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Socioeconomic Status as a Predictor of Chronic Obstructive Pulmonary Disease

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Walden University
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Abstract

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by

Hilaire Saint-Pierre

MBA, University of Phoenix, 2010

BA, Cambridge College, 2003

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health

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Abstract

While epidemiological research has generated new knowledge about the treatment and prevention of chronic obstructive pulmonary disease (COPD) through smoking secession, the socioeconomic status (SES) of people with the disease has been under investigated. Thus, the purpose of this study was to determine the predictability of association between SES and COPD. This study was based on the ecological theory, which states that health and disease may have multiple underlying factors. This study was a secondary analysis of archived data from the Inter-University Consortium for Political and Social Research (ICPSR). The dataset was collected for the National Survey of Midlife Development in the United States (MIDUS)" study. Variables were measured at the nominal, ordinal, and continuous levels. In this cross-sectional quantitative analysis, logistic regression was used to inform the research questions. The results showed that neither education, income, nor occupation was a predictor of COPD. The logistic regression reported the significance of the predictability of education, income, and occupation to be $\rho = 1.000$, $\rho = .498$, $\rho = .581$, respectively, with odd ratios and confidence interval of 1.007 (.987, 1.028), 1.018 (.948, 1.094), 1.429 (.684, 2.988). Neither education, income, nor occupation yielded a significant statistic value for a $\rho < .05$ or a $p < .01$. These non-significant results regarding the relationship between the SES of a person and COPD reaffirmed that cigarette smoking remains the known determinant of the disease. The social implications of these research findings are that more stringent laws and mandates need to be enacted to discourage easy access to the proven determining factor of COPD: cigarettes smoking.

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Dedication

First, I dedicate this dissertation project to my friend, life companion, my wife Marie Sophia Saint-Pierre for her moral and emotional support all throughout this long journey. This manuscript is also dedicated to my two lovely children, Pascal and Hilarishah J. Saint-Pierre for their support, encouragement, patience, and understanding that daddy was not always available to play and have fun with them when they needed and requested it. Additionally, I devoted this manuscript to my long-suffering, provider, caring, and loving mother, Marie Mariela Saint-Pierre, and to all my brothers and sister, Maria Vita Saint-Pierre Charles, Sainteles Saint-Pierre, Saintaubin Saint-Pierre, Ronal Saint-Pierre who have encouraged me one way or another to create a path for present and future generations to follow.

Acknowledgments

I would like to express my sincere gratitude to each and every one who provided guidance and support during this rather extensive journey to complete this dissertation. First of all, my Lord, God, Jesus Christ my savior without whom it would not have been possible to even start this long endeavor. Also, I would like to thank Dr. Joseph Robare, my committee chair for his diligence, prompt feedback, support, and patience. Additionally, I thank Dr. Lee Caplan, my committee member for his insight, guidance, constructive comments and feedbacks, and patience. Last, but not least, I thank Dr. James Rohrer, the University Research Review (URR) member and Dr. Rea, the program director for their patience dealing with processes issues when they arose. Again, I want to thank my beloved wife, Sophia and my two lovely children, Pascal and Hilarishah for their support and kindness.

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Chapter 1: Introduction to the Study

Background, Risk Factors, and Burdens of COPD

In the last few decades, research in various disciplines, including public health and social sciences, have recorded unprecedented breakthroughs (Cataldi et al., 2014). These breakthroughs contributed significantly to improving the health of populations. In fact, a reversal of disease trend has been observed as infectious diseases were controlled effectively. However, chronic diseases became more difficult to prevent and treat (Alwan et al., 2010; Gegick, Coore, & Bowling, 2013). Chronic diseases became so complex in the 20th century and the beginning of this century that various government agencies, health organizations, educational institutions, private groups, philanthropic charities, and researchers in diverse disciplines are dedicating tremendous resources to generate knowledge useful in mitigating the challenges and burdens of chronic diseases (Humble, 2005; Pleasants et al., 2015; Tang, & Nutbeam, 2006.

In the course of life and health, genetic structures, behavioral activities, and social mobility and transitions are interconnected in such a way that diseases, especially chronic ailments, require special attention at the population level (Li et al., 2011; Lynch & Smith, 2005). Primarily, this study sought to learn whether socioeconomic status (SES) is a predictor of chronic obstructive pulmonary disease (COPD), a disease that affects the upper and lower airways. According to Barr and Elmahallawy (2012), COPD is the sixth and fourth leading cause of death in the world and in the United States. This inquiry sought to improve the limited scientific evidence that is available to improve the prevention and management of the condition within groups. The identification of group

differences, characteristics of their social lives and SES may help health professionals, policy makers, and people to provide more effective alternative approaches for treating COPD (Lamontagne et al., Bosse, 2013). As a result, people with COPD could enjoy life with less morbidity and premature mortality.

According to the World Health Organization (WHO) and the National Institutes of Health (NIH), COPD is supported to have multiple underlying causes, including environmental, behavioral, genetic, and other pathophysiological conditions (Kheirallah, Miller, Hall, & Sayers, 2016). The WHO (2012) estimated that the number of deaths from COPD would increase from 5.4 million in 2005 to 8.3 million by 2030. In the United States and other developed countries, exposure to cigarette smoke accounts for many cases (El-Zein, Young, Hopkins & Etzel, 2012). Research suggests that pipes, cigars, and other forms of tobacco use and smoke, especially when inhaled, can cause significant damage to the lungs and respiratory system (Eriksson, Lindberg, Müllerova, Rönmark, & Lundbäck, 2013). Second-hand smoke, for example, is a causal risk factor that cannot go unmentioned when respiratory conditions are considered (Lutz et al., 2015). Other hazards include air pollution, dust, work-related chemical exposures and fumes (Cunningham, Ford, Rolle, Wheaton, Croft, 2015). The use of woods and coals are observed in the Middle East, Asia, Africa, and poor countries among nonsmokers (people who do not smoke cigarette) (Cho et al., 2015; Kurmi, Semple, Simkhada, Smith & Ayres, 2010). The WHO (2011) reported that almost three billion people in the world use wood and coal that produce the organic material biomass fuel, which is found to be a determinant of COPD. For them, indoor air pollution is a significant risk factor for

COPD. In fact, three studies were able to dissociate COPD caused by cigarette smoking and other pollutants from chemical exposure and dust (Holguin, Folch, Redd, & Mannino, 2005; Hu et al., 2010; Parker et al., 2014; Regan et al., 2010).

Research shows that there exists external and internal predisposing risk factors related to COPD. Although uncommon, a genetic condition known as alpha1 antitrypsin deficiency may play a predisposing role in the disease development (Obeidat et al., 2015). This protein is made naturally by some organs in the body, such as the liver. People with the deficiency showed a lower level of antitrypsin than normal. When this condition is present in people, even second-hand smoke or mild air pollution can accelerate the disease process and speed up the complications (Castaldi et al., 2010). Finally, people with improperly treated asthma may develop COPD when inflammation of the upper and lower bronchioles persists (Pleasants et al., 2014). In effect, regardless of the etiology of the disease, its burdens remain unchanged on at-risk populations.

At the turn of the century, COPD was the fifth leading cause of death worldwide. It is predicted to rank third after cancer and heart disease by 2030 (Mannino & Buist, 2007; Parker et al., 2014). This ranking is largely based on data from high and middle-income countries. If not for the underreporting and underdiagnosing of COPD due to lack of health care access and other factors, COPD might rank even higher (Dilektasli et al., 2016). In 2005, 3 million people worldwide died from COPD, which accounted for 5% of the total death worldwide (WHO, 2011). In the United States, the age-adjusted death rate was 27.2 per 100,000 population in 2000 (Mannino & Buist, 2007). Low-income

countries have such limited resources that epidemiological data on COPD cannot be generated scientifically and reliably (McDonald et al., 2014).

All the above mentioned factors such as environmental, genetic, and behavioral contributing risk factors of COPD are important and need to be studied. They are well documented in the literature and may require more than health promotion programs to reduce the prevalence and incidence of COPD. This study's aim was not only to contribute to the limited scientific data on the effects of SES on COPD, but also to identify groups that are at greater risk of developing COPD, as well as to explore some new strategies to prevent the disease in those populations.

Problem Statement

COPD is one of the top five leading causes of death worldwide, especially in developed countries. In fact, empirical evidence showed that 15 to 25 million people are living with this debilitating condition in the United States alone which claims tens of thousands of lives annually (Mannino & Buist, 2007; Pleasants, Herrick, Liao, 2013). For decades, social science researchers have dedicated various resources and efforts to mitigate the physical, emotional, psychological, and financial burdens of COPD on individuals and populations (Begum et al., 2016; Eisner et al., 2011; Eriksson et al., 2013). The financial yoke of COPD not only affects individuals and families, but also places a significant burden on the health care infrastructure of the United States. The issue is so important that several entities—including the WHO , Healthy People 2010 and 2020, the NIH, together with government agencies and private sector organizations—have consolidated their resources to identify the various risks associated with COPD.

They do so in an effort to alleviate its many burdens on people and health structures (Aryal, Diaz-Guzman, Mannino, 2014; WHO, 2011).

In recent decades, COPD has seen significant breakthroughs due to the use of new, powerful, and sophisticated software, research methodologies, and study designs (Cox et al., 2009; Lu et al., 2016). COPD is one of the diseases that has recorded many research efforts. However, it was only in the last decades that researchers began to explore the probability of (SES) being associated with health in general and COPD in particular (García-Polo et al., 2012). Though ethnicity has proven to be an important variable related to health risks, only a few studies have considered it in their research on COPD (Begum et al., 2016; Long, Bamba, Ling, & Shea, 2006).

More and more research investigations have found inconclusive findings between COPD and SES (García-Polo et al., 2012). Yet some studies have yielded conflicting findings (Monteiro, Carvalho, Velho, & Sousa, 2012). These conflicting findings in the literature warrant more research to find the truth around an ecological concept of risks that may underline COPD. Therefore, this conflict creates an opportunity for my research to contribute knowledge to help professionals and people to understand on a larger scale the implications of SES on COPD.

Purpose of the Study

The purpose of this research study was to advance knowledge on the relationship between SES and COPD and to contribute to the limited empirical evidence (Li et al., 2011). Findings from this statistical analysis study have shed light on what was not known about COPD and have helped to equip health professionals, organizations, and

policymakers to make appropriate decisions in mitigating potential SES risk factors of the disease. In this study, I used the same three indications that other researchers have used to measure SES: education, income, and occupation.

Research Question and Hypotheses

Research Question

Question 1:

Is SES (education, income, and occupation) a predictor of COPD?

Research Hypotheses

Null Hypothesis 1:

*H0*1: The Income of a person is not a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Alternative Hypothesis 1:

*HA*1: The Income of a person is a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Null Hypothesis 2:

*H*02: The education level of a person is not a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Alternative hypothesis 2:

*H*A2: The education level of a person is a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Null hypothesis 3:

*H*03: The occupation of a person is not a predictor of COPD while age, sex, and race/ethnicity are controlled for.

*H*A3: The occupation of a person is a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Question 2:

Is race/ethnicity a risk factor of COPD and SES among different ethnic groups – White, Black, Native American, and Asian?

Null hypothesis 4:

H04: There is no risk difference for COPD and SES among different ethnic groups – White, Black, Native American, and Asian.

Alternative hypothesis 4:

HA4: There is a risk difference for COPD and SES among different ethnic groups – White, Black, Native American, and Asian.

Theoretical and Conceptual Framework

Theoretical Foundation

As research processes evolved, especially with new statistical software, methods, and designs, theories have always served as an important part in research. Over the years, novel theoretical perspectives became available to bring about new knowledge in social sciences and public health epidemiology (Crosby & Noar, 2010). In fact, the epistemology of science has recognized and supported the ecological models theory as an effective and comprehensive model for the complex understanding of phenomenological events that correlate with human behavior and health risks (Baral, Logie, Grosso, Wirtz

& Beyrer, 2013; Glanz, K. & Bishop, 2010). The ecological models transcend the notions that specific health risks are responsible for particular health conditions.

Research shows that the fundamentals of the theory is that people's behavior is conditioned at numerous stages of influence. The theory conditions behavior at the "intrapersonal (biological, psychological), interpersonal (social, cultural), organizational, community, physical environmental, and policy" (Glanz, Rimer, & Viswanath, 2008, p. 466). In other words, people's behavioral attitudes, beliefs, and actions must be comprehended in the complete context that individuals live. In recent years, many authoritative documents and organizational leaders in the fields of social sciences and public health (Healthy People 2010 and 2020, Institute of Medicine, WHO) have embraced the ecological models to fit various scientific research procedures (Glanz et al., 2008). Thus, ecological theory is an appropriate fit for this research on COPD, race/ethnicity, and SES; it helps to understand the impact of education, income, and occupation of individuals and health risks in different ethnic groups in the United States, including White, Black, Hispanic, and Asian.

In the past 3 decades, the ecological theory of health behavior has seen increasing use in various fields of research and practice due to its comprehensive concepts and approaches to behavior change, which is an important factor in reducing serious health conditions and prevalent health burdens (Baral et al., 2013; Glanz et al., 2008). The primary concept of the theory is that it can influence behaviors on multiple levels. The ecological theory facilitates the complex understanding of health determinants interacting together to induce ill-health. Also, and perhaps more important, the ecological theory is

used very often by researchers to develop strategies, guidelines, and intervention approaches that aim to change behavioral mechanisms (Baral et al., 2013). This theory can achieve significant results when policy, social support, healthful choices, and education interact positively toward the same goal of healthy populations and communities (Lee et al., 2014; Glanz & Bishop, 2010).

Conceptual Framework

Of all the novelty of the ecological theory recorded in the last years (see exhibit A), research identified four core principles that underlined behavioral change (Glanz et al., 2008):

1. Multiple levels of factors influence health behaviors. The understanding is that factors at various levels can have a significant influence on health behavior to include "intrapersonal, interpersonal, organizational, community, and public policy" (p. 470).
2. Influences interact across levels. This assumes that variables at different levels may interact to modify behavioral outcomes. For instance, motivation can make the difference between health and ill-health (p.470).
3. Multi-level interventions should be most effective in changing behavior. This principle is based on the concept that single-level interventions are not powerful enough to create sustainable health change in populations. For example, the combination of policy, education, and environmental supports should yield better outcomes than one intervention alone (p. 470).

4. Ecological theory is mostly powerful when it is behavior-specific. In this instance, if a model is aimed at smoking behavior, research suggests that it is more likely to be effective than when multiple behaviors are targeted (p. 471).

Nature of the Study

In this dissertation activity, the quantitative nature of the study complied with the guidelines of research to help understand the implications of the different composites of SES (education level, Income level, and occupation) and race/ethnicity with the condition being considered, COPD. Quantitative analyses are commonly used in the social sciences field to inform research questions and test hypotheses (Bernard, 2012; Creswell, 2009; Lee et al., 2014). Such research entailed the identification or development of an instrument to collect, code, manipulate, analyze data, interpret, and disseminate findings. For this dissertation a secondary dataset was identified at the national level to answer the above research questions and hypotheses.

Definitions

In social sciences, terms are specifically defined by researchers to avoid confusion and misunderstanding of variables in a study. The following definitions of the terms used in this study are supported in the literature.

COPD: A preventable and treatable disease of the respiratory system with some significant pulmonary effects that may contribute to its severity in individuals. Its pulmonary component is characterized by airflow limitation that is not fully reversible (Hurst & Wedzicha, 2007).

SES: The relative position of an individual on a hierarchical social structure, based on their access to or control over wealth, prestige, and power (Tang, Rashid, Godley, Ghali, 2016).

Education: Different definitions exist for education, especially in health research. In this study, education is referred to the process of receiving knowledge or information in a school or university. In other words, the formal teaching in a school or university that improves skills and judgment and prepares individuals to be self-sustainable. In this research study, two categories of education were considered: 1) those with some high school education or a diploma, 2) and those with some college education or a degree (Kasai, & Bradley, 2011).

Income: This is a commonly used measure of socioeconomic status of an individual in this country and in others around the world by private and government organizations. Three categories or levels of income are often identified and applied in social sciences research: low-income, mid-income, and high income. More details about income brackets will be explored in the following sections (Bjorklund, Lindah, & Lindquist, 2010).

Occupation: The type of work a person does to earn his income. Because occupation has so many categories, only five groupings were considered in this study, including white-collar and blue-collar workers. White-collar workers are professionals and highly trained individuals. Whereas blue-collar workers are low-skilled and manual trade individuals (Klein et al., 2009).

Race-ethnicity: The American Office of Management and Budget recognizes five racial and ethnic groups when collecting national data: White, Black, Asian, and Hispanic origin. These groups will be the focus of this research study (Holvino, E. 2008; Sarkar, Fisher, Schillinger, 2006).

Assumptions

The following assumptions underlie this cross-sectional quantitative study:

1. Based on preliminary research, SES has had significant effects on people with COPD, especially those who never smoke.
2. If more knowledge or light was shed on the understanding and the implications of SES on COPD, the population may be better cared for based on a larger perspective than simply behavioral risk factors.

Scope and Delimitations

The scope of this study was limited to men and women between the ages of 40 and 100 living in the United States. People must be able to read and write and report on their health. Only the following races were included: African American, White, Asian, and Hispanic. Those younger than 40 were excluded because they were less likely to develop COPD; those older than 100 were excluded because they were likely to have confounding comorbid conditions. Also excluded in the study were people who were institutionalized for whatever the cause or people who were in the military services camps.

