……………………………...9

1.2.3 Health in Communities Proximate to Swine CAFOs………………………...10

1.2.4 Adverse Pregnancy Outcomes and Swine CAFOs…………………………...11

1.3 Thesis Outline 13

2. Data, Prelminary Anlayses and Modeling Swine CAFO Air Emission Exposure.........15

2.1 NCDBR..15

2.2 NCCAFO Permits 16

2.3 Study Area 17

2.3.1 Regional NCDBR Summary Statisitcs 19

2.3.2 Regional Swine CAFO Summary Statisitcs 21

2.4 Modeling Maternal Exposure to Swine CAFO Air Emissions. 23

2.4.1 Summary Statistics of Maternal Demographics by Estimated Swine Exposure Metrics........ 25

3. Regression Anlaysis 26

3.1 Ordinary Least Squres Regression 26

3.1.1 Results Baseline Regression 27

3.1.2 Results Binary Exposure Regression 29

3.1.3 Results Interaction Exposure Regression 31

3.1.4 Results Exponential Decay Exposure Regression 33

3.2 Robustness of Estimated Maternal Exposure to Swine CAFO Air Emissions 35

4. Conclusions 36

4.1 Proposed Mechanisms for the Adverse Impact of Swine CAFOs on Birth Weight 36

4.1.1 Mitigating Factors 38

4.2 Directions for Future Work 39

4.2.1 Field Sampling and Community Driven Reserach 41

4.3 Policy Implications 42

4.4 Conclusion 44

Appendix A 444

References 46

List of Tables

Table 1: Odors and VOCs associated with swine CAFOs. 10

Table 2: Conversion Factors for Generating Values of SSLW... 17

Table 3: NCDBR 2004-2008, summary statistics of unrestricted births by region. 19

Table 4: Percent of pregnancies near swine CAFOs.. 211

Table 5: Summary statistics for all mothers in the study area by swine CAFO exposure…………………………………………………………………………………25

Table 6: Baseline Regression Birth Weight Model……………………………………….. 28

Table 7: Binary Swine CAFO Exposure Birth Weight Models.. 30

Table 8: Interaction CAFO Exposure Birth Weight Models. 322

Table 9: Exponential Decay CAFO Exposure Birth Weight Models. 344

Table 10: Birth Weight and Methods of Estimating Exposure to Swine CAFOs…………35

List of Figures

Figure 1: Host, social, and environmental factors contribute to poor pregnancy outcomes [30].. 2

Figure 2: Rates of A) preterm birth and B) low birth weight among singleton live births in the United Sates from 1990-2006 [30, 45].. …..5

Figure 3: North Carolina hog production dramatically increased in the early 1990s [9]…7

Figure 4: Map of NC permitted swine CAFOs displaying values of SSLW (Steady State Live Weight) in hog pounds [10]………………………...7

Figure 5: Maps of NC counties describing swine production and poverty rates in 2009. The Southeastern coastal plain region (in rectangle) has some of the highest rates of poverty and swine production [9, 11]………………………………………………...……..8

Figure 6: Fate and transport of swine CAFO air Emissions……………………………...10

Figure 7: Regions of NC represented by the NCDBR. This study will be limited to the Coastal Plain Region [10, 57]. 18

Figure 8: Kernel density of births. Areas with a higher density of births are represented by the darker red. 200

Figure 9: Kernel density of Swine CAFOs. Areas with a higher density of swine production are represented by the darker blue. 211

Figure 10: Buffers around swine CAFO with radii of 1, 3 and 5 kilometers..................... 222

Figure 11: Producing the exponential decay estimate of maternal exposure to swine CAFO emissions. 233

Figure 12: Distance to swine CAFO plotted against decay metric at a)1km, b)3km, c)5km 333

Figure 13: Mechanisms by which swine emissions could decrease birth weight. *Bioaerosols include viable pathogens, endotoxin, and swine dander. 377

Acknowledgements

I would like to sincerely thank my advisor Marie Lynn Miranda. Marie Lynn's support and mentoring has aided my personal and professional development. Throughout my graduate school experience her insight has illuminated new paths for my career. I am grateful for financial support for this work provided by the USEPA in support of the Southern Center on Environmentally Driven Disparities in Birth Outcomes (RD-83329301).

Additionally, I would like to extend special thanks to the members of my board. Dean Urban, Jerry Reiter, and Tim Barzyk, your conversations and technical input have helped immensely.

To my colleagues at CEHI and ITEHP thank you so very much for sharing this journey with me. Your advice, technical support, and friendships are irreplaceable. To the ladies of the REACH project, thank you for the privilege of working with your community. I would like to extend special thanks to David Hinton and Meg Stephens for their accessibility, guidance, and overwhelming kindness.

Last but not least I would like to thank my family everything they have given me. You guys saw my potential when no one else did. In the future I hope to be a source of support instead of a source of stress. To my beloved Minnaloushe, your presence has enriched my life in unimaginable ways, many thanks.

1. Introduction

1.1 Background and Motivation

Air emissions from confined animal feeding operations (CAFOs) have been associated with poor health outcomes in neighboring communities, particularly respiratory symptoms [4, 5, 7, 12]. Prior research has indicated that air pollution may also be associated with poor birth outcomes [13-23]. However, the majority of these studies have been limited to urban areas [14, 24-29]. Associations of CAFO air emissions with low birth weight and preterm birth have not been adequately investigated. It is essential to address this data gap because adverse birth outcomes are associated with poor health outcomes later in life.

The majority of studies investigating the association of air pollution with adverse pregnancy outcomes estimate exposure by generalizing ambient air pollution over a large geographic region[14]. However, these generalizations may not appropriately reflect the air pollution exposure of an individual. Evidence indicates that varying the geographic scale of ambient air pollution generalizations (Example County vs. Census tract) alters the association with health outcomes [30, 31]. This study uses an exponential decay function of hog steady state live weight to estimate individual maternal exposure to swine CAFO air emissions, generating an alternative technique for evaluating exposure to agricultural air pollutants [30, 32].

1.1.1 Consequences of Adverse Pregnancy Outcomes

Adverse pregnancy outcomes include conditions such as low birth weight and preterm birth. Low birth weight is defined as any birth less than 2500g, while preterm birth is defined as any birth less than 37 weeks gestation [33]. Poor pregnancy outcomes are the result of combined host, social and environmental factors interacting with one another (See Figure 1). However, the mechanism by which these factors contribute to adverse pregnancy outcomes remains largely unclear, especially in certain susceptible subpopulations.

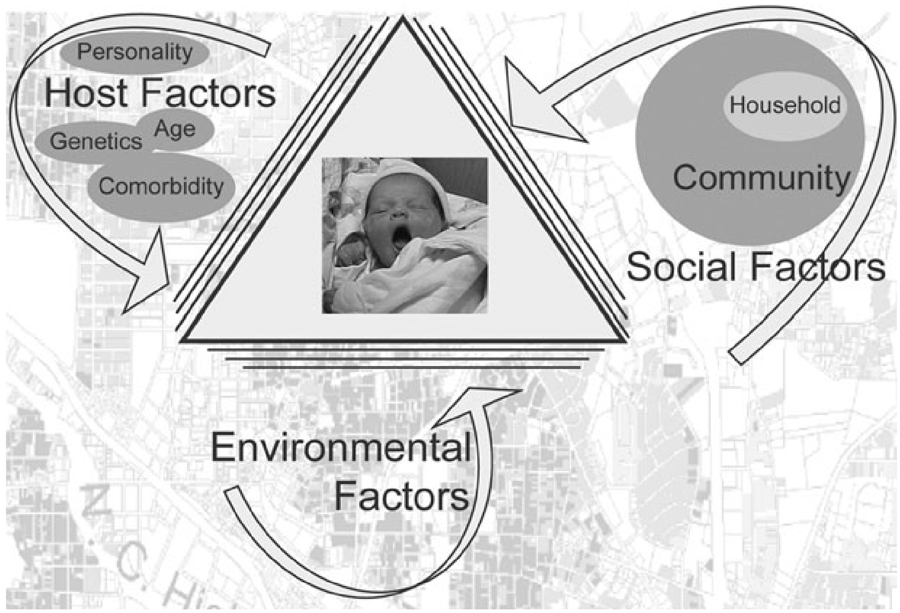

Figure 1: Host, social, and environmental factors contribute to poor pregnancy outcomes [31].

While advances in neonatology have led to the survival of increasingly preterm and low weight infants, as a group, these individuals have an increased risk for immediate and long term health challenges. Preterm birth is the leading cause of neonatal mortality [34]. In infancy, low birth weight and preterm individuals are at increased risk for developing respiratory distress syndrome, variable heart rate, cerebral palsy, mental retardation, blindness, and deafness [35-38]. In childhood, preterm and low weight infants are more likely than full weight term infants to have motor impairment, learning disabilities, and behavioral issues requiring additional educational resources [39-41]. Adults who were born preterm or low weight are disproportionately at risk for chronic diseases including diabetes, obesity, and cardiovascular disease [42, 43]. In 2005, the Institute of Medicine estimated that the annual cost of preterm birth in the United States was approximately 26 billion dollars [34]. This estimate is conservative since it only accounts for immediate medical expenses and does not incorporate the cost of disability and chronic disease later in life [34]. While acute health challenges face preterm and low birth weight infants, the long term consequences arising from poor pregnancy outcomes impose significant economic costs to healthcare, educational and social welfare systems.

1.1.2 Ethnic Disparities in Adverse Birth Outcomes

In the United States, disparities in rates of low birth weight and preterm births occur between racial groups. Ethnic disparities in adverse birth outcomes persist throughout the country over time [44, 45] (See Figure 2). In 2006, 9.7% of non-Hispanic white singleton births were preterm; comparatively, 16.5% of non-Hispanic black singleton births were preterm that year [46]. From 1990 through 2006 the rates of low weight and preterm births was almost three times higher in non-Hispanic blacks than non-Hispanic whites [46]. Within ethnic groups there is significant geographic variation in adverse birth outcomes. The rates of preterm and low birth weight are elevated in the Southeastern United States compared to other regions of the United States [2]. The increased rates of adverse birth outcomes in the Southeastern United States are present across all ethnicities, but are especially pronounced among African Americans [31]. Despite multiple programs and initiatives attempting to reduce poor pregnancy outcomes, the rates of low weight and preterm births continue to be elevated in minority groups [35, 45, 47-50]. It is hypothesized that environmental factors may significantly impact pregnancy outcomes. The persistant disparate rates of adverse pregnancy outcomes between ethnic groups may be due to environmental factors unaddressed by current programs. Additionally, prior studies indicate adverse environmental impacts on pregnancy are exacerbated by poverty, which minority women are more likely to face than non-Hispanic white women [51].

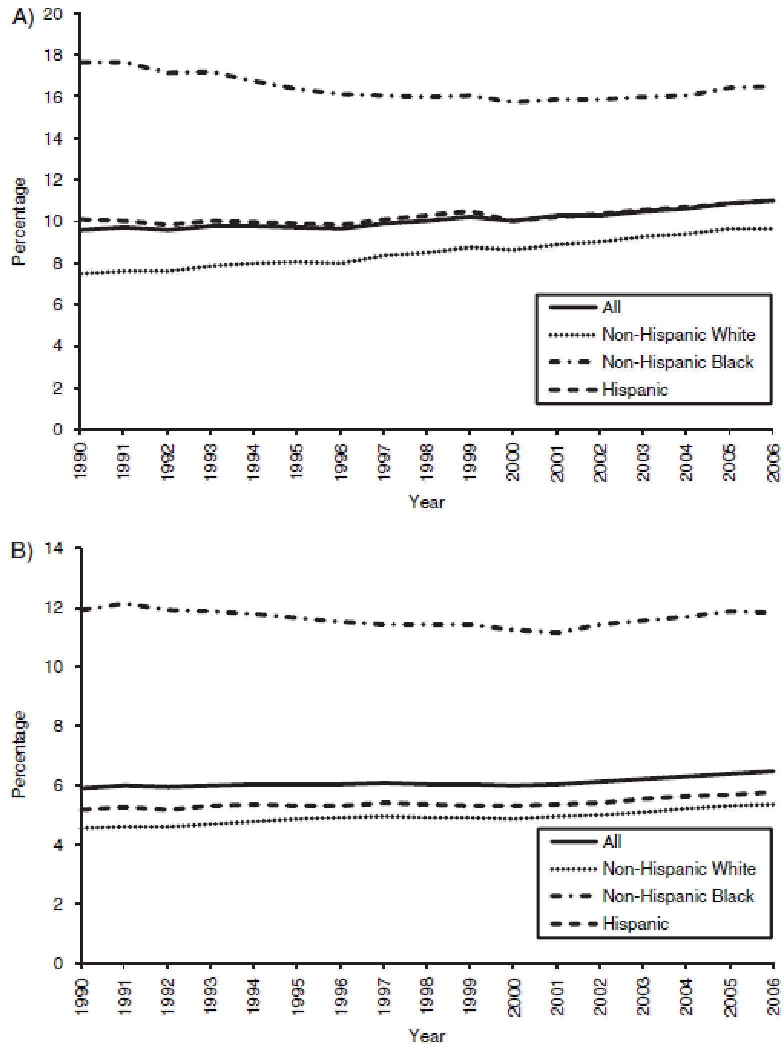

Figure 2: Rates of A) preterm birth and B) low birth weight among singleton live births in the United Sates from 1990-2006 [31, 46]

1.1.3 Environmental Disparities Between Ethnic Groups May Contribute to Ethnic Disparities in Birth Outcomes

Independently, poor socioeconomic status and air pollution have been associated with adverse birth outcomes. A variety of air pollutants are associated with low birth weight and preterm birth, including but not limited to: particulate matter, ozone, sulfur dioxide, nitrogen oxides, and carbon monoxide [13, 14, 16, 18-22]. Women of lower socioeconomic status are more likely to be exposed to air pollutants during pregnancy [51]. Only 33% of non-Hispanic whites live in areas failing to meet two or more of the national air quality standards, while 60% of Hispanics and 50% of non-Hispanic blacks live in areas failing to meet two or more of the national air quality standards [52]. In addition to increased exposure to ambient air pollutants, this cohort of women also may be more vulnerable to the health effects of air pollutants due to the physical manifestations of psychosocial stress attributed to poor socioeconomic status [51, 53-56]. The synergistic combination of increased exposure to air pollutants and psychosocial stress could contribute to the ethnic disparities in adverse birth outcomes.

