

University of Louisville

ThinkIR: The University of Louisville's Institutional Repository

Electronic Theses and Dissertations

5-2014

Coal ash and children's sleep : a community-based study.

Clara G. Sears

University of Louisville

Follow this and additional works at: <https://ir.library.louisville.edu/etd>



Part of the [Epidemiology Commons](#)

Recommended Citation

Sears, Clara G., "Coal ash and children's sleep : a community-based study." (2014). *Electronic Theses and Dissertations*. Paper 1294.

<https://doi.org/10.18297/etd/1294>

This Master's Thesis is brought to you for free and open access by ThinkIR: The University of Louisville's Institutional Repository. It has been accepted for inclusion in Electronic Theses and Dissertations by an authorized administrator of ThinkIR: The University of Louisville's Institutional Repository. This title appears here courtesy of the author, who has retained all other copyrights. For more information, please contact thinkir@louisville.edu.

COAL ASH AND CHILDREN'S SLEEP: A COMMUNITY-BASED STUDY

By

Clara G. Sears
B.A., Goshen College, 2012

A Thesis
Submitted to the Faculty of the
School of Public Health and Information Sciences
In Partial Fulfillment of the Requirements
for the Degree of

Master of Sciences

Department of Epidemiology and Population Health
University of Louisville
Louisville, Kentucky

May 2014

COAL ASH AND CHILDREN'S SLEEP: A COMMUNITY-BASED STUDY

By

Clara G. Sears
B.A., Goshen College, 2012

A Thesis Approved on

April 15, 2014

by the following Thesis Committee:

Dr. Kristina M Zierold, PhD, MS

Dr. Guy Brock, PhD

Dr. Barbara Polivka, PhD, RN

DEDICATION

This thesis is dedicated to

Dr. James Miller

Your persistence, encouragement, and humor will never be forgotten.

ACKNOWLEDGEMENT

I would like to thank Dr. Kristina Zierold, for her guidance and dedication to the community. I would also like to thank Dr. Zierold, Dr. Guy Brock and Dr. Barbara Polivka for serving on my committee and providing thoughtful feedback and insight. Thank you to Dr. Yolanda Yoder and all of the physicians and staff at Comprehensive Health Care for allowing me to come into the clinic.

ABSTRACT

COAL ASH AND CHILDREN'S SLEEP: A COMMUNITY-BASED STUDY

Clara G. Sears

April 15, 2014

Kentucky is the fifth largest producer of coal ash, a by-product of coal combustion. The small spherical coal ash particles contain heavy metals like arsenic, lead, mercury, and cadmium. Coal ash is currently classified as non-hazardous by the EPA, which allows it to be stored in open-air impoundments near low-income communities. The primary object of the study is to determine the prevalence of sleep disruptive behaviors in children exposed to coal ash, compared to a group of demographically similar non-exposed children. Parents or guardians from five neighborhoods surrounding a coal ash storage facility, and one non-exposed community, participated in a cross-sectional survey about the health and sleep of children living in their home. Delay in sleep onset ($p= 0.007$), frequent night awakenings ($p= 0.0001$), teeth grinding ($p= 0.03$), lip smacking ($p= 0.006$), snoring ($p= 0.002$), and complaint of leg cramps while resting ($p= 0.0004$) were significantly greater in the exposed group compared to the non-exposed group. When controlling for both health and environmental factors, the odds of frequent night awakenings were significantly greater in the exposed group compared to the non-exposed group (OR= 6.9, CI= 2.2-21). It is important to further evaluate the association between frequent night awakenings and coal ash

exposure because of the potential long-term cognitive and biological impacts on children.

TABLE OF CONTENTS

ACKNOWLEDGEMENT.....	iv
ABSTRACT.....	v
LIST OF TABLES.....	viii
I. INTRODUCTION.....	1
II. OBJECTIVES AND AIMS.....	3
III. LITERATURE REVIEW.....	5
1. Air Pollution Particulate Matter.....	5
2. Background Information on Coal Ash.....	7
3. Coal Ash Exposure and Potential Impact on Children's Sleep.....	11
4. EPA Policy.....	14
5. Kentucky Coal Ash.....	15
6. Description of Cane Run Station.....	16
IV. METHODS.....	19
V. RESULTS.....	30
VI. DISCUSSION.....	38
VII. CONCLUSION.....	44
REFERENCES.....	46
CURRICULUM VITA.....	51

LIST OF TABLES

1. Sleep Disruptive Behavior Variables.....	25
2. Potential Covariates.....	26
3. Logistic Regression Covariates.....	29
4. Demographics and General Behaviors (Ages 4-17).....	31
5. Diseases, Disorders, and Illnesses Reported.....	33
6. Activities within One Hour of Bedtime.....	34
7. Sleep Disruptive Behaviors.....	35
8. Sleep Disruptive Behavior Odds Ratios for Exposed vs. Non-exposed.....	36

I. INTRODUCTION

Coal ash, the waste product generated from burning coal, has varying concentrations of minerals and heavy metals including iron, arsenic, lead, mercury, and cadmium. Coal ash is comprised of fine spherical particles that can have diameters $< 10\mu\text{m}$. The current storage methods of coal ash allow the particles to contaminate surrounding impoverished communities through ambient air pollution [1]. The EPA estimates that there are approximately 300 coal ash landfills and 584 ash ponds in use, although the actual number may be much greater [2]. There are 45 units storing slurred, or wet, coal combustion residual that have been labeled “high hazard” meaning that failure of the structure could result in “loss of life, or serious damage to houses, industrial or commercial buildings, important public utilities, main highways or major railroads”. Of these 45 high hazard slurry units, eight are located within Kentucky, and two are in Louisville [3]. In addition to the storage ponds, there are two coal ash landfills in Louisville that have been found to pollute surrounding neighborhoods with fugitive dust emissions.

The chronic effect of stack coal ash on children has been evaluated in few studies, but no studies have specifically evaluated community concern to landfill ash. Multiple studies have found significant health outcomes including damage to internal organs, respiratory conditions, sleep-disruptive breathing and

chronic respiratory conditions related to exposure to similar fine particles, ambient air pollution, smoke, heavy metals and fibrous minerals [4-7].

Children's exposure to coal ash particles in ambient air is of particular concern. Children have higher rates of respiration relative to adults, which increases exposure to ambient air pollution, including coal ash particles, and elevates risk of adverse health effects [8]. Furthermore, childhood exposure to particles containing heavy metals can effect brain development and have long-term neurological implications [9]. The structure and composition of coal ash particles has created considerable concern in communities living near coal ash storage facilities.

II. OBJECTIVES AND AIMS

The primary object of the study is to determine the prevalence of sleep disruptive behaviors in children exposed to coal ash, compared to a group of demographically similar non-exposed children. Sleep disruptive disorders are defined as: delay or disruption in sleep/wake cycle, loud snoring, teeth grinding, sleep talking, or disruptive movements while sleeping.

The specific aims of the proposed study are:

Specific Aim 1: To determine the prevalence of sleep disruptive behaviors and identify potential covariates including bedtime routine, health, gender, and age, in a sample of children exposed to coal ash and a sample of non-exposed children.

Subaim 1a: Evaluate by questionnaire delay in sleep onset, frequent night awakenings, teeth grinding, leg jerking, head rolling, lip smacking, hand flapping, twitching, sleep walking, sleep talking, snoring, and complaint of leg cramps in children exposed to coal ash and non-exposed children.

Subaim 1b: Develop a composite score for sleep disruptive behaviors outlined in subaim 1a. Ordinal variables will be assessed as dichotomous and a numeric value will be assigned based on the number of sleep disruptive behaviors reported.

Subaim1c: Evaluate by questionnaire potential covariates including use of TV, video games, computer, cell phone, caffeine use, and health score

reported in children exposed to coal ash and non-exposed children. The health score is the number of illnesses reported that could potentially impact sleep.

Specific Aim 2: To evaluate the association between coal ash exposure and sleep disruptive behaviors controlling for potential confounders and covariates.

Subaim 2a: Use Wilcoxon Rank-sum test to assess the relationship between sleep disruptive behavior composite score and coal ash exposure (non-exposed or exposed).

Subaim 2b: Use logistic regression to assess the relationship between individual sleep disruptive behaviors and coal ash exposure (non-exposed or exposed) while adjusting for covariates in four different models.

It is hypothesized that children exposed to coal ash report a higher prevalence of each individual sleep disruptive behavior and have a greater sleep disruptive behavior composite score, compared to non-exposed children.

III. LITERATURE REVIEW

1. Air Pollution Particulate Matter

Any fumes or particles suspended in the ambient air are classified as particulate air pollution. Particulates can be produced through industrial or natural process. There are two types of particulate matter, primary and secondary. Primary particulate matter is a direct result of an industrial or natural process. One example of a process producing primary particles is emissions from smoke stacks. After the primary particulate matter is produced, additional chemical processes occur naturally as the particles travel through the atmosphere, yielding secondary particulate matter [10].

Particles are classified by aerodynamic properties. According to the EPA, particles with a diameter of 10 μm or less are inhalable. Particle ranging from 2.5 μm to 10 μm are considered coarse particles while particles with a diameter less than 2.5 μm are classified as fine particles. Ultrafine particles are a subset of fine particles with a diameter less than 0.1 μm . A majority of coarse particles are generated naturally from soils and other particles of the Earth's crust. Fine particles are mainly produced from combustion processes. These particles are able to absorb chemicals from the surrounding environment and attach them to their surfaces often generating secondary particles of sulfates and nitrates [10-12].

