2016

The Association of Smoking with Low Back Pain in Adult Americans: Analysis of the 2012 National Health Interview Survey

Bart Green
Walden University

Walden University

College of Health Sciences

This is to certify that the doctoral dissertation by

Bart Green

has been found to be complete and satisfactory in all respects,
and that any and all revisions required by
the review committee have been made.

Review Committee
Dr. Jeff Snodgrass, Committee Chairperson, Public Health Faculty
Dr. Patricia Risica, Committee Member, Public Health Faculty
Dr. Vincent Agboto, University Reviewer, Public Health Faculty

Chief Academic Officer
Eric Riedel, Ph.D.

Walden University
2016

Abstract

The Association of Smoking with Low Back Pain in Adult Americans: Analysis of the 2012 National Health Interview Survey

by

Bart N. Green

MSEd, University of Southern California, 2000

DC, Los Angeles College of Chiropractic, 1992

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health

Walden University

January 2016

Abstract

Back pain is a chronic disease epidemic and the most common chronic painful condition in Americans. It is associated with human suffering and enormous financial and social burdens. Smoking is a prevalent and harmful health behavior and is the greatest modifiable risk factor for many chronic diseases. Cigarette smoking is associated with back pain, but there is little research on this relationship among adults in the United States. Using biopsychosocial theory, this study examined (a) the prevalence of back pain (dependent variable) among smokers, former smokers, and never smokers (independent variable), and (b) the influence of age, sex, race, body mass index, level of physical activity, level of education, depression, and anxiety on predicting the likelihood of back pain. This cross-sectional secondary analysis of the 2012 National Health Interview Survey included over 34,000 respondents and utilized chi-square distribution, *t* test, one-way analysis of variance, and multiple logistic regression analysis. People who self-reported being anxious or worried, had been diagnosed with depression by a health care provider, were current or former smokers, obese, or failed to meet recommended levels of physical activity were more likely to have back pain. This study has implications for social change in the United States because it shows that anxiousness, depression, smoking, obesity, and low physical activity are risk factors for back pain in Americans. Further, it indexes the need for primary studies of the relationship between smoking and back pain to determine whether smoking is causal for back pain. These studies could lead to public health interventions that develop strategies to prevent back pain and thereby alleviate some of the social burden associated with this common and costly ailment.

The Association of Smoking with Low Back Pain in Adult Americans: Analysis of the 2012 National Health Interview Survey

by

Bart N. Green

MSEd, University of Southern California, 2000

DC, Los Angeles College of Chiropractic, 1992

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health

Walden University

January 2016

Dedication

Foremost I want to thank my wife, Claire Johnson, who mutually agreed to accept the same challenge and will finish her PhD simultaneously. What a journey this has been together! I thank her for the love, encouragement, and prodding necessary at times and, most importantly, for a genuine interest in my work and for the many brainstormings, planning meetings, war boardings, and mutual learning opportunities. This work is dedicated to her. This dissertation is also dedicated to our mutual friend and mentor, Joseph Keating, Jr, who encouraged, counseled, advised, and cared that we pursue the goal of PhD. While we lost him too soon, his influence and spirit stayed with us to the end and will always.

Acknowledgments

Behind any effort such as a dissertation, there is a team and there are supporters. I would like to thank my committee chair, Jeff Snodgrass for his invaluable assistance in seeing this dissertation through to completion by being willing to step up as my new committee chair and for the excellent and formative feedback provided along the way. I thank my former chair, Cassius Lockett for his unwavering patience, since it took me longer than most students to complete this process due to my other work and life commitments. Thanks are also due to Patricia Risica, my committee member, who also came on to the project late in the game during turnover in faculty and I greatly appreciate her input on the dissertation. I also owe a great debt to Monica Smith for coaching me for many hours on my SPSS data preparation and complex samples set up and analysis. There are also those friends and colleagues who are just good people and cared enough to ask me how things were progressing with the dissertation whenever I saw them. I thank them for asking, for letting me bore them to tears with epidemiology, and for genuinely caring. They do not know how much I appreciate them letting me discuss my research and how formative those discussions were, but I recognize them here: Louis Sportelli, Andy Dunn, Scott Gilford, Cindy Chapman, Ken Young, Sil Mior, Rick Branson, Bic Bakkum, Russ Linderman, and Gregg Schluter. I am blessed to have such intelligent and caring people in my life.

Table of Contents

List of Tables

List of Figures

Chapter 1: Introduction to the Study

Introduction

The public health consequences of the morbidity of back pain and mortality associated with smoking are staggering. Back pain appears to have a relationship with smoking, although the magnitude of this association has not been the subject of much investigation in the United States (Shiri, Karppinen, Leino-Arjas, Solovieva, & Viikari-Juntura, 2010). Together, back pain and smoking are two highly prevalent problems that negatively affect the health of the public.

Back pain is a chronic disease epidemic. As a highly prevalent human ailment (Evans, 2011), back pain is the most common chronic painful condition in Americans (Johannes, Le, Zhou, Johnston, & Dworkin, 2010), and is associated with reduced quality of life, human suffering, and enormous financial and social burdens. Yet, back pain has no cure. Smoking is a prevalent but harmful health behavior amongst Americans (Centers for Disease Control and Prevention, 2011) that represents the greatest modifiable risk factor for many chronic diseases (Mokdad, Marks, Stroup, & Gerberding, 2004). The "cure" for smoking is cessation. There are effective smoking cessation interventions supported by healthcare policy and public health programs (Wilson et al., 2012).