1.2 Swine CAFOs and Human Health

1.2.1 Trends in North Carolina Swine Production

In the State of North Carolina, the hog population has exponentially increased since the early 1990s (See Figure 3), while simultaneously localizing in the southern coastal plain region and decreasing in the rest of the state (See Figure 4) [57]. In less than ten years, North Carolina went from the 18th ranked state for swine production to the 2nd[11]. Historically, hog farming was a secondary commodity of farms focusing on row crops (corn, tobacco, soy…) and the entire production lifecycle from farrow to finish was completed in the same location. As recently as the 1970s, the average number of hogs on a NC swine farm was only 150 head [57]. Beginning in the mid-1980s, NC hog production shifted from traditional, small scale mixed commodity farming, to a large scale, vertically integrated, single commodity, contract farm model [57].

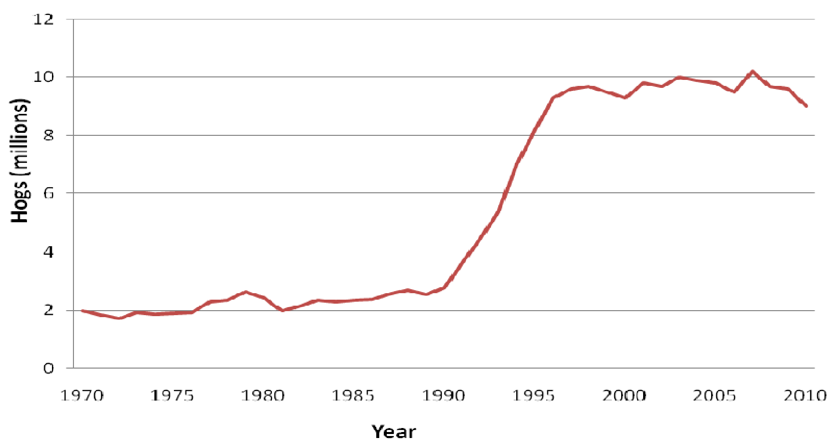

Figure 3: North Carolina hog production dramatically increased in the early 1990s [9].

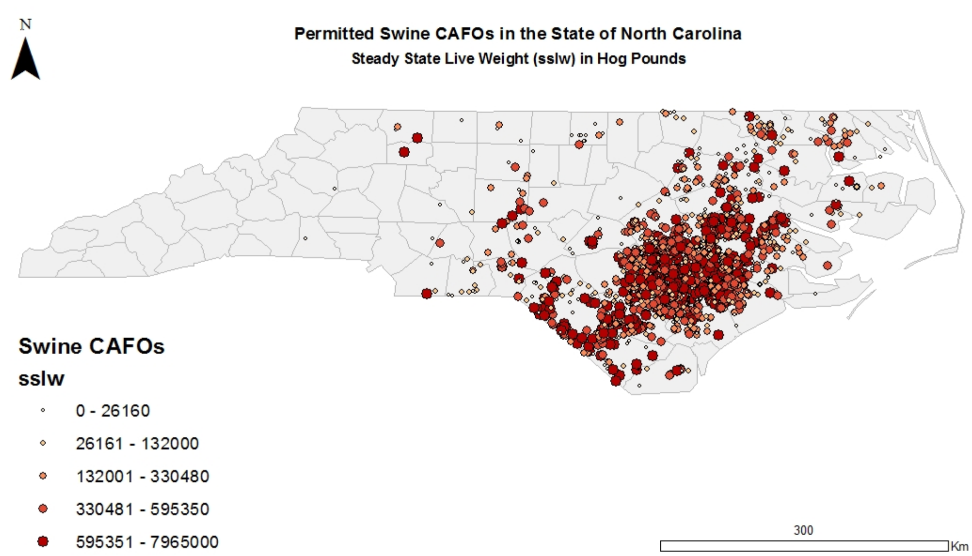

Figure 4: Map of NC permitted swine CAFOs displaying values of SSLW (Steady State Live Weight) in hog pounds [10].

In numerous states including North Carolina, CAFOs are disproportionately located in economically disadvantaged rural communities of color [58-60]. Consequently exposure to CAFO air emissions is an environmental justice issue [58-62]. Specifically, NC counties located in the Southeastern coastal plain are some of the most economically challenged in the state; additionally, these counties compose the most concentrated swine-producing region in the United States [9, 11](See Figure 3). Disparate exposures to CAFO air emissions may contribute to the persistent disparities in adverse birth across ethnic groups. Economically disadvantaged, rural communities of color may be at disproportionate risk for poor birth outcomes associated with CAFO air emissions.

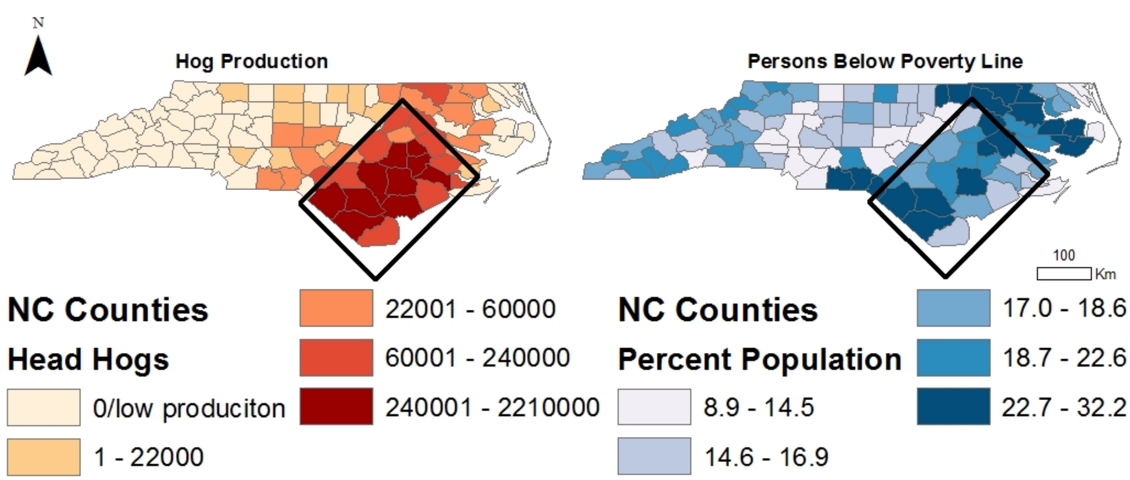

Figure 5: Maps of NC counties describing swine production and poverty rates in 2009. The Southeastern coastal plain region (in rectangle) has some of the highest rates of poverty and swine production [9, 11].

1.2.2 Air Emissions from Industrial Swine Production

Swine CAFOs generate air emissions from three distinct sources: confinement houses, waste lagoons, and field application of manure (See Figure 5). The air pollutants generated from CAFO swine farming include ammonia, hydrogen sulfide, methane, carbon monoxide, carbon dioxide, particulate matter, bioaerosols, and volatile organic compounds (VOCs) [63]. Passive air sampling has indicated that the concentrations of these air emissions are positively correlated with proximity to swine CAFOs [7, 12, 63]. Compared to pastured swine, CAFOs produce significantly higher levels of hydrogen sulfide, bioaerosols, and odorants per head [64]. A total of 331 different VOCs were identified in NC swine facilities [65]. The majority of VOCs associated with hog waste have unpleasant and irritating odors (See Table 2). Most of these compounds are present at concentrations below odor and irritation thresholds [65]. However, the cumulative exposure to hundreds of low level VOCs simultaneously is perceived as odorous and irritating to humans.

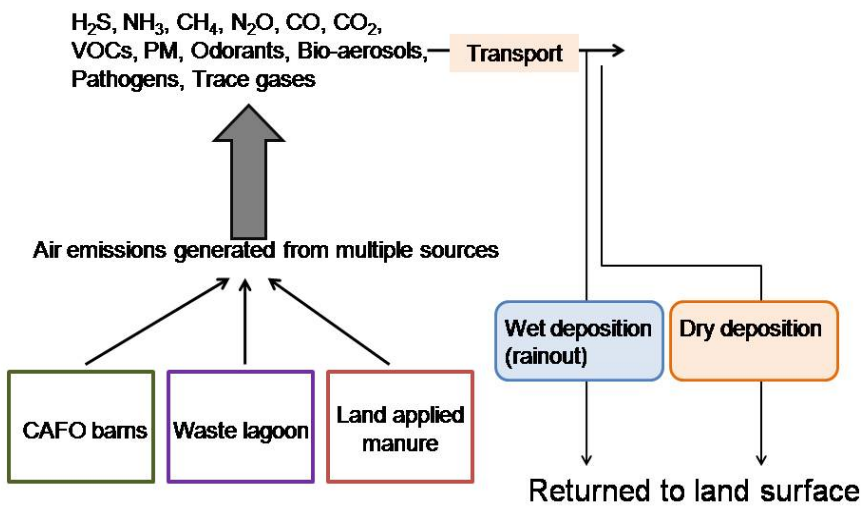

Figure 6: Fate and transport of swine CAFO air Emissions

Table 1: Odors and VOCs associated with swine CAFOs.

Chemical Name	Smell
Hydrogen sulfide	rotten eggs
Dimethyl sulfide	rotten vegetables
Butyric acid	rancid butter
Valeric acid	putrid, fecal
Skatole	fecal, nauseating
Indole	intensely fecal

1.2.3 Health in Communities Proximate to Swine CAFOs

While it is accepted that swine air emissions present an occupational hazard to agricultural workers, there is mounting evidence of swine CAFOs air emissions impacting the health of residents in nearby communities. Although community members are exposed to lower levels of CAFO air emissions than workers, it is essential to note that the proximate population does not choose to be exposed to swine CAFO emissions and may include individuals who may have a sensitivity to air quality including children, asthmatics, the elderly, and pregnant women [5, 66]. The intermittent nature of CAFO air emissions alters the normal process of long and short term adaption, increasing the likelihood of an individual becoming sensitized to these air emissions [67]. The most prominent health complaints of residents living near CAFOs are respiratory difficulties including nasal allergies, shallow respiration, and asthma [2, 4, 5, 8, 12]. Exposed individuals also experience other physical and mental health impacts. Including but not limited to: diarrhea, headaches, reduced mucosal immune function, staph infection, burning eyes, depression, difficulty concentrating, and anxiety [2, 3, 5, 8, 58, 66, 68-70]. Odorous emissions from the hog CAFOs have the potential to induce symptoms through several different mechanisms such as: a) exposure to pollutants at concentrations that induce irritation or other toxicological responses, b) innately or acquired aversion to odor at non-irritating concentrations, or c) co-exposure to pathogens and endotoxins [6, 63, 67].

1.2.4 Adverse Pregnancy Outcomes and Swine CAFOs

CAFO air emissions may be associated with poor health in residents of nearby communities. Of these health effects, respiratory symptoms have most often been associated with exposure to CAFO air emissions [8, 12, 66]. A study by Sneeringer *et al.*

suggested that doubling hog production in a region could lead to an increase in infant mortality, due to elevated levels of respiratory distress. This highlights the potential for swine CAFO air emissions to impact infant health and possibly pregnancy outcomes [71]. Furthermore, a Norwegian study demonstrated that women living near swine farms are at increased risk of preeclampsia compared to women living near grain farms or other livestock due to an immune response to increased pathogen exposure [72]. Emissions from industrial hog farming could be associated with poor infant health and adverse pregnancy outcomes.