Both coarse particles and fine particles have the ability to cause damage to human tissue when inhaled; however, fine particles are able to travel deeper into alveolar-gas exchange regions of the lung and pass through blood vessels to potentially affect various organ systems [10, 11]. Many of the early epidemiological studies conducted evaluated the short-term effects of exposure to air particulate matter. Overall, these studies found that short-term exposure was associated with increased risk of lower respiratory symptoms, decreased lung function, and chronic cough [12].

One of the first studies to evaluate the association between long-term exposure to particulate air pollution and health effects was the Harvard Six-Cities study. This was a prospective cohort study initiated in 1974 that estimated the effects of air pollution on mortality while controlling for smoking status, sex, age and additional risk factors. Results indicated that mortality rate was strongly associated with levels of fine particles in air pollution [13].

The EPA notes that children are among the population of people especially vulnerable to adverse health effects from inhalation of coarse and fine particles [11]. Studies have found that due to children's increased respiration rate and smaller lung size, children inhale a larger dose of particulate matter compared to adults. An early study conducted in Utah by Pope III et. al. (1992), evaluated the association between levels of particulate matter air pollution and respiratory effects in symptomatic and asymptomatic children. Over the course of about 3 months, children recorded their daily peak expiratory flow and symptoms. Level of air particulate matter was measured at two central sites

within a 4 km distance of all participants' homes. Findings indicate a significant negative association between peak expiratory flow and level of air particulate matter. In addition, increased levels of particulate matter were associated with lower respiratory symptoms in both asymptomatic and symptomatic children [8].

2. Background Information on Coal Ash

a. Coal Combustion Products Overview

Coal combustion used to produce electricity creates by-products known as coal combustion products (CCP) through a process called condense-volatilization. During this process, trace elements initially found in the coal are not released, but instead concentrated into small coal ash particles [14]. The concentration of trace elements including nickel, vanadium, arsenic, beryllium, cadmium, copper, zinc, lead, mercury, selenium, radon and molybdenum varies based on the amount originally contained in the coal [1, 15].

Coal ash comprises 60% of all generated CCPs. Around 20% of coal ash generated is bottom ash, a noncombustible ash that has a large particle size and remains in the bottom of the coal combustion furnace. A portion of the bottom ash (40%) is removed from the furnace, and used in wallboard, concrete, and agriculture. The remaining portion of the bottom ash is stored in landfills and holding ponds. Fly ash, the most predominate form of coal ash (80%), is composed of combustible material that is carried by a flue gas stream until it cools and condenses. Once it has condensed, it forms glassy spherules, known as cenospheres [15]. Studies have found that the exact size of fly ash particles varies, but the average range of respirable particles is between 1.98 and 5.64

μm . The spherical shape is provided by a glass matrix composed mostly of silicon, aluminum, iron, cadmium and oxygen [1].

Brown et al. (2011) conducted a study in the United Kingdom evaluating the mineralogy, trace elements, and microstructure of coal ash. Samples of coal fly ash were collected from multiple coal-burning power facilities in Poland, China, and the United Kingdom with the objective of expanding the current understanding of the geochemistry and structure of coal fly ash. It was found that the most profuse minerals in coal fly ash are quartz, mullite, and hermatite with the exact concentration of each mineral varying by location. The mullite composition of a particle depends heavily on the cooling process that occurs while being carried by the flue gas stream after combustion. As the mullite cools, it is recrystallized into fibers that are approximately 5 μm in length and 0.5-1 μm in width. At lower temperatures as the particle continues to cool, the mullite is surrounded by a quartz structure. This mineral composition and configuration is similar to other fibrous minerals, such as asbestos and zeolite erionite, that have been linked to serious health implications in the past. Brown et al. concludes that mullite fibers in coal ash particles are small enough to cause damage to the tracheo-bronchial and broncho-alveolar regions in the lungs based on the size and structure [1].

Brown et al. (2011) finding is supported by research conducted in animal models. Smith et al. (2012) exposed rats to coal ash particles suspended in air through the nasal inhalation only. After exposure to an occupationally relevant dose of coal ash for four hours over a three-day period, the rats accrued 32 μg of

coal ash per rat, of which 25% was found in the head, 20% was in the tracheobronchial region and 50% was found in the pulmonary tissue. Exposure to coal ash significantly increased neutrophils in blood, lung tissue, and bronchoalveolar lavage fluid. There was also a significant increase in macrophages found in the bronchoalveolar lavage fluid [16].

The Brown et al. (2011) conclusion that the small particle size of coal fly ash has potential to cause damage to lung and esophageal tissue is supported by the United States Environmental Protection Agency (EPA) assessment of potentially hazardous particle matter [1, 11]. The range in size of the coal fly ash falls within the range of inhalable particles defined by the EPA.

b. Metal Concentration of Coal Ash

The concentration of metals in fly ash particles increases the potential for environmental contamination from leached trace elements. Leachability of metals from fly ash particles can be affected by the pH of the surrounding environment and availability of metals in the fly ash particle matrix [14]. Flues (2013) found that the availability of metals in fly ash particles was different from the total concentration of metals. Availability refers to the concentration of metals that could contaminate soil and water due to extraction from a leaching agent. The availability of metals in coal ash particles varied from that of coal. A majority of metals, including cadmium and aluminum, had higher availability in coal than coal ash; however, coal ash contained higher availability of arsenic and molybdenum. The most available metals (greater than 40%) in the ash particles

were arsenic, cadmium, and molybdenum. Arsenic in particular was found to have a higher availability in coal ash particles than coal [14].

The leaching of available toxic metals was of particular concern after the failure of a storage dike-pond that occurred at the Tennessee Valley Authority Kingston Fossil plant in December of 2008. Over 4.1 million cubic meters of coal fly ash was released into the surrounding area covering 300 acres and contaminating the Emory River. A majority of the fly ash removal was completed by high intensity dredging which has potential for increasing release of toxic metals from fly ash particles [17]. Before dredging, Bednar et. al. (2013) analyzed water samples from the Kingston site and found toxic metals including arsenic, selenium, barium, manganese, and molybdenum. Dredging was not found to increase the total and reduced levels of these metals, but it did not completely remove them [18].

c. Use and Storage of Coal Ash

Sixty percent of the bottom ash and fly ash produced in the United States is stored in piles, landfills, or holding ponds [15]. These open forms of storage allow the fine fly ash particles to be re-suspended into the ambient air, creating significant fugitive dust emissions. In the Clean Air Act, the EPA defines these emissions as those that cannot “reasonably pass through a stack, chimney, vent, or other functionally-equivalent opening” [19]. Therefore, a fugitive emission can be any particulate matter, liquid, or gases emitted by a facility that is not confined [19]. As these particles travel through the air, they can also attract new surface particles such as polycyclic aromatic hydrocarbons (PAHs) [1].

3. Coal Ash Exposure and Potential Impact on Children's Sleep

The effect of chronic coal ash inhalation on children has not been well studied; however, studies evaluating children's exposure to particles of similar structure and concentrations of heavy metals raise concerns applicable to coal ash particle exposure.

After coal ash particles are inhaled, they are deposited in the lungs and able to alter immunological mechanisms. The particles build up and leach genotoxic compounds that activate macrophages and epithelial cells in the alveolar. This immune response creates elevated levels of inflammatory markers, cytokines, and reactive oxygen species that induce fibrosis [20]. After particulate matter accumulates in the lungs, it is able to penetrate into the capillaries, enter the bloodstream, and impact biological mechanisms beyond the respiratory system. Chronic exposure to air pollution and particulate matter has been found to cause chronic inflammation and elevated levels of cytokines in the body and brain increasing the risk for central nervous system (CNS) disease [21, 22].

In experimental models, fine and ultrafine particulate matter is able to pass directly through the nasal olfactory pathway into the circulatory system and brain. It is unclear exactly how the fine particulate matter damages the CNS once circulating in the body [21,22]. One hypothesis is that the large ratio of surface to volume allows the particles to infiltrate cell membranes, explaining how the particulate matter is able to pass through the lung tissue and the blood-brain barrier. The ability for the fine particulate matter to transverse the blood-brain barrier means that any surface components of the particulate matter also has

access into the brain and bloodstream, this is referred to as the Trojan Horse Effect [21]. In regards to coal ash particles specifically, the Trojan Horse Effect could potentially allow high concentrations of heavy metals access to the brain.

Lead is one of the more studied heavy metals in coal ash that would be able to penetrate the blood-brain barrier. Exposure to lead has been found to alter process of the suprachiasmatic nucleus (SCN) of the hypothalamus. Chronic lead exposure during development of the nervous system can induce cell apoptosis resulting in altered neurogenesis and morphology of the hippocampus. The SCN in the hypothalamus maintains the circadian rhythm in the body's tissues, which regulates the sleep/wake cycle. A study conducted in Wistar rats determined that gestational exposure to lead resulted in morphological alterations to the SCN and abnormal circadian pacemaker cells [23].

Due to increasing focus on the effects of particulate matter on the CNS, more recent studies have evaluated the association between sleep disruptive behaviors and exposure to air particulate matter. Abou-Khadra (2013) conducted a cross-sectional study in Egypt to evaluate the association between particulate matter with a dynamic diameter less than 10 μm (PM_{10}) and sleep disturbances in children. The study population was obtained from two different locations, one that was highly polluted by residential and industrial emissions, and one that was less polluted. Parents of children completed a questionnaire regarding their child's sleep behaviors and other demographic information. Sleep was assessed by questions on initiation and maintenance of sleep, sleep breathing and arousal

disorders, disorders of excessive sleep, and sleep hyperhidrosis. Average daily concentration of PM₁₀ exposure was collected from monitoring stations in each location. Using a generalized additive model and controlling for covariates, a statistically significant association was found between disorders of sleep initiation and maintenance and PM₁₀ levels (p= 0.012) [24].

