ASSOCIATION BETWEEN OCCUPATIONAL EXPOSURE TO DUSTS AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE: EXAMINATION OF DATA FROM THE NATIONAL HEALTH and NUTRITION EXAMINATION SURVEY 2007-2010.

by

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ABSTRACT

DAVID M. PARKS. Association between occupational exposure to dusts and chronic obstructive pulmonary disease: examination of data from the National Health and Nutrition Examination Survey 2007-2010 (under the direction of DR. AHMED ARIF).

Chronic obstructive pulmonary disease (COPD) is a debilitating respiratory condition affecting more than 15 million Americans for a prevalence of 6.2%. Relatively few studies have investigated the association of occupational exposures and COPD in the general public. The objective of this study was to determine the association of occupational exposure to mineral and organic dust with COPD. This study used secondary data from the National Health and Examination Survey 2007-2010. Multivariable logistic regression analysis was used to calculate odds ratios and 95% confidence intervals adjusted for age, gender, race, poverty-to-income ratio, body mass index, exposure to fumes, smoking status, and second hand smoke. The odds of COPD were 1.84 times greater (95% confidence interval [CI]: 1.36-2.50) for respondents exposed to mineral dust as compared to those who were not exposed to mineral dust. Compared to those who were never exposed to mineral dust, the odds of COPD increased from 1.66 (95%CI: 1.16-2.38) for respondents exposed for less than 10 years to 2.20 (95%CI: 1.25-3.88) for those who were exposed for 30 years or more, but no dose-response pattern was observed. Similarly, occupational exposure to organic dust was associated with 1.12 times the odds (95% CI: 0.87-1.44) of developing COPD. No dose-response pattern was evident for increasing number of years of exposure to organic dust. Appropriate intervention measures should be

used at workplaces to minimize exposures to dust to preserve workers' respiratory health

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CHAPTER 1: INTRODUCTION

Chronic obstructive pulmonary disease, commonly referred to as COPD, is a debilitating respiratory condition characterized by the presence of emphysema and chronic bronchitis (Centers for Disease Control and Prevention, 2015). Like other chronic diseases, COPD usually takes time to develop. Of the 24 million individuals in the United States that suffer from COPD half are undiagnosed and not aware they are living with the condition (National Institutes of Health, 2010).

Breathing can be a labored task for individuals with COPD and at times they may experience a quick onset of symptoms. These instances are referred to as exacerbations and can limit individuals from participating in intended activities (Wedzicha & Seemungal, 2007). Activities a healthy individual may consider routine such as attending sporting events, going to the park, and attending family functions can at times be overwhelming for individuals suffering from COPD. COPD exacerbations, characterized by shortness of breath, shallow breathing, wheezing, and coughing, can be triggered by a change in air-quality, overexertion, and/or a pulmonary infection (Wedzicha & Seemungal, 2007). If not treated properly these episodes can lead to fatigue and an altered state of mind caused by a lower than normal concentration of oxygen in the arterial blood, which is referred to as hypoxia and is why some individuals suffering from COPD use oxygen treatment (Kent, Mitchell, & McNicholas, 2011).

One form of COPD, emphysema, can take years to develop. In emphysema the inner walls of the alveoli, small air sacs in the lungs, weaken and air pockets develop. The alveoli may eventually rupture reducing the amount of surface area in the lungs and reduce the amount of air absorbed while inhaling (Medinas et al., 2009).

Another form of COPD is chronic bronchitis, a condition where the tubes that carry oxygen to the lungs, the bronchial tubes, are continuously inflamed accompanied with a cough and mucus. This inflammation can also lead to a condition known as wheezing, which often accompanies exacerbations and can occur as a result of retained secretions from chronic bronchitis (Glauser, Thompson, & Marland, 1982).

The National Health Interview Survey estimates 12.7 million, 5.7%, of American adults have COPD (American Lung Association, 2013). National data from the 2011 Behavioral Risk Factor Surveillance System reports the prevalence of COPD at 14.7 million or 6.2% of American adults (American Lung Association, 2013). The difference in the two prevalence rates is due to BRFSS using a different question, which describes COPD as chronic bronchitis and emphysema. Furthermore, BRFSS does not just ask respondents about the past year but instead asks if they have ever had a COPD diagnosis (American Lung Association, 2013). COPD prevalence rates for individual states range from 10.9% in Kentucky to 4% in Utah (Centers for Disease Control and Prevention, 2013b). Although women were more likely to be diagnosed with COPD, in 2009 the age-adjusted death rate in men was higher than that of women (American Lung Association, 2013). In 2011, over

ten million Americans reported a physician diagnosis of chronic bronchitis with almost 70% of cases occurring in those over 45 (American Lung Association, 2013). 4.7 million people reported being diagnosed with emphysema during their lives with more than 90% of the diagnoses being reported by individuals 45 years of age or older (American Lung Association, 2013).

The current study is of great public health importance because approximately twelve million American adults are diagnosed every year while another twelve million are thought to be living with COPD and do not know it. Furthermore, over 120,000 Americans die annually due to COPD (National Institutes of Health, 2010). In the United States, there is a great deal of concern over heart disease. However, deaths due to COPD increased 163% over 33 years, whereas deaths from cardiovascular disease decreased (Hurd & Lenfant, 2005).

Dusts in a work environment create breathing hazards and can lead to chronic respiratory problems such as emphysema and chronic bronchitis (Vallyathan, F.H., Brower, & Attfield, 1997). One study suggests the effects of occupational exposure to dust could be greater than that of smoking cigarettes (Balmes et al., 2003). The American Thoracic Society concluded that nearly 15% of COPD cases might be attributed to workplace exposures (Balmes et al., 2003). For example, many energy production occupations have been reported in previous studies to be high-risk occupations for the development of COPD (Cullinan, 2012; Esswein, Breitenstein, Snawder, Kiefer, & Sieber, 2013; Hnizdo & Vallyathan, 2003; Marine, Gurr, & Jacobsen, 1988; Mastrangelo, Tartari, Fedeli, Fadda, & Saia, 2003; Seixas, Robins, Attfield, & Moulton, 1992, 1993; Viegi, Scognamiglio, Baldacci, Pistelli, & Carrozzi,

2001). With the growth of the USA's efforts to become energy independent, more of these high-risk jobs are likely to be created.