My study explored the hypothesized relationship between back pain and smoking behavior in adult Americans using a large epidemiologic survey, the National Health Interview Survey (Division of Health Interview Statistics, 2013). If a significant measurement of association exists between smoking and back pain, then it will signal the

need for future research that investigates the temporality of this relationship and the potential for smoking to be causal for back pain.

This chapter introduces the topic of back pain, its rising prevalence, and the enormous amount of human suffering and financial burden it causes. I discuss the potential relationship between smoking and back pain, and offer a brief overview of the theoretical and conceptual frameworks used to support the hypotheses that were tested in this study. Chapter 1 also includes a brief description of the purpose of the study, the data source utilized in the methods, and the significance of the study.

Background

Chronic and costly medical conditions such as cardiovascular disease and chronic obstructive pulmonary disorder have been associated with cigarette smoking for many decades, but it was not until the late 1970s that scientists began to suspect a similar relationship between smoking and back pain (H. Andersson, Ejlertsson, & Leden, 1998). Epidemiological studies from investigators outside of the United States vary in their conclusions regarding an association between back pain and smoking, suggesting that the choice of covariables and confounders likely influence the relationship (Ackerman & Ahmad, 2007; Shiri et al., 2010).

Despite back pain and smoking being listed as objectives of Healthy People 2020 (U.S. Department of Health and Human Services Office of Disease Prevention and Health Promotion, 2010), there is exceedingly little epidemiological research done on this topic in the United States. Of the research that has been done, these data are over a decade old and combine the prevalence of neck pain and back pain with a plethora of

comorbidities and health behaviors making it difficult to tease out confounders for the potential relationship between smoking and back pain (Strine & Hootman, 2007). Against the backdrop of the increasing economic and social costs of back pain, my study attempts to accurately estimate the association of back pain (as opposed to pain in the entire spine) with smoking using current data and more targeted variables than previous research.

Chapter 2 presents a full discussion of the relevant literature relating to the individual and social burdens associated with back pain and smoking. However, it is worth pointing out briefly here that back pain is the most common chronic musculoskeletal problem in Americans (Johannes et al., 2010), and smoking is the most common preventable cause of death. These are serious public health concerns that continue to persist.

Knowledge Gap and Need for This Study

The study of back pain and smoking in the U.S. population is limited to one paper (Strine & Hootman, 2007). Of this research, the data are more than a decade old. The previous study lacks a deliberate epidemiologic approach that investigates variables common to both smoking and back pain. The previous study also combined data for back and neck pain, and it is important to study back pain only because its social and health care burdens may be different than those associated with neck pain. My study fills a void in the literature by addressing these issues using more contemporary data. More importantly, my study highlights the important link between smoking and back pain to guide future studies that may look at causal models in a more prospective manner.

Problem Statement

Numerous studies have reported the costs, mortality, and morbidity of smoking or back pain as independent public health problems. Comparatively little effort has been made to investigate the association between back pain and smoking in the U.S. population. The intention of this study is to investigate this association, thereby filling this critical gap in research.

Purpose of the Study

This quantitative cross-sectional study explored the association of self-reported smoking behavior and the presence of back pain in U.S. adults using data from the 2012 National Health Interview Survey, a nationally representative cross sectional epidemiologic survey. The independent variable was smoking. The dependent variable was back pain. The covariables were age, sex, body mass index (BMI), level of education, level of physical activity, race, depression, and anxiety.

Research Questions and Hypotheses

This study focused on adult Americans over the age of 18 years. My primary research objective was to examine the prevalence of back pain among current smokers compared to former smokers and never smokers. I considered the influence of confounders in the potential risk of back pain for smokers. My secondary objective for this study was to determine if certain covariates were confounders for the potential relationship of smoking and back pain. I used the following research questions and associated hypotheses (H_0 = null hypothesis, H_1 = alternative hypothesis):

1. What is the association between back pain in current smokers, former smokers, and never smokers?

   H_0: There is no significant difference in the prevalence of back pain between current smokers, former smokers, and never smokers as measured by the National Health Interview Survey.

   H_1: The prevalence of back pain is greater in current smokers than in former smokers and never smokers.

2. What is the association between back pain and the non-modifiable individual factor of sex and race?

   H_0: There is no significant difference in the prevalence of back pain between sex and racial groups.

   H_1: The prevalence of back pain is significantly different between sexes and races.

3. What is the association between back pain and the non-modifiable individual factor of age?

   H_0: There is no significant difference in the prevalence of back pain across ages.

   H_1: The prevalence of back pain is significantly different for certain ages.

4. What is the association between back pain and modifiable individual factors, including BMI, level of education, and level of physical activity?

   H_0: There is no significant relationship between back pain and BMI, education, or level of physical activity.

H_1: There is a relationship between back pain and BMI, education, and/or level of physical activity.

5. Do smoking, age, sex, BMI, level of education, level of physical activity, race, depression, or anxiety predict back pain?

   H_0: There is no statistical model that predicts back pain using the variables under study.

   H_1: A combination of variables predicts the occurrence of back pain.

Theoretical and Conceptual Frameworks for the Study

I used biopsychosocial theory to ground the research questions and a wheel of causation model to conceptualize the study's multivariable analysis.