Kheirandish-Gozal et. al (2013) conducted a study in Iran to assess the prevalence of habitual snoring (HS) in children and the potential contribution of air pollution. It was hypothesized that inflammatory process in the upper airway and adenotonsillar tissue may contribute to HS. A questionnaire was used to assess risk factors for HS, which was defined as loud snoring more than three times a week. The survey was administered to children living in five different areas of Tehran. In total, 4,322 questionnaires were completed (response rate 72%). The association between HS and air pollution was assessed using multivariate logistic regression. Age, gender, socioeconomic factors, and clinical features were considered as covariates in the model. Findings indicated that the prevalence of HS was significantly higher in areas with poorer air quality compared to areas with less air pollution (24.5 % vs. 7.2%; RR: 3.49; 95% CI: 2.67-6.69; p<0.0001) [25].

The impact of coal ash on sleep disruptive behaviors is unknown. Findings from studies evaluating the impact of air pollution on sleep disruptive behaviors demonstrate that fine particulate matter exposure can have negative impacts on biological mechanisms that effect sleep. These results raise

concerns about the potential impact coal ash particles could have sleep due to their dynamic diameter and heavy metal composition.

4. EPA Policy

Subtitle D of the Resource Conservation and Recovery Act (RCRA), from 1976 (Law94-580, 1976) classifies coal ash as a non-hazardous solid waste, which allows disposal of coal ash to occur in open-air impoundments and landfills. Based on this classification, the federal government does not regulate coal ash, instead each state is responsible for regulation [2, 26].

In 2010, the EPA proposed a new rule for the storage and disposal of coal ash suggesting two possible options. One of the proposed options calls for reclassification of CCP as hazardous under Subtitle C of the RCRA [2, 26]. The EPA defines hazardous waste as “waste with properties that make it dangerous or potentially harmful to human health or the environment” [27]. This revised classification would require that all landfills have ongoing inspections and permits [26]. The second proposed option would allow CCP to persist under Subtitle D as a non-hazardous material, but develop national standards for constructing and monitoring storage facilities. These standards would regulate location of storage facilities, composite liner requirements, groundwater monitoring, and action plans to address emissions from the unit. Any storage facility that failed to meet these regulations would be classified as an open dump and therefore prohibited under the RCRA [2].

A verdict on the 2010 proposed rule regarding reclassification of CCP or storage regulations has yet to be reached and the current regulation for coal ash

storage from the 1976 RCRA remains. One reason for the delay in the decision is that re-classification of CCP as hazardous waste would have far reaching ramifications. One ramification would be that classification as a hazardous waste would introduce a stigma surrounding CCP directly impacting all products that reuse CCP. Despite the current understanding that the reuse of CCP in products is not hazardous, according to the EPA re-classification of CCP as hazardous would, “discourage purchase and re-use of the waste” [26]. Reduction in sales of CCP waste for products would increase the amount of waste and cost of storage. Classifying CCP as hazardous would also allow citizens to take legal action against any facility violating the stricter regulations of Subtitle C [26].

Until a final decision on the rule has been made, the state regulations have a major influence on safety measure taken at storage facilities. In 2000, the EPA stated, “Given that states have been diligent in expanding and upgrading programs for surface impoundments and landfills, we believe they will be similarly responsive in addressing environmental concerns arising from this emerging practice” [26].

5. Kentucky Coal Ash

Kentucky produces approximately 9 million tons of coal ash per year, which is the fifth highest in the nation. Of Kentucky’s 43 coal ash ponds, 21 are over 25 feet tall or contain over 500 acre-feet of coal ash [28]. Current state regulations exempt coal combustion waste from being a hazardous waste, but classify it as a special waste. This allows the waste to be used as ingredients in manufacturing products like cement, concrete, structural fill, and roofing granules

[29]. Current state regulations do not require groundwater monitoring around ponds or landfills, composite liners for ponds or landfills, financial assurance for ponds or landfills, or an emergency action plan. Furthermore, coal ash ponds and landfills are not prohibited from being constructed in the water table. Due to these lack of regulations, it is estimated that 100% of arsenic, chromium, and mercury toxic waste released in the land is from coal ash disposal [28].

In February of 2012, Kentucky Representative Joni Jenkins of House District 44 introduced House Bill 404 that would declassify coal combustion waste as “special waste” and require liners, groundwater monitoring, toxic substance monitoring, and emergency action plans for impoundments that the EPA has designated as “high hazard”. The bill went to the Natural Resources & Environmental committee where it quickly died [30]. The Kentucky Chamber of Commerce issued a 2012 General assembly report for business in which it was noted that the HB 404 would have increased electric rates by classifying coal ash as hazardous waste, which would be bad for business [31]. Even if the EPA reaches a verdict by the January 29, 2013 deadline, *The Courier Journal* reports that, “Pending legislation supported by many in the Kentucky congressional delegation would largely shut the EPA out of regulating coal-burning wastes, leaving it to the states” [32].

6. Description of Cane Run Station

The Cane Run station is located in southwest Louisville, and is owned and operated by LG&E and KU Energy, LLC, which is a PPL Corporation. The power generating station was opened in fall of 1954, and currently operates 3 units,

constructed 1962-1969, and 5 newer combustion turbines. Cane Run Station occupies 510 acres and burns 1.3 million tons of coal each year. The coal is primarily high sulfur transported from western Kentucky and southern Indiana [33, 34].

The Cane Run Station has one landfill and one slurry pond for the storage of coal ash, but LG&E has identified 9 potential sources for fugitive dust and odor emissions [35]. In 2011 and 2013, LG&E was fined \$22,500 and \$113,250, respectively, for blowing ash and odors into nearby residential neighborhoods [36]. In response to a 2013 violation, LG&E submitted an emission control plan to the Air Pollution Control District. This document outlined potential sources of fugitive dust and odors, measures established to reduce emissions, and plans to control emissions [35].

The first unit identified as a potential emission source is the Unit 4/5 Sludge Processing Plant (SPP) on the southwest side of the station. A blower system delivers coal ash to the SPP from a bin located by the coal-fired boilers. Measures taken to reduce fugitive emissions from the SPP include a wet cyclone dust collector and a filtering system [35].

The second unit identified is a material storage yard that stores bituminous coal in the middle of the station. Lime, soda ash, and coal ash are delivered to the storage yard via pneumatic tanker trailer and blown into silos. Bag-houses are in place to reduce dust emissions during transfer to silos [35].

The third unit identified is the landfill that stores Poz-o-tec. Poz-o-tec contains calcium sulfite, sulfate, fly ash, and fixation lime. This lightweight

concrete mixture is transferred from the SPP to the landfill via excavator and dump truck. The landfill was designed to comply with Kentucky Division Waste Management structural requirements [35].

The fourth and fifth source of emissions recognized are unpaved and paved roads. Dry road conditions and material released during vehicle traffic may create fugitive dust emissions. In order to reduce emissions from these sources, LG&E has limited traffic to contractors and employees [35].

The sixth unit identified is a fly ash transfer line that runs both below and above the ground. “A recent dust event” was caused due to a break in the line located above ground [35].

The seventh and eighth possible fugitive dust emission sources noted are due to maintenance and construction activities. The three Generating units undergo routine maintenance, involving balancing the fans, multiple times a year. During maintenance fly ash can become dislodged and carried out of the stack into the ambient air. Ongoing construction increases traffic, hauling of materials, and excavating which may increase emission depending on the specific activity. LG&E recognizes the ash pond, e-pond, south basin, and east ditch as the ninth source that is able to produce odors due to “build-up of bacteria” [35].

IV. METHODS

This ongoing community-based mixed-method study was initiated by Dr. Kristina Zierold, PhD, MS in Fall of 2011. Institutional Review Board approval for this study was obtained from the University of Louisville.

1. Methods Background

a. Population

Approximately 1,600 adults reside in the neighborhoods included in the study. The communities in this study reside in two zip codes. According to the 2010 census, 26,465 people reside in the Cane Run neighborhood zip code region; 27.4% of the population is 18 years of age or under and 84.9% is white. Approximately 86.5% are a high school graduate or higher, and 12.5% live below the poverty line. The average household size is 2.54 [37].

The population of the Riverside Gardens zip code region is 40,746; 26.3% of which is 18 years old or younger and 63% is white. Approximately 82.2% of the population is a high school graduate or higher and 17.7% live below the poverty line. The average household size in this zip code region is 2.41 [37].

b. Initial “community leaders” recruitment

In September 2011, the principal investigator initiated contact with a community activist residing directly across from the coal combustion plant and “high-hazard” slurry pond. After the initial meeting, the University of Louisville provided a grant to the principal investigator to engage the community in a small

mixed-methods study. In May 2012, the research team met with 11 community leaders representing four neighborhoods around the coal ash facility to explain the objectives of the small study and answer questions.

c. Community Meeting

After the leader meeting, two community-wide dinner meetings were held in June 2012. The research team went door-to-door to invite members of the community to the dinner meetings and answer questions about the study. Flyers were also administered to residents throughout the neighborhoods.

d. Focus groups

In July-August 2012, community members participated in five different focus groups. Focus groups were held at a local neighborhood restaurant that had private facilities on Wednesday evenings. A semi-structured focus group guide was used to direct the discussion towards three areas: community, coal ash exposure, and health. All discussions were tape-recorded and transcribed verbatim for analyses.