Agricultural and textile jobs have also been shown to have a high exposure to dust (Dosman et al., 1980; Eduard, Pearce, & Douwes, 2009; Khan & Nanchal, 2007; Monso et al., 2004; Pahwa, Senthilselvan, McDuffie, & Dosman, 1994; Rushton, 2007; Schenker, 2000; Shi et al., 2010; Szczyrek et al., 2011; Wang et al., 2005). As we move from economic recession to a time of job creation, it is important to be aware of the potential hazards these jobs may have.

The costs associated with COPD are staggering. Nearly $50 billion is spent annually in healthcare costs related to COPD (American Lung Association, 2013). Of even greater concern is that the societal burden of COPD is anticipated to grow over the next thirty years (Viegi et al., 2001).

CHAPTER 2: LITERATURE REVIEW

The goal of this study was to examine whether occupational exposure to dusts is associated with increased odds for developing Chronic Obstructive Pulmonary Disease (COPD). This section provides an overview of relevant literature discussing the association between exposure to dusts and COPD.

To identify resources relating to the study topic, searches were conducted using the University of North Carolina at Charlotte's library database, Google Scholar, PubMed Central, and Cabarrus County's public schools research databases. Keywords used in these searches on exposure included the following: "Occupational exposure to dust" and "Occupational Hazards". Keywords used to search on exposure and outcome of interest included, "Dust and Chronic Bronchitis", "Dust and Emphysema", "Organic Dust exposure" and "Mineral Dust exposure". Data on recent statistics and trends were primarily obtained from the Centers for Disease Control and Prevention, National Institutes of Health, and the American Lung Association websites.

2.1 Overview of COPD

William Briscoe was the first person to use the term "Chronic obstructive pulmonary disease" in 1966 as part of the 9th Emphysema Conference held in Aspen Colorado (Petty, 2006). COPD is defined as "a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema" (Hnizdo &

Vallyathan, 2003). More recent definitions have used clinical criteria based on the ratio of FEV_1 and FVC (Vaz Fragoso et al., 2010; Vestbo, Hurd, & Rodriguez-Roisin, 2012). FEV_1, forced expiratory volume in 1 second, provides a measure of the volume of air forced out of the lungs in one second, and FVC, forced vital capacity, measures the amount of air one can forcibly breathe out after taking the deepest breath possible (Seixas et al., 1993).

Although there is an understood link between smoking and COPD (Balmes et al., 2003), it is now known that as many as 20% of COPD patients never smoked cigarettes (National Institutes of Health, 2010) which could suggest that COPD from occupational exposures is a problem (Blanc & Toren, 2007).

2.2 Mineral and Organic Dusts

Dust is everywhere. When items found in nature start to breakdown, tiny pieces are released into air. These tiny pieces include anything from dead skin cells to weathered rock. However, not all dust come from natural deterioration of substances. Such occupations as mining, farming, and construction generate considerable amounts of dust. The types of dust found in work environments include mineral dusts, which are mostly inorganic, and organic dusts.

The inhalation of mineral dust of respirable size is associated with lung diseases such as silicosis and asbestosis. Concrete workers, in particular, experience high levels of exposure to silica dust (Echt, Sieber, Jones, & Jones, 2002; Echt & Sieber, 2002; Ringen, Seegal, & Englund, 1995). Other occupational groups commonly experiencing disproportionally high levels of mineral dust exposure include coal miners, gold miners, and hydraulic fracturing workers (Esswein et al., 2013; Hnizdo, Sluis-Cremer, Baskind, & Murray, 1994; Marine et al., 1988; Seixas et

al., 1993; Vallyathan, Green, Brower, & Attfield, 1997).

Sources of organic dusts include baking flours, grains, wood, cotton, plants, and animals. Agricultural workers, especially cotton workers and grain workers, are exposed to high levels of organic dusts (Mastrangelo et al., 2003; Pahwa et al., 1994; Rushton, 2007; Shi et al., 2010; Szczyrek et al., 2011; Viegi et al., 2001; Wang et al., 2005).

In the United States, the Occupational Safety and Health Administration (OSHA) under the Occupational Safety and Health Act of 1970 is responsible for setting and enforcing occupational exposure limits (OELs) called permissible exposure limits (PELS) to protect worker's health. The National Institute for Occupation Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) also publishes their own non-regulatory OELs called Recommended Exposure Limit (REL) and Threshold Limit Values (TLVs®), respectively (Occupational Safety and Health Administration). The OSHA regulatory limit (PELS) for total dust exposure that is not otherwise regulated is 15 mg/m^3 (Occupational Safety and Health Administration, 2012). OSHA regulation 29 CFR 1926.55 sets the PEL for respirable silica to 0.1mg/m^3 however NIOSH has a recommended level of 0.05mg/m^3 (Centers for Disease Control and Prevention, 2014b; Occupational Safety and Health Administration, 2012).

The EPA characterizes dust as a form of pollution referred to as particulate matter (PM). PM is categorized based on the particle size which is defined as coarse (PM_{10}), fine fraction ($PM_{2.5}$), course fraction ($PM_{10-2.5}$) and ultrafine particles, which are defined as particles with a diameter less than 0.1 microns (United States

Environmental Protection Agency, 2015a). The Clean Air Act sets standards for PM and requires the EPA to review the standards periodically and update them as needed (United States Environmental Protection Agency, 2015b).

The size of the PM is very important as particles less than 2.5 microns in diameter (fine fraction particles) travel deeper into the lungs and ultimately into the alveoli, where they pose the greatest health risks (United States Environmental Protection Agency, 2014) . In emphysema the inner walls of the alveoli weaken and air pockets develop. The alveoli may eventually rupture reducing the amount of surface area in a person's lungs. Once the surface area of the lungs decreases, the amount of air absorbed per inhalation also decreases (Medinas et al., 2009).