Immunological responses may contribute to the incidence of preeclampsia; the most common condition leading to preterm birth [73, 74]. Specifically, exposure to viable pathogens, endotoxins, animal dander, and other bioaerosols can trigger a systematic immune response contributing to preeclampsia [70, 71]. All of these biologic components are present in swine CAFO air emissions.

In urban settings, air pollutants are associated with preterm birth and low birth weight [1, 13-16, 18-23, 27, 75-86]. The pollutants found in urban settings; including sulfur dioxide, particulate matter, carbon monoxide, and hydrogen sulfide are also major components of swine CAFO air emissions. Swine CAFO air emissions are a complex mixture of hundreds of toxicants, odorants, particulates, and pathogens [22, 87-90]. Despite evidence indicating the association of air pollutants with adverse birth outcomes, there is a dearth of work concerning the association of CAFO air emissions with adverse birth outcomes.

Individuals residing near swine CAFOs report a multitude of adverse health outcomes [7, 8, 12, 70, 91]. If swine CAFO air emissions are correlated with other adverse health conditions, this complex mixture of pollutants may also be correlated with poor pregnancy outcomes. Investigating the association of adverse birth outcomes with maternal exposure to swine CAFO emissions addresses the issue of ethnic disparities in birth outcomes due to environmental inequalities.

1.3 Thesis Outline

Using regression analysis and geospatial modeling techniques, this thesis addresses issues surrounding maternal exposure to swine CAFO air emissions and birth weight. Elucidating the relationship between exposure to swine CAFO air emissions and birth weight in North Carolina's coastal plain region is the main objective of this study. Using ordinary least squares regression, the relationship between birth weight and exposure to swine CAFO air emissions was evaluated using various metrics of estimated swine exposure.

Chapter 2, briefly summarizes the North Carolina Detailed Birth Record (NCDBR) and the North Carolina Confined Animal Feeding Operations (NCCAFO) databases. Maternal demographics and estimated swine exposure are summarized by the study region (Coastal Plain) and the rest of the state (Mountain/Piedmont). Additionally, this chapter presents regional variation in swine production and the methods of spatially modeling maternal exposure to swine CAFO air emissions.

Chapter 3 explores the relationships between standard maternal covariates maternal exposure to swine CAFO air emissions and birth weight using ordinary least squares regression. A baseline model without a variable for swine CAFO air emissions examines the associations between the standard covariates and birth weight in the study area. Regression models were constructed for each of the binary, interaction, and decay models of maternal exposure to swine CAFO air emissions. Generating multiple regression models investigates the robustness of the metrics of maternal exposure to swine CAFO air emissions.

Chapter 4 discusses the results from the ordinary least squares regression analysis. Plausible biological mechanisms behind population trends in birth weight are put forth. This chapter concludes by briefly exploring directions for future research and policy implications.

2. Data, Preliminary Analyses and Modeling Swine CAFO Air Emissions Exposure

2.1 NCDBR

The North Carolina Detailed Birth Record (NCDBR) contains information on the outcome of every live birth in the State of North Carolina, including birth weight, estimated gestational age, plurality, parity, medical complications, and known congenital abnormalities. Maternal and paternal demographics are also noted including educational attainment, age, ethnicity, and marital status. The Children's Environmental Health Initiative (CEHI) had already standardized and address-geocoded the NCDBR data from 1990 through 2008. This analysis is restricted to births that occurred from 2004 through 2008. Maternal exposure to swine CAFO air emissions is estimated using the geographic location of the maternal residence (at delivery). For a birth to be included in this analysis, the following criteria must be met: a)singleton pregnancy, b)no congenital abnormalities, c)maternal age 15-44, d)birth weight greater than 400 grams, e)gestational age 24-42 weeks, f)ethnicity non-Hispanic White, non-Hispanic Black, or Hispanic, and g) first through fourth births to a given mother (*n*=122084). All of the covariates used in the final regression analysis (except the estimated value of exposure to swine CAFO air emissions) were obtained from the NCDBR and were associated with birth weight in comparable studies [30].

2.2 NCCAFO Permits

All confined animal operations over a specific number of head must obtain a required water release permit from the NC Division of Water Quality accounting for waste management practices [10]. All swine producers with over 250 head are required to have an active permit. The water release permit records for all commodities were downloaded from the North Carolina Department of Environment and Natural Resources' Division of Water Quality's Animal Feeding Operations Unit. The swine CAFO facilities were extracted, standardized and address geocoded. Swine operations with missing data were excluded along with 2 facilities owned by North Carolina State University. Swine facilities owned by Universities were excluded since they are subjected to different water release regulations than commercial operations.

Once the swine CAFO permits were geocoded, a measure of steady state live weight (SSLW) in hog pounds was generated by multiplying the average live weight by the number of head [92] (See Table 2). Since waste is the greatest source of swine CAFO air emissions, it is advantageous to use SSLW instead of a raw count of head. The volume of waste generated per head varies significantly with different swine production stages. For example, a 10 pound piglet produces significant less waste then a 130 pound finished hog. Therefore, SSLW more accurately reflects the amount of air emissions generated by a swine CAFO than the number of head.

Table 2: Conversion Factors for Generating Values of SSLW.

Swine Type	Average Live Weight (lb/hog)
Sows (gestating/lactating)	400
Farrow-to-Wean	10
Wean-to-Feed	30
Feed-to-Finish	135
Boars	400

2.3 Study Area

This research is geographically limited to the State of North Carolina. Historically North Carolina has been a rural state, but over the last 30 years it has undergone rapid urbanization. The population of this state is one of the fastest growing nationally due to immigration [11]. Ethnically, NC is a diverse state. According to the US 2010 census, non-Hispanic whites are 68.5% of the population, non-Hispanic blacks are 21.5% of the population, while Hispanics, the most rapidly growing segment, are 8.4% of the population [11].

This study area is further restricted to the coastal plain region of North Carolina (See Figure 5). The Coastal Plain is the core region of NC hog production. The population demographics of the Coastal Plain region diverge from the demographics in the Mountains and Piedmont region. In general, the Coastal Plain region has higher rates of poverty, lower rates of education, and a higher percentage of minorities compared to the Mountain and Piedmont regions [11].

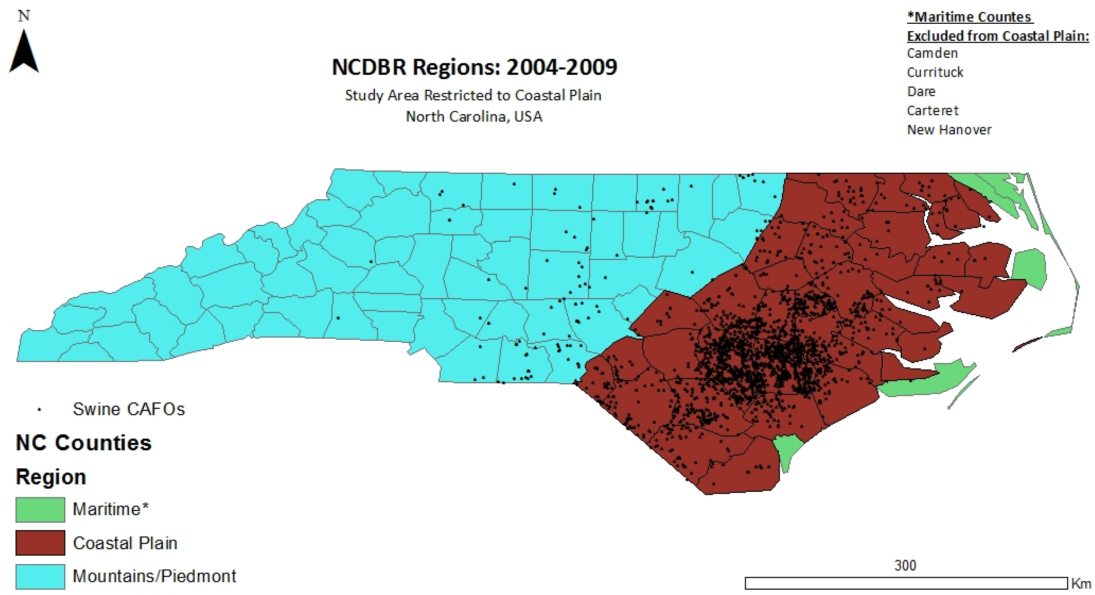

Figure 7: Regions of NC represented by the NCDBR. This study will be limited to the Coastal Plain Region [10, 11].

2.3.1 Regional NCDBR Summary Statistics

Demographic differences between the Coastal Plain and Mountain/Piedmont regions extend to pregnant women and birth outcomes. While most variables are similar between regions, on average, infants born in the Coastal Plain weigh 27.5 grams less than infants born in the Mountains/Piedmont region (See Table 3). Furthermore, mothers in the Coastal Plain are more likely to have demographics that have previously been negatively associated with birth weight compared to Mountain/Piedmont mothers. For example, 30.8% of births in the Coastal Plain were to non-Hispanic Black mothers compared to only 20.9% in the rest of the state (See Table 3).

Table 3: NCDBR 2004-2008, summary statistics of unrestricted births by region.

	Coastal Plain	Mountains/Piedmont
Total Births	122084	344846
Mean BWT (g±SD)	3289.3±578.7	3316.8±566.8
%LBW	7.3	6.5
Mean gestation (weeks±SD)	38.6±2.0	38.7±2.0
32-34	2.1	1.9
35-36	5.7	5.5
37-38	28.7	26.5
39-40	52.9	55.0
41-42	9.2	9.8
% Male	51.4	51.1
% First Born	43.0	42.4
% Trimester Prenatal Care		
First	83.3	83.3
Second	13.3	13.4
Third	2.0	1.9
None	0.6	0.6
Unknown	0.9	0.7
% Race/ethnicity		
NHW	55.5	61.3
NHB	30.8	20.9
HISP	13.8	17.8
% Maternal edu. (years)		
<9	5.6	6.8
9-11	15.3	15.4
12	34.7	26.0
13-15	26.4	20.9
>15	17.9	30.8
% Maternal age (years)		
15-19	13.6	11.1
20-24	34.7	24.8
25-29	27.8	27.4
30-34	16.2	23.7
35-39	6.6	11.2
40-44	1.6	1.9
% Tobacco use	11.9	10.4
% Married	58.1	61.8

Spatially the births included in the sample (unrestricted births, 2004-2008, coastal plain, n=122084) are not evenly distributed. While the Coastal Plain region is generally rural, births cluster around small cites (See Figure 8).

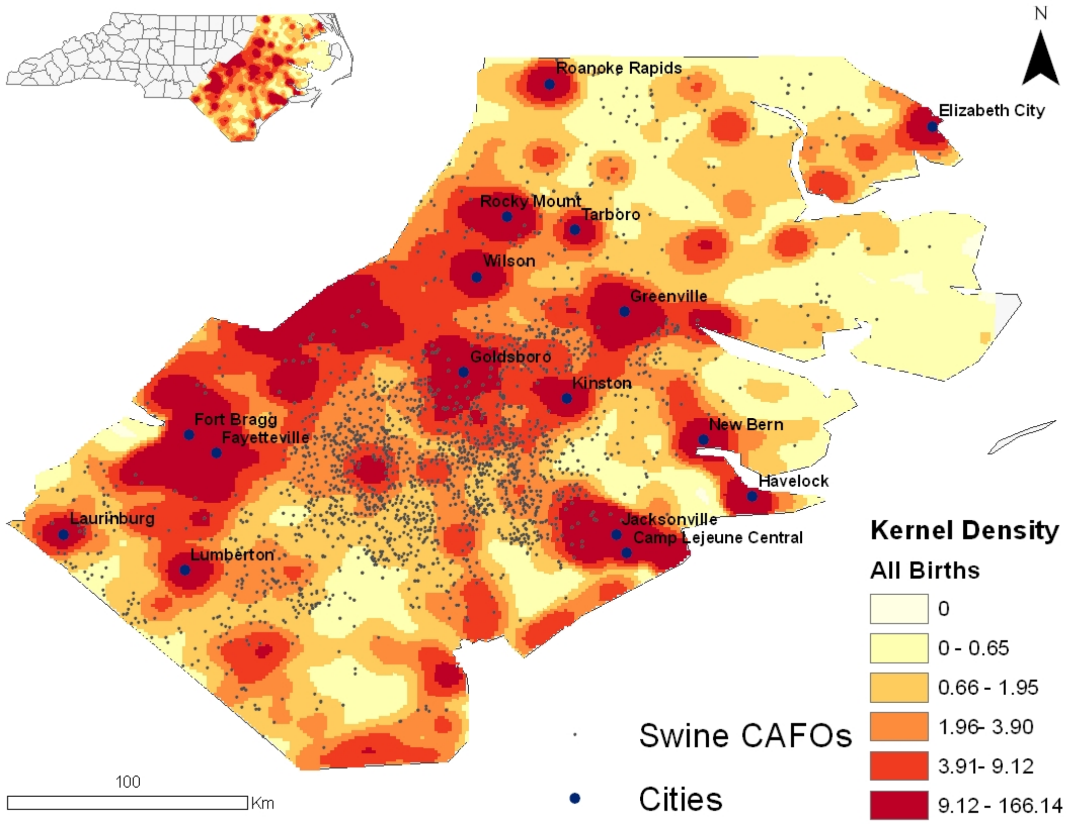

Figure 8: Kernel density of births. Areas with a higher density of births are represented by the darker red.