2. Specific Aim 1 Methods

1. Survey of Coal Ash Community

a. Survey Design

A cross-sectional questionnaire was developed using common themes and results from the focus group transcripts. Four members of the study team analyzed the focus group data and developed questions on behaviors related to exposure (30 questions), general health characteristics (3 Likert Scale, 6 true/false, and 28 symptom questions) and specific health outcomes (42

outcomes). Questions regarding behaviors related to exposure were in a yes/no or Likert Scale format. There was one open-ended question where participants were asked to describe how they know they are exposed to coal ash.

In addition to the adult questionnaire, the final questionnaire package contained sections on children's health and children's sleep. These sections were designed based on the community feedback and validated questionnaires.

The children's sleep section contained 19 questions: 3 about general characteristics, 5 about bedtime routine, 8 about sleep disruptive behaviors, 2 about traumatic events, and 1 chart about falling asleep during daily activities. All responses were formatted as a Likert scale. Questions in the children's sleep section were adapted from the Children's National Medical Center Child Sleep Questionnaire [38].

The children's health section contained a table of 21 common specific health conditions that may be associated with environmental exposures. The participant was asked to indicate if the child had any of the listed health conditions. The parent was also asked to compare the health of their child to another child of the same age.

In November 2012, a community meeting with seven leaders was held to pre-test the questionnaire and solicit feedback about revisions to improve comprehension.

b. Survey Method

Parents or legal guardians, 18 years or older, completed the questionnaire regarding their health and the health of their children residing in their homes. If

multiple adults from a household completed the questionnaire, the children's health section was only completed once.

Recruitment of participants varied in the four different neighborhoods due to unique characteristics. Methods of recruitment and reasoning for this variation are as follows:

Neighborhood 1- Riverside Gardens: This neighborhood had facilities that served as a central meeting location for community members.

Residents are very "close-knit" and have very active and well-known leaders on issues surrounding coal ash. With the help of these leaders, flyers were administered throughout the community instructing residents to come to a centrally located church on 1 of 4 dates to complete a questionnaire. Snack foods and beverages were provided as incentives. This neighborhood was the most active in community meetings and in the survey.

Neighborhood 2- Lazy Acres Mobile Home Park: This neighborhood was a mobile home park consisting of no centrally located indoor public facilities. There were no previously identified community leaders that assisted with recruitment or distribution of questionnaires in this neighborhood. After speaking with the mobile home park owner, research personnel distributed flyers door-to-door instructing residents to come to the centrally located outdoor neighborhood mailboxes on 1 of 2 dates to complete the questionnaire. Water bottles, hats, and screwdrivers were provided as incentives.

Neighborhood 3/4- Claremore Acres/Cane Run Road: This neighborhood is located directly across from the coal ash storage facility and did not have a centrally located public facility to administer the survey. Flyers providing details about the questionnaire were distributed to two leaders, who distributed them to residents. Residents were instructed to come to the driveway of a home located at one of the entrances of the neighborhood on 1 of 2 dates. Water bottles, hats, and screwdrivers were provided as incentives.

Neighborhood 5- Clark's Mobile Home Park: This neighborhood was a managed mobile home park. Research personnel approached the manager about administering the survey, but were refused entrance.

2. Survey of Non-Exposed group

For comparison, the children's health and sleep section was adopted to administer in a non-exposed group. The questionnaire contained the same number of questions and formatted responses. IRB approval was obtained from the University of Louisville for the revised survey protocol.

The non-exposed group was obtained from Orange County, Indiana. This area was selected due to similar demographics, rural setting, and distance from any coal ash storage facilities. Based on 2010 census data, the population of Orange County is 19,840; 27.3% of which is under 18 years old and 97.0% is white alone. An estimated 19.0% of the population is living below the poverty line, and about 79.5% are a high school graduate or higher. The average household size is 2.49 [37].

After approval from Dr. Yolanda Yoder and other co-physicians, recruitment of participants occurred in the waiting room of the Comprehensive Health Clinic in Paoli, IN. This clinic is a primary healthcare facility that provides a majority of the obstetrics and youth physical examinations in Orange County, Indiana. Parents or legal guardians, 18 years and older, were invited to complete a questionnaire about the health and sleep of their children under 18 years old that reside in their home. Incentives such as water bottles, hats, and screwdrivers were provided.

3. Data Entry

Completed questionnaires were stored in a locked secure location at the University of Louisville School of Public Health and Information Sciences. REMARK OMR software was used to scan the data, which was converted to Excel files. Questions not formatted with bubble responses were entered manually. The data was reviewed and cleaned by the PI before using for analysis.

4. Analysis of Cross Sectional Data

a. Subaim 1a

The variables assessed to address subaim 1 are listed in table 1. The prevalence of the individual sleep disruptive behaviors were calculated in the non-exposed and exposed groups. Exposure is defined as residing in one of the 4 neighborhoods surrounding the coal ash facility; non-exposure is defined as residing in Orange County, Indiana. Only children ages 4 to 17 were included in

this study because the sleep survey was designed to best analyze behaviors in this age group. The analysis column describes how the variables were analyzed in this study.

Table 1: Sleep Disruptive Behavior Variables

Variable	Question Type	Variable Type	Analysis
Delay in sleep onset	Likert (Q8)	Ordinal	Dichotomous (Never/Rarely vs. Sometime/Frequently/Always)
Frequent night awakenings	Likert (Q9)	Ordinal	Dichotomous (Never/Rarely vs. Sometime/Frequently/Always)
Teeth grinding	Likert (Q12)	Ordinal	Dichotomous (Never/Rarely vs. Sometime/Frequently/Always)
Leg Jerking	Yes/no (Q13)	Dichotomous	
Head rolling	Yes/no (Q13)	Dichotomous	
Lip smacking	Yes/no (Q13)	Dichotomous	
Hand flapping	Yes/no (Q13)	Dichotomous	
Twitching	Yes/no (Q13)	Dichotomous	
Sleep walking	Yes/no (Q13)	Dichotomous	
Sleep Talking	Yes/no (Q13)	Dichotomous	
Snoring	Yes/sometimes/no (Q8)	Ordinal	Dichotomous (Yes/sometimes vs. No)
Complain of leg cramps	Yes/sometimes/no (Q15)	Ordinal	Dichotomous (Yes/sometimes vs. No)

b. Subaim 1b

A composite sleep disruptive behavior score was developed based on the variables listed in table 1. Ordinal responses were assessed as dichotomous, “1” for a positive response and “0” for a negative response. A numeric score was assigned to each participant based on the number of sleep disruptive behaviors reported. The more sleep disruptive behaviors reported, the higher the sleep disruptive score. The median and range of the sleep disruptive scores was calculated for both the non-exposed and exposed groups.

c. Subaim 1c

The prevalence of covariates that could impact sleep were assessed. All covariates included in the questionnaire that may impact bedtime routine and sleep are listed in Table 2. The analysis column describes how the variables were analyzed in this study.

Table 2: Potential Covariates

Covariate	Definition	Question Type	Analysis
Bedtime routine	TV Use	Yes/no/sometimes (Q7)	Dichotomous (Yes/Sometimes vs. No)
	Computer use	Yes/no/sometimes (Q7)	Dichotomous (Yes/Sometimes vs. No)
	Cell Phone use	Yes/no/sometimes (Q7)	Dichotomous (Yes/Sometimes vs. No)
	Caffeine consumption	Yes/no/sometimes (Q7)	Dichotomous (Yes/Sometimes vs. No)
	Read book	Yes/no/sometimes (Q7)	Dichotomous (Yes/Sometimes vs. No)
	Shower or bath	Yes/no/sometimes (Q7)	Dichotomous (Yes/Sometimes vs. No)
	Listen to quiet music	Yes/no/sometimes (Q7)	Dichotomous (Yes/Sometimes vs. No)
	Eat large snack or meal	Yes/no/sometimes (Q7)	Dichotomous (Yes/Sometimes vs. No)
Gender		Male/Female (Q1)	Dichotomous
Age		Open ended (Q2)	Continuous
Secondhand Smoke (SHS) Exposure	Parent Smoke	Yes/no (Q23)	Dichotomous
	Person smoke in home	Yes/ no (Q24)	
Traumatic Event		Yes/no (Q17)	Dichotomous
Health score	List of conditions	(Q26)	Discrete, quantitative
Time spent outdoors		Open ended	continuous
Use of sleeping medications		Likert (Q16)	Dichotomous (Never/Rarely vs. Sometime/Frequently/Always)

The median age in the exposed and non-exposed group was compared using the Wilcoxon Mann-Whitney test. The gender ratio was compared using chi-square analysis. Smoking exposure was dichotomized. If a parent was a current smoker, or somebody else smoked in the house, the child was considered exposed to secondhand smoke.

3. Specific Aim 2 Methods

a. Subaim 2a

Using SAS, the Wilcoxon Rank-Sum test was conducted to evaluate if the sleep disruptive behavior composite scores are significantly different (p -value < 0.05) in the non-exposed and exposed groups.

b. Subaim 2b

Logistic regression was conducted using SAS to assess the relationship between each individual sleep disruptive behavior and coal ash exposure group (non-exposed or exposed). Head rolling, lip smacking, hand flapping, and sleepwalking were not analyzed individually using logistic regression due to low prevalence. Four adjusted models were created with different categories of covariates. The first adjusted model included age and gender as covariates. If age or gender were found to significantly impact the exposure odds ratio, or improve the fit of the model the variables were included in the subsequent models. The second model was adjusted for the health score; which was a composite score for the number of reported conditions that could potentially impact sleep. Conditions considered in the health score were asthma, allergies, cough, bronchitis, frequent respiratory infection, sinus problems, congestion,

learning difficulties, ADHD/ADD, developmental delay, emotional behavioral disorders, ear problems, headaches, and gastrointestinal problems. The third model was adjusted for environmental factors found to be significantly related to individual sleep disruptive behaviors in this study. Environmental factors that were found to be significantly associated with adverse sleep behaviors from previous studies were also considered [24 25]. Environmental factors included in the model were computer use one hour before bed, caffeine use one hour before bed, and smoking exposure. The fourth model was adjusted for both the health score and environmental factors. In each model potential interaction terms were also evaluated. Table 3 contains the covariates considered in each model for each individual sleep disruptive behavior. Smoking was not considered as a covariate in the model with frequent sleep awakenings as the outcome because: 1) smoking was not independently significantly associated with the outcome, 2) smoking was not significant in model three or four, 3) including smoking did not improve the overall fit of the model.