Limitations

This study had some important limitations that are worth mentioning. First, the criteria used for the inclusion of survey participants did not require health care utilization for COPD diagnosis. So misclassification of COPD might have been compromised. Selection bias is another potential limitation of the study. It is possible that there were important differences between the participants and non-participants that could have modified the results. Also, the distribution in race/ethnicity participants were so disproportioned that an equal distribution of participation of race/ethnicity might have shown the COPD risks in other groups other than the African American strum. Furthermore, there was a substantial number of participants who did not report their COPD status. Had they reported their COPD status, the results could have been different. Biomass, environmental dust, and air pollution were not able to be analyzed in this study in order to differentiate between different occupations and the participants who reported to have been diagnosed with COPD. The study population ages ranged from 36 to 100 years old. The older population could have had other comorbidities that could have been confused with COPD (Collins, Onwuegbuzie, & Jiao, 2006; Toschke, Lüdde, Eisele, & Von, 2005).

Significance

The significance of this research depends on the findings. Based on the results that SES was not found to be a predictor of COPD, cigarette smoking remains the primary determining factor known to be associated with the disease. This study is more proof to law makers to work with other sectors in enacting even more serious laws and

policies against cigarette smoking habits whether in public or in private homes. Thus, the forecasting incidence and prevalence rates of the disease might be positively impacted in preventing the disease in populations. Findings from this statistical analysis are expected to equip health professionals, organizations, and policymakers to make appropriate decisions to encourage healthy behaviors through education programs.

Summary

COPD is a complex respiratory system disease that places serious physical, financial, and emotional burdens on the American people and the world. Early research showed that cigarette smoking is a major determining factor of the disease. Yet, new research methods in the last few decades suggest that many diseases might have non-explored underlying factors. In fact SES is one factor that experts have looked at regarding a variety of diseases and found some association. In light of the latest information in the literature, this quantitative logistic regression analysis expects to shed light on whether SES is a predictor of COPD.

In Chapter two of this research study, a detailed review the literature of most recent studies available on the relationship between COPD and SES. The evidence shows that there is an existing gap in the literature, making this study important. Additionally, an in-depth theoretical framework served as the groundwork of the research study. Chapter three displayed the complete statistical plan, including the method and tests that are used to answer the research questions. Chapter four reports the research results and chapter five provides the interpretation of the results and answers of the research questions.

Chapter 2: Literature Review

Introduction

This research study investigated the relationship between COPD, the dependent variable, and SES, the independent variable on the U. S. population. The purpose of this research study was to contribute to the evidence on the relationship between SES and COPD, a disease that affect millions in this country alone and far more abroad particularly in low-income countries. Increased knowledge will help health care professionals, public health providers, policy makers, and patients better understand the risk factors underlying COPD and may help prevent or lower the prevalence and incidence of the disease.

It is urgent that researchers dedicate more time to this issue in order to bring the existing gap in the literature closer. The few journal articles that are found in the literature at times conflict with each other in their findings and conclusions. For instance, Zeng, Sun, and Zhong (2012) identified wide variations of research results across nations to be associated with the research designs used, the operational definition of COPD, and the diagnostic criteria applied data during the research. Conversely, in a meta-analysis, Castaldi et al. (2010) found no association in studies to substantiate any association of COPD with SES.

In this chapter, the following topics are covered: a) detailed search strategy, including the terms, databases, used and the reasons these terms were chosen over others. b) An extensive discussion of the study's framework: the major theoretical propositions

and hypotheses of the ecological models theory, c) the literature review on the key variables and constructs being investigated in the study

Literature Search Strategy

In identifying the literature for this review, I used the following databases: Academic Search Complete, CINAHL Plus with full text, Cochrane Central Register of Controlled Trials, Database of Abstracts of Reviews of Effects, Education Research Complete, Google Scholar, Health and Psychosocial Instruments, Health Technology Assessments, MEDLINE with full text, ProQuest Central, Science Direct.

The keywords and combinations I used in the process were as follows: SES, COPD, SES and COPD, race/ethnicity and COPD, health disparities and COPD, educational disparities and COPD, education and COPD, income disparities and COPD, income and COPD, social determinants and COPD, environmental determinants of COPD, SES and race/ethnicity and COPD, epidemiology of COPD, and trends in COPD. In addition, the acronym COPD is spelled out in every single search as an alternative search possibility. Since the search of the last five years yielded few articles, the search was expanded to the last seven years. The following table lists the number of articles from the last seven years for each search term generated by all nine databases previously mentioned. Some articles are found in more than one databases and the terms SES and COPD were constantly used together in all nine databases to limit the specificity of engine searches regarding the relatedness of both terms. Because some authors use SES and others socioeconomic status in publications, both terms are used in search engines. In effect, as it can be observed in the table below, the yielded different articles that the use

of only one term did not. Three thousand seven hundred twenty eight articles were found with the terms used within the last seven years. However, after careful examination of the articles, only 174 were qualified and fitted for my research. During the actual review of the articles, another handful was found to be less useful than others and was discarded.

Table 3

Number of hits for each keyword

Terms used	Number of hits
Socioeconomic status and COPD	39
SES and COPD	18
Education and COPD	07
Income and COPD	09
Occupation and COPD	13
Social determinants and COPD	11
Environmental determinants and COPD	10
Epidemiology of COPD	18
Trends in COPD	22
SES and chronic obstructive pulmonary disease	27

Theoretical Foundation

As research processes evolved, especially with new statistical software, methods, and designs, theories have always served as an important part in research. Over the years,

novel theoretical perspectives became available to bring about new knowledge in social sciences and public health epidemiology (Crosby & Noar, 2010). In fact, the epistemology of science has recognized and supported the ecological models theory as an effective and comprehensive model for the complex understanding of phenomenological events that correlate with human behavior and health risks (Baral et al., 2013; Glanz, K. & Bishop, 2010). The ecological models transcend the notions that specific health risks are responsible for particular health conditions.

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supports should yield better outcomes than one intervention alone (p. 470). 4) Ecological theory is mostly powerful when it is behavior-specific. In this instance, if a model is aimed at smoking behavior, research suggests that it is more likely to be effective than when multiple behaviors are targeted (p. 471).

Literature Review Related to Key Variables and Concepts

Various researchers have established the impact of SES on health in the last few decades. Topics they covered include, health literacy, heart disease, stroke, asthma, hospitalizations and others (Izquierdo et al., 2010; Gray, Edwards, Schultz, & Miranda, 2014; Seabrook & Avison, 2012). There are few studies in the literature on SES and COPD. Adler and colleagues (2002) have identified that SES indirectly impacts populations' health: SES is associated with people's health outcomes – low and no-income people are likely not to have health insurance and access to physicians. Eisner and Blanc (2011) demonstrated that peoples of low SES and those of the black race are more likely to have worse health outcomes - hospitalizations, symptoms of COPD, morbidity and mortality. Furthermore, a study in China showed that SES is an independent risk factor for COPD, and that low household income and education are associated with higher prevalence of COPD even in the never smokers (Schauer, Wheaton, Malacher, & Croft, 2014; Yin, Zhang, Li, Jiang, & Zhao, 2011). Similar findings were reported by Miravitlles, Naberan, Cantoni, and Azpeitia (2011) that lower quality of life as a result of low household income was associated with higher risks of developing COPD than in the skilled and educated.

Risks of COPD

The risk factors of COPD range from factors that are controllable, such as smoking, to uncontrollable factors, such as genetic predisposition. Both categories of risk factors include genetic, education, occupation, and behavior hazards. In fact, Regan and colleagues (2010) and others (Brogger, Steen, Eiken, Gulsvik, & Bakke, 2006; Nielsen, Barnes, Ulrik, 2015; Silverman, 2002) have explored the genetic and biological determinants of COPD in diverse scientific studies. Regan and colleagues agreed with other researchers that although smoking has a very strong association with COPD, not all smokers develop COPD in later life. Thus the importance of the interrelationship of genetic factors and modifiers that cause COPD in some but not in others. In this investigation, Regan and associates studied 10000 smokers, with two-thirds being non-Hispanic Whites and one-third being African Americans, to determine the level of severity of disease among the non-Hispanic Whites versus African Americans. The statistical power of the genetic variants would be guaranteed by the large pool of participants. The COPD cohort was established by a longitudinal-type follow up to determine mortality, comorbidity, and disease severity or levels. Pulmonary function test (PFT) and lung imaging were the two main tests used in the study. The authors concluded that an array of genetic variants may play a role, and that more specific studies are warranted to determine the specific COPD ties to the genetic factors.

A meta-analysis was performed to study the genetic association to COPD (Castaldi et al., 2010). The investigators found inconsistencies in studies that made it very difficult to interpret the findings. They identified 207 genetic variants that were found to

be associated with COPD, though the majority of the variants were inconsistently replicated for conclusive association except for *SERPINA1*, the gene that encodes the alpha-1 antitrypsin protein. However, the conclusion of the meta-analysis has found enough statistical significance and study power to show confidently strong association of SERPINA1 to COPD susceptibility. To support their findings, Castaldi and colleagues reviewed four previous meta-analyses studies that supported the fact that genetic variants are associated with COPD (Castaldi et al., 2010).

Education and Risk of COPD

Education plays a vital role in the epidemiology of COPD as it influences health decisions of populations. In the last three decades health education has played a pivotal role in research as a means to determine the effect of education on COPD (Bandura, 2004; Minkler, 1989; Crosby & Noar, 2010; Wheaton et al., 2016). In an early quantitative research study, a group of investigators evaluated the impact of education on coronary heart disease among three pairs of communities over a period of six years (Luepker et al., 1994; Vijayan, 2013). According to Vijayan and associates, many components of the risk reduction educational program were effective in targeted groups. However, they concluded that the area of research must continue to explore the various modifiers to determine the real impacts of education on the subjects. Within the same philosophical concept of education and health promotion, in a different study, peer education theory has evolved, and studies have been conducted to decipher its impact on population's health (Turner & Shepherd, 1999). The authors concluded that a lack of

evidence and statistical significance prevented them from speculating on the effectiveness of peer education on health outcomes.

A meta-analysis was conducted by another group of researchers to determine the benefits of disease-specific health education on the risk of COPD (Blackstock & Webster, 2007). Thirteen articles conformed to the inclusion criteria. The articles were assessed by two independently contracted individuals. These articles described ten randomized controlled trials. According to the investigators, the articles reported on a rather broad variety of outcomes, which in turn made it somewhat difficult to understand and interpret most measures considered in the review. However, they found that didactical education has minimal effects on physical function, quality of life, and healthcare utilization. The results lacked statistical significances due to insufficient power; suggesting the need for more studies in the area to determine true significance and impacts of education on COPD outcomes.

Although the empirical evidence from many studies of an association between health education or formal education and COPD is not very strong due to small sample sizes and other factors, its minimal associations in different studies substantiate that education is related to disease and specifically to COPD. In fact, studies show that mortality and morbidity increased in people with a low level of education (Pleasants, Riley, & Mannino, 2016; Meara, Richards, & Cutler, 2008). For instance, Meara and colleagues found that the more educated had a 30% greater life-expectancy than those with lower education. Even in the smokers, people with high levels of education had a 20% greater life-expectancy than their counterparts with low education, after controlling

for other confounders. Therefore, the investigators suggested that a better and larger effort be placed on educational programs that target the low education populations to mitigate and close the existing gap.

Lifestyle and Behavioral Risks of COPD

Since the beginning of established medicine and later public health, behavior has always played a decisive and consequential role in disease development. An innumerable number of studies can testify to that, especially with regards to COPD and other respiratory diseases and conditions (Choi, 2012; Mannino, 2016; Michalopoulos, 2005; Pleasants et al., 2006;). In 2004 worldwide, COPD claimed the lives of 1,772,580 people, caused 47,232,000 disabilities, and 520,000 hospitalizations, and was responsible for $24.9 billion in costs (Zaher, Halbert, Dubois, George, & Nonikov, 2004). Smoking is not only strongly associated with COPD and other lung diseases, but it is also associated with coronary heart disease. Though it is common knowledge that smoking causes debilitating diseases, not all smokers will develop COPD or other smoking-related conditions. More importantly, COPD is often diagnosed in people who never smoked (Bakr & Elmahallawy, 2012; Lamprecht et al., 2011; Zeng et al., 2012). For example, Hardie and colleagues (2002) found that the standard definitions of COPD may yield false diagnoses of COPD in some never-smokers. Because COPD diagnosis is based on less than 70% for the amount of volume of air exhaled in one second (FEV1) divided by the forced vital capacity (FVC) (FEV1/FVC) of a patient compared to his or her predicted volume based on age, gender, and height, diagnostic flaws may invade the results. For instance, Hardie and associates found that 208 healthy respondents to a survey of “never-

smokers" without any signs of cardiovascular and respiratory diseases had been diagnosed with COPD due to the measurement scales used to rate the severity of the disease. The investigators concluded that the criteria for the Global Initiative for Chronic Obstructive Lung Disease (GOLD) needed to be age-adjusted to reflect a non-bias method that is more reliable.

Why do some smokers develop COPD, while others do not? What are the implications of a person's anatomy and physiology in this complexity? Some researchers offer a valuable explanation as to the reason for that difference within smokers. In their study, Rusznak and colleagues (2000) shed light on the physiological condition of the human bronchial epithelium cells in respect to cigarette smoke effects in the lungs. Biopsy cultures were collected from people who never-smoked and had normal pulmonary function tests, from smokers with normal pulmonary function tests, and from smokers with COPD on biopsy. The biopsy materials were then exposed to 20 minutes of cigarette smoke-saturated-air. After 24 hours, measurements were taken "of transepithelial permeability and release of interleukin (IL)-1 β and soluble intercellular adhesion molecule-1 (sICAM-1)" (Rusznak et al., 2000, p. 1). The authors found that the cigarette smoke increased the permeability of cells in all three groups, with the largest permeability effects observed in the biopsy materials of those smokers with COPD with a mean increase of 85.5%, and the weakest effect found in smokers with normal pulmonary function tests with a mean of 25%, which was surprisingly lower than the effect in never-smokers (53%) ($p < 0.001$). The investigators concluded that the cigarette smoke, which contains inflammatory agents like ozone, nitrogen dioxide, and other harmful particles,

was associated with tumor necrosis and inflammation of tissues that may induce the development of COPD in the never-smokers.

Occupational and Environmental Risks of COPD

Anecdotal and empirical evidence suggest that the risks of developing respiratory diseases and airway inflammatory conditions are associated with second-hand smoke (SHS). In a quantitative study of 189 subjects wearing a nicotine badge for seven days following three months of previous environmental exposure to cigarette smoke, Eisner and colleagues (2005) measured the hair nicotine levels, health status, and airway disease severity of the participants. Sixty to 83% showed positive exposure to SHS on biopsy of the lung tissue. The highest level of nicotine as measured by the badge was found in subjects with the highest asthma severity (subjects that do not respond to bronchodilator medications), which depended on the exposure length of time (five years or more). The investigators found that the risk of exposure to SHS was comparable to the general population, and that exposure to SHS was associated with more severe cases of asthma exacerbations and hospitalizations. This study will increase the awareness of SHS impact on individuals, which may help to advance the agenda of prohibiting smoking in the United States. The political and economic implications of such an agenda are beyond the scope of this study.

In a multivariate analysis study of 1202 subjects of different ages, races, sex, and smoking histories, Blanc and associates (2009) measured the effects of vapors, gas, dust, and fumes (VGDF) on the airways of COPD and non-COPD persons. Cigarette smoking was controlled to determine the actual prevalence of airway diseases associated with

occupational hazards independently. Two important factors were calculated to determine relevant associations with VGDF, the odds ratio (OR) and the adjusted population attributable fraction (PAF). VGDF was found to increase the risk of COPD (OR 2.11; 95% CI 1.59 to 2.82 and a PAF of 31%, 95% CI 22% to 39%). There was a synergistic effect of workplace irritants and COPD (OR 14.1; 95% CI 9.33 to 21.2). Therefore, policies regarding the workplace environment should be enacted to restrict exposures that are harmful to employees and others.

Biomass smoke is another factor that experts study when COPD is being studied. A handful of studies yielded conflicting results regarding the association of biomass smoke and COPD (Harris & Debolt, 2008; Hu et al., 2010; Pastor, Aber, & Melillo, 1984) . A group of scientists conducted a meta-analysis of these studies (Hu et al., 2010). Two databases, MEDLINE, and EMBASE, were searched for studies of Latin American and Caribbean women and men that were exposed to biomass smoke. The meta-analysis found positive associations for both women (OR, 2.73; 95% CI, 2.28-3.28) and men (OR, 4.30; 95% CI, 1.85-10.01). Additionally, studies of Asian and non-Asian populations found positive associations (OR, 2.31; 95% CI, 1.41-3.78), (OR, 2.56; 95% CI, 1.71-3.83), respectively. Results also showed strong associations between risk of biomass and COPD in both cigarette smokers and non-cigarette smokers (OR, 2.65; 95% CI, 1.75-4.03), cigarette smokers (OR, 4.39; 95% CI, 1.40-4.66) and non-cigarette smokers (OR, 2.55; 95% CI, 2.06-3.15). The authors advised public health departments to develop measures to minimize the risks of biomass, especially in the rural areas and in third world countries where biomass is an essential part of cooking in some populations.

Physical Burdens and Functional Limitations of COPD

In the literature, it is very well established that burdens of COPD encompass psychological, financial, and physical. As a systemic disease, COPD affects the whole person, not simply the respiratory system. Research by Eisner and Blanc (2008) detailed some of the physical functional limitations associated with COPD. In a cohort study of 1202 subjects with COPD, the investigators used validated instruments to collect data and measure physical functionality. For example, "lower extremity function (Short Physical Performance Battery, SPPB), submaximal exercise performance (Six Minute Walk Test, SMWT), standing balance (Functional Reach Test), skeletal muscle strength (manual muscle testing with dynamometry), and self-reported functional limitation (standardized item battery)" (p. 11) were some physical functionalities that were measured. A multivariate analysis was conducted to control for confounders like age, sex, SES, smoking, and others. The following excerpt from the manuscript shows the consequences of the disease: COPD was found to be "associated with poorer lower extremity function (mean SPPB score decrement for COPD vs. referent -1.0 points; 95% CI -1.25 to -0.73 pts) and less distance walked during the SMWT (-334 feet; 95% CI -384 to -282 ft). COPD was also associated with weaker muscle strength in every muscle group tested, including both the upper and lower extremities ($p<0.0001$ in all cases) and with a greater risk of self-reported functional limitation (OR 6.4; 95% CI 3.7 to 10.9) (Eisner & Blanc, 2008, p.23).