2.3.2 Regional Swine CAFO Summary Statistics

The Coastal Plain region of North Carolina is the largest concentrated area of swine production nationwide. Of the 2368 swine CAFOs over 250 head, 2256 (95.3%) are in the Coastal Plain and only 112 (4.7%) are in the Mountains/Piedmont. Additionally, the largest CAFOs (over 100,000 head) are located in the Coastal Plain [10]. Women living in the Coastal Plain region are more likely to live in the vicinity of a swine CAFO then women living in the Mountains/Piedmont (See Table 4).

Table 4: Percent of pregnancies near swine CAFOs.

	Coastal Plain	Mountain/Piedmont
within 1km of Swine CAFO	3.85%	0.08%
within 3km of Swine CAFO	20.70%	1.18%
within 5km of Swine CAFO	37.79%	3.23%

Spatially the swine CAFOs included in this study (permitted facilities, coastal plain, 250+ head) are not homogenously distributed. Swine CAFOs tend to cluster in the sparsely populated South-Central Coastal Plain Region (See Figure 9).

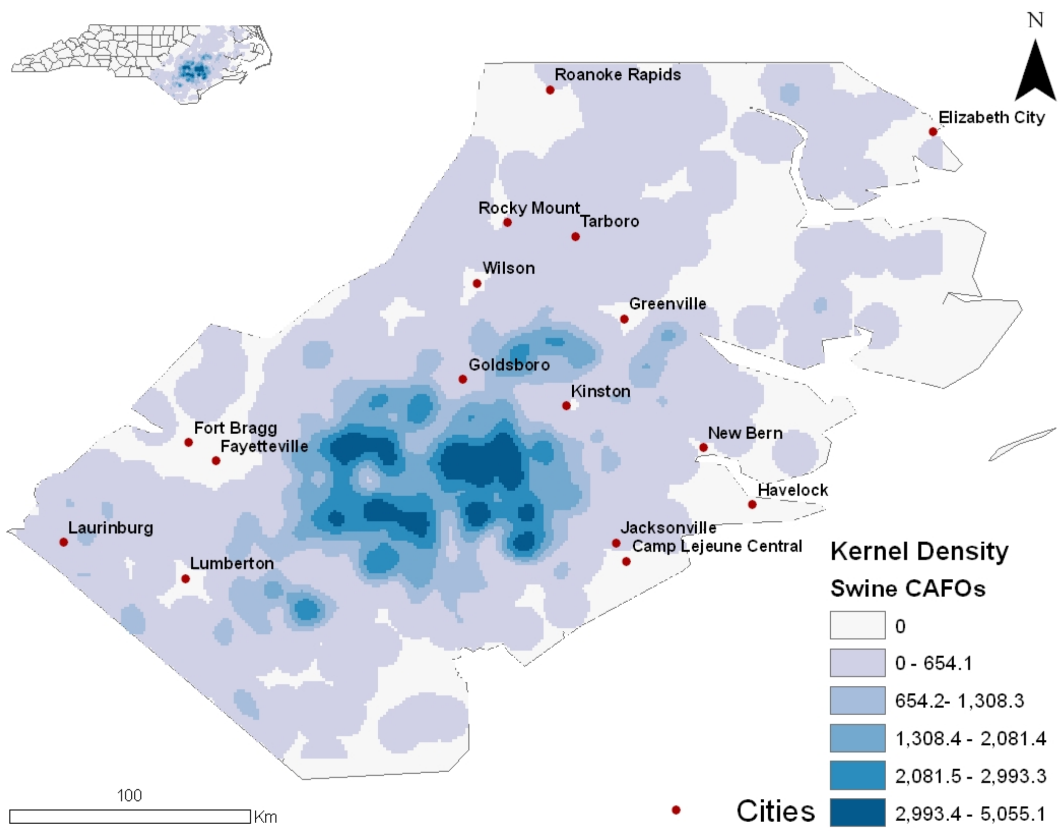

Figure 9: Kernel density of Swine CAFOs. Areas with a higher density of swine production are represented by the darker blue.

2.4 Modeling Maternal Exposure to Swine CAFO Air Emissions

Maternal exposure to swine CAFO air emissions was estimated by generating a series of 1, 3 and 5 kilometer buffers from the centroid of the swine CAFO (See Figure 10). The mothers who resided within these buffers were extracted and classified as "exposed" to swine CAFO air emissions. This initial extraction generated three sets of binary variables estimating maternal swine exposure. The variables were within 1km (yes/no), within 3km (yes/no), and within 5km (yes/no).

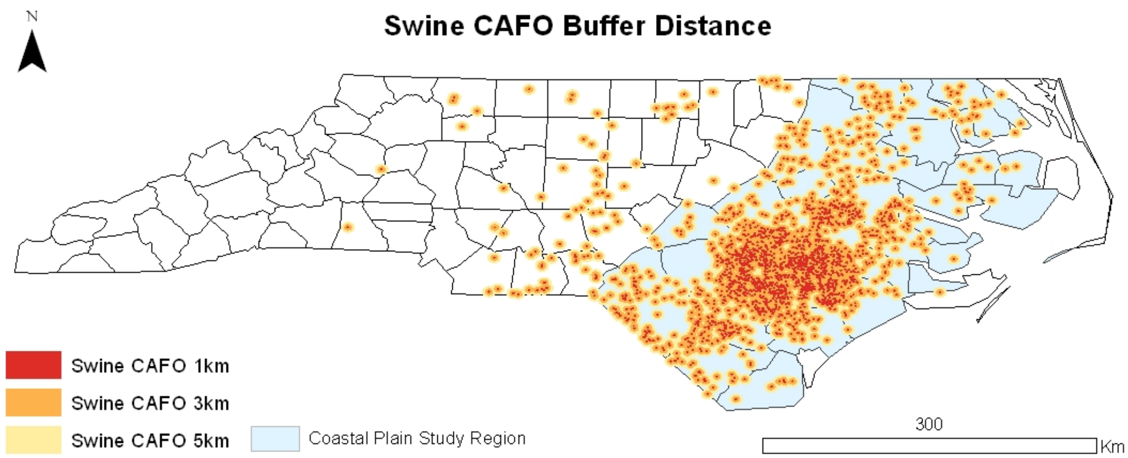

Figure 10: Buffers around swine CAFO with radii of 1, 3 and 5 kilometers.

The binary estimates of swine exposure were used to generate an interaction value weighted by SSLW. The value of SSLW for the particular CAFO was multiplied by the binary value. If multiple CAFOs existed within the radius, the SSLW of all the facilities were summed and multiplied by the binary value. The 5km radius had the most individuals that were exposed to multiple CAFOs.

Studies indicated that components of swine CAFO air emissions that are associated with adverse human health outcomes may decay exponentially [12, 63, 93]. Therefore, an additional set of estimated exposure values were generated using a critical distance of 1, 3, or 5km. In an exponential decay function, the value of k was solved under the assumption that 5% of the CAFO's air emissions remain at the critical distances of 1,3 and 5km (See Appendix A). For each individual, the total exposure value was generated by summing the outcome of the decay function from each individual CAFO and distance from maternal residence (See Figure 9).

Solving for k (decay coefficient):

5%=exp(k*critical distance)

Generating the exponential decay value of estimated exposure:
E_i= individual's total exposure to swine CAFOs (hog pounds)
w_j= weight (hog pounds of individual CAFO)
k=decay coefficient
d_{ij}=distance from CAFO individual's home (km)

$E_i = \sum w_j \exp(-k * d_{ij})$

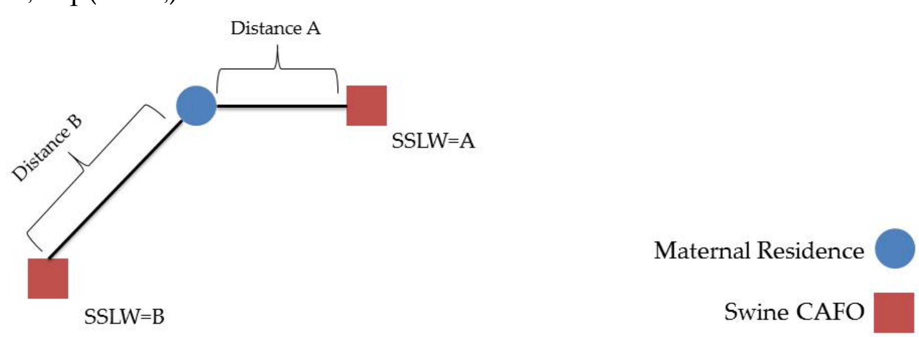

Figure 11: Producing the exponential decay estimate of maternal exposure to swine CAFO emissions.

2.4.1 Summary Statistics of Maternal Demographics by Estimated Swine Exposure Metrics

The standardized covariates were summarized for all the mothers in the study area and compared to individuals exposed to swine emissions (1km CAFO, 3km CAFO, and 5km CAFO) and individuals classified as unexposed to swine emissions (not 1km, not 3km, and not 5km). Overall, the variables were fairly consistent between the entire Coastal Plain Region and the exposed or unexposed individuals. Maternal race/ethnicity is a prominent exception to this generalization. For the entire study area, 30.8% of mothers are non-Hispanic black and only 13.8% are Hispanic. However, of the mothers residing within 1km of a CAFO only 17.0% of mothers are non-Hispanic black and 25.7% are Hispanic (See Table 5). This trend also emerges with mothers within 3 and 5km of a swine CAFO albeit to a lesser extent. So in this study area, more Hispanic mothers live in the vicinity of swine CAFOs than African American or White mothers, reflecting statewide demographic changes. In North Carolina, African Americans are migrating from rural areas to urban centers, while Hispanics (the fastest growing NC minority) are immigrating to rural areas to take advantage of jobs in the agricultural sector [11].

Table 5: Summary statistics for all mothers in the study area by swine CAFO exposure

	C.P Region	1km CAFO	not 1km	3km CAFO	not 3km	5km CAFO	not 5km
N Total Births	122084	4635	117449	24920	97164	45343	76741
Total Births(%)	100	3.8	96.2	20.4	79.6	37.1	62.8
Mean BWT (g)±SD	3289.3±578.7	3298.0±574.3	3288.9±578.9	3296.3±579.8	3287.5±578.4	3290.5±580.4	3288.6±577.6
Gestation (wks)							
<=34(%)	3.5	3.5	3.5	3.6	3.5	3.6	3.5
35-36(%)	5.7	5.2	5.7	5.4	5.8	5.5	5.8
>=37(%)	90.8	91.3	90.7	91	90.7	90.9	90.7
Race/ Ethnicity							
NHW(%)	55.5	57.3	55.4	57.6	54.9	56.5	54.9
NHB(%)	30.8	17	31.3	22.4	32.9	26.4	33.3
HISP(%)	13.8	25.7	13.3	20	12.2	17.1	11.8
Maternal Education(yrs.)							
<9(%)	5.6	13.5	5.3	10.3	4.4	8.6	3.8
9-11(%)	15.3	18.5	15.2	17.2	14.8	17	14.3
12(%)	34.7	30.1	34.9	33	35.1	33.8	35.3
13-15(%)	26.4	25.1	26.4	24.7	26.8	24.7	27.3
>15(%)	17.9	12.6	18.1	14.5	18.7	15.7	19.2
Maternal Age(yrs.)							
15-19	13.6	14.7	13.5	14.4	13.4	14.6	13
20-24	34.7	32.3	34.8	32.5	35.2	33	35.7
25-29	27.7	28.1	27.7	28.6	27.6	28.1	27.5
30-34	16.2	16.7	16.2	16.9	16.1	16.6	16
35-39	6.6	6.9	6.6	6.5	6.6	6.6	6.6
40-44	1.2	1.2	1.2	1.2	1.2	1.2	1.1
Prenatal Care							
First Tri.(%)	83.3	80.3	83.4	80.2	84.1	80.3	85.1
Second Tri.(%)	13.2	15.4	13.2	15.3	12.7	15.3	12
Third Tri.(%)	2	2.4	2	2.3	1.8	2.6	1.6
None(%)	0.6	0.7	0.6	0.6	0.6	0.7	0.5
Unknown(%)	0.8	1.3	0.8	1.2	0.8	1.1	0.7
Tobacco use(%)	11.9	12.9	11.8	12.8	11.6	12.7	11.4
Married(%)	59.1	57.7	59.1	57.9	59.4	56.9	60.4
First Born(%)	42.9	39.6	43.1	41	43.4	41.8	43.6
Male(%)	51.4	51.6	51.4	51.3	51.4	51.3	51.5

3. Regression Analysis

3.1 Ordinary Least Squares Regression

Multiple linear regression modeling was used to determine the association between the estimated swine CAFO exposure metrics (binary, interaction, and exponential decay at 1, 3, 5km) and birth weight. Using birth weight as a continuous outcome variable, the following demographic variables were controlled for: gestational age (<=34, 35-36 and >=37 weeks), maternal race/ethnicity (non-Hispanic white, non-Hispanic black, or Hispanic), maternal education (<9, 9-11, 12, 13-15, and >15 years), trimester prenatal care began (1, 2, 3, none, unknown), tobacco use during pregnancy (yes or no), marital status (married, unmarried), year of birth (2004, 2005, 2006, 2007, or 2008), firstborn (yes or no), and infant sex (male or female). These covariates were used because of their association with birth weight in the current literature [30, 50]. Birth year (a trend variable) was included since birth weight decreased over time in this study area.