Table 3: Logistic Regression Covariates

Sleep Disruptive Behavior	First Adjusted OR (95% CI)	Second Adjusted OR (95% CI)	Third Adjusted OR (95% CI)	Fourth Adjusted OR (95% CI)
Delay in sleep onset	Age Gender	Health score Age	Caffeine Computer SHS Age	Health score Caffeine Computer SHS Age
Frequent night awakening	Age Gender	Health score Age	Caffeine Computer Age	Health score Caffeine Computer Age
Teeth grinding	Age Gender	Health Score Age	Caffeine Computer SHS Age	Health score Caffeine Computer SHS Age
Leg jerking	Age Gender	Health Score Age Gender	Caffeine Computer SHS Age Gender	Health score Caffeine Computer SHS Age Gender
Twitching	Age Gender	Health Score Age	Caffeine Computer SHS Age	Health score Caffeine Computer SHS Age
Sleep talking	Age Gender	Health Score Age	Caffeine Computer SHS Age	Health score Caffeine Computer SHS Age
Snoring	Age Gender	Health Score Age	Caffeine Computer SHS Age	Health score Caffeine Computer SHS Age
Complaint of leg cramps	Age Gender	Health Score Age	Caffeine Computer SHS Age	Caffeine Computer SHS Age Health score Smoking/ Exposure

V. RESULTS

a. Subaim 1 Results

The demographic and general behavior comparison statistics are found in Table 4. Chi-square or Fisher exact p-values were calculated to compare prevalence between the exposed and non-exposed groups.

Table 4: Demographics and General Behaviors (Ages 4-17)

	Non-Exposed (n= 50)	Exposed (n= 61)	P-value
Mean Age	9.62 (4-17)	10.83 (4-17)	0.11
Gender			0.06
<i>Males</i>	66.0% (33)	47.5% (29)	
<i>Females</i>	34.0% (17)	52.4% (32)	
Relationship			0.005^{*F}
<i>Biological child</i>	86.0% (43)	57.4% (31)	
<i>Biological grandchild</i>	4.0% (2)	24.1% (13)	
<i>Adopted child</i>	6.0% (3)	7.4% (4)	
<i>Other</i>	4.0%(2)	11.1% (6)	
Time in home			0.04^{*F}
<i>All the time</i>	88.0% (44)	84.9% (45)	
<i>Only on weekends</i>	8.0% (4)	3.8% (2)	
<i>About 50% of time</i>	0.0% (0)	11.3% (6)	
<i>Sporadically</i>	2.0% (1)	0.0% (0)	
Years in community			0.07 ^{*F}
<i>0-5 years</i>	18.0% (9)	36.1% (22)	0.07 ^{*I}
<i>6-10 years</i>	44.0% (22)	41.0% (25)	
<i>11-15 years</i>	32.0% (16)	14.8% (9)	
<i>16-20 years</i>	6.0% (3)	8.2% (5)	
Average hours/day spent outdoors	2.52 (0-8)	5.69 (0-13)	<0.0001
Smoking Exposure	16.0% (8)	77.1% (47)	<0.0001
Experience a traumatic event	14.0% (7)	29.5% (18)	0.03
Average hours of sleep per night	8.38 (6-12)	8.02 (5-11)	0.10
Regular bedtime	82.0% (41)	80.3% (49)	0.82
Regular bedtime routine			0.08 ^{*F}
<i>Never</i>	4.0% (2)	3.3% (2)	
<i>Rarely</i>	6.0% (3)	8.2% (5)	
<i>Sometimes</i>	20.0% (10)	27.9% (17)	
<i>Frequently</i>	40.0% (20)	16.4% (10)	
<i>Always</i>	30.0% (15)	44.3% (27)	

^{*F} P-values for Fishers exact test due to low cell counts.

^{*I} Chi-square Test for Trend

Characteristics that were significantly different between the exposed and non-exposed were parent/child relationship, average hours/day spent outside, smoking exposure, and experience of a traumatic event.

Participants were asked to indicate their children's diseases or illnesses that had been diagnosed by a doctor. Table 5 contains the prevalence of specific health conditions and the health score, which was included as a covariate in logistic regression models.

Table 5: Diseases, Disorders, and Illnesses Reported

	Non-Exposed	Exposed	P-value
Asthma	18.0% (9)	26.2% (16)	0.30
Allergies	40.0% (20)	73.8% (45)	0.0003
Coughing	14.0% (7)	62.3% (38)	<0.0001
Frequent Bronchitis	0.0% (0)	19.7% (12)	0.0005*^F
Frequent Respiratory Infections	8.0% (4)	32.8% (20)	0.002*^F
Sinus Problems	16.0% (8)	55.7% (34)	<0.0001
Congestion	10.0% (5)	50.8% (31)	<0.0001*^F
Learning Difficulties	6.0% (3)	26.2% (16)	0.005*^F
ADHD/ADD	16.0% (8)	36.07% (22)	0.02*^F
Autism Spectrum Disorder	2.0% (1)	1.6%(1)	1.000* ^F
Developmental Delay	6.0% (3)	8.2% (5)	0.73* ^F
Emotional/ Behavioral Disorders	4.0% (2)	37.7% (23)	<0.0001*^F
Nose bleeds	4.0% (2)	18.0% (11)	0.04*^F
Ear problems	4.0% (2)	34.4% (21)	<0.0001*^F
Eye irritation/Red eyes	0.0% (0)	27.9% (17)	<0.0001*^F
Headaches	22.0% (11)	52.5% (32)	0.001
GI/Stomach problems	10.0% (5)	31.2%(19)	0.01*^F
Kidney problems	2.0% (1)	3.3% (2)	1.000* ^F
Skin rashes or sores	4.0% (2)	27.9% (17)	0.0008*^F
Cancer	0.0% (0)	0.0%(0)	1.000
Diabetes	2.0% (1)	0.0% (0)	0.45* ^F
Health Score	1.74 (1.7-7)	5.48 (0-14)	<0.0001

*^F P-values for Fishers exact test due to low cell counts.

Illnesses that were significantly different between the exposed and the non-exposed groups included allergies, coughing, frequent bronchitis, frequent respiratory infections, sinus problems, congestion, learning difficulties, ADHD/ADD, emotional/ behavioral disorders, nose bleeds, ear problems, eye irritation, headaches, GI/Stomach problems, and skin rashes. Overall, the exposed group reported significantly more illnesses and diseases than the non-exposed group.

Results from Chi-square analysis or Fisher exact test comparing bedtime routine are found in table 6.

Table 6: Activities within One Hour of Bedtime

	Non-Exposed (n= 50)	Exposed (n= 61)	P=value
Watch TV	98.0% (49)	96.7% (59)	1.000
Play video games	55.3% (26)	57.4% (35)	0.83
Use a computer	42.2% (19)	55.7% (34)	0.17
Do homework	61.7% (29)	50.8% (31)	0.26
Use cell phone	26.1% (12)	36.1% (22)	0.27
Read a book	78.7% (37)	78.0% (46)	0.93
Take a bath or shower	90.0% (45)	98.3% (59)	0.09
Drink caffeine (coke, energy drinks, ...)	17.8% (8)	25.0% (15)	0.38
Listen to quiet music	34.8% (16)	26.23% (16)	0.34
Eat a large meal or lots of snacks	21.7% (10)	46.7% (28)	0.008
Use medications to help sleep	2% (1)	32.2% (19)	<0.0001*^F
* ^F P-value for Fishers exact test due to low cell counts. Prevalence data include blank responses in the denominator.			

Children in the exposed group were significantly more likely to eat a large meal or snack, and/or use medications to help them sleep than children in the non-exposed group.

Table 7: Sleep Disruptive Behaviors

	Non-Exposed	Exposed	P-value
Delay in sleep onset	38.0% (19)	63.9% (39)	0.007
Frequent night awakenings	32.0% (16)	68.9%(42)	0.0001
Teeth grinding	30.0% (15)	50.8% (31)	0.03
Leg jerking	36.0% (18)	45.9% (28)	0.29
Head rolling	4.0% (2)	16.4% (10)	0.06 ^{*F}
Lip smacking	2.0% (1)	19.7% (12)	0.006^{*F}
Hand flapping	2.0% (1)	9.8% (6)	0.13 ^{*F}
Twitching	20.0% (10)	34.4% (21)	0.92
Sleep walking	6.0% (3)	14.8% (9)	0.22 ^{*F}
Sleep talking	30.0% (15)	45.9%(28)	0.09
Snoring	36.0% (18)	65.6% (40)	0.002
Complant of leg cramps while resting	24.0% (12)	57.4% (35)	0.0004
Average Sleep Disruptive Score	2.6 (0-9)	4.92 (0-11)	<0.0001
*F P-value for Fishers exact test due to low cell counts. Prevalence data include blank responses in the denominator			

Table 7 contains the prevalence of the individual sleep disruptive behaviors. The prevalence of delay in sleep onset, frequent night awakenings, teeth grinding, lip smacking, snoring, and complaining of leg cramps while resting was significantly greater in the exposed group compared to the non-exposed group.

b. Subaim 2 Results

Overall, participants in the exposed group reported significantly more sleep disruptive behaviors than in the non-exposed group (pvalue <0.001). The odds ratios for individual sleep disruptive behaviors in the exposed compared to the non-exposed are found in Table 8.