Another research study was conducted to characterize the physical functional limitations of COPD in remote organs from the lungs. This study was aimed to evaluate

the ecological model theory that COPD may cause general disability in persons. Eisner, Iribarren, and colleagues (2008) analyzed data from 2198 subjects ages 40-65 with COPD. They used previously validated instruments to measure physical functionality (FLOW) and linear regression to evaluate the model assumptions. The relation of tests and functional limitations were analyzed by logistic regression based on the nature of the variables, whether they were categorical or continuous. Results demonstrated that COPD levels of increased severity were associated with lower physical functional disabilities.

Additionally, studies showed increased association of mortality with COPD and the incidence of cardiovascular disease (CVD). In a retrospective matched cohort analysis, Sidney and others (2005) followed 45,966 participants for a four-year period in regards to hospitalization and mortality-related cases. The findings showed that CVD, a comorbidity in the presence of COPD, increased the relative risks of mortality with a relative rate of 2.09 for hospitalization (95% confidence interval [CI], 1.99 to 2.20) even after they adjusted for gender and other important factors. Also, they found that younger adults 65 or less were at greater risk of COPD than adults over 65 (Sidney et al., 2005,).

Psychological Burdens of COPD

COPD burdens are not limited to the physical functional limitations but also extend to more complex psychological forms that require expert analysis to elucidate them. Multiple empirical inquiries have demonstrated associations between COPD and psychological limitations. For instance, Ng and colleagues (2007) evaluated the impacts of comorbid depression on mortality, hospital admission, smoking, and other respiratory symptoms in COPD patients. In a prospective cohort study, 376 patients were followed

for one year to shed light on the baseline association of the comorbid depression and mortality after adjustments for relevant confounders. Multivariate analysis showed significant association between depression and mortality (hazard ratio, 1.93; 95% confidence interval, 1.04-3.58" (Ng et al., 2007). The investigators then concluded that those with comorbid depressive symptoms had poorer survival, persistent smoking, and longer hospital stay than those without the comorbid depressive symptoms.

Although COPD is less common in groups with higher education and income than in those with lower education and income, the likelihood of having the depressive co-morbidity seems to increase in people with higher education and income (Lin, Chen, & McDowell, 2005). A meta-analysis examined 44,963 Canadians ages 35 and older from surveys between 1996 and 1997. Logistic regression was used to determine whether an association existed between COPD and depressive symptoms while the investigators controlled for the main confounders, education, income, and gender. Additionally, a bootstrap procedure was used to weight the sample. The association was stronger in well-educated men (OR = 2.60, 95% CI = 1.55, 4.38) than in their counterparts with less education (OR = 1.19, 95% CI = 0.47, 3.05) (Lin et al., 2005). Also, an increased association was observed when comparing women with higher household income to their counterparts with lower income (OR = 4.57, 95% CI = 2.27, 9.19). In sum, men with higher education and income were found to be at greater risks of depression than their counterparts with lower education and income.

Because of depressive symptoms in people with COPD, researchers explored the impacts of pulmonary rehabilitation (iPR) programs that have the potential to mitigate the

quality of life of COPD patients (Garuti et al., 2003). In one prospective analysis, Garuti and colleagues studied the effectiveness of a rehabilitation program in 147 adult people with COPD. The sample was grouped into three based on age, and a standardized validated scale was used to measure dyspnea (D) and fatigue (F) after a six-minute walk. The quality of life was evaluated by using the St. George Respiratory Questionnaire (SGRQ). The authors reported that after an acute exacerbation of COPD during inpatient hospitalization, comprehensive rehabilitation programs may improve the anxiety and depression levels, symptoms, and health-related quality of life in moderate to severe COPD patients in all groups.

Economic Burdens of COPD

Globally, hospitalizations seem to be the major cost driver in COPD patients, contributing between 52% and 84% of total costs (TEO et al., 2012). In a study of two public health clusters in Singapore, data on patients 40 and older were analyzed to determine the cost burden of COPD. According to Teo and his colleagues, for these two clusters alone, the approximate mean cost was $9.9 million per year. In effect, the authors found that the proportion of hospitalization costs decreased from 75% in 2005 to 68% in 2009. This study should help the allocation of resources, especially in sectors of Singapore. In the United States, different reports portrayed different figures. However, authoritative institutions like the National Institute of Health (NIH) and the National Heart, Lung, and Blood Institute (NHLBI) have estimated the official appraisal to be $38.8 billion in 2005, including hospitalization, emergency room service, home healthcare services, and durable medical equipment (Foster et al., 2006). Although there

is no solid figure as to the loss of wages due to disabilities, some researchers and experts agree that many people have to retire early and others miss work days as a consequence of their COPD, both of which are not included in the current estimates (Fletcher et al., 2011).

Theoretical Foundation

In the past three decades, the ecological theory of health behavior have seen increasing use in various fields of research and practice due to its comprehensive concepts and approaches to behavior change, which is an important factor in reducing serious health conditions and prevalent health burdens (Glanz et al., 2008). The primary idea of the model is that an ecological model can influence behaviors on multiple levels. The ecological theory facilitates the complex understanding of health determinants interacting together to induce ill-health. Also, and perhaps more important, ecological theory are used very often by researchers to develop strategies, guidelines, and intervention approaches that aim to change behavioral mechanisms. This theory can achieve significant results when policy, social support, healthful choices, and education interact positively toward the same goal of healthy populations and communities (Glanz & Bishop, 2010).

The contemporary ecological theory of health behavior have their roots in earlier models that focused heavily on the effects of the environment on people's behavior and choices (Barker, 1968; McLeroy, Bibeau, Steckler, & Glanz, 1988). More recent models (Cohen, Scribner, & Farley, 2000; Stokols, 1992; Stokols, Grzywacz, McMahan, & Phillips, 2003) have focused on health promotion and behavior applications. Some

contemporary models (Fisher et al., 2005; Glass & McAtee, 2006; Stokols et al., 2003) helped to fit numerous health behaviors. The proliferation and diversity of the ecological theory is self-evident of its effectiveness and adaptability to understand complex social and public health issues. Skinner's (1953) operant learning theory and Bandura's (2001) theory of social learning and social cognitive theories are clear examples of the diversity and improvement observed through the years of the ecological models theory.

Of all the novelty of the ecological theory recorded in the last few decades, there are four ideas or principles that underline behavioral change and can influence people's health (Glanz et al., 2008):

1) Multiple levels of factors influence health behaviors. The understanding is that factors at various levels can have a significant influence on health behavior to include "intrapersonal, interpersonal, organizational, community, and public policy" (p. 470).

2) Influences interact across levels. This assumes that variables at different levels may interact to modify behavioral outcomes. For instance, motivation can make the difference between health and ill-health (Tang, Rashid, Godley, Ghali, 2016).

3) Multi-level interventions should be most effective in changing behavior. This principle is based on the concept that single-level interventions are not powerful enough to create sustainable health change in populations. For example, the combination of policy, education, and environmental supports should yield better outcomes than one intervention alone (Tang et al., 2016).

4) Ecological theory is mostly powerful when it is behavior-specific. In this instance, if a theory is aimed at smoking behavior, research suggests that it is more likely to be effective than when multiple behaviors are targeted (Glanz et al., 2008). See Appendix A for a visual representation of the ecological theory's multiple levels of interaction.

Application of Ecological Theory

This theory has been applied in different research fields and practices to inform behavioral changes in individuals and more so in population-wide applications increasingly in the later years of the last century. For instance, Sallis and colleagues (2006) have reviewed some articles that relate the environment, health policy, behavioral sciences, and transportation to the perspectives of physical activity in light of the ecological theory. The researchers wanted to understand the lack of physical activity in the population using a multilevel approach by integrating factors such as safe neighborhoods, street lighting, walkways, and transportation policy. Similar studies were conducted by other researchers using the ecological theory to guide health promotion and community interventions (Briske, Fuhlendorf, & Smeins, 2005; Fisher-Owens et al., 2008; Giles-Corti, Timperio, Bull, & Pikora, 2005;;).

Diabetes, a major source of health burdens, including mortality, when not properly managed shows short-lived and declined benefits when strategies exclude the environment in which people live in (Norris, Lau, Smith, Schmid, & Engelgau, 2002). The ecological recommendation for diabetes management should include regular physical exercise, community education, clinicians support and regular visits, and a targeted diet

that can sustain long-lasting disease management. Another behavior to which the ecological theory has been applied successfully was tobacco control and smoking cessation, not only individually but also population-based. This approach involved education of the people, the health care providers' supports to remind smokers of the risks factors associated with such behavior, telephone counseling, and use of mass media (Services, 2005).

Conceptual Framework

In their case study of the HIV epidemic, Baral et al. (2013) used the ecological theory with a slight modification to put emphasis on the importance of understanding inequalities and risk factors at multiple levels. Some behaviors like needle-sharing and unprotected sexual encounters, especially between men and men, may increase the risk of HIV among persons. Unlike the health belief model and the theory of planned action that place emphasis on the individual underlying motivations, the ecological theory calls for a holistic collection of research data to help reduce and prevent the disease. Therefore, every aspect of a person is at play including the intrapersonal, interpersonal, community, and public policy to mitigate the risk factors.

The investigators continued to assert that of all these factors mentioned above, individual ones, which are biological or behavioral, are more heavily weighted in the research scale than education and income prior to the application of the ecological theory. Just like community organizations and groups can promote health and well-being, it can also be a source of stigma for persons. Another source of risk factor identified by the researchers was the impact of laws whether they are at the local, state, or federal level -

they can encourage or discourage the spread of HIV especially among sex workers. For instance, laws could allow alternative medicine practitioners to conduct nature-based research without being prosecuted under false pretense as they are currently.

In another exploratory study of obesity, one of the most threatening behavioral epidemics in the United States, Alber and Hamilton-Hancock (2012) explored the importance of the ecological theory in facing the increasing challenge of obesity. Similar to the previous HIV study, the researchers studied four levels of risk integration to inform the risk factors of the disease: intrapersonal, interpersonal, community, and societal to include policy. The study evaluated the obesity rates in states compared to the number of policies that deal with obesity. The results showed clearly that more ecological studies need to be done to understand risk factors associated with diseases, in this case obesity. In effect, a correlation coefficient of -.30 that suggests an inverse relationship between policies and obesity with a significant p-value <.05 supports the findings (Alber & Hamilton-Hancock, 2012)

More researchers in the last few decades have built upon the original ecological theory to present novel strategies for health promotions within populations (Bando, Moreira, Pereira, & Barrozo, 2012; Coutts & Taylor, 2011; Ogden, Davis, Jacobs, Barnes, & Fling, 2005; Richard, Gauvin, & Raine, 2011). Other researchers use the ecological model as a filter to evaluate the effectiveness of health promotion programs. For instance, a preliminary ecologic study of Aboriginal health promotion by Reilly and colleagues (2011) in Northern Victoria, Australia discovered that some nutrition programs in various schools' sports clubs in the region were more effective than others.

The more effective and better performers in sports are those that are in higher alignment with the ecological model and those with poor alignment performed poorly. Also, researchers relate the ecological model to the concepts of "micro, meso, and macro" level systems, which represent the individual, group, and population, respectively (Wharf Higgins, Begoray, & MacDonald, 2009).

The complete understanding of every variation in the complex environment in which people live is important for sustainable behavioral change. Every aspect of life, including education, policy, income, physical alteration of communities, types of employment, and social status, combine to make the complex determinants of health exposures and risk factors. Thus, it is important for this research study to evaluate the effects of peoples' SES on the incidence of COPD, while considering the variation of race/ethnicity, age, and gender. The ecological framework and concept put in question not only individuals' responsibilities, but also all the perceived determinants that are found in the living context of the socially disenfranchised.

SES and Health Outcomes

The construct SES is widely used in the literature as a means of conducting empirical investigations and understanding natural phenomena in social sciences fields to include public health epidemiology, health care, and medicine. This construct is tightly related to the theory of ecological models, which takes into account the influences of external and internal factors that may help to understand health outcomes in different lights. For example, Prescott, Lange, and Vestbo (1999) conducted a research study to determine the effects of peoples' SES on their lungs functions and hospitalization in

COPD. Conducted in Copenhagen, a random sample study included 14,223 subjects ages 20-90 years. They used linear regression techniques to analyze the collected data and establish the relationship between variables.

Education and income were the main variables examined, while confounders like smoking and genetics were controlled for. The investigators found that education and household income were significantly associated with hospital admission even after adjustment for smoking. For medium and high SES versus low SES, the education and income indices in "females were 0.74 (0.55±1.02) and 0.27 (0.10±0.73), respectively. Corresponding relative risks in males were 0.47 (0.36±0.63) and 0.35 (0.17±0.70)" (p. 7), respectively. The study clearly demonstrated that SES is significantly associated with COPD.

In 2009, Eisner and colleagues studied the independent effects of SES and race/ethnicity on COPD severity status and functional limitations of patients in a cohort of 1202 members of the Kaiser Permanente Northern California Medical Care Plan with COPD. This study examined confounders (comorbidities, smoking, body mass index, and occupational exposures) that could skew the results and interpretation of the study outcomes. People with lower income and people with lower education had a greater risk for more severe levels of COPD than their counterparts with higher education and income: HR 1.5; 95% CI 1.01 to 2.1; and HR 2.1; 95% CI 1.4 to 3.4, respectively.

SES seems to be associated with every aspect of health and even death. In fact, a study was conducted in 2005 by a group of investigators who wanted to shed light on the relationship between SES and early death in populations (Power, Hyppönen, & Davey

Smith, 2005). This prospective study analyzed the socioeconomic position of women's childhood to find any correlation between their death and their childhood SES. In a study sample of 11855 British women aged 14 to 49 years, the study results confirmed the investigators' theory that the number of years that people spent in low SES during their childhood increased their risk of early mortality compared to their counterparts who had higher SES. According to the researchers, all-causes of death, including circulatory disease, coronary disease, respiratory disease, and chronic obstructive pulmonary disease, were controlled for to increase the validity of the findings. The results showed that women with low childhood socioeconomic positions died earlier than those with higher socioeconomic positions: women's age-adjusted hazard ratios were 1.95 (95% CI = 1.58, 2.40) and 2.32 (95% CI = 1.72, 3.14) for circulatory disease and other diseases.

Pharmaceutical breakthroughs in the medical setting can help to reduce health burdens, physical limitations, and psychological impairments imposed by disease on populations. Evidence shows that social inequalities have always been an influence on health outcomes, especially with respect to affording new medications and technologies. Fleischer, Diez Roux, Alazraqui, Spinelli, and De Maio (2011), performed a cross-sectional analysis of 427 subjects to determine the odds of low SES people accessing and using the novel medication tiotropium for their condition (COPD). Forty four subjects (10.3 %) reported the use of the new drug (OR 0.3; 95% CI 0.1–0.7; p = 0.005). Even after adjustments for confounders and covariates, the results remained the same to show that low SES is strongly associated with low income and fewer means to afford the new drug that could help lessen some of the disease burdens.

Allopathic and Pharmaceutical Management of COPD

Although the pharmaceutical industries have recorded many breakthroughs in the last few decades in medicine and technology, very limited successes in cure have seen the light of day. On the conventional side of medicine, various therapies, home medications, and treatments have been developed to help people with symptoms of their diseases, but nothing thus far is known to reverse the pathological and physiological conditions that COPD creates. Bronchodilators and anti-inflammatory drugs are the best things ever developed for patients with airway diseases and airflow limitations. On the natural and naturopathic side of medicine, which does not have a favorable stance with conventional medicine, many experts claim to have demonstrated that some naturally derived minerals can have reversal effects on several of the deadly and debilitating diseases of today, whether diseases occurred naturally, were man-made, or were laboratory created for purposes such as warfare. There are five categories of drugs that are commonly used in the treatment of COPD: short-acting bronchodilators, long-acting bronchodilators, corticosteroids, phosphodiesterase, and methylxanthines (Dhamija et al., 2011).

Bronchodilator Treatments

Conventional medicine throughout time has established schools of experts to conduct clinical trials on potential drugs to determine their effectiveness, efficacy, and safety as compared to placebos for specific diseases. Salmeterol xinafoate (a long-acting β_2 adrenergic agonist) in combination with Ipratropium Bromide (one of the first drugs that seemed to help COPD patients) were studied together as an inhaler. There were many studies in the literature that showed the efficacy of both independently in COPD,

but not jointly. In 1999, a group of experts did a randomized, placebo-controlled, double-blind, and parallel group clinical trial to determine their combined effects on COPD (Mahler et al., 1999). Sites throughout the country followed 411 symptomatic patients without any comorbidity at the time of the study. Over a 12-week period, three different groups were administered either one of the two drugs, both, drugs, or a placebo. Salmeterol was significantly ($p < 0.0001$) more effective than Ipratropium Bromide and placebo when lung functions were considered. Additionally, both drugs significantly reduced respiratory distress in the subjects. Interestingly, the investigators noted that the most improvement was observed in patients with reduced use of Albuterol, another commonly used drug for COPD. This study supports the fact that Salmeterol can be used as a first-line of defense inhaler in patients with airway disease like COPD.