A baseline model without a metric for estimated swine exposure was constructed to examine the association of the previously mentioned standard covariates and birth weight. Separate models were constructed for each of the 9 estimated exposure metrics. Three models incorporated the binary metric within 1km (yes or no), within 3km (yes or no), or within 5km (yes or no). Three models incorporated the SSLW weighted interaction metric as a continuous variable, 1km (binary*SSLW), 3km (binary*SSLW), 5km (binary*SSLW). The final three models incorporated the exponential decay metric assuming that 5% of the SSLW remained at the critical distances of 1, 3, and 5km.

3.1.1 Results Baseline Regression

The results of the baseline regression model were as expected. All of the variables were statistically significant and produced coefficients with the expected signs (See Table 6). Prenatal care had the weakest association with birth weight; however this finding is supported in the current literature. In prior studies, prenatal care has the weakest association with birth weight, while gestational age has the strongest association with birth weight [35, 94].

Table 6: Baseline Regression Birth Weight Model

	Baseline Model		
	Coef.	95% C.I.	
(Intercept)	3276.2	3264.9, 3287.6	***
Gestation (wks)			
<=34	-1562.3	-1576.2, -1548.3	***
35-36	-626.7	-637.7, -615.6	***
>=37			
Race/ Ethnicity			
NHW			
NHB	-198.1	-204.7, -191.5	***
HISP	-51.0	-60.0, -41.9	***
Maternal Education			
<9	-25.3	-38.4, -12.2	***
9-11	-27.3	-35.6, -19.9	***
12			
13-15	23.1	16.3, 30.0	***
>15	31.5	23.0, 40.0	***
Maternal Age			
15-19	-32.5	-42.6, -22.4	***
20-24	-19.0	-25.9, -12.1	***
25-29			
30-34	9.9	1.8, 18.1	*
35-39	15.4	4.1, 26.6	**
40-44	-5.5	-29.8, 18.8	
Prenatal Care			
First Tri.			
Second Tri.	-13.2	-21.0, -5.3	**
Third Tri.	-0.2	-18.8, 18.4	
None	9.5	-23.9, 43.0	
Unknown	-33.9	-62.2, -5.7	*
Non-Smoker	189.3	181.0, 197.7	***
Not Married	-36.8	-43.3, -30.2	***
Birth Year			
2004			
2005	-6.8	-15.1, 1.4	
2006	-16.8	-25.0, -8.7	***
2007	-11.6	-19.7, -3.5	**
2008	-14.3	-22.4, -6.2	***
First Born	-67.6	-73.4, -61.8	***
Male	123.9	118.8, 129.0	***

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

3.1.2 Results Binary Exposure Regression

The next series of models incorporates a binary variable for estimated swine CAFO exposure for mothers residing within 1, 3 or 5km of swine CAFO (yes/no). After incorporating the binary variable, the signs and relative significance of the other variables remained the same as in the baseline regression model (See Table 7). The binary CAFO exposure variables were not statistically significant for 3 and 5km. However, the 1km binary swine CAFO exposure term was statistically significant at a 90% confidence level. This outcome makes sense because the women living nearest to the swine CAFOs are likely to encounter the highest levels of air emissions. The significance level is not very high because swine CAFO emissions vary with the number of head and the waste management practices implemented. A simple binary variable for swine CAFO exposure does not account for the great variation in the number of animals in swine CAFOs (250 to 2 million head) and the radically different approaches to waste management.

Table 7: Binary Swine CAFO Exposure Birth Weight Models.

	1km Binary Model			3km Binary Model			5km Binary Model		
	Coef.	95% C.I.		Coef.	95% C.I.		Coef.	95% C.I.	
(Intercept)	3276.7	3265.4, 3288.1	***	3277.0	3265.8, 3288.7	***	3277.0	3265.5, 3288.6	***
Gestation (wks)									
<=34	-1562.2	-1576.2, -1548.3	***	-1562.0	-1576.2, -1548.3	***	-1562.0	-1576.2, -1548.3	***
35-36	-626.7	-637.8, -615.6	***	-626.7	-637.8, -615.6	***	-626.7	-637.8, -615.6	***
>=37									
Race/ Ethnicity									
NHW									
NHB	-198.4	-205.0, -191.8	***	-198.5	-205.1, -191.9	***	-198.3	-204.9, -191.7	***
HISP	-50.8	-59.8, -41.7	***	-50.8	-59.9, -41.8	***	-51.0	-60.0, -41.9	***
Maternal Education									
<9	-24.8	-37.9, -11.7	***	-24.7	-37.8, -11.6	***	-25.0	-38.1, -11.8	***
9-11	-27.1	-35.5, -18.8	***	-27.1	-35.5, -18.8	***	-27.2	-35.5, -18.8	***
12									
13-15	23.1	16.3, 30.0	***	23.1	16.2, 29.9	***	23.1	16.2, 29.9	***
>15	31.4	22.8, 39.9	***	31.3	22.7, 39.8	***	31.4	22.8, 39.9	***
Maternal Age									
15-19	-32.5	-42.7, -22.4	***	-32.6	-42.7, -22.5	***	-32.6	-42.7, -22.4	***
20-24	-19.1	-26.0, -12.2	***	-19.2	-26.1, -12.3	***	-19.1	-26.0, -12.2	***
25-29									
30-34	10.0	1.9, 18.1	*	10.0	1.9, 18.1	*	10.0	1.9, 18.1	*
35-39	15.4	4.2, 26.7	**	15.4	4.1, 26.6	**	15.4	4.2, 26.6	**
40-44	-5.4	-29.8, 18.9		-5.4	-29.8, 18.9		-5.4	-29.8, 18.9	
Prenatal Care									
First Tri.									
Second Tri.	-13.1	-21.0, -5.3	**	-13.1	-20.9, -5.2	**	-13.1	-20.9, -5.2	**
Third Tri.	-0.2	-18.8, 18.4		0.0	-18.6, 18.6		0.0	-18.6, 18.6	
None	9.6	-23.9, 43.0		9.6	-23.9. 43.0		9.6	-23.8, 43.1	
Unknown	-33.7	-62.0, -5.5	*	-33.6	-61.8, -5.3	*	-33.7	-61.9, -5.4	*
Non-Smoker	189.3	181.0, 197.7	***	189.3	181.0, 197.7	***	189.3	181.0, 197.7	***
Not Married	-36.7	-43.2, -30.2	***	-36.7	-43.2, 30.1	***	-36.7	-43.2, -30.1	***
Birth Year									
2004									
2005	-6.8	-15.1, 1.4		-6.8	-15.1, 1.4		-6.8	-15.1, 1.4	
2006	-16.8	-25.0, -8.6	***	-16.8	-25.0, -8.7	***	-16.8	-25.0, -8.7	***
2007	-11.6	-19.7, -3.5	**	-11.6	-19.7, -3.4	**	-11.6	-19.7, -3.4	**
2008	-14.3	-22.4, -6.2	***	-14.3	-22.4, -6.2	***	-14.3	-22.4, -6.2	***
First Born	-67.6	-73.4, -61.8	***	-67.6	-73.4, -61.8	***	-67.6	-73.4, -61.8	***
Male	123.9	118.8, 129.0	***	123.9	118.8, 129.0	***	123.9	118.8, 129.0	***
Exposure Est.	-12.2	-25.6, 1.3	.	-4.6	-11.0, 1.8		-2.1	-7.4, 3.3	

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

3.1.3 Results Interaction Exposure Regression

The following series of models incorporated an interaction variable for estimated swine CAFO exposure for mothers residing within 1, 3 or 5km of swine CAFO. The binary variables from the prior section were multiplied by the SSLW (hog lbs) of the proximate swine CAFO. In cases where multiple swine CAFOs are in the radius near the mother, the total SSLW of all the facilities were summed to generate a single interaction term. As in the binary models, the signs and significance of the non CAFO variables remained similar to the baseline model. However, the interaction metric for estimated swine CAFO exposure was negatively associated with birth weight and statistically significant at 1, 3, and 5km (See Table 8). The 1km interaction term had the greatest adverse impact on birth weight; however it had the widest 95% confidence interval because the number of mothers residing within 1km of a swine CAFO was exceedingly small. The coefficient for the interaction term was expressed as grams birth weight per 250,000 hog pounds putting birth weight in the context of swine values that actually reflect production.

Table 8: Interaction CAFO Exposure Birth Weight Models.

	1km Interaction Model			3km Interaction Model			5km Interaction Model		
	Coef.	95% C.I.		Coef.	95% C.I.		Coef.	95% C.I.	
(Intercept)	3277.0	3265.2, 3287.9	***	3277.0	3265.8, 3288.6	***	3277.0	3265.9, 3288.7	***
Gestation (wks)									
<=34	-1562.0	-1576.2, -1548.3	***	-1562.0	-1576.2, -1548.3	***	-1562.0	-1576.2, -1548.3	***
35-36	-626.6	-637.7, -615.5	***	-626.6	-637.7, -615.5	***	-626.6	-637.7, -615.5	***
>=37									
Race/ Ethnicity									
NHW									
NHB	-198.3	-204.9, -191.7	***	-198.6	-205.2, -192.0	***	-198.5	-205.1, -191.9	***
HISP	-50.8	-59.8, -41.8	***	-50.6	-59.6, -41.6	***	-50.6	-59.6, -41.6	***
Maternal Education									
<9	-25.0	-38.1, -11.9	***	-24.4	-37.6, -11.2	***	-24.4	-37.5, -11.3	***
9-11	-27.2	-35.5, -18.8	***	-27.0	-35.4, -18.7	***	-27.0	-35.4, -18.7	***
12									
13-15	23.1	16.3, 30.0	***	23.1	16.3, 30.0	***	23.1	16.3, 30.0	***
>15	31.4	22.9, 39.9	***	31.3	22.7, 39.8	***	31.3	22.8, 39.8	***
Maternal Age									
15-19	-32.5	-42.6, -22.4	***	-32.6	-42.7, -22.5	***	-32.6	-42.7, -22.5	***
20-24	-19.1	-26.0, -12.2	***	-19.2	-26.1, -12.3	***	-19.2	-26.1, -12.3	***
25-29									
30-34	10.0	1.8, 18.1	*	10.0	1.9, 18.1	*	10.0	1.9, 18.1	*
35-39	15.4	4.2, 26.6	**	15.5	4.2, 26.7	**	15.5	4.2, 26.7	**
40-44	-5.4	-29.7, 18.9		-5.4	-29.8, 18.9		-5.5	-29.8, 18.9	
Prenatal Care									
First Tri.									
Second Tri.	-13.2	-21.0, -5.3	**	-13.1	-21.0, -5.3	**	-13.1	-20.9, -5.2	**
Third Tri.	-0.2	-18.8, 18.4		0.0	-18.6, 18.6		0.0	-18.6, 18.6	
None	9.5	-24.0, 42.9		9.5	-24.0, 42.8		9.4	-24.0, 42.9	
Unknown	-33.8	-62.0, -5.5	*	-33.6	-61.8, -5.3	*	-33.6	-61.8, -5.3	*
Non-Smoker	189.3	181.0, 197.7	***	189.3	181.0, 197.7	***	189.3	180.9, 197.6	***
Not Married	-36.7	-43.2, -30.2	***	-36.6	-43.2, -30.1	***	-36.6	-43.1, -30.1	***
Birth Year									
2004									
2005	-6.8	-15.1, 1.4		-6.8	-15.1, 1.4		-6.8	-15.1, 1.4	
2006	-16.8	-25.0, -8.7	***	-16.8	-25.0, -8.6	***	-16.8	-25.0, -8.6	***
2007	-11.6	-19.7, -3.5	**	-11.6	-19.7, -3.4	**	-11.6	-19.7, -3.5	**
2008	-14.3	-22.4, -6.2	***	-14.3	-22.4, -6.2	***	-14.3	-22.4, -6.2	***
First Born	-67.6	-73.4, -61.8	***	-67.6	-73.4, -61.8	***	-67.6	-73.4, -61.8	***
Male	123.9	118.8, 129.0	***	123.9	118.8, 129.0	***	123.9	118.8, 129.0	***
Exposure Est. per 250,000 hog lbs	-4.6	-9.8, 0.6	.	-1.1	-2.1, -0.2	*	-0.4	-0.8, -0.04	*

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

3.1.4 Results Exponential Decay Exposure Regression

The final series of models incorporates a decay variable representing estimated maternal exposure to swine CAFO emissions. The value of SSLW for each CAFO was decayed to 5% at 1, 3, and 5km. When mothers were exposed to more than one swine CAFO the total decay values were summed. As in the models incorporating other swine exposure metrics, the signs and significance of the non CAFO variables remained similar to the baseline model. The decay metric for estimated swine CAFO exposure was negatively associated with birth weight and statistically significant at 3 and 5km (See Table 9). However, the decay metric for 1km was not statistically significant. Of the decay values, the 1km exposure metric had the lowest exposure values, had the fewest number of "exposed" individuals, and had the least amount of variation compared to the 3 and 5km models (See Figure 10). Thus these somewhat anomalous statistical results may derive from the small cell size problem associated with the 1km exposure metric.