Table 8: Sleep Disruptive Behavior Odds Ratios for Exposed vs. Non-exposed

Sleep Disruptive Behavior	OR (95% CI)	First OR (95% CI)	Second OR (95% CI)	Third OR (95% CI)	Fourth OR (95% CI)
Delay in sleep onset	2.89 (1.3 - 6.3)	3.03 (1.3 - 6.9)	1.26 (0.5 - 3.2)	4.34 (1.4-13)	2.04 (0.6 - 7.1)
Frequent night awakenings	4.70 (2.1 - 11)	5.52 (2.3 - 14)	6.07 (2.1 - 17)	6.56 (2.5-17)	6.9 (2.2 - 21)
Teeth grinding	2.41 (1.1 - 5.3)	2.33 (1.0 - 5.3)	1.37 (0.5 - 3.6)	2.57 (0.9-7.2)	1.5 (0.5 - 5.0)
Leg jerking	1.51 (0.7 - 3.2)	1.63 (0.7 - 3.6)	0.62 (0.2 - 1.7)	1.3 (0.5-3.6)	0.61 (0.2 - 2.0)
Twitching	2.10 (0.9 - 5.0)	2.74 (1.1 - 6.9)	1.39 (0.5 - 4.1)	4.05 (1.3-13)	2.43 (0.7 - 8.9)
Sleep talking	2.0 (0.9 - 4.3)	2.64 (1.1 - 6.1)	1.23 (0.5 - 3.3)	2.83 (1.0-7.9)	1.37 (0.4 - 4.5)
Snoring	3.39 (1.6 - 7.4)	3.97 (1.7 - 9.2)	1.29 (0.5 - 3.5)	4.18 (1.5-12)	1.5 (0.4 - 5.1)
Complaint of leg cramps	4.26 (1.9 - 9.7)	4.27 (1.8 - 10)	1.33 (0.5 - 3.8)	3.0 (1.1 - 8.5)	1.73 (0.3 - 8.4)
OR: Not adjusted First Adj. OR: Age and Gender Second Adj. OR: Health Score Third Adj. OR: Environmental (Smoking, Using computer, Drinking caffeine) Fourth Adj OR: Environmental and Health Score					

The unadjusted odds of delay in sleep onset (OR= 2.89), frequent night awakenings (OR= 4.7), teeth grinding (OR= 2.4), snoring (OR= 3.39), and complaint of leg cramps (OR= 4.26) were significantly greater in the exposed group than the non-exposed group. After considering environmental factors, the odds of delay in sleep onset (OR= 4.34), frequent night awakenings (OR= 6.56), twitching (OR= 4.05), sleep talking (OR= 2.83), snoring (OR= 4.18), and complaint of leg cramps (OR= 3.00) were significantly greater in the exposed group than the non-exposed group. The health score was found to be a potential confounder because it is significantly related to the exposure and each individual

sleeps disruptive behavior. The odds of frequent night awakenings were significantly greater in the exposed (OR= 6.9) compare to the non-exposed group when adjusting for both environmental factors and the health score.

VI. DISCUSSION

The most prevalent sleep disruptive behavior in the exposed group were frequent night awakenings (68.9% in exposed group, compared to 32% in the non-exposed group). Delay in sleep onset (63.9% in exposed group, compared to 38% in the non-exposed group) and snoring (65.6% in exposed group compared to 36% in the non-exposed group) were the second and third most prevalent sleep disruptive behaviors. The sleep disruptive score was also significantly higher in the exposed children compared to the non-exposed children. When adjusting for health and environmental factors, the odds of frequent night awakenings were significantly greater in the exposed group compared to the non-exposed group (OR= 6.9, CI= 2.2-21). These findings are consistent with Abou- Khadra (2013) findings that PM_{10} is significantly associated with disorders of initiating and maintaining sleep ($p= 0.012$) in school-aged children [24].

The diagnosis of diseases and disorders are important to consider when evaluating the association between coal ash exposure and sleep disruptive behaviors. For all individual sleep disruptive behaviors, addition of the health score into the logistic regression model decreased the odds of sleep disruptive behaviors related to exposure. The health score may act as a confounder in the association between coal ash exposure and sleep disruptive behaviors because the variable is significantly associated with both the exposure and each outcome,

independently. However, it is also possible that some diseases and disorders, reflected in the health score, act as intermediates in potential causal pathways between coal ash exposure and sleep disruptive behaviors. For example, studies evaluating allergic rhinitis or ADHD, and the correlation with sleep disordered breathing, demonstrate the interchangeability of the cause-effect relationship between disease and sleep disruption [40, 41]. Medical treatment focused on one factor or the other, has improved outcomes for both.

Adenotonsillectomy, a treatment for sleep disordered breathing, has been found to reduce symptoms of ADHD including hyperactivity and inattention [40]. On the other hand, treatment of allergic rhinitis, a common illness, was found to reduce symptoms of sleep disordered breathing [41]. It can be hypothesized that 1) exposure to coal ash increases odds of infection, which is associated with sleep disruptive behaviors; or 2) exposure to coal ash increases odds of sleep disruption, independent of disease.

A recent *in vitro* and *in vivo* study supports the hypothesis that coal ash particles can induce respiratory infections capable of causing sleep disruption. The Borcharding et al. study (2013) assessed the ability of coal fly ash, in concentrations relevant to human daily exposure, to induce growth of pathogenic bacteria that could potentially cause respiratory infections [42]. In this study, the effects of coal fly ash on the growth of *Pseudomonas Aeruginosa* (PA01) were evaluated in mouse models (*in vivo*) and human lung cells (*in vitro*). In the mouse models, three coal fly ash samples were found to significantly decrease clearance of the PA01 bacterial growth in the lung tissue. The increase in

bacterial growth induced by coal fly ash exposure was not found to be associated with an increase in inflammation in the tissue of respiratory organs. This is inconsistent with previous hypotheses that a decrease in bacterial clearance is a result of inflammation induced by air pollution particulate matter. Based on this finding, Borcharding et al. (2013) next evaluated if PA01 proliferation is a result of cellular damage to human epithelia airway cells caused by exposure to coal fly ash. Production of hydrogen peroxide, a reactive oxygen species, was measured after cells were exposed to PA01 and coal fly ash. In human epithelia cells PA01 clearance was significantly inhibited by coal fly ash, however, no increase in hydrogen peroxide production was found in cells exposed to both coal fly ash and PA01 relative to cells only exposed to PA01. Based on these findings, it was further hypothesized that coal fly ash impairs innate immunity in the airway by inhibiting antimicrobial peptide activity. Antimicrobial peptide normally breaks down the cell wall of pathogenic bacteria and prevents microbial respiration. It was found that antimicrobial peptide activity was significantly impaired in cell cultures treated with coal fly ash and PA01. These findings led Borcharding et al. (2013) to conclude that coal fly ash has the ability to increase nutrient bioavailability for bacterial growth and inhibit antimicrobial peptide activity in the airway [42]. Increased bacterial growth could contribute to the creation of a biofilm supportive of microbial proliferation that can cause damage to epithelial cells. It has been suggested in a clinical study that biofilm growth is associated with adenoid hypertrophy and reoccurring acute otitis media, which may increase risk of obstructive sleep apnea [43].

There are few studies evaluating the hypothesis that particulate matter exposure is associated with sleep disruptive behaviors in children independent of diseases. The exact mechanism through which exposure to PM, such as coal ash particles, impacts sleep in children is not known. Findings from studies evaluating the impact of environmental tobacco exposure and air pollution may contribute to hypotheses regarding potential mechanisms since tobacco smoke contains small particulate matter and metals, similar to coal ash. One hypothesis is that PM in secondhand cigarette smoke irritates the upper airway, which disrupts breathing and causes more nighttime awakenings [44]. PM from air pollution in the airway may also induce chronic inflammation, produce inflammatory mediators, disrupt barriers that prevent PM from circulating throughout the organ systems, and potentially impair cognitive function [22, 24]. Coal ash particles may similarly cause irritation of the upper airway and/or systemic inflammation that disrupts sleep breathing and normal sleep patterns.

Based on the information collected in this study, the direction of the causal pathway between the disease and sleep disruption is unclear. The additive effect of having a number of diseases and disorders may cause more stress on the body and contribute to disruption of sleep; on the other hand, disruption of sleep may cause extended stress on the body, increasing odds of presenting with a greater number of reported diseases or disorders. The health impacts of coal ash exposure are unknown, but disease prevalence in this study suggest that exposure is significantly associated with some of the most prevalent diseases and disorders reported. Further research, with larger sample sizes, is

needed to assess the association between coal ash exposure and specific health conditions in order to understand the systemic relationship between coal ash exposure, disease, and sleep disruption.