Methylxanthine Treatments

Theophylline, the commonly used trade name for Methylxanthine, is another first-line drug that is frequently used in the treatment of COPD along with other bronchodilators when the first-line treatments do not seem to work. However, researchers believe that its combination with Salmeterol may have greater effects than either one used alone. In a double-blind investigation, ZuWallack and colleagues (2001) randomly assigned some of 934 patients to receive Salmeterol in combination with Theophylline while others received only one of the two drugs. Results showed that all three groups improved over their baseline. However, simultaneous multi-drug use had greater improvement than any other group ($p \leq 0.045$) in pulmonary function, quality of life as documented and recorded periodically by subjects, and exacerbation on simple tasks.

Thus, the general recommendation of Theophylline and Salmeterol combined to be used for maximum benefits in COPD patients remained unchanged.

Corticosteroid Treatments

As inflammation of the airway and flow limitations are persistent symptoms of COPD, anti-inflammatory corticosteroids in combination with other bronchodilators can help to keep airway swelling down. Frequently used corticosteroid inhalers include Fluticasone (Flovent), Budesonide (Pulmicort), and Prednisolone (Prednisone). A randomized double-blind clinical trial on 120 patients was conducted to determine the efficacy and safety of the drug Pulmicort. Three groups were stratified: one placebo, one Pulmicort, and one Pulmicort with a long-acting bronchodilator. Results showed significant improvement from the baseline lung functions of patients from 25 to 75% (P=.037). The most improvement was recorded in the small airway functions of patients that were administered both the corticosteroid and the bronchodilator (Tse et al., 2013).

Phosphodiesterase-4 Inhibitors

This medication is an anti-inflammatory drug, which comes as a pill and is used with long-acting bronchodilators to reduce inflammation, thereby increasing the air movement in the airways of patients with COPD. Roflumilast (Daliresp) is an example of this inhibitor medication. Various studies had been conducted in multi-centers around the world and nationally before these drugs were approved. In fact, Giembycz and Field (2010) did a study in Europe and showed the benefits of Roflumilast as a maintenance drug in the care of COPD. When used in conjunction with other bronchodilators, lung functions increased significantly, while shortness of breath (SOB), dyspnea exacerbation,

and hospitalization were reduced. Although all COPD drug management is beneficial, all of the drugs have some mild to moderate side effects that are outweighed by their benefits.

Homeopathic and Naturopathic Management of COPD

History tells that early medicine was simply natural and homeopathic until the end of the 1800's and the beginning of the 1900's when politics in the United States and the world changed medical schools from homeopathic to allopathic. The healthcare systems suddenly depended on prescription drugs monopolies, instead of natural alternatives. Systematically, naturopathic schools and practitioners either became extinct or prosecuted and imprisoned for their practices (McCabe, 2003). Although the majority of the literature on naturopathic medicine had been destroyed for decades after the allopathic reforms of the early 1900's, the timid re-emergence of naturopathic medicine has been gaining support from all walks of life in developed and developing countries for the last few decades. Some medically-trained allopathic doctors, naturopaths, natural practitioners and others have dedicated years of research to prove the efficacy and safety of natural remedies for some important diseases and health conditions. For the purpose of this discussion, only a few natural treatments for COPD will be considered: Ozone (O_3), Hydrogen Peroxide ($H_2 O_2$), and Chlorine Dioxide ($NaClO_2$).

Natural practitioners, including a handful of allopathic physicians, homeopaths, and naturopaths, claim to have reversed heart disease, cancer, diabetes, HIV, and COPD to name a few (Lee, Rhee, & Lee, 2009; Altman, 2007). Although there is evidence in the literature to support their claims, the scientific world has a hard time accepting the facts

and call their stories – “anecdotes” and “quackery.” While natural practitioners agree that more studies need to be conducted in the field of natural sciences to secure their place in the scientific discussions, the fact is that diseases are cured, and disabilities are reversed for users of natural medicine (Humble, 2011; McCabe, 2003)

Ozone - Bio-Oxidative Therapy

Research studies in in the field of naturopathy agrees that ozone is a water-soluble gas that has some unique characteristics (Coogan et al., 2012; Hoffmann et al., 2012; Xu et al., 2010). Research shows that ozone acts like a powerful antibacterial, germicidal, fungicidal, and an oxidant that leaves no toxic traces of residue or by-product, because it converts to simple oxygen after releasing its third electron. Ozone is used commercially in developed and developing countries including the United States. It is used in swimming pools as a cleanser, in the food industry as a disinfectant, in wastewater, and in treatment of toxic waste to neutralize or eliminate heavy metals. Ozone is simply a more powerful form of oxygen that was in use in the medical realm about one hundred years ago until it was discredited by the chemical-forming giants of today (McCabe, 2003). According to McCabe, in 1976, the Food and Drug Administration (FDA), against all the evidence in the literature, declared ozone as toxic and banned it from use in the medical industry. Clinical trials show that as an oxidant, ozone neutralizes or deactivates virus, bacteria, fungus, pathogens, and toxins in the human body. Thus, it permanently reverses the effects of disease by cleansing the blood from the pathogens, as no disease can thrive in the presence of oxygen-rich blood.

The administration of ozone is rather simple and can be done at home or in a clinic or hospital. In fact, countries like Germany, other European nations, and Cuba, use ozone openly and legally in hospitals, clinics, and at home to cure numerous deadly diseases. Some methods of administration, such as blood "ozonation," require the intervention of medical professionals, but other methods, such as rectal or vaginal "insufflation," may be done at home. Blood "ozonation" is done on an out-patient basis. The intra-arterial application is done by removing some blood from a patient, infusing the blood with a mixture of ozone and oxygen, and administering the blood back to the patient (Travagli, Zanardi, Silvietti, & Bocci, 2007). Some diseases may require a combination of other natural medicines, but according to research and reports no additional natural remedies are required for the treatment of COPD. Typically, rectal "insufflations" are done at home by injecting about 100 to 800 ml of a mixture of ozone and oxygen through the rectum; a process that takes approximately two minutes to complete. According to reports, millions of people around the world, including the United States of America and Mexico, are healed and cured of their diseases through the use of ozone.

Hydrogen Peroxide (H_2O_2) and COPD Treatment

Similar to ozone, hydrogen peroxide is a supercharged mineral that is used to neutralize toxins, pathogens, bacteria, and viruses by oxidation. Oxidation is simply defined as blowing holes through the membranes of toxins (those that have membranes in their structure). For those without membranes, as soon as the H_2O_2 comes into contact with pathogens, it releases one oxygen electron that disrupts the normal course of the

pathogens and then the remaining compound turns into water (H_2O) (Bocci & Paolo, 2004). Hydrogen peroxide comes in many grades. However, the 35% food-grade is the only solution that can be used internally in the human body. It is naturally produced in the atmosphere and can be found in small traces in rain and snow. In 1888, Dr. Love, a respected authority in medicine, reported to have cured cancer of the womb, scarlet fever, and other diseases, including airway diseases, with oxygen-water (H_2O_2). This impressed the Surgeon General of the United States to the point that he endorsed Dr. Love's book published in 1904. That book is on display in the Library of Congress today bearing the Surgeon General of the United States' stamp of approval.

Hydrogen peroxide just like ozone may be applied in various ways, including intravenous infusion (IV), oral ingestion, and insufflation. In any case, a small diluted amount of the 35% food-grade solution is used. For instance, 5cc of 3% peroxide are mixed with 500cc of water before it is infused. For oral ingestion, drops of 35% food-grade solution are mixed with a glass of water for the therapy. Hydrogen peroxide is never to be ingested in its pure form, because it is a powerful irritant and may cause serious damage to the airways and the intestines (Bocci, 2011). According to Bocci, bio-oxidative therapy (the process in which an extra oxygen electron is released) is effective in increasing the CD4, the one clinical indicator that when it reaches a set low-level, people are diagnosed with HIV. This is maybe the strongest supportive evidence that claims hydrogen peroxide can reverse HIV. Although it has also been claimed that COPD has been reversed with the use of bio-oxidative therapy, the evidence is not so conclusive with the limited body of literature available.

Oxidative Therapy with Chlorine Dioxide ($NaClO_2$)

Chlorine (Cl) had been the chemical of choice to disinfect waters in various municipalities in this country and other developed and developing countries until research found that some chlorine by-products may be associated with cancer and other forms of serious illnesses (Drake & Villhauer, 2011). Therefore, alternatives like chlorine dioxide (ClO_2) have been slowly replacing chlorine on the stage of disinfectants. However, some experts were concerned, because very limited research studies had established chlorine dioxide as safe as or any safer than chlorine. Various inconclusive studies have been conducted since then, but chlorine dioxide has been widely accepted as the disinfectant of choice over chlorine in the last 2 decades (Grootveld, Silwood, Gill, & Lynch, 2000). Private Citizens and natural health practitioners have gone a step further to claim that chlorine dioxide has the chemical ability to inactivate disease-causing pathogens and viruses, especially in HIV patients. In fact, a few articles in the literature have found that the measure of CD4 in patients with HIV has markedly increased as they have been exposed to chlorine dioxide therapy (Knapp & Battisti, 2001; Farr & Walton, 1993). In addition, COPD patients have seen significant improvements in their FEV_1 and FVC to the point that people have been "undiagnosed" of COPD (Humble, 2011).

According to Humble, chlorine dioxide may be applied the same way as ozone and hydrogen peroxide, intravenously and orally. The intravenous protocol requires a trained professional to mix the exact dose with distilled water or normal saline to deliver the treatment safely without harm. The most commonly administered route is by ingestion in the comfort of people's homes. The right dosage of chlorine dioxide must be

diluted in a cup of distilled water two to three times a day for a few months, depending on the disease severity and co-morbidities involved during the treatment. Others have reported that COPD may take up to six months of treatment before being cured. In the case of airway disease, in addition to drinking the solution, it is important that people inhale for about one second the fumes from the reaction of the chlorine dioxide with citric acid or other approved reagents. Humble and other experts in the field agree that more scientific studies need to be conducted to support those findings.

Nutrition and COPD

Although researchers agree that no particular diet is a risk factor for COPD, they have come to the understanding that people with the condition may improve their respiratory functions by consuming some types of foods or avoiding others, including irritants which change the physiology of the respiratory muscles (Rogers et al., 1984). For example, Ferreira, Brooks, Lacasse, and Goldstein (2001) presented a systematic overview of randomized controlled trials to shed light on the benefits of specific diet to COPD patients. The theory that people with marginal ventilatory reserve may benefit from a high-calorie diet and have a decrease in systemic inflammatory effects, including diet-induced respiratory infections, was supported. Fruits, vegetables, fish, wine, and whole grains were shown to be associated with improvement of lung functions in COPD patients. Conversely, decreased lung functions were observed in people who did not consume those same foods (Kamholz, et al. 2006).

Summary and Conclusions

In studying the potential association between the SES of people and the debilitating, incurable disease of COPD, a thorough analysis must be considered. The literature review presented evidence that COPD is linked to some predictors, including cigarette smoking, genetic factors, environmental and industrial pollution, and behavioral factors.

It is well established through research that diseases place serious burdens on systems and people. Recorded evidence showed that behavior and genetics are not the sole causes of disease that place burdens on people. To identify other possible factors this research study will evaluate whether SES is a predictor of COPD. This study will contribute to and advance the knowledge about COPD and SES. It is well known that SES is associated with various diseases and conditions. However, the evidence is not conclusive regarding an association between COPD and SES, due to limited number of studies available in the literature.

Chapter 3: Research Method

Introduction

This chapter detailed the various aspects of the research design and methodologies used to test the hypotheses and interpret the data. In this section, details are provided on the primary data collection. A step by step process, including coding and recoding of nominal, ordinal, interval, and ratio variables must be presented. Furthermore, all the intended descriptive and inferential statistics on data will be detailed.

Research Design and Rationale

Similar to every research investigation in any given field, including social sciences, observational or experimental studies require the use of variables to understand phenomena and their interrelationships (Aponte, 2010). Variables are constructs that researchers used to measure and understand associations between complex natural phenomena that would otherwise be difficult to study (Grispen, Ronda, Dinant, de Vries, & van der Weijden, 2011). This study was a secondary analysis of archived data from the Inter-University Consortium for Political and Social Research (ICPSR). The dataset number ICPSR 04652 was originally collected for the "National Survey of Midlife Development in the United States (MIDUS)" study (Ryff et al., 2012). The purpose was to understand and investigate the role of behavioral, psychological, and social factors associated with age differences in physical and mental health between different ethnic groups – Black, Asian, American native, and White middle aged to late life from 35 to 86 year-old persons. The following table lists and describes the main variables used in this secondary data analysis:

Table 2

Inclusive Variable Names and Descriptions

Variable	Description
COPD	A disease of the lungs
Age	Number of years after birth
Education	High school/college achievement of a person
Sex	Gender male or female
Income	Annual family earnings or revenue
Occupation	The type of job a person does to generate revenue
Race/Ethnicity	A person's cultural background
Smoking	A person's history of smoking cigarettes

Methodology

The cross-sectional design is often used in social sciences to describe complex patterns of relationship between variables (Frankfort-Nachmias & Nachmias, 2007). This design has no time constraints and few limitations (discussed later in the chapter) compared to others. Most of those limitations may be overcome by applying some statistical techniques.

Population

This nationally representative longitudinal follow up survey of the continental United States was used to understand people's health changes over the periods between 2004 and 2006 when the data were collected. In 1995, the survey began across the nation with face-to-face interviews, mail-in, and phone of 4963 adult subjects ages 35 to 86

(Ryff et al., 2012). A probability sample design was used to find the participants. People who were age eligible but who lived in military camps, institutions, and other group homes were excluded from the survey population. According to the investigators, the response rate was still 68% in spite of the categories that were excluded in the survey.

Using the features of SPSS statistical software, the entire dataset will be used to test my main investigative hypothesis whether SES is a predictor of COPD as measured by education, occupation, and income. The earning income of people generally reflects their social status, but it is necessary to study the variable "occupation" as part of this research study (Franzini & Fernandez-Esquer, 2006).

Sampling and Sampling Procedures

In determining the sample size for this project, the computer software G*Power 3.1.5 was used. For the software to calculate the sample size accurately, certain information must be provided: 1) a two-tailed t-test is chosen to increase the power of relationship detection; 2) multiple logistic regression analysis will be used based on the categorical nature of the variables. The sample size was calculated to be 134. However, to increase the generalizability of the findings, all 4963 cases in the dataset will be used because a low sample size can limit the inferences of research findings to other populations. The following parameters were used to determine the sample size:

Effect size = .3

α err prob. = 0.05

Power (1-β err prob.) = 0.95

The effect size of .3 (30%) is a conventional number that signifies a medium-size effect in the real world (the smaller the effect size, the larger the sample size must be to detect the effect). In this research study, a p value of less than 0.05 and a 95% confidence interval will be used to determine statistical significance. Although the sample size was correctly calculated for the study, other challenges such as missing values in the data must be mitigated.

Missing data in the secondary analysis, for that matter in any research procedure, is a hurdle that must be dealt with carefully because of the serious implications it may have on the integrity of the research findings (Grispen et al., 2011). According to Herzig and associates (2009), when missing data are not appropriately taken into consideration, bias may be introduced, especially when imputation, a method that assigns one or more values for each missing response in a dataset, is used (Langkamp, Lehman, & Lemeshow, 2010). In fact, research has identified three other methods besides imputation to deal with missing data, including available case analysis commonly known as pairwise deletion, the weighting technique, and the complete case or listwise deletion. Of all the four methods, listwise deletion is the most widely used and reliable, although it has its limitations (Herzig, Howell, & Marcantonio, 2009). This technique calls for the deletion of the case that has a missing response. Thus, this process decreases the sample size. Therefore, it may threaten the statistical power of the data to generalize the findings to the population among other things.

In this research investigation, a multivariate analysis was applied on the data to mitigate the problem of missing data. Because this technique has some limitations, it may

also threatened the validity and reliability of the results (Langkamp et al., 2010). Although this technique can introduce selection bias into the study and potentially reduce the sample size available, it was the most appropriate to use in this case. With the multivariate analysis method, if the sample size remains representative of the population, the inferential power of the study and its generalizability are protected (Monteiro et al., 2012). The final reason for the use of this technique is that it is the simplest thing to do as I manipulate the data for analysis. Low sample size issues is the reason why it is important, especially in primary data collection procedures, to add 10-15% extra sample size to compensate for these anticipated problems (Gemmeke, Van Hamme, Cranen, & Boves, 2010).

Operationalization for Each Variable

Table 3

Brief Descriptions of variables

Variable name	Variable construct	Categories	Measure	IV/DV
COPD/Emphy.	B1SA11A	1 = Yes 2 = No	Nominal	DV
Education	B1PB1	1 = 0 – 7 grade 2 = 8/Junior High 3 = 9 – 12 grade 4 = GED 5 = High School Diploma	Ordinal	IV
Age	B1PAGE_ME	1 = 35 - 49 2 = 50 - 64 3 = 65 - 79 4 = 80 - 95+	Ordinal	Co-variate
Race/ethnicity	B1PF7A	1 = White 2 = Black 3 = Native American 4 = Asian 5 = Native Hawaiian	Nominal	Co-variate
Sex	B1PRSEX	1 = Male 2 = Female	Nominal	Co-variate
Income	B1SG8A	1 = $0 - 49,000 2 = $50,000 – 99,000 3 = $100,000 or more	Nominal	IV
Occupation	B1POCMAJ	1 = Executive/Adm. 2 = Professional 3 = Technician 4 = Sales 5 = Adm. Support 6 = Service Occ. 7 = Farming, Fish 8 = Production 9 =Operator	Nominal	IV
Smoking	B1PA38A	1 = Yes 2 = No	Nominal	Co-variate

For clarity and easy reference and understanding, the table above details the new variable names after transformation, their categories, and how they are measured and whether they are dependent variables (DV), independent variables (IV), or co-variate variables.