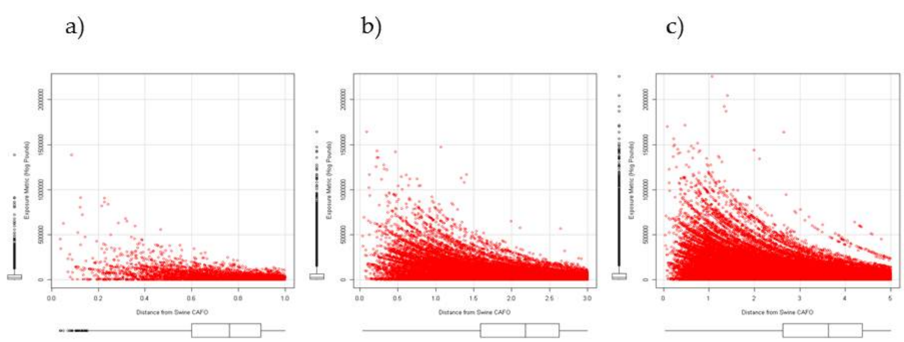

Figure 12: Distance to swine CAFO plotted against decay metric at a)1km, b)3km, c)5km.

Table 9: Exponential Decay CAFO Exposure Birth Weight Models.

	1km Decay Model			3km Decay Model			5km Decay Model		
	Coef.	95% C.I.		Coef.	95% C.I.		Coef.	95% C.I.	
(Intercept)	3276.0	3265.1, 3287.8	***	3277.0	3265.8, 3288.5	***	3277.0	3266.0, 3288.8	***
Gestation (wks)									
<=34	-1562.0	-1576.2, -1548.3	***	-1562.0	-1576.2, -1548.3	***	-1562.0	-1576.1, -1548.3	***
35-36	-626.6	-637.7, -615.5	***	-626.6	-637.7, -615.5	***	-626.6	-637.7, -615.5	***
>=37									
Race/ Ethnicity									
NHW									
NHB	-198.3	-204.8, -191.7	***	-198.6	-205.2, -192.0	***	-198.6	-205.2, -192.0	***
HISP	-50.8	-59.8, -41.8	***	-50.6	-59.6, -41.6	***	-50.6	-59.6, -41.5	***
Maternal Education									
<9	-25.0	-38.1, -11.9	***	-24.5	-37.6, -11.4	***	-24.3	-37.4, -11.2	***
9-11	-27.2	-35.5, -18.8	***	-27.0	-35.4, -18.7	***	-27.0	-35.4, -18.6	***
12									
13-15	23.1	16.3, 30.0	***	23.1	16.3, 30.0	***	23.1	16.3, 30.0	***
>15	31.4	22.9, 39.9	***	31.3	22.7, 39.8	***	31.2	22.7, 39.8	***
Maternal Age									
15-19	-32.5	-42.7, -22.4	***	-32.6	-42.7, -22.5	***	-32.6	-42.7, -22.5	***
20-24	-19.1	-25.9, -12.1	***	-19.1	-26.0, -12.2	***	-19.2	-26.1, -12.3	***
25-29									
30-34	10.0	1.8, 18.1	*	10.0	1.9, 18.1	*	10.0	1.9, 18.1	*
35-39	15.4	4.2, 26.6	**	15.5	4.3, 26.7	**	15.5	4.2, 26.7	**
40-44	-5.4	-29.8, 18.9		-5.4	-29.7, 18.9		-5.4	-29.8, 18.9	
Prenatal Care									
First Tri.									
Second Tri.	-13.2	-21.0, -5.3	**	-13.1	-21.0, -5.3	**	-13.1	-21.0, -5.3	**
Third Tri.	-0.2	-18.8, 18.4		-0.1	-18.7, 18.5		0.0	-18.6, 18.6	
None	9.5	-24.0, 42.9		9.4	-24.0, 42.9		9.4	-24.0, 42.9	
Unknown	-33.9	-62.1, -5.6	*	-33.6	-61.8, -5.3	*	-33.5	-61.8, -5.3	*
Non-Smoker	189.3	181.0, 197.7	***	189.3	181.0, 197.7	***	189.3	180.9, 197.6	***
Not Married	-36.7	-43.2, -30.2	***	-36.6	-43.1, -30.1	***	-36.6	-43.1, -30.1	***
Birth Year									
2004									
2005	-6.9	-15.1, 1.4		-6.8	-15.1, 1.4		-6.8	-15.1, 1.4	
2006	-16.8	-25.0, -8.7	***	-16.8	-25.0, -8.6	***	-16.8	-25.0, -8.6	***
2007	-11.6	-19.7, -3.5	**	-11.6	-19.7, -3.5	**	-11.6	-19.7, -3.5	**
2008	-14.3	-22.4, -6.2	***	-14.3	-22.4, -6.2	***	-14.3	-22.4, -6.2	***
First Born	-67.6	-73.4, -61.8	***	-67.6	-73.4, -61.8	***	-67.6	-73.4, -61.8	***
Male	123.9	118.8, 129.0	***	123.9	118.8, 129.0	***	123.9	118.8, 129.0	***
Exposure Est. per 250,000 hog lbs	-26.0	-57.5, 5.2		-6.5	-11.5, -1.5	*	-2.6	-4.5, -0.6	*

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

3.2 Robustness of Estimated Maternal Exposure to Swine CAFO Air Emissions

The relationship between estimated swine CAFO exposure and birth weight was robust over the metric used and geographic space. Regardless of which swine CAFO exposure metric (binary, interaction or exponential decay) was incorporated into the regression model, they were all associated with a decrease in birth weight and most of these variables were statistically significant (See Table 11). Furthermore, the relationship between swine CAFO exposure and birth weight was consistent over variation in the radii length (1, 3, 5km).

Table 10: Birth Weight and Methods of Estimating Exposure to Swine CAFOs.

Method of Estimating Maternal Swine CAFO Exposure	Coefficient BW(g) per 250,000 hog lbs	95% C.I	
Binary (1km)	-12.2	-25.6, 1.3	.
Binary (3km)	-4.6	-11.0, 1.8	
Binary (5km)	-2.1	-7.4, 3.3	
Interaction (1km)	-4.6	-9.8, 0.6	.
Interaction (3km)	-1.1	-2.1, -0.2	*
Interaction (5km)	-0.4	-0.8, -0.04	*
Exponential Decay (1km)	-26.0	-57.5, 5.2	
Exponential Decay (3km)	-6.5	-11.5, -1.5	*
Exponential Decay (5km)	-2.6	-4.5, -0.6	*

Signif. codes: 0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

4. Conclusions

The regression modeling indicates that maternal exposure to swine CAFO emissions is associated with a statistically significant decrease in birth weight. However, the decrease in birth weight (g) per exposure metric (250,000 hog lbs) was of limited clinical significance.

4.1 Proposed Mechanisms for the Adverse Impact of Swine CAFOs on Birth Weight

Complex emissions from swine CAFOs could potentially decrease birth weight via a variety of mechanisms (See Figure 11). Swine CAFOs generate air emissions from lagoons, field application of waste and directly from barns. Particulates generated from swine CAFOs may be especially biologically active since odorants and bioaerosols agglomerate onto the particles [65]. Despite the initial focus on air emissions, swine CAFO waste could potentially contaminate drinking water. Many individuals living in the Coastal Plain rely on well water. Swine waste has the potential to contaminate well water through lagoon failure and inappropriate field application.

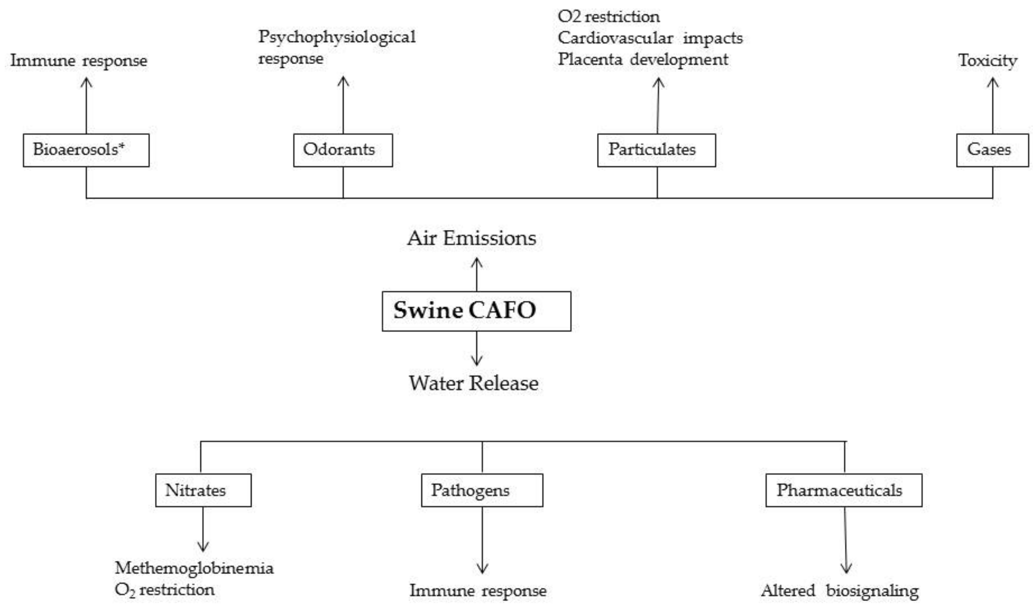

Figure 13: Mechanisms by which swine emissions could decrease birth weight. *Bioaerosols include viable pathogens, endotoxin, and swine dander.

The association of swine CAFOs with a decrease in birth weight could be due to factors other than emissions from the facility itself. While the addition of CAFOs in rural communities slightly increases the average individual income, they are also associated with greater financial disparities [58, 59, 62]. Additionally, swine CAFOs inhibit the social capital of rural communities [2, 8]. Prior studies have found an association between decreased birth weight and communities with increased levels of economic inequality and lack of social support [45]. It is possible that CAFOs drive socioeconomic changes in rural communities which may in turn contribute to the decrease in birth weight.

4.1.1 Mitigating Factors

The association between swine CAFOs and decreased birth weight was statistically significant, but of limited clinical significance. Mitigating maternal factors may have dampened the association of swine CAFOs with decreased birth weight. The population of exposed mothers was generally younger and less educated than unexposed mothers (See Table 5). Most importantly, Hispanic women were the most prominent minority in exposed women, while non-Hispanic black women were the most prominent minority in unexposed women. In regression models, maternal race was one of the most influential variables associated with birth weight. Compared to non-Hispanic white births, non-Hispanic African American race was associated with a decrease of 198.1g decrease in birth weight, while Hispanic ethnicity was associated with a 51.0g decrease in birth weight (See Table 6). Typically Hispanic women have better birth outcomes than non-Hispanic black women, despite economic similarities. The racial/ethnic demographics of the exposed women could mask the relationship between exposure to swine CAFOs and pregnancy outcomes. Stratifying the regression models by race/ethnicity would provide insight to racially disparate associations with birth weight.

4.2 Directions for Future Work

Using data already available to our research group, future work should focus on incorporating temporal variables, racial/ethnic factors and more realistic models of exposure to swine CAFO emissions.