2.3 Key Epidemiological Studies on Dust and COPD

It has been reported that exposure to dust can lead to respiratory dysfunction (Marine et al., 1988). Both biological dust (8.86 2.29-34.3) and mineral dust (3.80 1.21-12.0) were shown to increase odds for the development of COPD (Mastrangelo et al., 2003). A 5-year follow up of a cohort study found that exposure to organic dusts is associated with chronic bronchitis (relative risk 1.41 95%CI 1.28-1.55) (Bunger, Schappler-Scheele, Hilgers, & Hallier, 2007). Exposure to mineral dusts, like respirable crystalline silica, has been linked to several diseases including silicosis, lung cancer, kidney disease, and tuberculosis (Esswein et al., 2013).

Marine and colleagues (Marine et al., 1988) studied British coal-miners, focusing on the contribution of smoking and respirable dust to respiratory dysfunction. Four indices were used to evaluate respiratory function: FEV_1<80%, chronic bronchitis, chronic bronchitis with FEV_1<80%, and FEV_1<65%. As dust

exposure increased the prevalence of respiratory dysfunction, as measured by the four indices, also increased. Although the results provide insight into the effect of exposures to dusts on pulmonary function, because the study population from Marine et al. (1988) examined individuals that are not representative of the general public in the United States, their results can not be generalized to the US population.

Rothenbacher et al. (1997) examined chronic respiratory disease morbidity among men working in a German construction industry. The prevalence of COPD was 6.1% among all participants.

Blue-collar professionals in occupations with high likelihood of dust exposures includes carpenters, painters/varnishers, and plasterers, had almost twice the prevalence rate ratios as compared to white-collar professionals. However, the results were not statistically significant. The study had several limitations (Rothenbacher et al., 1997). First, it looked only at construction workers in a southern region of Germany. The data were collected from multiple clinics and the workers compensation board, but the diagnostic criteria for COPD were not the same among all clinics, which could introduce misclassification bias. Furthermore, information was not collected on smoking status among all the employees and the referent group was comparatively small. Not accounting for the smoking status of the study participants could bias results away from the null.

There have also been cross-sectional studies that explored associations between exposures to dust and airway obstructions (Hnizdo et al., 1994; Rodriguez et al., 2014). Hnizdo and colleagues reported no correlation between exposure to mineral dust and COPD, stating "the degree of emphysema found at necropsy was

not associated with a statistically significant impairment of lung function or with dust exposure (Hnizdo et al., 1994). The lack of association observed in the study could possibly be due to the fact it tested corpses, which may not give accurate results because small airways could not be established from the necrotic tissues examined. Being unable to examine these airways could have led to reporting of negative results. Furthermore, no information was provided on the level of dust exposure.

A cross-sectional study explored the link between occupational exposure to dusts, gases, and fumes(VGDF) with COPD (Rodriguez et al., 2014). Researchers conducted face-to-face interviews to determine the occupational history of participants while quality of life, dyspnea, and chronic bronchitis were determined through the use a validated questionnaire. Although it was not statistically significant, chronic bronchitis symptoms and scores from the St. George's questionnaire on symptoms were high among participants with high exposures to mineral dusts, gases, and fumes.

Another study exploring the link between occupation exposure to dusts, gases, and fumes with COPD explored used a case control design (Blanc et al., 2009). 302 controls were matched to the cases based on age, sex, and race and analyzed. Demographic data and self reported dusts exposures were assessed through the use of structured telephone interviews. In addition to the self reported exposure, a job exposure matrix, categorizing occupations as having low, intermediate, or high probability of exposure to materials associated with COPD, was used to the assess levels of exposure. Pulmonary function was assessed based on FEV_1 FVC ratio. Results showed that among those with exposures to VGDF alone had double the odds

of COPD as compared to those non-smokers without occupational exposures years (OR=1.98, 95% CI: 1.26-3.09).

Mineral dust exposure is only part of the problem. Biological dust has also been shown to affect pulmonary function. Several studies have researched biological dusts as occupational hazards (Dosman et al., 1980; Eduard et al., 2009; Khan & Nanchal, 2007; Monso et al., 2004; Pahwa et al., 1994; Rushton, 2007; Schenker, 2000; Shi et al., 2010; Szczyrek et al., 2011; Wang et al., 2005). In one cross-sectional study of grain-workers, the prevalence of chronic bronchitis in those exposed to dust, 23.1%, (p less than 0.01) was higher than in control subjects 3.3 % (p less than 0.01) (Dosman et al., 1980).

These studies often use study populations that are job specific. This limits the external validity of the studies as those results are only generalizable to participants in that specific occupation. These studies are also limited because they explore one form of dust exposure. Exploration of one, and not the other, does not account for any modification from a combined exposure to both. This study explored exposures to both inorganic mineral dusts as well as organic dusts.

The current literature exploring occupational exposure to dusts considers specific occupations. My study adds to the limited literature on studies conducted using a nationally representative population from the United States. This in itself serves to help minimize any bias that might be introduced by honing in on specific occupations.

2.4 Possible Biological Mechanism

Respiratory irritants can be deposited in the respiratory tract and other

adjacent pathways. Those which end up being deposited in the gas exchanging parts of the lungs cause injuries to the airway mucosal cells and the physiological responses cause secretions of mucus along with airway narrowing to the pulmonary tissue cells (Taylor, 1996). Obliteration within bronchioles has been shown to occur after the inhalation of irritants (Taylor, 1996).