Theoretical Foundation

Because the body, mind, and social interactions are inextricably linked with both smoking and pain, I used Engel's biopsychosocial model (Engel, 1977) for the theoretical foundation of this study. Briefly, the biopsychosocial model attempts to interrelate the biological, psychological, and social factors that affect health (Alonso, 2004; Shorter, 2005). Biological factors are physical or chemical changes to the body; psychological factors are represented by personal growth and mental health; and social factors are social interactions and health/disease context (Engel, 1977). This theoretical model is a good fit for investigating the association between smoking and back pain since there are several determinants for why people smoke and why they may have back pain. I offer a more detailed description of this model in Chapter 2.

Conceptual Framework

Building on the theoretical foundation provided by Engel's biopsychosocial model, I used the "wheel" of human-environment interactions developed by Mausner and colleagues (Mausner & Bahn, 1974) as a conceptual model to formulate a framework for the study's multivariable analysis. The wheel of causation is a model for conceptualizing health or disease from a multicausal perspective. At the center of the model is the host and genetic factors that affect health. Surrounding the host are factors from the biological, social, and physical environments that affect the host population (Fos, 2011). The wheel of causation allows one to consider numerous variables in the design of a research study. Derived from the wheel model, the analytical model for my study's multiple variable analysis included biologic, psychosocial, and outcome variables.

Nature of the Study

The study was a quantitative cross-sectional secondary data analysis. The sample was American adults responding to the 2012 National Health Interview Survey, which is a large-scale in-person interview with data from 75,000 to 100,000 individuals that provides a statistically representative sample of the U.S. civilian non-institutionalized population. The design of this study was a cross-sectional analysis which allowed for the computation of odds ratios to detect the measurement of association between the variables under study. The independent variable was smoking. The dependent variable was back pain. The covariates were age, sex, BMI, level of education, level of activity, race, depression, and anxiety. A thorough description of the methods used in this study is presented in Chapter 3.

Definitions

Age: The number of years a person has been alive. This study investigates adults, defined as people considered to be greater than 18 years of age.

Anxiety: Is the anticipation of future threat, and is associated with muscle tension and vigilance in preparation for future danger and cautious or avoidant behaviors (American Psychiatric Association, 2013). When a patient has back pain, he or she may be fearful of re-injury or worsening pain and may become anxious due to this association (Leeuw et al., 2007).

Back pain: Often defined as pain that exists below the neck and above the gluteal folds, in the thoracic and/or lumbar area (Schmidt et al., 2007). Pain extending from the back into one of the legs is sometimes accepted as part of the definition of back pain (Bejia et al., 2005).

Body mass index: Calculated by dividing weight in kilograms by height in meters squared (kg/m2) (Goodman & Anise, 2006).

Depression: One of several depressive disorders, including disruptive mood dysregulation disorder, major depressive disorder, depressive disorder due to another medical condition, and others. For this study, depression, or depressive symptoms, include "sad, empty, or irritable mood, accompanied by somatic and cognitive changes that significantly affect the individual's capacity to function" (American Psychiatric Association, 2013).

Intervertebral disc: A structure found in the spine of vertebrate animals. One disc resides between each vertebral bone. Intervertebral discs are essential load bearing,

stability producing spinal structures constructed of a tough, fibrous external portion called the annulus fibrosis and a softer inner gelatinous matrix called the nucleus pulposus, which is 70-80% water (Akmal et al., 2004).

Pain: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage." (International Association for the Study of Pain, 2011). Acute pain, usually lasting no more than 12 months (depending upon which study one reads), is relieved when the pain-inducing stimulus is removed (Ditre, Brandon, Zale, & Meagher, 2011). Chronic pain is an unpleasant experience that last longer than acute pain and does not necessarily subside when the pain-inducing stimulus is removed.

Physical activity: Movement by the human during leisure time, that includes exercise, sports, and physically active hobbies (National Center for Health Statistics, 2013).

Race: A person's identification with one or more racial groups, as delineated in NHIS.

Sex: Defined as either male or female.

Smoking: The use of cigarettes.

Vertebral body: The largest part of the vertebral bone made of spongy bone that contains numerous blood vessels. One intervertebral disc is located between two vertebrae, at the location of the vertebral body (Cramer, Darby, & Cramer, 2014).

Assumptions

I made several assumptions in this study. Chief among these was that back pain is a serious public health problem that is worthy of investigation. Secondly, I assumed that smoking is a health behavior with negative consequences. Finally, I assumed that the responses in the NHIS were true and accurate, and that the NHIS was truly a nationally representative survey.

Scope and Delimitations

For this study I included only adults (age > 18 years). The NHIS used a representative sample of the U.S. population. The study was very large and used a multistage area probability design with a response rate that exceeded 90%. The survey had been thoroughly assessed using psychometrics and was administered by highly trained data collectors who were assessed via quality control measures.

Limitations

The NHIS survey instrument did not assess pain severity, and the NHIS did not provide a clear definition of "back" to the respondent. Thus, some respondents may have though that "back" included the neck and/or buttocks, even though there were separate questions for the neck. Duration of pain was also not assessed in the NHIS. The NHIS was based upon self-report, so validity of data was limited in this regard because it was subject to misclassification bias. Because my study used a cross-sectional research design, it was impossible to determine if smoking is a cause for low back pain since there was no control group, the design was retrospective, and the study was observational in nature (Schoenbach & Rosamond, 2000). A further limitation inherent to my study's

research design was that the sample was comprised only of living people. Therefore, the results may have been biased by not including those people who have died (Friis & Sellers, 2009). This study was intentionally limited to the population of U.S. adults, and therefore generalizations should not necessarily be made beyond U.S. adults.