Data Analysis Plan

SPSS 22 was used to analyze the data using descriptive and inferential tests. The same statistical software was used to replace missing values identified during data cleaning. Although the 'listwise deletion' technique, which drops all cases with missing data, is commonly used, it is not often recommended by experts in the field. I therefore used logistic regression, which automatically dropped all missing cases.

First, descriptive statistics were calculated for the variables. Because all the variables were categorical, the frequency distribution was necessary to describe them. Second, inferential statistics were calculated. The Logistic Regression will test the hypothesis that SES, including all three of its components, income, education, and occupation, is a predictors of COPD. Additionally, an analysis of covariates, age, gender, and race/ethnicity was performed. The covariate variables were carefully chosen based on empirical studies that have already established associations between them and respiratory and airway conditions.

Threats to Validity

Measuring human behaviors in social sciences is a complex process. Investigators, for that reason, must be concerned with the issue of research validity before they infer study results to other groups. The validity of measurement can influence both

the internal and external end-results and conclusions drawn from data. According to Frankfort-Nachmias and Nachmias (2007), three principal types of validity issues that must be controlled during research are content validity, empirical validity, and construct validity. Due to the nature of this secondary analysis, the actual threats to validity that experimental research procedures would carry are not present here. For example, interaction effects of multiple-treatments interference that experimental investigations have, are absent in a secondary analytic approach.

The content validity stipulates that the measurement instrument (questionnaire) covers every aspect of a concept being studied without leaving out anything relevant to the phenomenon. The empirical validity is concerned more about the end-point relationship between the instrument and the measured outcomes. In other words, science believes that if an instrument has content validity, the research conclusions should be valid. Construct validity is related to the theoretical framework used to conduct the investigation. With respect to all three types of validity threats, the primary research study from which my secondary dataset was created does not have any validity threats. Therefore, there are no validity threats to this current secondary analysis.

Strengths and Limitations

This research survey has many strengths based on the secondary analysis approach of the available data. First, the cross-sectional design allows one to test the hypotheses and answer questions that can augment knowledge of policy-makers and professionals that can better care for COPD people in the United States and in the world. Second, the design helps to generalize study results beyond the sample size because a

sufficient sample size is used and randomly selected. Third, it allows researchers to control the influence of confounding variables that may hamper the validity of study results. Finally, it allows researchers to study large numbers of people while using a small sample size that is representative of the population.

Although the cross-sectional design of this research survey has many strengths, it also has a few weaknesses or limitations. First, the initial observational study relied heavily on self-reported data, which in turn may introduce some inaccuracy or bias into the study. Identified as recollection bias, this issue is especially worrisome when education and income levels are self-reported (Collins, Onwuegbuzie, & Jiao, 2006). There is no way for us to try and verify the accuracy of what is reported by participants. Second, a limitation of the cross-sectional design is that researchers cannot infer causal effects, but can only suggest the association between variables (Toschke, Lüdde, Eisele, & Von Kries, 2005).

Ethical Procedures

An institutional review board (IRB) was helps ensure that no infringement of the rules and guidelines of ethics are observed. For instance, it is prohibited to publish the real identity of participants in research or to cause harm to vulnerable populations. The Walden University IRB approved the proposal (No. 06-20-16-0225947). In secondary data analysis, manipulation of exposures is not present. As a precautionary measure, the investigators of the original study did not ask participants for their names or addresses. Also, the data is securely protected at the ICPSR databases from misuse and misappropriation.

The ICPSR is a virtual, public-access data-sharing service usually offered free of charge to members of the public to promote scientific research-based studies in social, behavioral, and scientific disciplines. Data in the databases are reviewed by professional data curators before they are posted for members' use. The ICPSR does not require a permission request from members to use databases, but rather only a signed "Terms of Use" agreement to abide by the rules (signed agreement exhibited in the appendix B section). However, the responsibilities of the user-member are two-fold. First, it is absolutely required that datasets used for publishing manuscripts be properly cited in the text (format usually provided by the ICPSR). Second, the digital object identifier (DOI) that will be attached to the dissertation once published or citation of the document must be provided to the ICPSR administrator so that the manuscript may be attached to the dataset as a user-reference.

Summary

This secondary data observational study uses the free access to the public ICPSR database and the appropriate dataset to answer the research questions pertaining to the relationship of the independent variable COPD and the dependent variable construct SES to include income, education, and occupation. A national sampling of the 3617 cases identified in the study of Americas' Changing Lives in Waves I, II, III, IV, and V, in 1986, 1989, 1994, 2002, and 2011, respectively, was used and manipulated to test the hypotheses under investigation. The data analysis plan, which includes descriptive and inferential statistics through the application of the Logistic Regression, is an appropriate fit to answer the research questions and examined the hypotheses based on the

measurement levels and nature of the variables, SES and COPD. Due to the fact that this is a secondary analysis study and also that the researchers in the initial study complied with the research guidelines (anonymity, informed consent, etc.), minimal threats to validity and ethical issues need to be addressed. In the following chapter, the results of the data analyses are presented, including the descriptive and inferential statistics of the logistic regression test used to answer the research questions.

Chapter 4: Results

Introduction

This chapter summarizes the tests results of the logistic regression used to determine the predictability of SES in relation to COPD. This study explored the perceived predictive effect of the independent variable, SES, on the dependent variable, COPD, while controlling for age, sex, smoking, and ethnicity. The purpose of this research study was to determine whether SES was a predictor of developing COPD later in life. Findings from this statistical analysis are expected to help health professionals, organizations, and policymakers make appropriate decisions in preventing and lowering SES risk factors of the disease.

Research Questions and Hypotheses

Question 1:

Is SES (education, occupation, and income) a predictor of COPD?

Null hypothesis 1:

*H*01: The Income of a person is not a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Alternative hypothesis 1:

*H*A1: The Income of a person is a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Null hypothesis 2:

*H*02: The education level of a person is not a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Alternative hypothesis 2:

*H*A2: The education level of a person is a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Null hypothesis 3:

*H*03: The occupation of a person is not a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Alternative hypothesis 3:

*H*A3: The occupation of a person is a predictor of COPD while age, sex, and race/ethnicity are controlled for.

Question 2:

Is there is difference in the relationship between COPD and SES by ethnic group – White, Black, Native American, and Asian?

Null hypothesis 4:

*H*04: There is no difference in the relationship between COPD and SES by ethnic group – White, Black, Native American, and Asian.

Alternative hypothesis 4:

*H*A4: There is a difference in the relationship between COPD and SES by ethnic group.

To answer these questions, analyses were done on a dataset archived in ICPSR. The data was originally used to understand the psychological and mental health status of 35–86-year-olds in the United States. As discussed in Chapter 3, the data collection was representative of the American population by design. A probability sample was drawn to guarantee the external validity of the results. Representation of all ethnic groups was well

calculated to compensate for deficits in response, especially among African American responders (Ryff et al., 2012). In the next section, I report on the descriptive and inferential statistics that are appropriate to test the hypotheses and answer the research questions.

Results

Univariate Descriptive Statistics

The sample in the study consisted of 4963 participants, approximately 47% of whom were male (n = 2316).

Regarding the smoking status of the participants, approximately 49% (n = 2,419) reported to have smoked cigarettes regularly, while only 26% reported not to have smoked cigarettes regularly (n = 1,274). The remaining 25% did not report their cigarette smoking history.

Table 4

Education Distribution of Participants

Education level	Frequency	Percentage
9 – 12 Grade	230	4.6
GED	61	1.2
HS Diploma	1266	25.5
1 – 2 yrs. of college	885	17.8
3 or more yrs. college	309	4.2
AS Degree	390	7.9
Bachelor's Degree	957	19.3
Grad School	152	3.1
Master's Degree	498	10.0
PHD/Terminal Degree	229	4.6
Missing/Do not know	7	.1

Regarding education, about 33% (n = 1636) of participants had an education level between no school at all and a high school diploma. Regarding college education, 52.3% (n = 2541) of participants attended or had a bachelor's degree. Some 14.6% (n = 879) had some graduate school education or received a doctoral degree. The most frequently occurring education level was high school.

Table 5

Frequencies and Percentages of occupation categories

Occupation	Frequency	Percentage
Executive	815	16.4
Professional	709	14.3
Technician	126	2.5
Adm. Support	447	9.0
Sales Occ.	334	6.7
Service Occ.	298	6.0
Farming/Fishing	67	1.3
Precision Production	293	5.9
Operator/Laborer	249	5.0
Missing/INNAP	1625	32.7

Occupation was distributed among nine categories. The top three occupations were executive, professional, and administrative support and they accounted for 39.7% (*n* = 1971) of the participants, while the middle three and the bottom four were identically 18.2 % (*n* = 907) of the sample. Non-respondents were 32.7% (*n* = 1625).

Table 6

Income distribution of participants

Income	Frequency	Percentage
$0 - $9,999	1,068	21.4
$10,000 - $19,999	337	6.8
$20,000 - $29,999	359	7.2
$30,000 - $39,999	348	7.0
$40,000 - $49,999	296	6.0
$50,000 - $59,999	428	4.3
$60,000 - $79,999	296	6.0
$80,000 - $99,999	179	3.6
$100,000 - $149,999	160	3.3
$150,000 - $200,000	100	2.0

Income participants were distributed in 10 categories. Participants that earned $49,999 or less accounted for 58.7% (n = 2408) of the sample. Earnings between $50,000 and $99,999 accounted for 19.2% (n = 908) while participants that earned more than $100,000 accounted for 5.3% (n = 260).

Table 7

Frequencies and Percentages of Ethnicity Categories

Ethnicity	Frequency	Percentage
White	4473	90.1
Black/African American	229	4.6
Native American	77	1.6
Asian	27	.5
Native Hawaiian	7	.1
Other	126	2.5
Don't Know	19	.4
Refused	5	.1

Approximately 90% of the participants were White (n = 4,473), and all the remaining groups combined constituted about 10%.

Regarding the dependent variable, 9.3% (n = 463) of participants reported they were diagnosed with COPD/Emphysema, while 72.1% (n = 3578) reported that they were not diagnosed with the disease.

Univariate Descriptive Statistics for Quantitative Variables

The two scale variables that are described here are the age and income of participants. The table and figure below present the means and standard deviations of participants' age and income or wages. As observed in the table, the means and standard deviations of age and income were relatively similar for both male and female. However,

on the average, males were slightly older than females and males earned significantly higher income or wages than females on average.

Table 8

Means and Standard Deviations for Age and Income for Each Gender

	Male		Female	
	M	SD	M	SD
Age	55.42	11.88	54.19	11.86
Wages	19.24	12.28	12.83	10.44

Multivariate Logistic Regression

By conducting logistic regression, we determined the individual contribution of each predictor variable on the outcome variable and the amount of variance explained by each predictor variable. Research suggests that a score of the overall prediction of the model fitness that is higher than 85% is considered a good fit for the data. In this study, the overall prediction was 91% in the table below (Field, 2011).

Table 9

Iteration History

Iteration [a,b,c]		-2 Log Likelihood	Coefficients Constant
Step 0	1	1121.470	1.647
	2	1044.460	2.187
	3	1041.318	2.326
	4	1041.309	2.334
	5	1041.309	2.334

a. Constant is included in the model.
b. Initial -2 log Likelihood: 1041.309.
c. Estimation terminated at iteration number 5 because parameter estimates changed by less than .001.

Table 10

Classification [a, b]

Observed			Predicted COPD Yes	Predicted COPD No	% Correct
Step 0	COPD	Yes	0	154	0
		No	0	1589	100.0
	Overall %				91.2

a. Constant is included in the model
b. The cut value is .500

The above tables, 9 and 10, report the model fit when only the constant is included. That said, only COPD was included and all the independent variables and covariates were omitted. The first table, (i.e. Iteration) tells that the log-likelihood of the model is 1,041.309, which represents the fit of the most basic model to the data. Because it is crucial that the model maximize its prediction of the observed data, the logistic regression model predicted that every responder belongs to the category in which most observed cases fell. In the second table, there were 1,589 cases who did not have COPD and only 154 had COPD. Therefore, if the logistic regression model predicted that every person did not have COPD, then the prediction would be correct 1,589 times out of 1,743 or approximately 91% (See table 9). However, if the logistic regression model had predicted the inverse, then the prediction would be correct only for 154 times out of 1,743 or about 10% (See table 12). Thus out of the two options, it is better to predict that all respondents did not have COPD because this results in a greater number of correct predictions. Overall, the model correctly classifies 91% of respondents, which is therefore a very good fit.

Table 11

Variables not in the equation

		Score	df	Sig
Step 0 Variables:	Sex	7.916	1	.005
	Smoking	.257	1	.612
	Occupation	6.482	8	.593
	Occupation (1)	3.792	1	.052
	Occupation (2)	1.377	1	.241
	Occupation (3)	.032	1	.858
	Occupation (4)	.304	1	.581
	Occupation (5)	.233	1	.630
	Occupation (6)	.129	1	.719
	Occupation (7)	.897	1	.324
	Occupation (8)	.973	1	.324
	Race	9.699	5	.084
	Race (1)	.045	1	.833
	Race (2)	6.646	1	.10
	Race (3)	.259	1	.611
	Race (4)	2.239	1	.135
	Race (5)	.552	1	.457
	Age	1.178	1	.278
	Income	3.677	1	.055
	Education	5.964	11	.876
	Education (1)	.001	1	.976
	Education (2)	.297	1	.586
	Education (3)	.013	1	.911
	Education (4)	.001	1	.980
	Education (5)	2.609	1	.106
	Education (6)	.373	1	.541
	Education (7)	1.303	1	.254
	Education (8)	1.539	1	.215
	Education (9)	.135	1	.713
	Education (10)	.529	1	.467
	Education (11)	.074	1	.785
Overall Statistics		32.220	28	.266

The model of statistics reported the constant b_0, which equals to 2.334 with a significance $p = .000$ and an odds ratio of 10.318. Table 13 reported that the residual chi-

square statistic as referred to in the table as 'Overall statistics' was 32.220 which was not significant at $p < .05$. This means that the coefficients for the variables that are not in the model are not significantly different from zero. In other words, the inclusion of one or more of those variables to the model would not affect its predictive power. Had the probability for the residual chi-square been significant at $\rho \leq .05$, it would mean that the inclusion of those variables in the model would have made a significant contribution to its predictive power.

On the other hand, table 13 lists each of the predictors with a score value, Roa efficiency statistics. When the null hypothesis is true, the Wald value in table 13 should have been identical to the score statistic and the likelihood ratio statistics. In this analysis they differ from one another, which tells us that the null hypothesis is not true. In the table, sex and race had significant score statistics at $p < .01$ and would make a potential contribution to the model. For, they were both selected to be included in the model with their respective high score statistics of (7.916 and 6.646).

Table 12

Omnibus Test of Model Coefficients

		Chi-Square	df	Sig
Step 1	Step	30.637	28	.333
	Block	30.637	28	.333
	Model	30.637	28	.333

The -2LogLikelihood (-2LL) is found to be less than the -2LL (1010.672) found in table 11 (1041.309) when only the constant was included in the model. This decrease in the value indicates that the model accurately predicts the outcome variable. The model is now more precise in predicting if someone was diagnosed or not with COPD than it was before. As observed in table 14, the chi-square value 30.637 represents the difference between the two -2LL. Yet because the chi-square value is not significant ($p < .333$) at ρ .05, it can safely be reported that the model did not make a better prediction than when the constant only was included. Therefore, the model indicates that the unexplained data in the model was minimal, which makes it stronger.

Answer to Question 1 and Hypotheses 1, 2 and 3

In table 13 below, question one is answered by failing to reject the null hypotheses. The logistic regression reports the significance of the predictability of income, education, and occupation to be, $\rho = .498$, $\rho = 1.000$, $\rho = .581$ respectively with an Exp (B) and confidence interval of 1.007 (.987, 1.028), 1.018 (.948, 1.094), 1.429 (.684, 2.988). Neither one has yielded a significant statistic value for a $\rho > .05$ or a $p > .01$. Therefore, based on the results of the data, we can report that the socioeconomic status (SES) of a person has no influence on the development of COPD. In other words, there is no risk factors associated with the disease COPD and the level of education, the type of occupation, and the income level of a person while controlling for age, sex, race/ethnicity, and smoking.

Answer to question 2 and Hypothesis 4

This logistic analysis was also conducted to determine whether there was a risk difference for COPD between ethnic groups that included White, Black, Native American, and Asian. Based on the results reported in table 13 below, the null hypothesis, which stated that there is no risk difference for COPD between different ethnic groups must be rejected. According to the report in the table, the Black ethnicity showed significant statistics, $\rho = .028$, Exp. (B) .340 with a CI of (.130, .890) to support the true hypothesis that there is a risk difference by race/ethnicity in developing COPD. The model also showed that sex ($\rho = .006$), Exp. (B) .581 with a CI of (.393, .859) showed a significant statistical value, which means that of the two possible genders, male is more susceptible than female to develop COPD simply based on their sex.

Table 13

Variables in the equation.