Respiratory irritants are differentiated from sensitizers by their mechanisms. Irritants typically have a quicker onset and more of a toxic effect as compared to sensitizers that induce inflammation and more of an allergic style reaction (Taylor, 1996). Airway sensitizers fall into 4 main categories: chemicals, animal origin, plant origin, and microorganisms (van Kampen, Merget, & Baur, 2000). The major pathological reaction caused by organic dust is inflammation (Rylander, 2004). Through activation of macrophages, there is a secretion of inflammatory cytokines, which serve as messenger cells, and that signal for the migration of inflammatory cells into the lungs and the airways (Rylander, 2004). Inflammation is the human body's immune response to speed up the healing process. In this case the inflammation can lead to secretions, coughing, and swelling.

2.5 Summary

The current study aimed at exploring a possible association between occupational exposures to dusts as a precursor for the development of COPD. Many studies have investigated single occupations as risk factors of COPD, however relatively few studies have been done with a specific aim of determining the contribution of occupational exposures to COPD in the general public (Balmes et al., 2003).

CHAPTER 3: HYPOTHESES

This study examined the association between occupational exposure to mineral and organic dusts and COPD using the National Health and Nutrition Examination Survey (NHANES) 2007-2008 and 2009-2010. The study hypotheses were:

1. Occupational exposures to mineral dusts are associated with increased odds for developing COPD.
2. The odds of COPD increases with the increasing number of years of occupational exposure to mineral dusts.
3. Occupational exposures to organic dusts are associated with increased odds for COPD.
4. The odds of COPD increases with the increasing number of years of occupational exposure to organic dusts.

CHAPTER 4: RESEARCH METHODOLOGY

4.1 Study Design and Population

This study used data from the NHANES 2007-2010. NHANES was first conducted in the early 1960s and was designed to assess the health and nutritional status of adults and children in the USA. Although NHANES is conducted annually by the National Center for Health Statistics, a division of the Centers for Disease Control and Prevention (CDC), results are reported every two years (Centers for Disease Control and Prevention, 2013a). NHANES annually examines a nationally representative sample of about 5,000 persons from 15 counties across the country selected with a complex survey design (Centers for Disease Control and Prevention, 2014a).

NHANES is conducted in two stages. In the first stage, trained personnel conduct computer-assisted interviews with participants in their homes. In the second stage, a special assessment team conducts laboratory tests and physical examinations on the participants (Centers for Disease Control and Prevention, 2013a).

The interview portion includes questions about demographics, socioeconomic status, dietary habits, and health-related questions (Centers for Disease Control and Prevention, 2013a). The physical examination is comprised of physiological, medical and dental measurements as well as blood and urine laboratory tests conducted by medical staff (Centers for Disease Control and Prevention, 2013a). These

measurements are performed in specially equipped mobile centers, which travel throughout the country (Centers for Disease Control and Prevention, 2013a).

4.2 Merging and Appending

NHANES makes data available publicly on their website in small data files. First, the data files, including variables relevant to this study, were downloaded separately for 2007-2008 and 2009-2010 data cycles. The data files include demographic (DEMO), medical conditions (MCQ), occupation (OCQ), smoking - cigarette use (SMQ), and body measures (BMX). Next, the data files for the years 2007-2008 and 2009-2010 were merged separately using the unique identifier SEQN. Finally, the two two-year datasets were appended to create a four-year dataset, 2007-2010. For the 2007-2008 NHANES, 10,149 individuals were interviewed but only 9,762 were examined (Center for Disease Control and Prevention, 2013). For the 2009-2010 NHANES, 10,537 were interviewed but only 10,253 were examined (Center for Disease Control and Prevention, 2013).

Since COPD affects mostly middle-aged population, the analysis was restricted to respondents ages 40 and older, leaving the final sample size available for the analysis at 8,160 respondents.

4.3 Measurement of the Exposure Variable

The exposure variable in this study is occupational exposure to dusts. Data on this variable was obtained through the occupational screener questionnaire, from the 2007-2008 and 2009-2010 NHANES. The occupational questionnaire evaluates occupational dusts exposures and differentiates between exposures to mineral dusts and organic dusts. NHANES item number OCQ510 asked respondents if they ever

had work exposure to mineral dust and if in any job they had been exposed to dust from rock, sand, concrete, coal, asbestos, silica, or soil. NHANES question OCQ530 asked respondents if they ever had work exposure to organic dust and further qualified the exposure by asking if in any job had they been exposed to dust from baking flours, grains, wood, cotton, plants or animals. The response choices given were yes, no, refused, and don't know. The responses were coded 0=no and 1=yes. All other responses were coded as missing data. Frequency of exposure was determined by the respondents' answers to NHANES questions OCQ520 and OCQ 540. These two questions were follow-up questions for those who reported having been exposed to mineral dusts or organic dusts in NHANES questions OCQ510 and OCQ 530. OCQ 520 and OCQ 540 asked respondents for the total number of years for all jobs where they had been exposed. Respondents who reported not having been exposed were labeled never exposed and coded as 1. Other groups included <10 years of exposure (coded as 2), 10-19 years of exposure (coded as 3), 20-29 years of exposure (coded as 4), and 30 or more years of exposure was coded as 5. This process was conducted for both OCQ 520 and OCQ 540.

4.4 Measurement of the Outcome Variable

The outcome variable was measured by responses to NHANES questions MCQ160G and MCQ160K from the medical conditions questionnaire. Respondents were asked if a doctor, or another health professional, had ever told them they had emphysema or chronic bronchitis. The possible responses were yes, no, refuse, and not sure. The binary variable COPD was formed by coding positive responses to

question MCQ160G or MCQ160K as 1 and coding negative responses to both MCQ160G and MCQ160K as 0. Any other response was coded as missing data.

4.5 Sample Size and Power

Since the sample size was fixed, power calculation was conducted using the epi-calc power analysis calculator for cross-sectional studies (Soe, Sullivan, Dean, & Mir, 2015). Approximately 8,160 subjects of age 40 and above were available for the analysis. Based on the cross-tabulation of mineral dust exposure variable and COPD, a total of 2,288 respondents exposed to mineral dust and 4,716 were not exposed to mineral dust. The remaining 1,156 respondents were accounted for as missing data. Using the prevalence of COPD in the exposed group as 4.1% (Matheson et al., 2005) and the prevalence of COPD in the non-exposed group as 3.3% (Matheson et al., 2005) the power was calculated as 39.5%. Similarly, there were 1,513 respondents exposed to organic dust and 5,508 were not exposed to organic dust. The remaining 1,139 were accounted for as missing data. Using the prevalence of COPD in the exposed group as 5.4% (Matheson et al., 2005) and the prevalence of COPD in the non-exposed group as 2.2% (Matheson et al., 2005) the power was calculated as 100%.