In this study, I attempted to limit the effects of confounding by variables related to back pain and/or smoking by including known factors (age, sex, depression, anxiety, BMI, level of education) in the statistical analysis. There is a small chance however that an unknown confounder could have distorted the estimates for the measurement of association between smoking and back pain (Goldberg, Scott, & Mayo, 2000).

Significance

This study contributes to a better understanding of how smoking is associated with back pain because current measurements of association for the U.S. population are unavailable. Should significant associations between back pain and smoking in Americans be revealed in this study, it could stimulate further research to investigate if smoking is causal for back pain or if back pain stimulates smoking behavior. Since no cure for back pain exists, if smoking is causal for back pain, then a primary prevention approach to lower the prevalence of back pain and its associated suffering and social burden through smoking cessation could have large social implications.

Summary

Back pain and smoking are independently associated with colossal health, financial, and social burdens. That each may be related to one another has received little study in the United States. This quantitative cross-sectional study explored the

association of smoking and back pain in U.S. adults using data from the 2012 NHIS, a nationally representative cross-sectional epidemiologic survey. The independent variable was smoking. The dependent variable was back pain. The covariables were age, sex, BMI, level of education, level of activity, race, depression, and anxiety. Chapter 1 has provided an overview of the study. Chapter 2 presents an exhaustive review of the literature pertaining to the association of back pain and smoking, the various theories available to support or negate this relationship, and the methods used to obtain and synthesize the literature reviewed. Chapter 3 presents the methods used to conduct the study. Chapter 4 presents the results of this research and Chapter 5 provides a comprehensive discussion of the interpretation of the data, including the consideration of study limitations and potential social implications of this research.

Chapter 2: Literature Review

Introduction

This quantitative study explored the association of self-reported smoking behavior and the presence of back pain in U.S. adults using data from the 2012 NHIS, a nationally representative cross-sectional epidemiologic survey. The independent variable was smoking. The dependent variable was back pain. The covariates were age, sex, BMI, level of education, level of physical activity, race, depression, and anxiety.

The association of back pain and smoking is an important and relevant public health problem. Back pain affects every culture around the globe with a prevalence exceeding 70% (Waddell, 1987), and smoking is reported in all countries regardless of socioeconomic status, and is associated with numerous morbidities (World Health Organization, 2013). Back pain and smoking are associated with physical impairments that may be permanent. While back pain is rarely associated with death, smoking is considered the most common preventable cause of death (Mokdad et al., 2004). Studies in the field of pain medicine have shown that there is a relationship between pain expression and smoking in many individuals. Often psychological comorbidities, including anxiety and depression, are intertwined with the behavior of smoking and with pain (Ditre et al., 2011; Gatchel, Peng, Peters, Fuchs, & Turk, 2007). How these variables interact and affect the back pain experience is an area in need of further research.

In this Chapter, I present the literature I reviewed to inform my research. First, I discuss the literature search methods and present the number of relevant primary sources. Second, I discuss in detail the theoretical foundation used in this study. Finally, using the

classic epidemiological categories of population, frequency, distribution, determinants, control, and burden, I review the literature pertaining to the association of back pain and smoking.

Literature Search Strategy

PubMed was the primary library database I searched for this review of the literature, and I used it from the earliest entries in the database through December 2013. I combined the term “smoking” with the term “back pain” in the advanced search function of PubMed so as to include a search of medical subject headings (MeSH) of PubMed as well as the titles and abstracts of indexed articles. My search was limited to articles published in English because a pilot literature search demonstrated that the overwhelming majority of articles on this topic were published in English, and access to and translation of articles published in other languages was beyond the resources allocated for this study. Thus, I organized the PubMed search string as follows: “smoking"[MeSH Terms] OR "smoking"[All Fields]) AND ("back pain"[MeSH Terms] OR ("back"[All Fields] AND "pain"[All Fields]) OR "back pain"[All Fields]) AND English[lang].

I also performed a search for dissertations pertinent to the topic in the ProQuest dissertations database using the same search terms and strategy as was employed in the PubMed search. Using the search terms “smoking” and “back pain” present in the abstracts of indexed dissertations and theses, I also searched for and retrieved dissertations from the Networked Digital Library of Theses and Dissertations. I located additional articles by reading the references found in the articles retrieved from the above sources and crosschecking them to the articles I had already obtained, and by contacting

authors who have published in this area. I considered articles for final inclusion if they specifically addressed the potential association between smoking and back pain and included the description or testing of plausible theories for this relationship, measurement of association, prevalence/incidence, and the burden of disease.

All published research designs were considered eligible for inclusion in the synthesis of literature except for case reports, conference abstracts, and letters to the editor. I excluded case reports and letters to the editor because of their low level of evidence in an area of scholarship that already possesses numerous articles of more complex designs. Further, I excluded conference proceedings consisting only of abstracts because of the high rate of biomedical conference presentations that never reach full publication (Dumville, Petherick, & Cullum, 2008; Scherer, Langenberg, & von Elm, 2007); however, I did include full papers published in conference proceedings included as bound volumes of journals. Commentaries from non-peer reviewed sources (e.g., trade magazines) and other non-scholarly sources were excluded, as were writings not specific to the relationship of smoking with back pain.