	B	S.E	Wald	Sig	Exp(B)	95 % CI for Exp (B) Lower	Upper
Step 1*							
Sex	-543	199	7.413	.006	.581	.393	.859
Smoking	.061	.184	.109	.742	1.063	.741	1.525
Occu. (1)	.357	.376	.900	.343	1.429	.684	2.988
Occu. (2)	-.035	.385	.008	.928	.966	.454	2.054
Occu. (3)	.250	.565	.196	.658	1.284	.424	3.887
Occu. (4)	.270	.425	.404	.525	1.310	.570	3.014
Occu. (5)	.176	.376	.219	.640	1.192	.571	2.491
Occu. (6)	.207	.408	.259	.611	1.231	.553	2.736
Occu. (7)	1.086	1.067	1.036	.309	2.963	.366	23.994
Occu. (8)	-.344	.388	.789	.374	.709	.331	1.515
Income	.007	.010	.460	.498	1.007	.987	1.028
Race (1)	.153	.535	.081	.775	1.165	.408	3.328
Race (2)	-1.079	.491	4.828	.028	.340	.130	.890
Race (3)	-.683	1.106	.381	.537	.505	.058	4.417
Race (4)	-1.924	1.252	2.359	.125	.146	.013	1.701
Race (5)	.452	.740	.373	.542	1.571	.369	6.692
Age	.007	.009	1.590	.207	.988	.971	1.006
Edu. (1)	-.018	.037	.246	.620	1.018	.948	1.094
Edu. (2)	-.007	1.104	.000	.995	.993	.114	8.643
Edu. (3)	-.347	.562	.380	.537	.707	.235	2.127
Edu. (4)	-.165	.816	.041	.840	1.179	.238	5.836
Edu. (5)	-.118	.380	.097	.755	.888	.422	1.870
Edu. (6)	-.377	.376	1.003	.317	.686	.328	1.434
Edu. (7)	-.189	.484	.152	.697	.828	.320	2.139
Edu. (8)	-.205	.451	.206	.650	1.227	.507	2.970
Edu. (9)	-.029	.371	.006	.937	1.030	.498	2.130
Edu. (10)	-.463	.484	.914	.339	.630	.244	1.626
Edu. (11)	- .441	.380	1.349	.246	.643	.305	1.355

*Variables entered on step 1: occupation, education, age, smoking, income

The logistic regression analysis was conducted to predict if the education, income, and occupation of a person was a risk factor to develop COPD. As mentioned, the outcome variable of interest was COPD and income, education, and occupation were the

independent variables. The Hosmer-Lemeshow goodness-of-fit was not significant at $\rho > .05$ (.377), which is indicative of a suitable model. As observed in table 13, neither one of the three independent variables resulted in a significant test at $\rho > .05$ while the model controlled for sex, smoking, race, and age. However, sex (male) ($\rho = .006$) and race (Black) ($\rho = .028$) were found to be significantly predictable of COPD. The table also reports the unstandardized B both for sex (-.543), Wald of 7.413, and an odds ratio of .581 with a 95% confidence interval of (.393, .859); and race for Black (-1.079), Wald of 4.828and an odds ratio of .340 with a 95% CI of (.130, .890). In other words, for every 58.1% increase in the odds ratio of males, it is expected an increase in Exp (B) of equal proportion.

Summary

The test statistics revealed that the model was an appropriate fit for the data, but all the independent variables, income, occupation, and education were found to be poor predictors of the dependent variable, COPD while controlling for age, sex, race, and smoking. Therefore, based on the statistical reports, it can be affirmed with confidence that SES is not a predictor of COPD as hypothesized. On the other hand, question two, based on the same statistics, the black ethnic group was found to have more difference to develop COPD than all other groups. In chapter 5, the results are discussed in greater detail.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

The purpose of this quantitative study was to determine if SES would predict the development of COPD. According to the results, income, education, and occupation are not associated with COPD. Therefore the alternative hypothesis could be accepted. In the following paragraphs, more detailed interpretation is presented.

Interpretation of the Findings

The findings of this study went along to support a handful of some previous research studies that found no association between SES and COPD (Blackstock & al. 2007; Turner & Sheperd, 1999). Also, other studies have found only one component of SES (occupation) to be associated with (Blanc, 2009). In a meta-analysis, Hu and associates (2010) found an association between COPD and occupation only, not SES. In regards to income, this study also confirmed many previous studies that did not find any associations with COPD. There are three possible reasons worth mentioning related to the non-significance findings of the study. First, one possible reason why the results of the analysis did not show any predictive effects between COPD and SES could be related to the degrees of freedom that was necessary in the analysis. When many variables are projected from a dataset, more degrees of freedom are required than that of a single variable, which may be done at the cost of precision. Hence, a lower t-statistics may result, which leads to higher *p*-values (non-significance) than expected. In other words, because all the variables in this study were used in the model at once, the precision of

predicting any association might have lowered the t-statistics of the results, which might have affected the significance of the results – in this study higher *p*-values.

The second possible reason for the results to show no significance between SES and COPD was that the independent variables are close related to each other. In trying to individually measure the effect of different variables that are similar, the variables may find it difficult to show significance to COPD. In this case, all three independent variables, income, occupation, and education were competing to show significance with the dependent variable, COPD, which could cause all of them to lose their effectiveness. Thus, the effect of either one variable was imprecisely estimated, which led to larger p-values than predicted (Hayes & Matthes, 2009). The third possible reason, could be related to the model fitting of the regression analysis. Because only the constant (COPD, the dependent variable) was inserted into the model, the univariate model may have been subject to uncontrollable variations such as omission bias, which in turn, may have affected the *p*-values' behavior to be larger than predicted during the analysis of the independent variables.

Limitations of the Study

This study had some important limitations that are worth mentioning. First, the criteria used for the inclusion of survey participants did not require health care utilization for COPD diagnosis. So misclassification of COPD might have been compromised. Selection bias is another potential limitation of the study. It is possible that there were important differences between the participants and non-participants that could have modified the results. Also, the distribution in race/ethnicity participants were so

disproportioned that an equal distribution of participation of race/ethnicity might have shown the COPD risks in other groups other than the African American strum. Furthermore, there was a substantial number of participants who did not report their COPD status. Had they reported their COPD status, the results could have been different. Biomass, environmental dust, and air pollution were not able to be analyzed in this study in order to differentiate between different occupations and the participants who reported to have been diagnosed with COPD. The study population ages ranged from 36 to 100 years old. The older population could have had other comorbidities that could have been confused with COPD (Collins, Onwuegbuzie, & Jiao, 2006; Toschke, Lüdde, Eisele, & Von, 2005).

Recommendations

Based on the results of this research study, I would make several recommendations for future research on the research questions. I would recommend that future research include sizable proportions of African Americans, Native Americans, Hispanic, Asian, and Whites in the sample that represent their respective racial/ethnic groups in the population at large. Also, I would recommend that healthcare utilization be used to diagnose participants based on tests. That would prevent misclassification in diagnoses. Because comorbidity can be a major factor in this study, participants' age should be limited to avoid misclassification of COPD. Furthermore, since gender was found to be associated with COPD, I would recommend that future studies stratify their samples based on gender. Finally, I would suggest that controlled variables be introduced in the data analysis one by one so that the effect of each one can be captured.

Implications

Positive Social Change

As this research study results showed, the black population is more likely to develop COPD in later life than any other ethnic group in the study. Cigarette-smoking as evidenced in the literature is a determining factor of COPD. Thus, this research study may be used to guide programs, policy makers, and legislative committees to enact more stringent laws, regulations and mandates to discourage easy access to cigarettes and decrease exposures to second-hand smoke. Strategies that might need implementation and reinforcement in that respect can be more tax-increase laws on cigarette purchases, smoke-free public places, and education programs that are geared to raise the awareness of the damaging effects of smoking on people's health. Though statistics revealed that the effects of smoking and COPD are more apparent in the black population, such laws and constraints of access to cigarettes may lower the risk of COPD in every smoking group regardless its ethnicity. These efforts may create social changes for both the smoking and non-smoking populations.

Conclusion

Although a number of studies have been done on this topic of SES and COPD, this study was the first to use secondary data from a population-based survey. Even though these research findings did not show any association between the dependent and independent variables COPD and SES, this study was an addition to the body of literature regarding these variables. Although income, occupation, and education were not found to be predictors of COPD in this study, there were a few studies that found them to be

predictors. These studies used primary data collection. In corroboration with some previous findings, this study found that race/ethnicity is a predictor of COPD more so in the African American group than in the White, Native American, or Asian groups.

References

Alber, J., & Hamilton-Hancock, D. (2012). Using the socio-ecological model to analyze U.S. policies for managing obesity. *International Journal of Health, Wellness & Society*, *2(3*), 75–87.

Alwan, A., MacLean, D. R., Riley, L. M., Tursan d'Espaignet, E., Mathers, C. D., & Stevens, G. A. (2010). Chronic diseases: chronic diseases and development. Monitoring and surveillance of chronic non-communicable diseases: progress and capacity in high-burden countries. *Lancet*, *376*, 1861–8.

Aponte, J. (2010). Key elements of large survey data sets. *Nursing Economic$*, *28*(*1*), 27–36. Retrieved from https://ezproxy.southtexascollege.edu/login?url=http://search.proquest.com/docview/236939356?accountid=7069

Aryal, S., Diaz-Guzman, E., Mannino, D.M. (2014). Influence of sex on chronic obstructive pulmonary disease risk and treatment outcomes. Int J Chron Obstruct Pulmon Dis. 2014 Oct 14; 9:1145-54. Epub 2014 Oct 14.

Azad, M. B., Lissitsyn, Y., Miller, G. E., Becker, A. B., HayGlass, K. T., & Kozyrskyj, A. L. (2012). Influence of socioeconomic status trajectories on innate immune responsiveness in children. *PLoS ONE*, *7*(6), 1–9. http://doi.org/10.1371/journal.pone.0038669

Bakr, R. M., & Elmahallawy, I. I. (2012). Prevalence characteristics of COPD in never smokers. *Egyptian Journal of Chest Diseases and Tuberculosis*, *61*(3), 59–65. http://doi.org/10.1016/j.ejcdt.2012.10.035

Bando, D. H., Moreira, R. S., Pereira, J. C. R., & Barrozo, L. V. (2012). Spatial clusters of suicide in the municipality of São Paulo 1996-2005: an ecological study. *BMC Psychiatry*, *12*(1), 124–131.

Bandura, A. (2001). Social cognitive theory: An agentic perspective. *Annual Review of Psychology*, *52*(1), 1–26.

Bandura, A. (2004). Health promotion by social cognitive means. *Health Education and Behavior*, *31*(2), 143–164.

Baral, S., Logie, C. H., Grosso, A., Wirtz, A. L., & Beyrer, C. (2013). Modified social ecological model: a tool to guide the assessment of the risks and risk contexts of HIV epidemics. *BMC Public Health*, *13*(1), 1–8. http://doi.org/10.1186/1471-2458-13-482

Barker, R. G. (1968). *Ecological Psychology: Concepts and methods for studying the environment of human behavior*. Stanford University Press. Retrieved from https://books.google.com/books?hl=en&lr=&id=p1arAAAAIAAJ&oi=fnd&pg=PA1&dq=Ecological+Psychology:+Concepts+and+methods+for+studying+the+environment+of+human+behavior&ots=heZrALzUX8&sig=reWOlZ0669PTxn3rTnO9007EM98#v=onepage&q=Ecological%20Psychology%3A%20Concepts%20and%20methods%20for%20studying%20the%20environment%20of%20human%20behavior&f=false

Bernard, H. R. (2012). *Social research methods: Qualitative and quantitative approaches*. Sage Publications, Incorporated.

Blackstock, F., & Webster, K. E. (2007). Disease-specific health education for COPD: a systematic review of changes in health outcomes. *Health Education Research*, *22*(5), 703–717.

Blanc, P. D., Iribarren, C., Trupin, L., Earnest, G., Katz, P. P., Balmes, J. … Eisner, M. D. (2009). Occupational exposures and the risk of COPD: dusty trades revisited. *Thorax*, *64*(1), 6–12. http://doi.org/10.1136/thx.2008.099390

Bocci, V., & Paolo, N. D. (2004). Oxygenation-ozonization of blood during extracorporeal circulation (EBOO). Part III: a new medical approach. *Ozone: Science and Engineering*, *26*(2), 195–205.

Bocci, V. (2011). The Potential Toxicity of Ozone: side effects and contraindications of ozone therapy. In *Ozone* (pp. 75–84). Springer. Retrieved from http://link.springer.com/chapter/10.1007/978-90-481-9234-2_7

Briske, D. D., Fuhlendorf, S. D., & Smeins, F. E. (2005). State-and-transition models, thresholds, and rangeland health: a synthesis of ecological concepts and perspectives. *Rangeland Ecology & Management*, *58*(1), 1–10.

Brogger, J., Steen, V. M., Eiken, H. G., Gulsvik, A., & Bakke, P. (2006). Genetic association between COPD and polymorphisms in TNF, ADRB2 and EPHX1. *European Respiratory Journal*, *27*(4), 682.

Begum, F., Ruczinski, I., Li, S., Silverman, E. K., Cho, M.H., Lynch, D.A. … Parker, M.M., (2016). Identifying a delition affecting total lung capacity among subjects. *Genetic Epidemiology*. 2016 Jan; 40(1):81-8. doi:10.1002/gepi.21943

Begum, F., Ruczinski, I., Hokanson, J.E., Lutz, S.M., Parker, M.M., Cho, M.H. Silverman, E.K. (2016). Hemizygous Deletion on Chromosome 3p26.1 Is Associated with Heavy Smoking among African American Subjects in the COPDGene Study. *PLoS One. 2016 Oct 6; 11(10):e0164134. Epub 2016 Oct 6.* http://dx.doi.org/10.1371/journal.pone.0164134

Castaldi, P. J., Cho, M. H., Cohn, M., Langerman, F., Moran, S., Tarragona, N. … Trikalinos, T. A. (2010). The COPD genetic association compendium: a comprehensive online database of COPD genetic associations. *Human Molecular Genetics*, *19*(3), 526–534. doi: *10.1093/hmg/ddp519*

Castaldi, P.J., Dy, J., Ross, J., Chang, Y., Washko, G.R., Curran-Everett, D. … Crapo J. D. Thorax. (2014). Cluster analysis in the COPD gene study identifies subtypes of smokers. *Thorax*. May; 69(5):415-22. doi:10.1136/thoraxjnl-2013-203601

Cho, M.H., Castaldi, P.J., Hersh, C.P., Hobbs, B.D., Barr, R.G., Tal-Singer, R. … Van Beek, E. J. (2015). A Genome-wide association study of emphysema and airway. *American Journal and Respiratory Critical Care Medicine*. 2015 Sep 1; 192(5):559-69. *http://dx.doi.org/10.1164/rccm.201501-0148OC*

Choi, S. H. (2012). *Smoking behavior and the impact on sleep quality and health-related quality of life among operating engineers*. The University of Michigan. Retrieved from http://deepblue.lib.umich.edu/handle/2027.42/91494

Cohen, D. A., Scribner, R. A., & Farley, T. A. (2000). A structural model of health behavior: a pragmatic approach to explain and influence health behaviors at the population level. *Preventive Medicine*, *30*(2), 146–154.

Collins, K. M. T., Onwuegbuzie, A. J., & Jiao, Q. G. (2006). Prevalence of mixed-methods sampling designs in social science research. *Evaluation & Research in Education*, *19*(2), 83–101.

Coutts, C. J., & Taylor, C. (2011). [Commentary on] putting the capital "E" environment into ecological models of health. *Journal of Environmental Health*, *74*(4), 26–29.

Cox, E., Martin, B. C., Van Staa, T., Garbe, E., Siebert, U., & Johnson, M. L. (2009). Good research practices for comparative effectiveness research: approaches to mitigate bias and confounding in the design of nonrandomized studies of treatment effects using secondary data sources: The International Society for Pharmacoeconomics and Outcomes Research Good Research Practices for Retrospective Database Analysis Task Force Report—Part II. *Value in Health*, *12*(8), 1053–1061. http://doi.org/10.1111/j.1524-4733.2009.00601.x

Creswell, J. W. (2009). *Research design: qualitative, quantitative, and mixed methods approaches*. Retrieved from https://books.google.com/books?hl=en&lr=&id=EbogAQAAQBAJ&oi=fnd&pg=PR1&dq=Research+design:+qualitative,+quantitative,+and+mixed+methods+approaches.+&ots=caiLqPPCF4&sig=58_Pa_991zAD9c2-v3Zm5eK7hPc#v=onepage&q=Research%20design%3A%20qualitative%2C%20quantitative%2C%20and%20mixed%20methods%20approaches.&f=false

Crosby, R., & Noar, S. M. (2010). Theory development in health promotion: are we there yet? *Journal of Behavioral Medicine*, *33*(4), 259–263.

Cunningham, T.J., Ford, E.S., Rolle, I.V., Wheaton, A.G., Croft, J.B. (2015). Associations of Self-Reported Cigarette Smoking with Chronic Obstructive Pulmonary Disease and Co-Morbid Chronic Conditions in the United States. COPD: Journal of Chronic Obstructive Pulmonary Disease. 2015 Jun; 12(3):276-86. Epub 2014 Sep 10. http://dx.doi.org/10.3109/15412555.2014.949001

Demakakos, P., Nazroo, J., Breeze, E., & Marmot, M. (2008). Socioeconomic status and health: The role of subjective social status. *Social Science & Medicine*, *67*(2), 330–340. doi.org/10.1016/j.socscimed.2008.03.038

Dhamija, P., Bansal, D., Srinivasan, A., Bhalla, A., Hota, D. & Chakrabarti, A. (2011). Patterns of prescription drug use and incidence of drug–drug interactions in patients reporting to medical emergency. Fundamental and Clinical Pharmacology. doi: 10.1111/j.1472-8206.2011.00990.x.

Dilektasli, A.G., Porszasz, J, Casaburi, R., Stringer, W.W., Bhatt, S.P., Pak, Y. … Hansen, J.E. (2016). A novel spirometry measure identifies mild COPD unidentified by standard criteria. *Chest*. 2016 Jul 22; 150(5): 1080-1090. doi:10.1016/j.chest.2016.06.047

Drake, D., & Villhauer, A. L. (2011). An in vitro comparative study determining bactericidal activity of stabilized chlorine dioxide and other oral rinses. *Journal of Clinical Dentistry*, *22*(1), 1.