4.6 Confounders

Eight variables were used as confounders. The age of the respondents was categorized into 4 groups: 40-49 years, 50-59 years, 60-69 years, and 70 or more years. The groups were coded 1, 2, 3, and 4 respectively. Gender was coded as 0=female or 1=male. The race/ethnicity variable was coded as Mexican Americans=1, other Hispanics =2, non-Hispanic white=3, non-Hispanic black=4, and

other race/ethnicity=5. Poverty-to-income ratio was used as a measure of socio-economic status (Center for Disease Control and Prevention, 2013). Responses below 1 were considered as living below the poverty (coded as 1) and those at or above 1 were considered living above the poverty level and were coded as 0. Respondent's Body Mass Index was categorized into 3 groups: <or=24.9 (normal), 25.0-29.9 (overweight), and 30 or more. BMI was coded 1, 2, and 3 respectively. An exposure to exhaust fumes was coded as 1 and no exposure was coded as 0. A categorical variable using responses to NHANES questions SMQ020 and SMQ 040 was created to classify respondents' smoking status. Question SMQ 020 asked respondents if they had smoked at least 100 cigarettes in their life and the responses listed were yes, no, refused, do not know, and missing. SMQ 040 asked respondents if they currently smoked cigarettes and the responses listed were everyday, some, not at all, refused, don't know, and missing. If respondents responded no for SMQ 020 they were grouped as non-smokers and coded 1. If respondents replied yes to SMQ020 and not at all to SMQ040 they were grouped as ex-smokers and coded 2. Any response of yes for SMQ 020 coupled with either a response of everyday or some for SMQ 040 were grouped as current smokers and coded as 3. Second hand smoke was derived from respondents' answers when asked if anyone smoked cigarettes, cigars, or pipes inside the house. Possible answers were yes, no, refused, do not know, or missing. Responses were coded 0=no, 1=yes. For all questions any response other then what was coded for was considered to be missing data and coded accordingly.

4.7 Constructing Weights

NHANES data are released in 2-year cycle. Since the analysis included four-year data, the 2007-2008 and 2009-2010 sample data sets were combined and four-year (2007-2010) weights were constructed using the following formula.

If sddsrvyr in (5, 6) then INT4YR = ½* INT2YR;

*SDDSRVYR is the survey cycle variable that identifies what years the data were collected from. In this case sddsrvyr 5 = 2007-2008 and sddsrvyr 6 = 2009-2010; WTINT2YR is the interview weight variable (Center for Disease Control and Prevention, 2013a).

A sample weight is an estimation of the number of people in the target population that a particular respondent represents. If an individual represents 10,000 in their subdomain then that persons sample weight is said to be 10,000. The formula simply shows that a respondent's 4-year weight would be half of their 2-year weight value. If the weight for the respondent illustrated above were their 2-year weight, their 4-year sample weight would then be 5000. These weights are sometimes referred to as final sample weights because they have been adjusted for differences in sampling rates, response rates, and different coverage rates among people in the sample. These adjustments are what allow the final sample weights to produce accurate national estimates from the sample (Center for Disease Control and Prevention, 2013a).

4.8 Statistical Analysis

The study population was described using frequencies and weighted percentages. Unadjusted odds ratios and 95% confidence intervals were calculated using univariable logistic regression analyses to examine the crude association between the exposure to mineral and organic dust and COPD as well as the crude association between the number of years of exposure to mineral and organic dust and COPD. In addition, other factors associated with COPD were identified.

Multivariable logistic regression analysis was used to calculate the adjusted odds ratio and 95% confidence intervals to determine the exposure-disease relationship while controlling for confounding. To select confounders for inclusion in the multivariable logistic regression analysis, a purposeful method of variable selection was used (Hosmer, Lemeshow, & Sturdivant, 2013). This method examined each variable for significance at different levels before including them in the final model. First, all variables significant at $p>=0.20$ at the univariate level were selected as candidate for inclusion in the multivariable logistic regression model. Next, variables with p-value >0.05 were removed from the model one-by-one until only the variables significant at $p<0.05$ remained in the multivariable logistic regression model. The main exposure variable remained in the final regardless of its statistical significance. Finally, the model was tested for fit using the Hosmer and Lemeshaw goodness of fit test (Hosmer et al., 2013). All analyses were conducted using Stata version 13.0 statistical software package (Stata Inc, College Station, TX) taking into account the complex survey design of NHANES.

CHAPTER 5: RESULTS

5.1 Descriptive Analysis

The data were summarized using frequency distributions. The weighted frequency percentages were calculated and recorded in table 1. The prevalence of COPD was 8.3%. Only 29% of the respondents reported ever being exposed to mineral dusts while at work while 19.6% reported exposure to organic dusts at work. Just over 32% of the population was 40-49 years old, 29.2% was 50-59 years old, 19.5% was 60-69 years old and 19% was 70 years old or older. Slightly more than 47% of the population was comprised of male subjects. Approximately 73.6% of the study population was Non-Hispanic White, 10.5% was Non-Hispanic Black, while 6.2% was Mexican American and the rest (Other Hispanic and Other Race) represented 9.7% of the 8160 individuals in the analysis. Participants with a normal BMI (<=24.9) comprised 27% of the study population, while 35.5% were overweight (25.0-29.9) and 36.1% were obese (>=30). Nearly 82% of the respondents reported their family poverty-to-income ratio to be at or above poverty and 10.1% were below poverty. 67.2% of the study population reported occupational exposure to exhaust fumes while only 29.3% reported exposure from other fumes. Just over half (51.7%) of the respondents were non-smokers, while 30.2% reported smoking in the past and 18.1% of respondents reported being current smokers. 83.6% of the respondents stated they were exposed to second-hand smoke. Close to 13% of respondents

reported their length of exposure to occupational mineral dust as less than 10 years. 6% said they were exposed from 10-19 years. Almost 5% had a length of exposure ranging from 20-29 years and 5.4% of respondents reported having been exposed to occupational mineral dusts for 30 or more years. Less than 10 years of occupational exposure to organic dusts was reported among 8.5 % of the respondents. 10-19 years of occupational exposure to organic dusts was reported in 4.2% of the respondents while only 3% were exposed from 20 to 29 years. 3.8 % of the respondents reported 30 or more years of occupational exposure to organic dusts.