I conducted an initial search in December 2011, with updates each month thereafter until December 2013, and saved abstracts of the citations that obviously or possibly met the review criteria. I retrieved the full papers of each abstract and then independently reviewed each article to verify that it met the inclusion criteria. Papers that did not meet the criteria were discarded, and I noted reasons for doing so. I did not perform quality scoring because the articles reviewed were not homogenous and data could not be pooled.

My initial search of PubMed yielded 532 citations. After reading the titles and abstracts, I excluded 317 citations. Efforts to retrieve the full papers of the remaining 215 citations garnered 211 full papers, representing a rate of retrieval of 98% of the target papers. The ProQuest search yielded 15 citations with three relevant dissertations available in this area of inquiry, and the Networked Library of Theses and Dissertations yielded 8 citations with one being relevant to my study.

I performed additional less formal literature reviews for several reasons. I conducted one search for studies to provide context for various sections within Chapters 1, 2, and 5. I also included additional materials provided by colleagues familiar with my work in this area. Additional searches and similar contributions by knowledgeable colleagues garnered materials that provided a meaningful theoretical foundation and conceptual framework for my research.

Theoretical Foundation

Numerous theories exist to explain disease and its causes. Because of the fundamental shortcomings of the epidemiologic triangle of disease model as it relates to diseases or disorders that are multifactorial (such as smoking and back pain), I used biopsychosocial theory to guide this study. In the subsections that follow, I review alternative theories and the reasons why I chose not to include them.

Epidemiologic Triangle of Disease Model

The epidemiologic triangle is a core model used in epidemiology to explain disease causation. The three points of the epidemiologic triangle (Figure 2.1) are the host, the agent, and the environment (Friis & Sellers, 2009). The host is the organism that gets

the disease, the agent is the disease causing organism, and the environment is that realm in which the interaction between the host and agent transpires (Friis & Sellers, 2009). Whether the host actually gets the disease is dependent upon a variety of factors that are inherent to each point in the triangle. For example, host resistance is a factor determining whether the host is susceptible, agent infectivity is a factor bearing on how likely the agent is to cause infection, and environment plays a role in the final point of the triangle by determining if conditions are optimal for an infection. Since the triangle is typically used in models of infectious disease causation, a vector is included in the model.

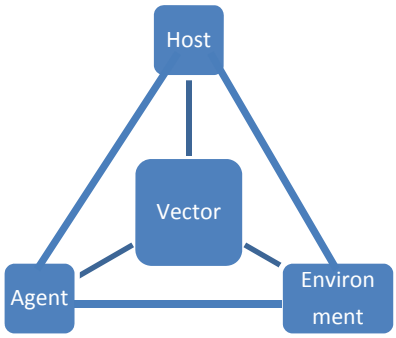

Figure 2.1. The epidemiologic triangle.

While the epidemiologic triangle is perhaps best known as a model used to explain the pathogenesis of infectious disease, it has also been applied to injury models in work done by Drs. John Gordon and James Gibson during the mid-1900s, and served as a basis for the emergence of multicausal models of mass disease (Gordon, 1954; Runyan, 2003). In the injury model, the hosts remain the people who get the disease (injury), the agent is represented by physical energy, and the environment is represented by the physical and atmospheric conditions where the injury is sustained (Rockett, 2009).

While the injury epidemiologic triangle model is more applicable to chronic non-infectious diseases than its predecessor, it still lacks room for considerations of the sociological and psychological factors involved in most non-infectious, non-accidental health concerns such as cancer, diabetes, or back pain. One criticism of the triangle, or any single disease mechanism model (Joffe, Gambhir, Chadeau-Hyam, & Vineis, 2012), is that it is heavily based upon the Henle-Koch approach to disease causation which requires that (a) one agent to be present in every cause of disease, (b) that the agent to be able to only cause one disease, and (c) that healthy hosts who are exposed to the agent get the disease (Friis & Sellers, 2009). Further, the triangle is heavily influenced by the concept of dualism prevalent in biomedical theory, which posits that the mind and body of the host are separate and function independently (Urdang, 2002), and that the mind is not influential on the disease/injury experience. As Dr. Scott Swisher (1980), past Associate Dean of Research at Michigan State University, so eloquently stated, "This is the theme that man is not reducible to a series of interacting biological systems that are totally describable in physicochemical terms" (Swisher, p. 113). Thus, the triangle model holds that disease, illness, or dysfunction can be explained through mechanisms or pathways that are entirely biological or physical in nature.

With regard to smoking and back pain, using the triangle model, the host would be represented by humans who experience back pain, the agent would be represented by exposure to inhaling cigarette smoke, and the environment might include the various other factors affecting the host. However, as the back pain experience is more than biological, since it indeed involves pain, there is little room in the triangle model for the

psychological and sociological factors inherent in the experience of pain. For this, we turn to other theoretical foundations to serve as the lens for this dissertation.