Exposure to secondhand smoke is another important factor to consider when assessing the relationship between coal ash exposure and sleep disruptive behavior. There was a significant difference in secondhand smoke exposure between the coal ash exposure groups. However, previous population surveys suggest that these groups may not be that different when it comes to smoking. According to the Community Health needs assessment (2012) conducted by the only hospital in the non-exposed county, smoking prevalence among the patients in the hospital's regional market was 28% [45]. This prevalence is comparable to the Louisville Metro smoking prevalence of 23.9% found in the 2012 Louisville Metro Health Status Report [46]. This suggests that the stark difference in prevalence of secondhand smoke exposure in this study may be partially impacted by survey bias, selection bias, and random error exacerbated by the small sample size. Differences in survey methods and question design between the exposed and non-exposed groups may have impacted participant's responses to questions about smoking. The exposed group completed questions about parent or guardian smoking in a separate adult health survey while the non-exposed group completed questions about smoking exposure within the children's health questionnaire. The way the question was phrased may have impacted participant's interpretation and response. In addition, participants in the exposed group were recruited with community involvement while participants in

the non-exposed group were randomly selected in a waiting room. Participants in the exposed group who recruited their friends and close neighbors are more likely to have similar social behaviors, like smoking.

VII. CONCLUSION

This is the first community-based study assessing sleep disruptive behaviors in children exposed to coal ash. Coal ash storage is a significant concern in Kentucky where storage regulations are limited, and the amount to be stored is ever growing. This study is designed based on an impoverished community's concerns and will improve understanding about environmental exposure and children's sleep. Findings from this study suggest that coal ash exposure is significantly associated with sleep disruptive behaviors that impact maintenance of sleep. It is important to further evaluate this association because chronic sleep disruption in children can have long term impacts on cognitive and biological development.

One limitation of this study is that the lack of funding limited the incentives and resources available to conduct the survey. In two neighborhoods, the survey was conducted outdoors during the summer months, which may have contributed to lower response rates. Some community members also expressed fear that participation in the survey could result in an increase in their electric bill, and felt that it would not make a significant difference because the city has forgotten about them. Furthermore, questionnaires had to be shortened and no biometric measurements or environmental data could be collected. This limited biometric covariates that may impact the association between coal ash exposure and some sleep disruptive behaviors. Despite these drawbacks, data collected from this

study can direct future more in-depth investigations into specific sleep disruptive behaviors and investigators will be able to better identify and collect all necessary covariates. Further research in these specific areas is necessary to identify the impact of coal ash exposure on biological sleep mechanism in order to identify plausible causal pathways.

Sleep disruptive behaviors likely have numerous causal factors. The exposed and non-exposed populations in this study were selected to control for some socio-economic and demographic characteristics that may impact sleep. However, one major difference between the two groups is the urban setting of the exposed group and rural setting of the non-exposed group. The composition of particulate matter in these two settings is likely different beyond the presence, or absence, of coal ash particles. Noise and other sources of pollution not considered in this study may also impact sleep disruptive behaviors. Future studies utilizing quantifiable measures of PM will be better able to assess and control for differences in PM composition between the exposed and non-exposed groups.

In conclusion, exposure to coal ash was found to be significantly associated with frequent night awakenings when controlling for both health and environmental factors. Findings from this study can help guide future studies assessing coal ash exposure and sleep disruptive behaviors that prevent children from getting adequate sleep.

REFERENCES

1. Brown, P., T. Jones, & K. BeruBe. (2011). The internal microstructure and fibrous mineralogy of fly ash from coal-burning power stations. *Environ Pollut*, 159(12): 3324-33. doi: 10.1016/j.envpol.2011.08.041.
2. United States Environmental Protection Agency. (2010). *Hazardous and Solid Waste Management System; Identification and Listing of Special Wastes; Disposal of Coal Combustion Residuals from Electric Utilities*. Retrieved from: <http://www.regulations.gov/> - !documentDetail;D=EPA-HQ-RCRA-2009-0640-0352.
3. United States Environmental Protection Agency. (2012). *Coal combustion residues (CCR) - Surface Impoundments with high hazard potential ratings*. Retrieved from: <http://www.epa.gov/osw/nonhaz/industrial/special/fossil/ccrs-fs/index.htm>.
4. Gilmour, I.M., Jaakkola, M.S., London, S.J., Nel, A.E., & Rogers, C.A. (2006). How Exposure to Environmental Tobacco Smoke, Outdoor Air Pollutants, and Increased Pollen Burdens Influences the Incidence of Asthma. *Environmental Health Perspectives*, 114(4): 627-633. doi: 10.1289/ehp.8380.
5. Li, S., Williams, G., Jalaludin, B., & Baker, P. (2012). Panel studies of air pollution on children's lung function and respiratory symptoms: a literature review. *J Asthma*, 49(9): 895-910.
6. Sinclair, A.H., Edgerton, E.S., Wyzga, R., & Tolsma, D. (2010). A Two-Time-Period Comparison of the Effects of Ambient Air Pollution on Outpatient Visits for Acute Respiratory Illnesses. *Journal of the Air & Waste Management Association*, 60(2): 163-175. doi: 10.3155/1047-3289.60.2.163.
7. Castaneda, J.L., Kheirandish-Gozal, L., Gozal, D., Accinelli, R.A. (2012). Effect of reductions in biomass fuel exposure on symptoms of sleep apnea in children living in the peruvian andes: A preliminary field study. *Pediatr Pulmonol*. doi: 10.1002/ppul.22720.
8. Pope III, A.C., Dockery, D.W.. (1992). Acute health effects of PM10 pollution on symptomatic and asymptomatic children. *Am Rev Respir Dis*, 145(5):1123-8.

9. Myers, G.J., Davidson, P.W., Weitzman, M., & Lanphear, B.P. (1997). Contribution of Heavy Metals to Developmental Disabilities in Children. *Mental Retardation and Developmental Disabilities Research Reviews*, 3: 239-245.
10. Dockery, D.W. (2009). Health effects of particulate air pollution. *Ann Epidemiol*, 19(4): 257-63. doi: 10.1016/j.annepidem.2009.01.018.
11. United States Environmental Protection Agency. (2012). *Particulate Matter (PM) Research*. Retrieved from: <http://www.epa.gov/airscience/air-particulatematter.htm>.
12. Pope III, A.C. (2000). Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? *Environ Health Perspect*, 108(suppl 4): 713-723.
13. Dockery, D.P.I., A., Xu, X., Spengler, J.D., Ware, J.H., Fay, M.E., Ferris, B.G., et al. (1993). An association between air pollution and mortality in six U.S. cities. *The New England Journal of Medicine*, 329(24):1753-1759.
14. Flues, M., et al. (2013). Toxic elements mobility in coal and ashes of Figueira coal power plant, Brazil. *Fuel*, 103: 430-436. doi: 10.1016/j.fuel.2012.09.045.
15. Jones, K.B., L.F. Ruppert, and S.M. Swanson. (2012). Leaching of elements from bottom ash, economizer fly ash, and fly ash from two coal-fired power plants. *International Journal of Coal Geology*, 94: 337-348. doi: 10.1016/j.coal.2011.10.007.
16. Smith, K. R., Veranth, J. M., Kodavanti, U. P., Aust, A. E., & Pinkerton, K. E. (2006). Acute pulmonary and systemic effects of inhaled coal fly ash in rats: comparison to ambient environmental particles. *Toxicol Sci*, 93(2): 390-9. doi: 10.1093/toxsci/kfl062.
17. Ruhl, L., Vengosh, A., Dwyer, GS. Hsu-Kim, H., Deonarine, A., Bergin, M., & Kravchenko, J. (2009). Survey of the potential environmental and health impacts in the immediate aftermath of the coal ash spill in Kingston, TN. *Environ Sci Technol*, 43: 6326-6333.
18. Bednar, A.J., et al. (2013). Characterization of metals released from coal fly ash during dredging at the Kingston ash recovery project. *Chemosphere*, 92(11):1563-70. doi: 10.1016/j.chemosphere.2013.04.034.

19. Environmental Protection Agency. (1996). *Protection of Environment*, Title 40 Electronic Code of Federal Regulations Pt. 70.1 and 70.2. 2014 ed. Retrieved from: http://www.ecfr.gov/cgi-bin/text-idx?c=ecfr&SID=3a7e222592f9ef7e6e1496363b640130&tpl=/ecfrbrowse/Title40/40cfr70_main_02.tpl.
20. Borm, P.J.A. (1997). Toxicity and occupational health hazards of coal fly ash (CFA): A review of data and comparison to coal mine dust. *Ann. occup. Hyg.*, 41(6): 659-676.
21. Block, M.L. & Calderon-Garciduenas, L. (2009). Air pollution: mechanisms of neuroinflammation and CNS disease. *Trends Neurosci*, 32(9): 506-16. doi: 10.1016/j.tins.2009.05.009.
22. Calderon-Garciduenas, L., et al. (2007). Pediatric respiratory and systemic effects of chronic air pollution exposure: nose, lung, heart, and brain pathology. *Toxicol Pathol*, 35(1): 154-62. doi: 10.1080/01926230601059985.
23. Rojas-Castaneda, J. C., Viguera-Villasenor, R. M., Rojas, P., Chavez-Saldana, M., Gutierrez-Perez, O., Montes, S., & Rios, C. (2011). Alterations induced by chronic lead exposure on the cells of circadian pacemaker of developing rats. *Int J Exp Pathol*, 92(4): 243-50. doi: 10.1111/j.1365-2613.2011.00761.x.
24. Abou-Khadra, M.K. (2013). Association between PM10 exposure and sleep of Egyptian school children. *Sleep Breath*, 17(2): 653-7. doi: 10.1007/s11325-012-0738-7.
25. Kheirandish-Gozal, L., Ghalebadi, M., Salehi, M., Salarifar, M. H., & Gozal, D. (2013). Neighbourhood air quality and snoring among school-aged children. *Eur Respir J*. doi: 10.1183/09031936.00113113.
26. *Environmental Protection Agency; Notice of Regulatory Determination on Wastes from the Combustion of Fossil Fuels*. 65 Federal Register 99. (22 May 2000). pp. 32214.
27. United States Environmental Protection Agency. *Hazardous Waste Regulations*. 2012; Retrieved from: <http://www.epa.gov/wastes/laws-regs/regs-haz.htm>.
28. Evans, L., Becher, M., & Lee, B. (2011). *State of Failure: How states fail to protect our health and drinking water from toxic coal ash*. Retrieved from: http://earthjustice.org/sites/default/files/StateofFailure_2013-04-05.pdf.