Eisner, M. D., Klein, J., Hammond, S. K., Koren, G., Lactao, G., & Iribarren, C. (2005). Directly measured second hand smoke exposure and asthma health outcomes. *Thorax*, *60*(10), 814–821. http://doi.org/10.1136/thx.2004.037283

Eisner, M. D., Blanc, P. D., Omachi, T. A., Yelin, E. H., Sidney, S., Katz, P. P. … Iribarren, C. (2011). Socioeconomic status, race and COPD health outcomes. *Journal of Epidemiology and Community Health*, *65*(1), 26–34.

Eisner, M. D., Blanc, P. D., Yelin, E. H., Sidney, S., Katz, P. P., Ackerson, L. … Iribarren, C. (2008). COPD as a systemic disease: impact on physical functional limitations. *The American Journal of Medicine*, *121*(9), 789–796.

Eisner, M. D., Iribarren, C., Yelin, E. H., Sidney, S., Katz, P. P., Ackerson, L. … Blanc, P. D. (2008). Pulmonary function and the risk of functional limitation in chronic obstructive pulmonary disease. *American Journal of Epidemiology*, *167*(9), 1090–1101.

El-Zein, R.A, Young, R. P., Hopkins, R. J., Etzel, C. J. (2012). Genetic predisposition to chronic obstructive pulmonary disease. *Cancer Prevention Research*. 2012 Apr; 5(4):522-7. DOI: 10.1158/1940-6207.CAPR-12-0042

Eriksson, B., Lindberg, A., Müllerova, H., Rönmark, E., & Lundbäck, B. (2013). Association of heart diseases with COPD and restrictive lung function – Results from a population survey. *Respiratory Medicine*, *107*(1), 98–106. http://doi.org/10.1016/j.rmed.2012.09.011

Farr, R., & Walton, C. (1993). Inactivation of human immunodeficiency virus by a medical waste disposal process using chlorine dioxide. *Infection Control*, *14*(09), 527–529.

Ferreira, I. M., Brooks, D., Lacasse, Y., & Goldstein, R. S. (2001). Nutritional intervention in COPD: a systematic overview. *CHEST Journal*, *119*(2), 353–363.

Field, A. (2009). *Discovering statistics using SPSS*. Sage Publications, Inc. Thousand Oaks, CA.

Fisher, E. B., Brownson, C. A., O'Toole, M. L., Shetty, G., Anwuri, V. V., & Glasgow, R. E. (2005). Ecological approaches to self-management: the case of diabetes. *American Journal of Public Health*, *95*(9), 1523.

Fisher-Owens, S. A., Barker, J. C., Adams, S., Chung, L. H., Gansky, S. A., Hyde, S., & Weintraub, J. A. (2008). Giving policy some teeth: routes to reducing disparities in oral health. *Health Affairs*, *27*(2), 404–412.

Fleischer, N. L., Diez Roux, A. V., Alazraqui, M., Spinelli, H., & De Maio, F. (2011). Socioeconomic gradients in chronic disease risk factors in middle-income countries: evidence of effect modification by urbanicity in Argentina. *American Journal of Public Health*, *101*(2), 294–301. http://doi.org/10.2105/AJPH.2009.190165

Fletcher, M. J., Upton, J., Taylor-Fishwick, J., Buist, S. A., Jenkins, C., Hutton, J. … Walker, S. (2011). COPD uncovered: an international survey on the impact of chronic obstructive pulmonary disease [COPD] on a working age population. *BMC Public Health*, *11*(1), 612. http://doi.org/10.1186/1471-2458-11-612

Franzini, L., & Fernandez, M. E. (2006). The association of subjective social status and health in low-income Mexican-origin individuals in Texas. Science Direct 63(3), 788 - 804. doi.org/10.1016/j.socscimed.2006.01.009

Foster, T. S., Miller, J. D., Marton, J. P., Caloyeras, J. P., Russell, M. W., & Menzin, J. (2006). Assessment of the economic burden of COPD in the U.S.: a review and synthesis of the literature. *COPD: Journal of Chronic Obstructive Pulmonary Disease*, *3*(4), 211–218. http://doi.org/10.1080/15412550601009396

Frankfort-Nachmias, C., & Nachmias, D. (2007). *Research methods in the social sciences*. Worth Publishers.

García-Polo, C., Alcázar-Navarrete, B., Ruiz-Iturriaga, L. A., Herrejón, A., Ros-Lucas, J. A., García-Sidro, P. … Miravitlles, M. (2012). Factors associated with high healthcare resource utilization among COPD patients. *Respiratory Medicine*, *106*(12), 1734–1742. http://doi.org/10.1016/j.rmed.2012.09.009

Garuti, G., Cilione, C., Dell Orso, D., Gorini, P., Lorenzi, M. C., Totaro, L. … Clini, E. (2003). Impact of comprehensive pulmonary rehabilitation on anxiety and depression in hospitalized COPD patients. *Monaldi Archives for Chest Disease*, *59*(1), 56–61.

Gegick, S., Coore, H.A., Bowling, M.R. (2013). Chronic obstructive pulmonary disease: epidemiology, management, and impact on North Carolina. *N C Medical Journal*. 2013 Sep-Oct; 74(5):411-4. Retrieved from, http://classic.ncmedicaljournal.com/wp-content/uploads/2013/09/74510.pdf

Gemmeke, J. F., Van Hamme, H., Cranen, B., & Boves, L. (2010). Compressive sensing for missing data imputation in noise robust speech recognition. *Selected Topics in Signal Processing, IEEE Journal Of*, *4*(2), 272–287.

Giembycz, M. A., & Field, S. K. (2010). Roflumilast: first phosphodiesterase 4 inhibitor approved for treatment of COPD. *Drug Design, Development and Therapy*, *4*, 147–158.

Giles-Corti, B., Timperio, A., Bull, F., & Pikora, T. (2005). Understanding physical activity environmental correlates: increased specificity for ecological models. *Exercise and Sport Sciences Reviews*, *33*(4), 175–181.

Glanz, K., & Bishop, D. B. (2010). The role of behavioral science theory in development and implementation of public health interventions. *Annual Review of Public Health*, *31*, 399–418.

Glanz, K.., Rimer, B. K., & Viswanath, K. (2008). *Health behavior and health education: theory, research, and practice* (4th Ed.). Jossey-Bass, San Francisco, CA.

Glass, T. A., & McAtee, M. J. (2006). Behavioral science at the crossroads in public health: extending horizons, envisioning the future. *Social Science & Medicine*, *62*(7), 1650–1671.

Gray, S. C., Edwards, S. E., Schultz, B. D., & Miranda, M. L. (2014). Assessing the impact of race, social factors and air pollution on birth outcomes: a population-based study. *Environmental Health: A Global Access Science Source*, *13*(1), 1–16. http://doi.org/10.1186/1476-069X-13-4

Grispen, J. E. J., Ronda, G., Dinant, G. J., de Vries, N. K., & van der Weijden, T. (2011). To test or not to test: a cross-sectional survey of the psychosocial determinants of self-testing for cholesterol, glucose, and HIV. *BMC Public Health*, *11*(1), 112.

Grootveld, M., Silwood, C., Gill, D., & Lynch, E. (2000). Evidence for the microbicidal activity of a chlorine dioxide-containing oral rinse formulation in vivo. *The Journal of Clinical Dentistry*, *12*(3), 67–70.

Hardie, J. A., Buist, A. S., Vollmer, W. M., Ellingsen, I., Bakke, P. S., & Mørkve, O. (2002). Risk of over-diagnosis of COPD in asymptomatic elderly never-smokers. *European Respiratory Journal*, *20*(5), 1117–1122.

Harris, D. & Debolt, S. (2008). Relative crystallinity of plant biomass: study on assembly, adaptation and acclimation. PLOS - One. doi.org/10.1371/journal.pone.0002897

Hayes, A. F., Matthes, J. (2009). Computational procedures for probing Interactions in OLS and logistic regression: SPSS and SAS implementations. Behavior Research Methods, 41 (3), 924-936. doi:10.3758/BRM.41.3

Herzig, S. J., Howell, M. D., Marcantonio, E. R. (2009). Acid-suppressive medication use and the risk for sospital-acquired pneumonia. Jama, 301(20):2120-2128. doi:10.1001/jama.2009.722

Hoffmann, B., Luttmann-Gibson, H., Cohen, A., Zanobetti, A., de Souza, C., Foley, C. . . . Gold, D. R. (2012). Opposing effects of particle pollution, ozone, and ambient temperature on arterial blood pressure. *Environmental Health Perspectives, 120*(2), 241-6. Retrieved from

https://ezproxy.southtexascollege.edu/login?url=http://search.proquest.com/docview/928054980?accountid=7069

Holguin, F., Folch, E., Redd, S. C., & Mannino, D. M. (2005). Comorbidity and mortality in COPD-related hospitalizations in the United States, 1979 to 2001. *CHEST Journal*, *128*(4). Retrieved from http://journal.publications.chestnet.org/article.aspx?articleid=1083826

Holvino, E. (2008). Intersections: The Simultaneity of Race, Gender and Class in Organization Studies. doi:10.1111/j.1468-0432.2008.00400.x. Blackwell Publishing. Retrieved from http://www.chaosmanagement.com/images/stories/pdfs/GWO%20Simultaneityfinal5-08.pdf

Hu, G., Zhou, Y., Tian, J., Yao, W., Li, J., Li, B., & Ran, P. (2010). Risk of COPD from exposure to biomass smoke: a metaanalysis. *Chest*, *138*(1), 20–31. http://doi.org/10.1378/chest.08-2114

Humble, J. V. (2011). *The master mineral solution of the third millennium* (5th Ed.). Retrieved from https://www.amazon.com/Mineral-Solution-Millenium-Updated-Supplement/dp/B005LC3KW2

Hurst, J. R., & Wedzicha, J. A. (2007). What is (and what is not) a COPD exacerbation: thoughts from the new GOLD guidelines. *Thorax*, *62*(3), 198–199. http://doi.org/10.1136/thx.2007.077883

Izquierdo, C., Oviedo, M., Ruiz, L., Sintes, X., Vera, I., Nebot, M. … Domínguez, A. (2010). Influence of socioeconomic status on community-acquired pneumonia outcomes in elderly patients requiring hospitalization: a multicenter observational study. *BMC Public Health*, *10*, 421–429.

Kamholz, B.W., Hayes, A.M., Carver, C.S. et al. (2006). Identification and evaluation of cognitive affect-regulation strategies: development of a self-report measure. Cognitive therapy and research, 30: 227. doi:10.1007/s10608-006-9013-1

Kasai, T. Bradley, T. D. (2011). Obstructive Sleep Apnea and Heart Failure: Pathophysiologic and Therapeutic Implications. *Journal of the American College of Cardiology*. (57) 2, pages 119 – 127. http://dx.doi.org/10.1016/j.jacc.2010.08.627

Kheirallah, A.K., Miller, S., Hall, I.P., Sayers, I. (2016). Translating Lung Function Genome-Wide Association Study (GWAS) Findings*: New Insights for Lung Biology*. *Advances in Genetics*. 2016; 93:57-145. Epub 2016 Feb 9. http://dx.doi.org/10.1016/bs.adgen.2015.12.002

Klein, L. W., Miller, D. L., Balter, S., Laskey, W., Haines, D., Norbash, A., Mauro, M. A. and Goldstein, J. A. (2009), Occupational health hazards in the interventional laboratory: Time for a safer environment. Cathet. Cardiovasc. Intervent., 73: 432–438. doi:10.1002/ccd.21801

Knapp, J. E., & Battisti, D. L. (2001). Chlorine dioxide. *Disinfection, Sterilization, and Preservation*, *5*, 215–228.

Kurmi, O. P., Semple, S., Simkhada, Simkhada, P. Smith, W. C. S., Ayres, J. G. (2010).

COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis. *Thorax. 2010; 65:221-228 doi:10.1136/thx.2009.124644*

Lamontagne, M., Couture, C., Postma, D.S., Timens, W., Sin, D.D., Paré, P.D. … Bossé, Y. (2013). Refining susceptibility loci of chronic obstructive pulmonary disease with lung eqtls. *PLoS One. 2013; 8(7):e70220. Epub 2013 Jul 30.* http://dx.doi.org/10.1371/journal.pone.0070220

Lamprecht, B., McBurnie, M. A., Vollmer, W. M., Gudmundsson, G., Welte, T., Nizankowska-Mogilnicka, E. … Buist, S. A. (2011). COPD in never smokers: results from the population-based burden of obstructive lung disease study. *CHEST Journal*, *139*(4), 752–763.

Langkamp, D. L., Lehman, A., & Lemeshow, S. (2010). Techniques for handling missing data in secondary analyses of large surveys. *Academic Pediatrics*, *10*(3), 205–210. http://doi.org/10.1016/j.acap.2010.01.005

Lee, J.H., McDonald, M.L., Cho, M.H., Wan, E.S., Castaldi, P.J., Hunninghake, G.M. … Lomas, D.A., (2014). DNAH5 is associated with total lung capacity in chronic obstructive pulmonary disease. *Respiratory Research*. 2014 Aug 20; 15:97. Epub 2014 Aug 20. *doi***:** 10.1186/s12931-014-0097-y

Lee, D. H., Rhee, J. G., & Lee, Y. J. (2009). Reactive oxygen species up-regulate p53 and Puma; a possible mechanism for apoptosis during combined treatment with trail and wogonin. *British Journal of Pharmacology*, *157* (7), 1189–1202.

Li, C., Balluz, L.S., Okoro, C.A., Strine, T.W., Lin, J.M., Town, M. … Valluru, B. (2011). Surveillance of certain health behaviors and conditions among states and selected local areas --- Behavioral Risk Factor Surveillance System, United States, 2009. MMWR Surveill Summ. (2011). 2011 Aug 19; 60(9):1-250.Retrieved from, http://www.cdc.gov/MMWr/preview/mmwrhtml/ss6009a1.htm

Lin, M., Chen, Y., & McDowell, I. (2005). Increased risk of depression in COPD patients with higher education and income. *Chronic Respiratory Disease*, *2*(1), 13–19.

Long, J. A., Bamba, M. I., Ling, B., & Shea, J. A. (2006). Missing race/ethnicity data in veterans health administration based disparities research: a systematic review. *Journal of Health Care for the Poor and Underserved*, *17*(1), 128–40.

Lu, Q., Jin, C., Sun, J., Bowler, R., Kechris, K., Kaminski, N., Zhao, H. Statistics in biosciences, (2016). Post-GWAS prioritization through data integration provides novel insights on COPD. 2016 Jun 13; 2016: 1-17. doi:10.1007/s12561-016-9151-2

Luepker, R. V., Murray, D. M., Jacobs, D. R., Mittelmark, M. B., Bracht, N., Carlaw, R. … Folsom, A. R. (1994). Community education for cardiovascular disease prevention: risk factor changes in the Minnesota Heart Health Program. *American Journal of Public Health*, *84*(9), 1383–1393.

Lutz, S.M., Cho, M.H., Young, K., Hersh, C.P., Castaldi, P.J., McDonald, M.L. … Parker, M., l (2015). A genome-wide association study identifies risk loci for spirometric measures among smokers of European and African ancestry. *BMC*

Genetics. 2015 Dec 3; 16:138. Epub 2015 Dec 3. doi**:** 10.1186/s12863-015-0299-4

Lynch, J., & Smith, G. D. (2005). A Life course approach to chronic disease epidemiology. *Annual Review of Public Health*, *26*(1), 1–35. http://doi.org/10.1146/annurev.publhealth.26.021304.144505

Mahler, D. A., Donohue, J. F., Barbee, R. A., Goldman, M. D., Gross, N. J., Wisniewski, M. E. … Anderson, W. H. (1999). Efficacy of salmeterol xinafoate in the treatment of COPD. *Chest*, *115*(4), 957–965. http://doi.org/10.1378/chest.115.4.957

Mannino, D. M., & Buist, A. S. (2007). Global burden of COPD: risk factors, prevalence, and future trends. *Lancet*, *370*(9589), 765–773.

McCabe, E. (2003). *Flood your body with oxygen* (1st Edition edition). Breath of God Ministry publication, Miami Shores, FL.

McDonald, M.L., Cho, M.H., Sorheim, I.C., Lutz, S.M., Castaldi, P.J., Lomas, D.A. … Vestbo, J., (2014). Common genetic variants associated with resting oxygenation in chronic obstructive pulmonary disease. *American Journal and Respiratory Cell Molecular Biology*. 2014 Nov; 51(5):678-87. DOI: http://dx.doi.org/10.1165/rcmb.2014-0135OC

McLeroy, K. R., Bibeau, D., Steckler, A., & Glanz, K. (1988). An ecological perspective on health promotion programs. *Health Education & Behavior*, *15*(4), 351–377. http://doi.org/10.1177/109019818801500401

Meara, E. R., Richards, S., & Cutler, D. M. (2008). The gap gets bigger: changes in mortality and life expectancy, by education, 1981–2000. *Health Affairs*, *27*(2), 350–360.

Michalopoulos, A. (2005). Smoking and COPD. *Tobacco Induced Diseases*, *3*(1), 30.

Minkler, M. (1989). Health education, health promotion and the open society: an historical perspective. *Health Education & Behavior*, *16*(1), 17–30.