Biopsychosocial Model

Advocates of holism have tried to incorporate social and cultural issues as well as personal experience into medical theory since it is the opinion of many theorists and philosophers that illness or disability "… have profound and widespread biopsychosocial effects" (Urdang, 2002, p. 65). Interest in a more holistic approach to understanding health and its associated factors existed as early as the days of Hippocrates, but the "biopsychosocial model" was developed over many years by George L. Engel, MD (Shorter, 2005) and given wide dissemination in his seminal article on the topic (Engel, 1977), published in *Science*, which gave rise to the popularity of the model (Cohen, 2000). There were several reasons that Engel's model resonated with scientists, practitioners, and the public. First, Engel positioned the then-prevailing dualistic biomedical model as a failed construct that needed to be replaced, an argument that was a direct challenge to organized medicine, brash, and garnered great attention during the period of liberation known during the 1970s. Second, the publication of the model in a major scholarly journal with wide distribution during a time when the world was just beginning to recognize that pharmacological remedies were not effective in a vast number of chronic diseases aligned with the way that many people were thinking (Shorter, 2005).

The biopsychosocial model has at its heart an appreciation for the interplay of biological, psychological, and social factors that affect health (Alonso, 2004; Shorter,

2005). Physical or chemical changes to the body constitute the biological contribution, personal growth and mental health are the psychological influence, and social interactions and context represent the final aspect (Engel, 1977). Engel provided an understanding of the determinants for disease, especially for those diseases or disorders for which the biomedical model was not a good fit (Lindau, Laumann, Levinson, & Waite, 2003). Through his model Engel attempted to transcend the prevailing dualistic separation of mind and body prevalent in the dominant biomedical model of the period (Jacob, 2003).

Engel claimed that his model was based upon the general systems theory as espoused by Ludwig von Bertalanffy (Engel, 1977; Malmgren, 2005). General systems theory posits that there are multiple systems at work in any phenomenon and that these systems also interact with one another and that there are some universal principles that apply to all of the systems under consideration (Bertalanffy, 1972). General systems theory is a holistic (some might even suggest vitalistic) attempt to understand the functions of organisms or organized entities in a matter that considers numerous processes simultaneously and in a hierarchy (Puustinen, Leiman, & Viljanen, 2003). As von Bertalanffy stated, “Since the fundamental character of the living thing is its organization, the customary investigation of the single parts and processes cannot provide a complete explanation of the vital phenomena.” (Bertalanffy, 1972) He later elaborates that from this first conceptualization, general systems theory developed to include social groups and even technology in systems theory (Bertalanffy, 1972).

Von Bertalanffy described general systems theory as a model that helped to understand general aspects of reality in a manner that had previously been overlooked,

such that a holistic and intricately woven set of interactions within and between various levels of organization (Engel, 1977). Accordingly, a change in any level of human functioning affects all other levels (Novack et al., 2007). For example, it is well known that chronic psychological stress can lead to a number of harmful biologic outcomes, such as heart disease and other conditions, as mediated by hormonal and neurological systems, as thoroughly explained by Hans Selye and other researchers investigating the general adaptation syndrome theory (Novack et al., 2007). Thus, viewed from the biopsychosocial model (Figure 2.2), all diseases or disorders of humans contain biological, psychological, behavioral and social interactions.

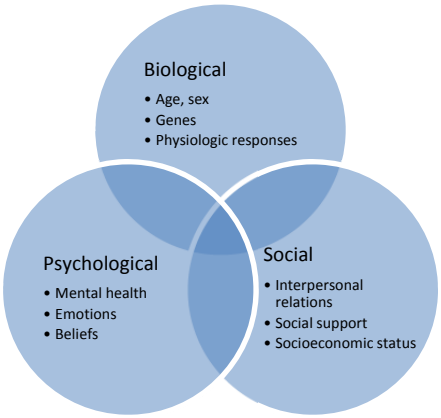

Figure 2.2. The biopsychosocial model demonstrates how various factors interrelate to affect physical and mental health.

Determinants of Health

As Engel (1977) argued in his papers, the biopsychosocial model provided a more accurate "basis for understanding the determinants of disease"(p. 132) than previous models. Public health has a venerable position, holding that consideration of social determinants of health is vital to alleviating human suffering and achieving a more

socially just balance of human health. Determinants of health are factors that may affect the health of the population (Johnson & Green, 2009). In a population there are usually social determinants, involving socioeconomic factors as well as individual determinants, such as non-modifiable risk factors and health behaviors (Baker, Metzler, & Galea, 2005). Figure 2.3 depicts the interaction of the determinants of health.