The decay function of estimated swine CAFO air emission exposure could be adjusted to CAFO air emissions fluctuate seasonally. During the summer months (June, July, and August), swine production peaks and CAFOs generate the most air emissions [12, 58, 64]. Additionally, maintenance activities such as waste lagoon agitation or field application significantly increases air emissions over a very short period of time [92]. Air pollution exposure during the first trimester may interfere with the proper development of the placenta restricting nutrient and oxygen delivery to the developing fetus. Air pollution exposure during the third trimester may interfere with weight gain and contribute to preeclampsia, while a sudden severe air event may trigger preterm birth within two weeks of the event [77, 84, 95, 96]. Maternal exposure to high levels of swine CAFO air emissions during vulnerable developmental windows could cause a more significant decrease in birth weight than constant low level exposure. Incorporating a temporal term indicating overlap of peak swine production with windows of developmental sensitivity into the model would refine the conclusions drawn from the regression model.

Hispanic birth outcomes are more similar to NHW than NHB [97, 98]. Additionally, the data set used in this study had a disproportionately high percentage of Hispanic women compared to the Coastal Plain study region and the entire State of North Carolina. Implementing a racially stratified model or using an interaction term (maternal race*CAFO exposure) could elucidate the racial differences in the association of birth weight with exposure to swine CAFOS.

The exponential decay model of maternal exposure to swine CAFO air emissions could be adjusted for the maximum association with birth weight. The radii and the value of k can be tuned for new exponential models of exposure to swine CAFO air emissions. Additionally, the extremely large CAFOs could be weighted to have a greater correlation with decreased birth weight. An element of directionality could also be added to the CAFO exposure metric. The swine CAFO exposure metrics (binary, interaction, and exponential decay) used in this study did not include any directionality. Prevailing wind direction or temporally sensitive wind roses could be used generate an exposure metric that accurately reflects the dispersion of swine CAFO air emissions. Alternatively, if it seems that maternal exposure to CAFO contaminated well water is more strongly associated with decreased birth weight, the exposure metric could be adjusted to be drinking water centric. By linking maternal address to property parcel data, it would be possible to assign a binary value for drinking water source (well or city water) to each mother. Additionally, maps of watersheds and water tables could be obtained from USGS to determine the upstream CAFO burden on each pregnant mother.

4.2.1 Field Sampling and Community Driven Research

Limited field data presents the largest barrier to generating and validating models of human exposure to CAFO emissions. While a few studies have measured concentrations of swine air emissions at specific distances, it is not enough information to generate an accurate model of human exposure to swine CAFO air emissions. However, establishing a trusting community-based research relationship with exposed communities could help clarify the relationship between exposure to swine CAFO air emissions and decreased birth weight. Since pregnant women may spend a significant amount of time outside the home, interviewing mothers about their daily activities would provide insight into their exposure to CAFO air emissions outside of the home. While prior work has made key contacts in the rural African American community, they have not focused on making these connections with the Hispanic community.

A cooperative relationship between researchers and communities impacted by swine farms would be mutually beneficial. Community members can bring overlooked issues to the attention of the academic community and aid in collecting valuable data. On the other hand, researchers could bring national attention to the issues facing communities near CAFOs and implement outreach programs improving the health and wellbeing of rural residents.

4.3 Policy Implications

While this particular study did not find a clinically significant association between birth weight and CAFOs, other studies have indicated that swine CAFOs are associated with chronic human health issues [8]. The crux of the matter is that CAFOs are regulated like small family farms. Regulations need to be specifically tailored to the industrial scale corporate facilities that are the modern CAFOs. Many options exist for minimizing the impact of swine CAFOs on human health. In the case of applying waste to spray fields, the volume of waste must be appropriate to the amount of nitrogen absorbed by crops. Additionally, limiting filed application to the early morning with little wind and rain, air emissions and runoff could be minimized. Decreasing sprayer pressure would reduce the amount of particulates suspended in the air. Taking into account the human factor, it would also be useful to avoid field application of waste during weekends and holidays when nearby resident are more likely to be home. Stricter regulation of hog lagoons would ensure that waste lagoons are a sufficient size for the number of hogs in a specific facility. Requiring lined and covered lagoons could reduce the incidence of watershed contamination. Swine dust emissions from the barns themselves could be addressed by filtering air as it exits the exhaust fans. Reducing the density of swine permitted in a facility would have the dual benefit of decreasing air emissions and improving animal welfare. Most farrowing pens are small to allow the sow to lie down and to turn around. These restrictive pens have recently come under criticism in the United States and are banned in several other countries. Increasing the size of farrowing pens would be more humane for sows and reduce the concentration of animals.

However, the political environment of North Carolina makes it extremely difficult to enforce strict regulations on swine CAFOs. Alternatively, initiatives could be taken to mitigate the impact of swine CAFOs and protect the health of pregnant women in the Coastal Plain region. The North Carolina Agromedicine Institute already has a great deal of experience working with public health issues in rural areas and could extend their programming to include programs for pregnant women[99]. The Southern Extension and Research Activity-37 (SERA-37, the New Hispanic South) is an effort to expand Cooperative Extension's capacity to understand and reach the growing Latino community in the South [100]. SERA-37 could advance pregnancy health and educational opportunities for Latino mothers living in areas of intensive swine production.

4.4 Conclusion

Using ordinary least squares regression modeling, this study demonstrates a statistically but not clinically significant association with exposure to swine CAFOs and a decrease in birth weight. As the metric of estimated maternal exposure to swine CAFO air emissions moved from strictly binary to continuous (from binary to interaction terms to exponential decay), the association between swine CAFO exposure and decreased birth weight was strengthened.

While several other studies have found associations between exposure to swine CAFO air emissions and adverse health outcomes, this study is the first to investigate the correlation between these emissions and decreased birth weight. Prior studies investigating air pollution and low birth weight focus on urban areas and automotive emissions. This analysis sought to understand the relationship between a rural source of air pollution and birth weight. This paper suggests that continued investigation of environmental contributions to pregnancy outcomes in rural areas is warranted.

Appendix A

Exposure to Swine CAFO air emissions (sslw exposure metric)= weight(sslw)exp(-k*dist)

Dist. (km)	Percent emissions remaining	k
1	5%	-2.9957
3	5%	-0.9986
5	5%	-0.5991

References

1. Binkova, B., et al., *Ambient air pollution and pregnancy outcomes: a review of the literature.* Environmental Health Perspectives, 2005. **113**(4): p. 375.

2. Greger, M. and G. Koneswaran *The public health impacts of concentrated animal feeding operations on local communities*. 2010. **33**, 11-20.

3. Cole, D., L. Todd, and S. Wing, *Concentrated Swine Feeding Operations and Public Health: A Review of Occupational and Community Health Effects.* Environmental Health Perspectives, 2000. **108**(8): p. 685.

4. Mirabelli, M.C., et al., *Asthma symptoms among adolescents who attend public schools that are located near confined swine feeding operations.* Pediatrics, 2006. **118**(1): p. 370.

5. Mitloehner, F.M. and M.B. Schenker, *Environmental exposure and health effects from concentrated animal feeding operations.* Epidemiology (Cambridge, Mass.), 2007. **18**(3): p. 309-311.

6. Nimmermark, S., *Odour influence on well-being and health with specific focus on animal production emissions.* Annals of agricultural and environmental medicine : AAEM, 2004. **11**(2): p. 163-173.

7. Schinasi, L., et al., *Air pollution, lung function, and physical symptoms in communities near concentrated Swine feeding operations.* Epidemiology (Cambridge, Mass.), 2011. **22**(2): p. 208-215.

8. Wing, S. and S. Wolf, *Intensive Livestock Operations, Health, and Quality of Life among Eastern North Carolina Residents.* Environmental Health Perspectives, 2000. **108**(3): p. 233-238.

9. National_Agricultural_Statistics_Services. *Hog Production for the State of North Carolina*. 2010; Available from: http://www.nass.usda.gov/QuickStats/PullData_US.jsp.

10. N.C._Division_of_Water_Quality. *Animal Feeding Operations Active Permits*. 2010; Available from: http://portal.ncdenr.org/web/wq/aps/afo/perm.

11. U.S_Census_Bureau. *State and County QuickFacts: North Carolina*. 2010; Available from: http://quickfacts.census.gov/qfd/states/37000.html.

12. Wing, S., et al., *Air Pollution and Odor in Communities near Industrial Swine Operations.* Environmental Health Perspectives, 2008. **116**(10): p. 1362-1368.

13. Bell, M.L. and K. Belanger, *Ambient Air Pollution and Low Birth Weight in Connecticut and Massachusetts.* Environmental Health Perspectives, 2007. **115**(7): p. 1118-1124.

14. Bobak, M., *Outdoor Air Pollution, Low Birth Weight, and Prematurity.* Environmental Health Perspectives, 2000. **108**(2): p. 173-176.

15. Bosetti, C., et al., *Ambient particulate matter and preterm birth or birth weight: a review of the literature.* Archives of toxicology, 2010. **84**(6): p. 447-460.

16. Chen, L., et al., *Air pollution and birth weight in northern Nevada, 1991-1999.* Inhalation toxicology, 2002. **14**(2): p. 141-157.

17. Gehring, U., et al., *Traffic-related air pollution, preterm birth and term birth weight in the PIAMA birth cohort study.* Environmental Research, 2011. **111**(1): p. 125-135.

18. Gouveia, N., S.A. Bremner, and H.M.D. Novaes, *Association between Ambient Air Pollution and Birth Weight in São Paulo, Brazil.* Journal of Epidemiology and Community Health (1979-), 2004. **58**(1): p. 11-17.

19. Ha, E.H., et al., *Is air pollution a risk factor for low birth weight in Seoul?* Epidemiology (Cambridge, Mass.), 2001. **12**(6): p. 643-648.

20. Hansen, C., et al., *Low levels of ambient air pollution during pregnancy and fetal growth among term neonates in Brisbane, Australia.* Environmental Research, 2007. **103**(3): p. 383-389.

21. Liu, S., et al., *Association between Gaseous Ambient Air Pollutants and Adverse Pregnancy Outcomes in Vancouver, Canada.* Environmental Health Perspectives, 2003. **111**(14): p. 1773-1778.

22. Maisonet, M., et al., *A review of the literature on the effects of ambient air pollution on fetal growth.* Environmental Research, 2004. **95**(1): p. 106-115.

23. van den Hooven, E.H., et al., *Residential traffic exposure and pregnancy-related outcomes: a prospective birth cohort study.* Environmental health : a global access science source, 2009. **8**(1): p. 59-59.

24. Gouveia, N., S.A. Bremner, and H.M.D. Novaes, *Association between ambient air pollution and birth weight in Sao Paulo, Brazil.* Journal of Epidemiology and Community Health, 2004. **58**(1): p. 11-17.

25. Ha, E.H., et al., *Is air pollution a risk factor for low birth weight in Seoul?* Epidemiology, 2001. **12**(6): p. 643-648.

26. Leem, J.H., et al., *Exposures to air pollutants during pregnancy and preterm delivery.* Environmental Health Perspectives, 2006. **114**(6): p. 905-910.

27. Ritz, B. and M. Wilhelm, *Ambient air pollution and adverse birth outcomes: Methodologic issues in an emerging field.* Basic & Clinical Pharmacology & Toxicology, 2008. **102**(2): p. 182-190.

28. Shah, P.S., T. Balkhair, and P. Knowledge Synth Grp Determinants, *Air pollution and birth outcomes: A systematic review.* Environment International, 2011. **37**(2): p. 498-516.

29. Wang, X.B., et al., *Association between air pollution and low birth weight: A community-based study.* Environmental Health Perspectives, 1997. **105**(5): p. 514-520.

30. Gray, S.C., S.E. Edwards, and M.L. Miranda, *Assessing exposure metrics for PM and birth weight models.* Journal of exposure science & environmental epidemiology, 2010. **20**(5): p. 469-477.

31. Miranda, M.L., P. Maxson, and S. Edwards, *Environmental contributions to disparities in pregnancy outcomes.* Epidemiologic reviews, 2009. **31**(Journal Article): p. 67.

32. Nethery, E., et al., *From measures to models: an evaluation of air pollution exposure assessment for epidemiological studies of pregnant women.* Occupational and Environmental Medicine, 2008. **65**(9): p. 579-586.

33. Alexander, G.R., et al., *A united states national reference for fetal growth.* Obstetrics & Gynecology, 1996. **87**(2): p. 163-168.

34. Behrman, R.E., et al., *Preterm birth causes, consequences, and prevention: causes, consequences, and prevention*. 2007, Washington, D.C: National Academies Press.

35. Ashdown-Lambert, J.R., *A review of low birth weight: predictors, precursors and morbidity outcomes.* The Journal of the Royal Society for the Promotion of Health, 2005. **125**(2): p. 76-83.