5.2 Univariate Analysis

Table 2 displays the unadjusted odds ratios and the corresponding 95% confidence intervals that were calculated and summarized for each individual variable in comparison to a referent group. Respondents with occupational exposure to mineral dusts (OR=1.63, 95% CI:1.35-1.98) as well as respondents with exposure to organic dusts (OR=1.43, 95% CI:1.17-1.76) had increased odds of COPD when compared to respondents that reported no exposure. Individuals from each age group, 50-59 years (OR=1.19, 95% CI: 0.84-1.68), 60-69 years (OR=2.34, 95% CI: 1.68-3.28) and 70+ years (OR=1.91, 95% CI: 1.42-2.56), were all shown to be at increased odds for COPD as compared to individuals that are 40-49 years old. Although all age groups were at increased odds, only the results for respondents age 60 and older were statistically significant. Males were reported to have decreased odds (OR=0.65 95% CI: 0.50-0.84) for developing COPD when compared to female respondents. As compared to Mexican Americans, other hispanics (OR=2.06, 95% CI: 1.27-3.33), non-hispanic whites (OR=2.79, 95% CI: 1.96-3.97), non-hispanic blacks (OR=2.22,

95% CI: 1.46-3.36), and other (OR=1.98, 95% CI: 1.21-3.24) are all at increased odds for developing COPD. Considering BMI as a possible risk factor, respondents that were overweight were found to have nearly the same odds (OR=0.92, 95% CI:0.73-1.17) for developing COPD as compared to respondents with a normal BMI. Respondents who were obese were reported to have increased odds (OR=1.41, 95% CI: 1.12-1.79) of developing COPD in comparison to those with a normal BMI score.

Family poverty income ratio was used to assess social economic status among the respondents. When compared to individuals at or above the poverty level, individuals below poverty level (OR=2.04, 95% CI: 1.54-2.71) have just over twice the odds of developing COPD. Respondents that have had occupational exposure to exhaust fumes (OR=1.65, 95% CI: 1.28-2.12) have 1.65 the odds of developing COPD as compared to respondents with no exposure to exhaust fumes, while exposure to other fumes (OR=1.34, 95% CI 1.12-1.62) showed respondents to have 1.34 the odds of developing COPD when compared to those who are unexposed. Ex-smokers (OR=2.28, 95% CI: 1.78-2.93) and current smokers (OR=3.47, 95% CI: 2.75-4.37) both have considerably higher odds for developing COPD in comparison to non-smokers. Individuals exposed to second-hand smoke (OR=2.48, 95% CI:1.95-3.15) also have increased odds, 2.48 times higher than those unexposed, for developing COPD. Increased frequency of exposure to mineral dusts showed an overall linear dose response relationship but an increased frequency of exposure to organic dust did not show a linear increase. Individuals with any length of exposures did report increased odds for developing COPD, but the results were only significant among individuals reporting 10-19 years of exposure.

5.3 Multivariate Logistic Regression Adjusted Analysis

Tables 3 and 4 record the odds ratios associated with mineral and organic dust exposure as well as the frequency level of the exposures. The results reflect adjustments made for confounders such as age, sex, race/ethnicity, BMI, poverty-to-income ratio, exposure to exhaust fumes, smoking status, and exposure to second-hand smoke. Those respondents with reported exposures to mineral dust have 1.84 the odds of developing COPD. Increased odds were reported from each of the length of exposure groups, however the suspected linear increase in dose response was not witnessed. Increased odds for developing COPD were recorded among respondents that reported occupational exposure to organic dusts (OR=1.12, 95% CI: 0.87-1.44). However, these results were not statistically significant. Respondents that reported 10 or more years of exposure to organic dusts did have higher odds of developing COPD as compared to respondents that reported never having been exposed, but these results lacked statistical significance.

CHAPTER 6: DISCUSSION

6.1 Summary of Findings

After adjusting for confounders, the current study noted that individuals with occupational exposure to mineral dusts as well as those with occupation exposure to organic dusts had higher odds of developing COPD as compared to those who were unexposed. Not all of these results however were found to be statistically significant.

6.2 Comparison to findings from the previous research

Although studies have looked at relationships between the exposures to mineral and organic dust and COPD, the majority of these studies included exposures to vapors, and gas (VGDF) as part of the exposures (Blanc et al., 2009; Boschetto et al., 2006; Cullinan, 2012). Blanc et al. (2009) reported that self-reported VGDF exposure was significantly associated with more than double the odds of COPD (OR 2.11, 95% CI 1.59-2.82). Another study stated that VGDF was associated with COPD (OR 2.5 95% CI 1.9-3.4) (Blanc et al., 2009).