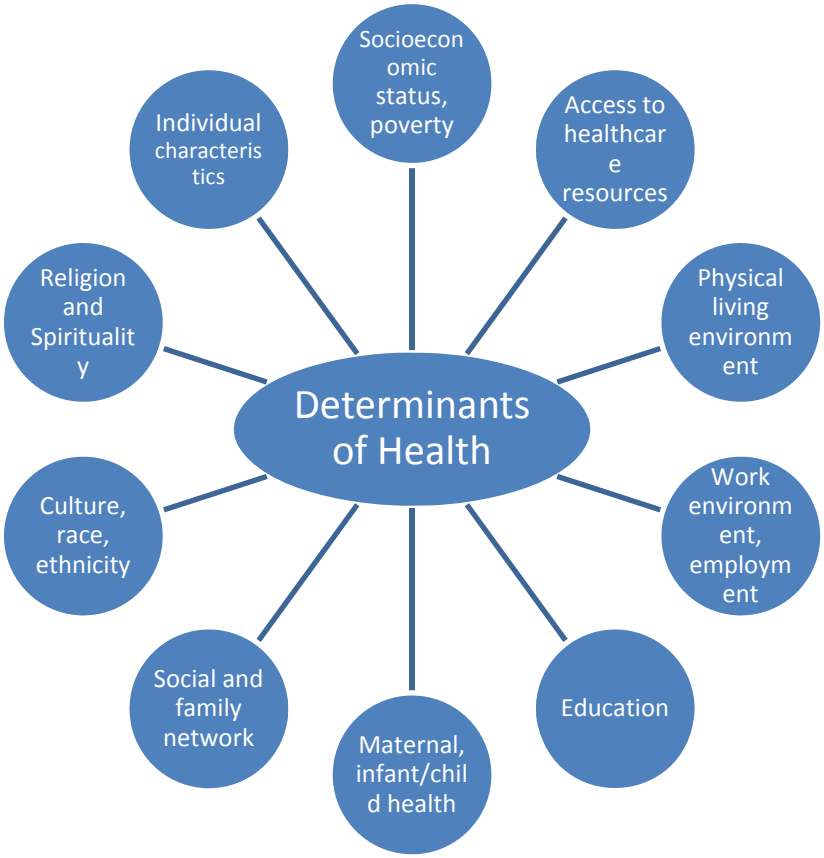

Figure 2.3. Various factors serve as determinants of health in some health problems, such as back pain. Determinants of health include psychosocial variables. From Public health,

wellness, prevention, and health promotion: considering the role of chiropractic and determinants of health, by C.D. Johnson and B.N. Green, 2009, *Journal of Manipulative and Physiological Therapeutics, 32,* 407.

When considering the complexities involved in the back pain experience and smoking behavior, not only for individuals but also for the social networks where individuals function, the determinants of health model provides a compelling picture of how important it is to consider multicausation. Further contemplation of the extremely high prevalence of back pain, its lack of control, the colossal financial burden associated with it, and the enormity of human suffering involved with it makes a clear case for the use of multicausal models considering biological, psychological, and social determinants of illness. Merely looking at back pain and smoking through a biological model seems naïve.

Previous Use of Theories

Concepts of integrating biological, psychological, and social factors in the causation of disease or dysfunction are not new to epidemiology. In the early 1960s, celebrated epidemiologist John Cassel, MD, MPH advocated the use of sociological theories in epidemiological research. In particular, he pointed out that the then-prevalent germ theory of disease was an inappropriate model for epidemiological investigations of chronic illnesses, such as rheumatic disorders, cardiovascular disease and somatic complaints (Cassel, 1964). Cassel favored theories of multiple causation that involved variables of biological, psychological and social origin, particularly the study of how psychological distress affected health (Cassel, 1974), leading to a great deal of seminal work in the generalized susceptibility hypothesis (Cassel, 1975). In his work, Cassel uses

the host-agent-environment model to argue for a fuller and more accurate theoretical perspective of disease causation than permissible with the germ theory (host-agent) of disease (Krieger, 2001).

Several more recent studies on pain and disability highlight the use of multicausal theoretical models and the biopsychosocial approach. For example, the interplay between pain and depression was investigated to cross validate a biopsychosocial model in subjects suffering from rheumatoid arthritis. When using a research theoretical model that incorporated data on each of the three parts of the biopsychosocial model, the researchers found that physical disability, helplessness and coping strategies had a significant effect on the patients' pain and depression (Covic, Adamson, Spencer, & Howe, 2003). Janwantanakul and colleagues investigated spinal pain and its association with psychosocial variables in office workers and found that some of the psychosocial factors were associated with a higher prevalence of pain (Janwantanakul, Pensri, Jiamjarasrangsi, & Sinsongsook, 2009). Mitchell and coworkers used a biopsychosocial framework for their study on back pain in nursing students and found that lifestyle, psychological, and physical variables were associated back pain (Mitchell et al., 2009). Most recently, in 2011, the biopsychosocial model was used in a study that looked at psychosocial predictors of low back pain chronicity (Hancock, Maher, Laslett, Hay, & Koes, 2011).

Because of the multicausal biopsychosocial nature of back pain, secondary data analysis has been used in several studies that investigate its potential association with smoking. In a recent cohort study in Sweden, for example, investigators used a statistical model that included biologic (age, sex, smoking status, body mass index), psychological

(sense of coherence), and social (marital status, level of education, form of employment, sick leave) factors in more than 800 men (Holmberg & Thelin, 2010). In England, Palmer and co-workers used a case-control design to investigate risk factors for back pain in patients presenting for a magnetic resonance imaging scan. This team queried participants in all three areas of the biopsychosocial model, to include loading to the spine, psychosocial factors, exposure to driving and smoking, mental health, and beliefs about back pain (Palmer et al., 2008). Also recently published, a Canadian study by Alkherayf and colleagues constructed a cross-sectional study of more than 70,000 participants using data from the Canadian Community Health Survey. They included variables representing biologic (age, sex, smoking status, body mass index) and social (level of activity, level of education) factors (Alkherayf, Wai, Tsai, & Agbi, 2010). Summarily, recent epidemiologic publications vindicate the use of a biopsychosocial theoretical framework in secondary data analysis of back and spine pain and provide a good foundation for the present study.