36. Doussard-Roosevelt, J.A., *Vagal Regulation of Heart Rate in the Prediction of Developmental Outcome for Very Low Birth Weight Preterm Infants.* Child Development, 1997. **68**(2): p. 173-86.

37. Hack, M., N.K. Klein, and H.G. Taylor, *Long-Term Developmental Outcomes of Low Birth Weight Infants.* The Future of Children, 1995. **5**(1): p. 176-196.

38. Kuban, K.C. and A. Leviton, *Cerebral palsy.* The New England journal of medicine, 1994. **330**(3): p. 188-195.

39. Avchen, R.N., K.G. Scott, and C.A. Mason, *Birth weight and school-age disabilities: a population-based study: 889.* American journal of epidemiology, 2001. **154**(10): p. 895-901.

40. Lorenz, J.M., et al., *A quantitative review of mortality and developmental disability in extremely premature newborns: 1.* Archives of pediatrics & adolescent medicine, 1998. **152**(5): p. 425-435.

41. Saigal, S., et al., *School-age outcomes in children who were extremely low birth weight from four international population-based cohorts.* Pediatrics, 2003. **112**(4): p. 943-950.

42. Barker, D.J.P., et al., *Growth In Utero And Serum Cholesterol Concentrations In Adult Life.* BMJ: British Medical Journal, 1993. **307**(6918): p. 1524-1527.

43. Weiss, J.L., et al., *Obesity, obstetric complications and cesarean delivery rate--a population-based screening study.* American journal of obstetrics and gynecology, 2004. **190**(4): p. 1091-1097.

44. NationalCenterforHealthStatistics. *Vital Stats*. Centers for Disease Control and Prevention 2006; Available from: http://www.ced.gov/nchs/vitalstats.html.

45. Blumenshine, P., et al., *Socioeconomic disparities in adverse birth outcomes: a systematic review.* American journal of preventive medicine, 2010. **39**(3): p. 263-272.

46. NationalCenterforHealthStatistics. *Natality Public Use Files 1990-2006*. Centers for Disease Control and Prevention 2006; Available from: http://www.cdc.gov/nchs/data_access/Vitalstatsonline.htm.

47. Greg, R.A. and C.K. Carol, *The Role of Prenatal Care in Preventing Low Birth Weight.* The Future of Children, 1995. **5**(1): p. 103-120.

48. Halfon, N., et al., *Preventing low birth weight: is prenatal care the answer?* Journal of Maternal-Fetal and Neonatal Medicine, 2003. **13**(6): p. 362-380.

49. Sandman, C.A., et al., *Psychological Adaptation and Birth Outcomes: The Role of Personal Resources, Stress, and Sociocultural Context in Pregnancy [1].* Health Psychology, 1999. **18**(4): p. 333-345.

50. St-Laurent, J., et al., *Biopsychosocial determinants of pregnancy length and fetal growth.* Paediatric and Perinatal Epidemiology, 2008. **22**(3): p. 240-248.

51. Weck, R.L., T. Paulose, and J.A. Flaws, *Impact of environmnet factors and poverty on pregnancy outcomes.* CLINICAL OBSTETRICS AND GYNECOLOGY, 2008. **51**(2): p. 349-359.

52. Miranda, M.L., P. Maxson, and S. Edwards, *Environmental Contributions to Disparities in Pregnancy Outcomes.* Epidemiologic Reviews, 2009. **31**(1): p. 67-83.

53. Rini, C.K., et al., *Psychological adaptation and birth outcomes: The role of personal resources, stress, and sociocultural context in pregnancy.* Health Psychology, 1999. **18**(4): p. 333-345.

54. St-Laurent, J., et al., *Biopsychosocial determinants of pregnancy length and fetal growth.* Paediatr Perinat Epidemiol, 2008. **22**(3): p. 240-8.

55. Yi, O., H. Kim, and E. Ha, *Does area level socioeconomic status modify the effects of PM10 on preterm delivery?* Environmental Research, 2010. **110**(1): p. 55-61.

56. Zilko, C.E.M., *Economic contraction and birth outcomes: an integrative review.* Human Reproduction Update, 2010. **16**(4): p. 445-458.

57. Furuseth, O.J., *Restructuring of hog farming in North Carolina: Explosion and implosion.* PROFESSIONAL GEOGRAPHER, 1997. **49**(4): p. 391-403.

58. Wing, S., D. Cole, and G. Grant, *Environmental Injustice in North Carolina's Hog Industry.* Environmental Health Perspectives, 2000. **108**(3): p. 225-231.

59. Wilson, S.M., et al., *Environmental injustice and the Mississippi hog industry.* Environmental Health Perspectives, 2002. **110 Suppl 2**(Suppl 2): p. 195-201.

60. Stretesky, P.B., J.E. Johnston, and J. Arney, *Environmental Inequity: An Analysis of Large-Scale Hog Operations in 17 States, 1982–1997.* Rural Sociology, 2003. **68**(2): p. 231-252.

61. Taquino, M., D. Parisi, and D.A. Gill, *Units of Analysis and the Environmental Justice Hypothesis: The Case of Industrial Hog Farms.* Social Science Quarterly, 2002. **83**(1): p. 298-316.

62. Wing, S., et al., *Integrating epidemiology, education, and organizing for environmental justice: community health effects of industrial hog operations.* American journal of public health, 2008. **98**(8): p. 1390-1397.

63. Wilson, S.M. and M.L. Serre, *Use of passive samplers to measure atmospheric ammonia levels in a high-density industrial hog farm area of eastern North Carolina.* ATMOSPHERIC ENVIRONMENT, 2007. **41**(28): p. 6074-6086.

64. Thorne, P.S., A.C. Ansley, and S.S. Perry, *Concentrations of Bioaerosols, Odors, and Hydrogen Sulfide Inside and Downwind from Two Types of Swine Livestock Operations.* Journal of Occupational and Environmental Hygiene, 2009. **6**(4): p. 211-220.

65. Schiffman, S.S., J.L. Bennett, and J.H. Raymer, *Quantification of odors and odorants from swine operations in North Carolina.* Agricultural and Forest Meteorology, 2001. **108**(3): p. 213-240.

66. O'Connor, A.M., et al., *The association between proximity to animal feeding operations and community health: a systematic review.* PloS one, 2010. **5**(3): p. 9530.

67. Schiffman, S.S. and C.M. Williams, *Science of odor as a potential health issue.* Journal of environmental quality, 2005. **34**(1): p. 129-138.

68. Avery, R.C., et al., *Odor from Industrial Hog Farming Operations and Mucosal Immune Function in Neighbors.* Archives of Environmental Health, 2004. **59**(2): p. 101.

69. Schiffman, S.S., et al., *Symptomatic effects of exposure to diluted air sampled from a swine confinement atmosphere on healthy human subjects.* Environmental Health Perspectives, 2005. **113**(5): p. 567.

70. Tajik, M., et al., *Impact of odor from industrial hog operations on daily living activities.* New solutions : a journal of environmental and occupational health policy : NS, 2008. **18**(2): p. 193-205.

71. Sneeringer, S., *Does Animal Feeding Operation Pollution Hurt Public Health A National Longitudinal Study of Health Externalities Identified by Geographic Shifts in Livestock Production.* American Journal of Agricultural Economics, 2009. **91**(1): p. 124-124.

72. Nordby, K.-C., L.M. Irgens, and P. Kristensen, *Immunological exposures in Norwegian agriculture and pre-eclampsia.* Paediatric and Perinatal Epidemiology, 2006. **20**(6): p. 462-470.

73. Ruckhaberle, K.E. and L. Baumann, *INFECTIONS AND PREMATURE DELIVERY.* ARCHIVES OF GYNECOLOGY AND OBSTETRICS, 1991. **250**(1-4): p. 690-695.

74. Murphy, D.J., *Epidemiology and environmental factors in preterm labour.* Best practice & research. Clinical obstetrics & gynaecology, 2007. **21**(5): p. 773-789.

75. Bobak, M. and D.A. Leon, *Pregnancy Outcomes and Outdoor Air Pollution: An Ecological Study in Districts of the Czech Republic 1986-8.* Occupational and Environmental Medicine, 1999. **56**(8): p. 539-543.

76. Brauer, M., *A cohort study of traffic related air-pollution impacts on birth outcomes (vol 116, pg 680, 2008).* Environmental Health Perspectives, 2008. **116**(12): p. A519-A519.

77. Darrow, L.A., et al., *Ambient air pollution and preterm birth: a time-series analysis.* Epidemiology (Cambridge, Mass.), 2009. **20**(5): p. 689-698.

78. Gehring, U., et al., *Traffic-related air pollution and pregnancy outcomes in the Dutch ABCD birth cohort study.* Occupational and Environmental Medicine, 2011. **68**(1): p. 36-43.

79. Jiang, L.L., et al., *A time series analysis of outdoor air pollution and preterm birth in Shanghai, China.* BIOMEDICAL AND ENVIRONMENTAL SCIENCES, 2007. **20**(5): p. 426-431.

80. Leem, J.-H., et al., *Exposures to air pollutants during pregnancy and preterm delivery.* Environmental Health Perspectives, 2006. **114**(6): p. 905-910.

81. Llop, S., et al., *Preterm birth and exposure to air pollutants during pregnancy.* Environmental Research, 2010. **110**(8): p. 778-785.

82. Nordentoft, M., et al., *Intrauterine growth retardation and premature delivery: the influence of maternal smoking and psychosocial factors.* American journal of public health, 1996. **86**(3): p. 347-354.

83. Rogers, J.F., et al., *Association of very low birth weight with exposures to environmental sulfur dioxide and total suspended particulates.* American journal of epidemiology, 2000. **151**(6): p. 602-613.

84. Sagiv, S.K., et al., *A time-series analysis of air pollution and preterm birth in Pennsylvania, 1997-2001.* Environmental Health Perspectives, 2005. **113**(5): p. 602-606.

85. Wilhelm, M. and B. Ritz, *Residential proximity to traffic and adverse birth outcomes in Los Angeles county, California, 1994-1996.* Environmental Health Perspectives, 2003. **111**(2): p. 207-216.

86. Yorifuji, T., et al., *Residential proximity to major roads and preterm births.* Epidemiology (Cambridge, Mass.), 2011. **22**(1): p. 74-80.

87. Dorman, D.C., et al., *Fertility and developmental neurotoxicity effects of inhaled hydrogen sulfide in Sprague-Dawley rats.* Neurotoxicology and Teratology, 2000. **22**(1): p. 71-84.

88. Faas, M.M. and G.A. Schuiling, *Pre-eclampsia and the inflammatory response.* European Journal of Obstetrics & Gynecology and Reproductive Biology, 2001. **95**(2): p. 213-217.

89. Patel, P., et al., *The endogenous production of hydrogen sulphide in intrauterine tissues.* Reproductive biology and endocrinology : RB&E, 2009. **7**(1): p. 10-10.

90. Sjörgren, B., et al., *Increase in interleukin-6 and fibrinogen in peripheral blood after swine dust inhalation.* Scandinavian journal of work, environment & health, 1999. **25**(1): p. 39-41.

91. Mirabelli, M.C., et al., *Race, poverty, and potential exposure of middle-school students to air emissions from confined swine feeding operations.* Environmental Health Perspectives, 2006. **114**(4): p. 591.

92. The University of Iowa, a.I.S.U., *Iowa Concentrated Animal Feeding Operations Air Quality Study: Final Report*, 2002. p. 1-221.

93. Wilson, S.M. and M.L. Serre, *Examination of atmospheric ammonia levels near hog CAFOs, homes, and schools in Eastern North Carolina.* Atmospheric Environment, 2007. **41**(23): p. 4977-4987.

94. Swamy, G.K., et al., *Maternal age, birth order, and race: differential effects on birthweight.* Journal of epidemiology and community health, 2012. **66**(2): p. 136-142.

95. Lee, B.E., et al., *Exposure to air pollution during different gestational phases contributes to risks of low birth weight.* Human reproduction (Oxford, England), 2003. **18**(3): p. 638-643.

96. Mohorovic, L., *First two months of pregnancy--critical time for preterm delivery and low birthweight caused by adverse effects of coal combustion toxics.* Early human development, 2004. **80**(2): p. 115-123.

97. Bruckner, T.A., et al., *From paradox to disparity: trends in neonatal death in very low birth weight non-Hispanic black and white infants, 1989-2004.* The Journal of pediatrics, 2009. **155**(4): p. 482-487.e1.

98. Chung, J.H., et al., *Ethnic differences in birth weight by gestational age: at least a partial explanation for the Hispanic epidemiologic paradox?* American journal of obstetrics and gynecology, 2003. **189**(4): p. 1058-1062.

99. *North Carolina Agromedicine Institute*. 2012 [cited 2012; Available from: http://www.ecu.edu/cs-dhs/agromedicine/.

100. SERA-37. *The New Hispanic South*. 2008; Available from: http://sera37.wordpress.com/.