Miravitlles, M., Naberan, K., Cantoni, J., & Azpeitia, A. (2011). Socioeconomic status and health-related quality of life of patients with chronic obstructive pulmonary disease. *Respiration*, *82*(5), 402–408. http://doi.org/10.1159/000328766

Monteiro, A., Carvalho, V., Velho, S., & Sousa, C. (2012). Assessing and monitoring urban resilience using COPD in Porto. *Science of the Total Environment*, *414*, 113–119. http://doi.org/10.1016/j.scitotenv.2011.11.009

Nielsen, M., Bårnes, C.B., Ulrik, C.S. (2015). Clinical characteristics of the asthma-COPD overlap syndrome--a systematic review. *International Journal of Chronic Obstructive Pulmonary Disease*. 2015 Jul 27; 10:1443-54. Epub 2015 Jul 27. doi: 10.2147/COPD.S85363

Ng, T. P., Niti, M., Tan, W. C., Cao, Z., Ong, K. C., & Eng., P. (2007). Depressive symptoms and chronic obstructive pulmonary disease: effect on mortality, hospital readmission, symptom burden, functional status, and quality of life. *Archives of Internal Medicine*, *167*(1), 60–67.

Norris, S. L., Lau, J., Smith, S. J., Schmid, C. H., & Engelgau, M. M. (2002). Self-management education for adults with type 2 diabetes: a meta-analysis of the effect on glycemic control. *Diabetes Care*, *25*(7), 1159–1171.

Obeidat, M., Hao, K., Bossé, Y., Nickle, D.C., Nie, Y., Postma, D.S. … Hogg, J.C. (2015). Molecular mechanisms underlying variations in lung function: a systems genetics analysis. Lancet Respiratory Medicine. 2015 Oct; 3(10):782-95. Epub 2015 Sep 21. http://dx.doi.org/10.1016/S2213-2600(15)00380-X

Ogden, J. C., Davis, S. M., Jacobs, K. J., Barnes, T., & Fling, H. E. (2005). The use of conceptual ecological models to guide ecosystem restoration in South Florida. *Wetlands*, *25*(4), 795–809.

Parker, M.M., Foreman, M.G., Abel, H.J., Mathias, R.A., Hetmanski, J.B., Crapo, J.D. … Beaty, T.H. COPDGene Investigators (2014). Admixture mapping identifies a quantitative trait locus associated with FEV1/FVC in the COPD Gene Study. (2014). *Genetic Epidemiology*. 2014 Nov; 38(7):652-9. Epub 2014 Aug 11. doi: 10.1002/gepi.21847

Pastor, J., Aber, J. D., McClaugherty, C. A. & Melillo, J. M. (1984). Above ground production and N and P cycling along a nitrogen mineralization gradient on Blackhawk Island, Wisconsin. Ecology 65 (1), 256 – 268. doi: 10.2307/1939478

Patel, B. D., Loo, W. J., Tasker, A. D., Screaton, N. J., Burrows, N. P., Silverman, E. K., & Lomas, D. A. (2006). Smoking related COPD and facial wrinkling: is there a common susceptibility? *Thorax*, *61*(7), 568–671.

Pleasants RA, Herrick H, Liao W. (2013). The prevalence, characteristics, and impact of chronic obstructive pulmonary disease in North Carolina. *N C Med J*. 2013 Sep-Oct; 74(5):376-83. Retrieved from, https://www.researchgate.net/profile/Roy_Pleasants/publication/259646136_Pleasants_R_Herrick_H_Liao_W_Chronic_obstructive_lung_disease_in_North_Carolina_Prevalence_characteristics_and_impact_North_Carolina_Medical_Journal_2013745376-383/links/0deec52d144e971cfc000000.pdf

Pleasants, R.A., Heidari, K., Wheaton, A.G., Ohar, J.A., Strange, C., Croft, J.B. … Kraft, M. (2015). *COPD*: *Journal of Chronic Obstructive Pulmonary Disease*. Targeting Persons With or At High Risk for Chronic Obstructive Pulmonary Disease by State-based Surveillance. 12(6):680-9. Epub 2015 Sep 14. Retrieved from, http://www.tandfonline.com/doi/abs/10.3109/15412555.2015.1043424

Pleasants, R.A., Ohar, J.A., Croft, J.B., Liu, Y., Kraft, M., Mannino, D.M…. Herrick, H.L. (2014). Chronic obstructive pulmonary disease and asthma-patient characteristics and health impairment. COPD: *Journal of Chronic Obstructive Pulmonary Disease*. 2014 Jun; 11(3):256-66. Epub 2013 Oct 23. Retrieved from, http://www.tandfonline.com/doi/abs/10.3109/15412555.2013.840571

Pleasants, R.A., Riley, I.L., Mannino, D.M. (2016). Defining and targeting health disparities in chronic obstructive pulmonary disease. *Int J Chron Obstruct Pulmon Dis. 2016 Oct 4; 11:2475-2496. Epub 2016 Oct 4.* doi: 10.2147/COPD.S79077

Power, C., Hyppönen, E., & Davey Smith, G. (2005). Socioeconomic position in childhood and early adult life and risk of mortality: a prospective study of the mothers of the 1958 British birth cohort. *American Journal of Public Health*, *95*(8), 1396–1402. http://doi.org/10.2105/AJPH.2004.047340

Prescott, E., Lange, P., & Vestbo, J. (1999). Socioeconomic status, lung function and admission to hospital for COPD: results from the Copenhagen City Heart Study. *European Respiratory Journal*, *13*(5), 1109–1114.

Reilly, J. J. & Kelly, J. (2011). Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: International Journal of Obesity 35, 891–898; doi:10.1038/ijo.2010.222

Regan, E. A., Hokanson, J. E., Murphy, J. R., Make, B., Lynch, D. A., Beaty, T. H., … Crapo, J. D. (2010). Genetic epidemiology of COPD (COPD Gene) study design. *COPD: Journal of Chronic Obstructive Pulmonary Disease*, *7*(1), 32–43. http://doi.org/10.3109/15412550903499522

Richard, L., Gauvin, L., & Raine, K. (2011). Ecological models revisited: their uses and evolution in health promotion over two decades. *Annual Review of Public Health*, *32*, 307–326.

Rogers, R. M., Dauber, J. H., Sanders, M. H., Claypool, W. D., Openbrier, D., & Irwin, M. (1984). Nutrition and COPD: state-of-the-Art Mini review. *CHEST Journal*, *85*(6_Supplement), 63S–66S.

Rusznak, C., Mills, P. R., Devalia, J. L., Sapsford, R. J., Davies, R. J., & Lozewicz, S. (2000). Effect of cigarette smoke on the permeability and IL-1 β and sICAM-1

release from cultured human bronchial epithelial cells of never-smokers, smokers, and patients with chronic obstructive pulmonary disease. *American Journal of Respiratory Cell and Molecular Biology*, *23*(4), 530–536. http://doi.org/10.1165/ajrcmb.23.4.3959

Ryff, C., Almeida, D. M., Ayanian, J., Carr, D. S., Cleary, P. D., Coe, C., Davidson, R., Krueger, R. F., Marge. National survey of midlife development in the United States (MIDUS II), 2004-2006 (ICPSR 4652). Retrieved from: https://www.icpsr.umich.edu/icpsrweb/NACDA/studies/4652?paging.startRow=1&keyword=relationships&recency=QUARTER

Sallis, J. F., Cervero, R. B., Ascher, W., Henderson, K. A., Kraft, M. K., & Kerr, J. (2006). An ecological approach to creating active living communities. *Annu. Rev. Public Health*, *27*, 297–322.

Sarkar, U., Fisher, L., Schillinger, D. (2006). Is self-efficacy associated with diabetes self-management across race/ethnicity and health literacy? *Diabetes Care*. 2006 Apr; 29(4): 823-829. Retrieved from http://dx.doi.org/10.2337/diacare.29.04.06.dc05-1615

Schauer, G.L., Wheaton, A.G., Malarcher, A.M., Croft, J.B. (2014). Smoking prevalence and cessation characteristics among U.S. adults with and without COPD: findings from the 2011 Behavioral Risk Factor Surveillance System. *COPD: Journal of Chronic Obstructive Pulmonary Disease*. 2014 Dec; 11(6):697-704. Epub 2014 May 19. http://dx.doi.org/10.3109/15412555.2014.898049

Seabrook, J. A., & Avison, W. R. (2012). Socioeconomic status and cumulative disadvantage processes across the life course: implications for health outcomes. *Canadian Review of Sociology*, *49*(1), 50–68. http://doi.org/10.1111/j.1755-618X.2011.01280.x

Services, T. F. on C. P. (2005). *The guide to community preventive services: what works to promote health?* Oxford University Press.

Sidney, S., Sorel, M., Quesenberry, C. P., DeLuise, C., Lanes, S., & Eisner, M. D. (2005). COPD and incident cardiovascular disease hospitalizations and mortality: Kaiser Permanente Medical Care Program. *CHEST Journal*, *128*(4), 2068–2075.

Silverman, E. K. (2002). Genetic epidemiology of COPD. *CHEST Journal*, *121*(3_suppl), 1S–6S.

Skinner, B. F. (1953). *Science and human behavior*. Simon and Schuster. Retrieved from http://books.google.com/books?hl=en&lr=&id=Pjjknd1HREIC&oi=fnd&pg=PA1&dq=science+and+human+behavior&ots=iOxgvpB4nJ&sig=Izcr3Zd_KT2UFfAVShwINmsA_rY

Smith, B. J., Tang, K. C., & Nutbeam, D. (2006). WHO health promotion glossary: new terms. *Health Promotion International*, *21*(4), 340–345.

Standard Occupational Classification (SOC) System. (n.d.). Retrieved November 21, 2013, from http://www.bls.gov/soc/

Stokols, D. (1992). Establishing and maintaining healthy environments: toward a social ecology of health promotion. *American Psychologist*, *47*(1), 6.

Stokols, D., Grzywacz, J. G., McMahan, S., & Phillips, K. (2003). Increasing the health promotive capacity of human environments. *American Journal of Health Promotion*, *18*(1), 4–13.

Tang, K., Rashid, R., Godley, J., Ghali, W. A. (2016). Association between subjective social status and cardiovascular disease and cardiovascular risk factors: a systematic review and meta-analysis. *BMJ Open* 2016;6:e010137 doi:10.1136/bmjopen-2015-010137

Teo, W. S. K., Tan, W. S., Chong, W. F., Abisheganaden, J., Lew, Y. J., Lim, T. K., & Heng, B. H. (2012). Economic burden of chronic obstructive pulmonary disease. *Respirology*, *17*(1), 120–126.

Toschke, A. M., Lüdde, R., Eisele, R., & Von Kries, R. (2005). The obesity epidemic in young men is not confined to low social classes—a time series of 18-year-old German men at medical examination for military service with different educational attainment. *International Journal of Obesity*, *29*(7), 875–877.

Travagli, V., Zanardi, I., Silvietti, A., & Bocci, V. (2007). A physicochemical investigation on the effects of ozone on blood. *International Journal of Biological Macromolecules*, *41*(5), 504–511.

Tse, H. N., Raiteri, L., Wong, K. Y., Yee, K. S., Ng, L. Y., Wai, K. Y., … Chan, M. H. (2013). High-dose N-acetylcysteine in stable COPD: the 1-year, double-blind, randomized, placebo-controlled HIACE study. *CHEST Journal*, *144*(1), 106–118.

Turner, G., & Shepherd, J. (1999). A method in search of a theory: peer education and health promotion. *Health Education Research*, *14*(2), 235–247.

Vijayan, V.K. (2013) Presence of depression & its risk factors in patients with chronic obstructive pulmonary disease. *Indian J Med Res*. 2013 Feb; 137(2):251-69. Retrieved from, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4069734/

Wharf Higgins, J., Begoray, D., & MacDonald, M. (2009). A Social ecological conceptual framework for understanding adolescent health literacy in the health education classroom. *American Journal of Community Psychology*, *44*(3/4), 350–362. http://doi.org/10.1007/s10464-009-9270-8

Wheaton, A.G., Pleasants, R.A., Croft, J.B., Ohar, J.A., Heidari, K., Mannino, D.M. … Strange C. J. (2016). Gender and asthma-chronic obstructive pulmonary disease overlap syndrome. *Journal of Asthma*. Sep; 53(7):720-31. Epub 2016 Apr 6. http://dx.doi.org/10.3109/02770903.2016.1154072

World Health Organization. (2012). Health systems financing: the path to universal coverage. 2010. *The World Health Report URL: http://www. who. int/whr/2010/en/[accessed 2014-10-18][WebCite Cache ID 6TPqanY1w]*.

Xu, X., Yavar, Z., Verdin, M., Ying, Z., Mihai, G., Kampfrath, T. … Sun, Q. (2010). Insights into the mechanisms and mediators of the effects of air pollution exposure on blood pressure and vascular function in healthy humans. *Arteriosclerosis, Thrombosis, and Vascular Biology*. 2010; 30:2518-2527. http://dx.doi.org/10.1161/ATVBAHA.110.215350

Yin, P., Zhang, M., Li, Y., Jiang, Y., & Zhao, W. (2011). Prevalence of COPD and its association with socioeconomic status in China: findings from China Chronic

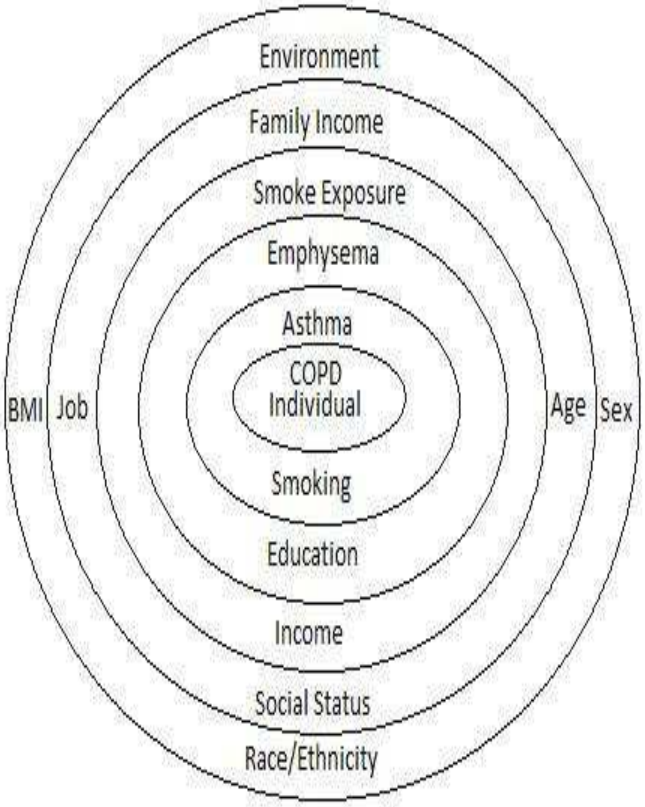
Disease Risk Factor Surveillance 2007. *BMC Public Health*, *11*(1), 586. http://doi.org/10.1186/1471-2458-11-586

Zaher, C., Halbert, R., Dubois, R., George, D., & Nonikov, D. (2004). Smoking-related diseases: the importance of COPD. *The International Journal of Tuberculosis and Lung Disease*, *8*(12), 1423–1428.

Zeng, G., Sun, B., & Zhong, N. (2012). Non-smoking-related chronic obstructive pulmonary disease: A neglected entity? *Respirology*, *17*(6), 908–912. http://doi.org/10.1111/j.1440-1843.2012.02152.x

Zu Wallack, R. L., Mahler, D. A., Reilly, D., Church, N., Emmett, A., Rickard, K., & Knobil, K. (2001). Salmeterol plus theophylline combination therapy in the treatment of COPD. *Chest*, *119*(6), 1661–1670. http://doi.org/10.1378/chest.119.6.1661

Appendix A: Ecological Model That Affects COPD



Appendix B: Terms of Use Agreement

ICPSR signed agreement of dataset use

On 2015-06-09, Hilaire Saint-Pierre agreed to the terms below pursuant to the download of folder 13372031.

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When sharing data or other materials in these approved ways, you must include all accompanying files with the data, including terms of use. More information on permission to redistribute data can be found on the ICPSR Web site.

Citing Data

You agree to reference the recommended bibliographic citation in any publication that employs resources provided by ICPSR. Authors of publications based on ICPSR data are required to send citations of their published works to ICPSR for inclusion in a database of related publications (bibliography@icpsr.umich.edu).

Disclaimer

You acknowledge that the original collector of the data, ICPSR, and the relevant funding agency bear no responsibility for use of the data or for interpretations or inferences based upon such uses.

Violations

If ICPSR determines that the terms of this agreement have been violated, ICPSR will act according to our policy on terms of use violations. Sanctions can include:

- ICPSR may revoke the existing agreement, demand the return of the data in question, and deny all future access to ICPSR data.
- The violation may be reported to the Research Integrity Officer, Institutional Review Board, or Human Subjects Review Committee of the user's institution. A range of sanctions are available to institutions including revocation of tenure and termination.
- If the confidentiality of human subjects has been violated, the case may be reported to the Federal Office for Human Research Protections. This may result in an investigation of the user's institution, which can result in institution-wide sanctions including the suspension of all research grants.
- A court may award the payment of damages to any individual(s)/organization(s) harmed by the breach of the agreement.

Definitions

Authorized user

A faculty member, staff member, or student at a member institution

ICPSR Inter-university Consortium for Political and Social Research

Member institution an institutional member of ICPSR Official/Designated Representative An individual appointed to represent a university's interests in ICPSR. This individual is also charged with providing user support to campus users. Promise of confidentiality. A promise to a respondent or research participant that the information the respondent provides will not be disseminated without the permission of the respondent; that the fact that the respondent participated in the study will not be disclosed; and that disseminated information will include no linkages to the identity of the respondent. Such a promise encompasses traditional notions of both confidentiality and anonymity. Names and other identifying information regarding respondents, proxies, or other people on whom the respondent or proxy provides information, are presumed to be confidential.

Research subject

A person or organization observed for purposes of research. Also called a respondent. A respondent is generally a survey respondent or informant, experimental or observational subject, focus group participant, or any other person providing information to a study or on whose behalf a proxy provides information.