29. United States Department of Energy, Office of Electricity Delivery & Energy Reliability. (2013). *Current Regulations Governing Coal Combustion By-Products- Kentucky*. Retrieved from: http://www.netl.doe.gov/technologies/coalpower/ewr/coal_utilization_byproducts/states/kentucky.html.
30. Jenkins, J. (2012). *HB404* (BR1155). Kentucky Legislature. Retrieved from: <http://www.lrc.ky.gov/record/12rs/HB404.htm>.
31. The Kentucky Chamber of Commerce. (2012). *2012 Kentucky General Assembly Results for Business*. Retrieved from: <http://www.kychamber.com/sites/default/files/2012resultsforbusiness.pdf>.
32. Bruggers, J., (2014). *Parties negotiate deal on coal ash, as judge extends a deadline*. *The Courier Journal*. Retrieved from: <http://blogs.courier-journal.com/watchdogearth/2014/01/02/parties-negotiate-deal-on-coal-ash-as-judge-extends-a-deadline/> .
33. LG&E and KU. (2014). *Power Plant Information: Cane Run Station*. Retrieved from: http://lge-ku.com/plant_info.asp.
34. LG&E and KU. (2014). *Neighbor to Neighbor: Cane Run Generating Station*. Retrieved from: http://www.lge-ku.com/neighbor2neighbor/cane_run_plantinformation.asp.
35. Revlett, G.H. (2013). *Plant-wide Odor, Fugitive Dust, and Maintenance Emissions Control Plan Cane Run Generating Station*. LG&E. p. 1-13.
36. Evans, L. (2013). *Coal Ash in Kentucky*. Retrieved from: <http://earthjustice.org/sites/default/files/files/ky-coal-ash-factsheet-1113.pdf>.
37. United States Census Bureau. (2010). American Factfinder. Retrieved from: <http://factfinder2.census.gov/faces/nav/jsf/pages/index.xhtml>.
38. Children's National Medical Center. (2009). Child Sleep Questionnaire. Retrieved from: <http://www.childrensnational.org/files/PDF/DepartmentsAndPrograms/Sleep-Medicine/SL-Child-Qs.pdf> .
39. Harper, L. M., Parry, S., Stamilio, D. M., Odibo, A. O., Cahill, A. G., Strauss, J. F., 3rd, & Macones, G. A. (2012). The interaction effect of bacterial vaginosis and periodontal disease on the risk of preterm delivery. *Am J Perinatol*, 29(5): 347-52. doi: 10.1055/s-0031-1295644.

40. Sedky, K., Bennett, D. S., & Carvalho, K. S. (2013). Attention deficit hyperactivity disorder and sleep disordered breathing in pediatric populations: A meta-analysis. *Sleep Med Rev.* doi: 10.1016/j.smr.2013.12.003.
41. Lin, S. Y., Melvin, T. A., Boss, E. F., & Ishman, S. L. (2013). The association between allergic rhinitis and sleep-disordered breathing in children: a systematic review. *Int Forum Allergy Rhinol.* doi: 10.1002/alr.21123.
42. Borcharding J.A., Chen, H., Caraballo J.C., Baltrusaitis J., Pezzulo A.A., et al. (2013). Coal fly ash impairs airway antimicrobial peptide and increases bacterial growth. *PLoS ONE*, 8(2): e57673. doi: 10.1371/journal.pone.0057673.t001.
43. Szalmas, A., Papp, Z., Csomor, P., Konya, J., Sziklai, I., Szekanecz, Z., & Karosi, T. (2013). Microbiological profile of adenoid hypertrophy correlates to clinical diagnosis in children. *Biomed Res Int*, 2013: 629607. doi: 10.1155/2013/629607.
44. Yolton, K., Xu, Y., Khoury, J., Succop, P., Lanphear, B., Beebe, D. W., & Owens, J. (2010). Associations between secondhand smoke exposure and sleep patterns in children. *Pediatrics*, 125(2): e261-8. doi: 10.1542/peds.2009-0690.
45. Eggers, R., Berry, S., Moore, S., Poteet, A., & Hodgkins, C. (2012). *Indiana University Health Paoli Hospital Community Health Needs Assessment*. Retrieved from: <http://iuhealth.org/images/glo-coe/paoli.pdf>.
46. Nesbitt, L., Harris, M., Hall, C., Pallam, H., & Chen, Y. (2012). *Louisville Metro Health Status Report*. Retrieved from: http://www.louisvilleky.gov/NR/rdonlyres/D83AC3D6-0C08-440C-B993-50BB258C573F/0/HealthreportFINAL_12712.pdf.

CURRICULUM VITAE

Clara G. Sears
1065 E. St. Catherine St.
Louisville KY, 40204
Phone: (812)653-1976
Email: cgsear01@louisville.edu

Education:

- B.A. **Goshen College**, Goshen, IN, April 2012
Biology (Minor: Business)
- M.S. **University of Louisville**, Louisville, KY
Expected graduation- May 2014
Epidemiology

Research Experience:

March 2014- Present

Graduate Research Assistant, University of Louisville, Department of Communications, Louisville, KY- Evaluating perception, language, and communication about Tobacco Use of Appalachian Youth.

September 2012- Present

Research Assistant, University of Louisville, Department of Epidemiology and Population Health, Louisville, KY- Mixed-methods community based research evaluating health in low-income neighborhoods near coal ash storage facilities.

March 2012- December 2012

Graduate Co-Investigator at the University of Notre Dame, Eck Institute for Global Health, Notre Dame, IN- Developing a Health Information Exchange- Driven Indicator Prioritization Process to Reduce Perinatal Mortality in North Central Indiana.

January 2012- March 2012

Research assistant at the University of Notre Dame, Institute for Latino Studies, Notre Dame, IN- Literature review and grant writing for research on health disparities, Hispanic/ Latino populations, and environmentally sustainable business.

September 2011- December 2011

Educational research assistant for Dr. James Miller, Biology Department, Goshen College, Goshen, IN- Development of laboratory procedures for studying cell membrane transport and spirometry in an educational facility.

May 2010-August 2010

Undergraduate research assistant at the University of Louisville, Department of Pediatrics, Louisville, Kentucky - Neuropsychological and biomedical functioning in children with developmental disabilities.

January 2009- April 2009

Undergraduate research assistant for Dr. James Miller, Biology Department, Goshen College, Goshen, IN - Cell membrane transport in mammalian and avian erythrocytes.

May 2008-August 2008

Research Intern for Dr. William McGregor, James Graham Brown Cancer Center, Department of Pharmacology and Toxicology, University of Louisville, Louisville, KY – Laboratory assistant for research on Cancer, Cancer Management, and Molecular Targets.

Teaching Assistance Experience:

Courses January 2011- December 2012, Goshen College

Assisted in grading, instruction, laboratory preparation, and lab supervision for:

-Biology 303, **Vertebrate Physiology** (1 semester)

-Biology 301, **Genetics** (1 semesters)

Presentations:

Sears, C, Reed, J., Maust, J., & Sider, J. November 2011. Sugar Consumption: Past and Present. *Biology Senior Seminar*. Goshen College, Goshen, IN.

Research Poster:

Zierold, K.M. & Sears, C. March 2014. Are Healthcare Providers asking the Right Questions of People living near Environmental Hazards? *American Academy of Health Behavior*, Charleston, SC.

Zierold, K.M. & Sears, C., March 2014. Exposure Reducing Behaviors among Residents Living near a Coal Ash Storage Site. *American Academy of Health Behavior*, Charleston, SC.

Sears, C. & Zierold, K. November 2013. A community-based approach to assessing emotional and behavioral disorders in children chronically exposed to coal ash. *American Public Health Association*. Boston, MA.

Sears, C. & Zierold, K. September 2013. Coal ash exposure and health in West Louisville, KY: a community-based mixed-methods study. *Research! Louisville, 2013*. Louisville, KY.

Tomcheck, S.D., Sears, L. & Sears, C. May 2012. Characteristic of toddlers screening false positive on the modified checklist for autism in toddlers. *2012 International Meeting for Autism Research*. Toronto, Canada.

Service and Outreach:

Goshen College, Goshen, IN. AIDS Awareness Benefit. December 2011 and December 2012. Organized and lead the campus community benefit to raise money for AIDS relief and educational programs in Africa.

Central America Study and Service Term in Guatemala. *Semilla: The Latin American Anabaptist Seminary*. May-August 2011. Volunteered with community health education programs at Asociacion Medica Integral San Lucas, San Juan, Guatemala.

IRS- Volunteer Income Tax Assistance Program. Goshen College, Goshen, IN. January-April 2010. Completed Income Tax Forms for low-income residents of Elkhart County, Indiana.

Paoli Community Schools, Paoli, IN. Moderate to Severe Special Education Grades K-6. May-August 2010 and May-August 2011. Assisted teachers in classroom and helped students complete daily tasks.