In the current study, the odds of COPD were significantly elevated in the univariable analysis for those exposed to organic dust as compared to those not exposed to organic dust (OR 1.43 95% CI 1.17-1.76). The odds ratio became statistically non-significant when adjusted for confounders (OR 1.12 95% CI 0.87-1.44). A few studies listed biological dust as an exposure much like the organic dust that served as an exposure in this study (Mastrangelo et al., 2003; Matheson et al.,

2005; Rodriguez et al., 2014). Although the results were not statistically significant, Rodriguez et al. (2014) interestingly reported a decrease in symptoms of chronic bronchitis with prolonged exposures to mineral dust (≤15 years OR 3.0 95% CI 1.5-6.1 / >15 years OR 1.3 95% CI 0.6-2.9). Mastrangelo et al. (2003) in a case-control study of workers reported significantly higher odds of COPD in individuals exposed to mineral dust (OR 3.80 95% CI 1.21-12.0) as compared to the referent group of office workers. However, a different study did not find a significant relationship between exposure to mineral dust and COPD (OR 1.13 95% CI 0.57-2.27) (Matheson et al., 2005). In the three exposure groups studied, biological dust, gas/vapor/fume, and mineral dust, mineral dust had the lowest OR among the three (Mastrangelo et al., 2003). Mastrangelo et al. (2003) also stated that the risk for COPD could have been overestimated due to preferential referral of patients with respiratory illness and past occupational exposure to their Institute.

The current study observed the dose-response pattern in the relationship of frequency of exposure to mineral dust to COPD, but only for respondents with <10 years (OR=1.66, 95% CI 1.16-2.38) of exposure to 10-19 years of exposure (OR=2.33, 95% CI 1.53-3.57). The odds ratio decreased to 1.48 (95% CI 0.87-2.49) for respondents with 20-29 years of exposure before rising again for respondents with 30 or more years of exposure. It is possible that the drop in the odds ratio observed for workers with 20-29 years of exposure is due to The Healthy Worker Effect. Some respondents may have dropped out of the workforce due to the illness and the ones who are left are healthy enough to continue working past 30 years.

The current study did not find any significant association between exposure to organic dust and COPD. In addition, no dose-response pattern in the association between the length of exposure to organic dust and COPD was observed. These results are in contrast to what are reported by other researchers (Mastrangelo et al., 2003; Monso et al., 2004; Pahwa et al., 1994). Monso et al. (2004) showed a higher prevalence of COPD among subjects with high dust exposure (1^{st} and 2^{nd} quartile 7.9%/4^{th} quartile 31.6%). These results were statistically significant even after adjustments for covariates were made (OR 6.60 95% CI 1.10-39.54). In a different study, the annual loss reported in FEV_1 (FVC) among grain workers that had been working less than 5 years was 9.2mL (21.1mL), but among those with 20 or more years the loss was 52.6mL (60.8mL) (Pahwa et al., 1994). One explanation for the discrepant results might be in the measurement of the exposure variable. The current study relied on self-reported information, which is commonly accompanied by recall bias. Not having a measure in which to verify the exposure could be linked to the different results from this study as compared to the results from previous studies. It is possible that the lack of a dose-response relationship is due to the healthy worker effect. Another possible explanation could be the presence of a threshold effect where once an individual has had a certain amount of exposure, further exposure may not affect the occurrence of COPD. There was an increase in the odds ratios from the first group (OR 1.11, 95% CI 0.82-1.50) to the second group (OR 1.89, 95% CI 1.25-2.84), however the threshold effect then shows no further increase in the next two frequency of exposure groups (OR 1.64, 95% CI 0.92-2.92) (OR 1.57, 95% CI 0.94-2.66).

6.3 Strengths and Limitations

Most of the studies that have been conducted on exposure to dust and COPD have focused on certain occupations. The use of NHANES for this study is a strength because this study examines data from a nationally representative population. Other than the use of data from the third NHANES and one other study using data from 1997-2004 NHANES, to my knowledge, there has not been a study using this national population based questionnaire (Bang, Syamlal, & Mazurek, 2009; Hnizdo, Sullivan, Bang, & Wagner, 2002).

Many studies that have looked at occupational exposure to dust and respiratory problems may have biased findings because of the healthy worker effect, a type of selection bias. Because individuals that are sick are no longer working, data is often only collected from healthy workers. Respondents' that self- reported having COPD may have left their jobs therefore only leaving the healthy workers to continue working and report more years of exposure to dusts. This healthy worker effect may have resulted in differential misclassification bias and the odds ratio may bias towards or away from the null.

In the NHANES questionnaires respondents were asked to self-report a diagnosis from either a doctor or another health care professional. Because the information is self-reported there is no way to check medical records and verify the diagnosis. This may have resulted in non-differential misclassification bias. Furthermore, recall bias may have led to further misclassification among respondents that were asked to report on incidents that happened years ago.

Although confounders were controlled through the use of a multivariable logistic regression analysis model, it is possible that some residual confounding could have taken place.

Unlike a prospective cohort study, where individuals are disease free to start and are followed until disease develops, cross-sectional studies rely on respondents to provide accurate data. Typically a researcher wants to establish that an exposure of interest is indeed what leads to the outcome. Reverse causation occurs when the outcome of interest is present prior to the exposure of interest. Because this was a cross-sectional study, all the information was gathered and analyzed at once. Not having an established causal timeline could lead to reverse causation and false reporting of the study results.

6.4 Future Research

Research should be expanded to further explore the effects of irritants and sensitizers. Taylor (1996) reported that although irritants tend to cause acute lung injuries, chronic airway limitations have been reported after short exposures to toxic concentrations of irritants. This exposes a need for studies that not only explore whether individuals were exposed, but future researchers should also look into the concentration of exposures.

The results from the current study did show individuals with exposures to mineral dust to have increased odds for developing COPD however with a low power it is hard to be sure if these results are real. Future researchers should continue to explore a possible correlation between mineral dusts and COPD. The low power of the current study makes the results inconclusive.

More research needs to be conducted on exposures to organic dusts. Although this study was unable to find a statistically significant association between occupational exposure to organic dusts and COPD, the odds ratios were elevated and warrant the need for more research.

6.5 Conclusions

Although the outcomes from this analysis did not produce the results anticipated, this research can potentially have a big impact in the public health field of COPD. Previous research has expressed a need for more population based studies like this one, however the results of this study imply these studies may not be as advantageous as first thought. The research methods used in the current study were sound. If the exposure were a big problem among a general population the current study would have identified it. Although the analysis for occupational exposures to mineral dust provided some significant results, due to the low power I am unable to conclude whether or not these results are real or by chance. Even with high power, when measured on a population-based level, the lack of any significant differences among individuals with occupational exposures to organic dusts suggest that exposures to dusts may not be a widespread problems. At a population-based level the exposure does not lead to COPD. These results may help in providing an explanation for the lack of population based studies on occupational exposure to dusts.

Among a large population there could be multiple exposures to dust and being unable to identify a specific source can be troublesome. Because of this, the results of the current study provide information that support further studies among

occupations that have already proven to be high risk or occupations that are thought to be high risk. Identifying a specific source of exposure allows researchers to hone in on and address the issues with occupational exposures and COPD.

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Table 1:Weighted descriptive analysis of variables

Variables	N	Weighted %
Mineral Dusts		
No	4716	61.5
Yes	2211	29.0
Organic Dusts		
No	5508	71.1
Yes	1502	19.6
COPD		
No	7400	91.3
Yes	727	8.3
Age Groups		
40-49	2057	32.2
50-59	1876	29.2
60-69	1960	19.5
70+	2267	19.0
Sex		
Male	3989	47.2
Female	4171	52.8
Race/Ethnicity		
Mexican American	1322	6.2
Other Hispanic	854	3.9
Non-Hispanic White	4060	73.6
Non-Hispanic Black	1572	10.5
Other- Including Multi-Racial	352	5.8
BMI (Values from NIH)		
Normal (<=24.9)	1925	27.0
Overweight (25.0-29.9)	2800	35.5
Obese (>=30)	3025	36.1
PIR (FPIR-family poverty income ratio)		
1.00 and above= at or above poverty	5962	10.1
0.00-0.99=Below Poverty	1365	81.8
Fumes		
Yes	1798	23.6
No	5228	67.2
Other Fumes		
Yes	2272	29.3
No	4752	61.6
Smoking		
Non-smoker	4079	51.7
Ex-smoker	2534	30.2
Current smoker	1542	18.1
Second Hand Smoke		
Yes	1375	16.0

No	6732	83.6
Frequency of exposure to Mineral Dusts		
Never Exposed	4716	61.5
<10yrs	945	12.6
10-19yrs	521	6.0
20-29yrs	383	4.9
30+yrs	428	5.4
Frequency of exposure to Organic Dusts		
Never Exposed	5508	71.1
<10yrs	654	8.5
10-19yrs	348	4.2
20-29yrs	216	3.0
30+yrs	284	3.8

N may not sum to total due to missing observations

Table 2: Univariate analysis

Variables	**Odds Ratio (95% CI)**
Mineral dusts	
No	Referent Group
Yes	1.63 (1.35-1.98)
Organic dusts	
No	Referent Group
Yes	1.43 (1.17-1.76)
Age	
40-49	Referent Group
50-59	1.19 (.84-1.68)
60 -69	2.34 (1.68-3.28)
70+	1.91 (1.42-2.56)
Sex	
Female	Referent Group
Male	0.65(0.50-0.84)
Race/Ethncity	
Mexican American	Referent Group
Other Hispanic	2.06 (1.27-3.33)
Non-Hispanic White	2.79 (1.96-3.97)
Non-Hispanic Black	2.22 (1.46-3.36)
Other- Including Multi-Racial	1.98 (1.21-3.24)
BMI (Values from NIH)	
Normal (<=24.9)	Referent Group
Overweight (25.0-29.9)	0.92 (0.73-1.17)
Obese (>=30)	1.41 (1.12-1.79)
PIR*ses*(FPIR-family poverty income ratio)	
1.00 and above= at or above poverty	Referent Group
0.00-0.99=Below Poverty	2.04 (1.54-2.71)

Exhaust/Fumes	
No	Referent Group
Yes	1.65 (1.28-2.12)
Other Fumes	
No	Referent Group
Yes	1.34 (1.12-1.62)
Smoking	
Non-smoker	Referent Group
Ex-smoker	2.28 (1.78-2.93)
Current-Smoker	3.47 (2.75-4.37)
Second Hand Smoke	
No	Referent Group
Yes	2.48 (1.95-3.15)
Frequency of exposure to Mineral Dusts	
Never Exposed	Referent Group
<10 yrs	1.41 (1.09-1.84)
10-19 yrs	1.86 (1.36-2.56)
20-29 yrs	1.47 (0.91-2.36)
30+ yrs	2.09 (1.38-3.17
Frequency of exposure to Organic Dusts	
Never Exposed	Referent Group
<10 yrs	1.11 (0.82-1.50)
10-19 yrs	1.89(1.25-2.84)
20-29 yrs	1.64 (0.92-2.92)
30+ yrs	1.57 (0.94-2.66)

Table 3: Multivarite adjusted analysis: mineral dusts and lenght of exposure

Variable	**Odds Ratio (95% CI)**
Mineral Dusts	1.84 (1.36-2.50)
Length of Exposure	
Never Exposed	Referent Group
<10 yrs	1.66(1.16-2..38)
10-19 yrs	2.33 (1.53-3.57)
20-29 yrs	1.48 (0.87-2.49)
30+ yrs	2.20 (1.25-3.88)

Table 4: Multivariate adjusted analysis: organic dusts and length of exposure

Variable	**Odds Ratio (95% CI)**
Organic Dusts	1.12 (0.87-1.44)
Length of Exposure	
Never Exposed	Referent Group
<10 yrs	0.85 (0.61-1.20)
10-19 yrs	1.53 (0.93-2.52)
20-29 yrs	1.25 (0.63-2.48)
30+ yrs	1.19 (0.63-2.24)

Analysis adjusted for confounding variables in tables 3 and 4
Confounders- Age, Gender, Race/Ethnicity, Family Poverty to Income Ratio, Body Mass Index, Exposure to Exhaust Fumes, Smoking Status, Second Hand Smoke