Rationale for Use of Selected Theories

Emmons reminds us that health behaviors must be viewed within a social context if we are to more fully understand the various causal factors leading to illness or dysfunction and how to best prevent disease (Emmons, 2000). With many disorders, such as back pain, there are multiple interacting variables and factors that may be causal or contributory (Borrell-Carrio, Suchman, & Epstein, 2004). Thus, when observing humans, their exposure to various health behaviors, such as smoking, and an outcome, such as back pain, models that engage biological, psychological, and social factors are likely to

provide better theoretical grounding than “one organism, one disease” theories (Borrell-Carrio et al., 2004).

The biopsychosocial model is, “The most widely accepted and most heuristic perspective to the understanding of chronic pain” (Gatchel et al., 2007). Perhaps the greatest appeal of the biopsychosocial model is for illnesses that are deemed “functional,” where a distinct physical pathology is difficult to identify (Shorter, 2005; Weiner, 2008). Back pain is one such disorder (Waddell, 1992). Pain is a phenomenon experienced differently by each individual and influenced by various biological, psychological and sociological factors (Gatchel et al., 2007; Turk & Okifuji, 2002). The biomedical model has dominated the research enterprise in musculoskeletal pain research, particularly as it relates to spine pain (Jull & Sterling, 2009). However, patients who experience pain often have emotional disorders, functional deficits, and health behaviors that are not conducive to wellness and such factors are better studied using a model that is biopsychosocial more than biomedical in its approach (Gatchel et al., 2007).

Back pain is complex and multicausal and therefore not well fit to the biomedical model (Weiner, 2008). It was first proposed in 1987 that the biopsychosocial model be used in back pain care and research (Waddell, 1987). Since then it has become generally accepted that thoughts and emotions have the ability to affect sensory information that is coming into the brain and thereby alter the musculoskeletal pain experience (Bergman, 2007). Further, it is also known that stress and one’s interactions socially can influence pain perception and sensory processing (Bergman, 2007). Von Korff et al. argue for the use of a biopsychosocial model in the epidemiologic study of chronic back pain, stating:

> The dimensions of pain dysfunction identified as components of chronic pain syndrome include: vegetative signs of depression; psychological distress and demoralization; preoccupation with the pain experience: impairment of interpersonal relationships; excessive use of health care and pain medications; significant activity limitations in work, family and social life; and adoption of a chronic sick role. (Von Korff, Dworkin, & Le Resche, 1990)

Thus, the biopsychosocial model is a good fit for studying the dynamic nature of the back pain experience. In fact, one authoritative team has said, "The interaction of smoking and pain can be conceptualized as a prototypical example of the biopsychosocial model." (Ditre et al., 2011) There has been a tendency in spine pain research to focus on either the biological component or the psychosocial factors affecting pain (Hancock et al., 2011; Jull & Sterling, 2009). However, studies employing a balanced approach to the various factors modulating back pain are more difficult to find. In fact, one author has even suggested that back pain be renamed to "biopsychosocial pain syndrome" since so many factors are involved in the back pain experience (Kikuchi, 2008).

The proposed study incorporated biological, psychological, and social variables shown in previous research to affect the prevalence of back pain. However, in previous research these variables were considered independently or in different combinations than those planned for the current study. Thus, this project made use of an evolving area of theory in spine pain research and addressed a gap in the research on back pain.

Conceptual Framework

Epidemiologists are concerned with population health and its determinants. Engel, as a clinician and medical educator was chiefly focused on making the doctor-patient experience more meaningful. Each viewpoint is interested in how a change in one variable affects the other variables under consideration. It is the view of the epidemiologist to look at these variables from a population health perspective (Krieger, 2011). Thus, why does a change in one or more variables have an effect on the health of the population?

The "wheel of causation" was used as a conceptual model to integrate the salient features of the epidemiologic triangle, biopsychosocial model, and social determinants of health. The wheel of causation model was proposed by Mausner and Kramer in 1985 (Friis & Sellers, 2009) and depicts disease in a multicausal manner, with the host near the center. At the core of the host are genetic factors that may affect health. Surrounding the host are factors from the biological, social and physical environments that affect the host population (Fos, 2011).

Epidemiological studies have demonstrated an association between smoking and back pain, although the potential mechanisms at work between these two variables remain unclear (Balague, Mannion, Pellise, & Cedraschi, 2012). As is often the case with health, the relationship between exposure to a potentially harmful health behavior (such as smoking) and disease (or back pain) outcome is often multifactorial (Krieger, 1994). The wheel of causation allows the researcher who is using secondary data analysis to

consider numerous variables and develop a meaningful framework for the analysis of variables.

In the wheel of causation for my study the host is represented by adults from the United States. At the center of the adult population are genetic tendencies to experience back pain, or pain in general. Until recently, the role of genetics in the understanding of pain has been underexplored (Mogil et al., 1996). Genetics likely play a role in the perception and expression of pain (Young, Lariviere, & Belfer, 2011) however it is currently unknown which mutations influence the various types of pain perceived by humans (Gatchel et al., 2007). Since this is a nascent area of spine research, that genetics play a role in the back pain experience is acknowledged, even though we are not confident regarding genetic determinants of back pain. Variables from the biologic environment include age, sex, and body mass index. Variables from the physical environment include smoking, labor, and level of activity/exercise. Variables from the social environment include socioeconomic status, level of education, and psychological inputs, such as anxiety or depression. Figure 2.4 depicts a wheel of causation for back pain and variables related to the present study.

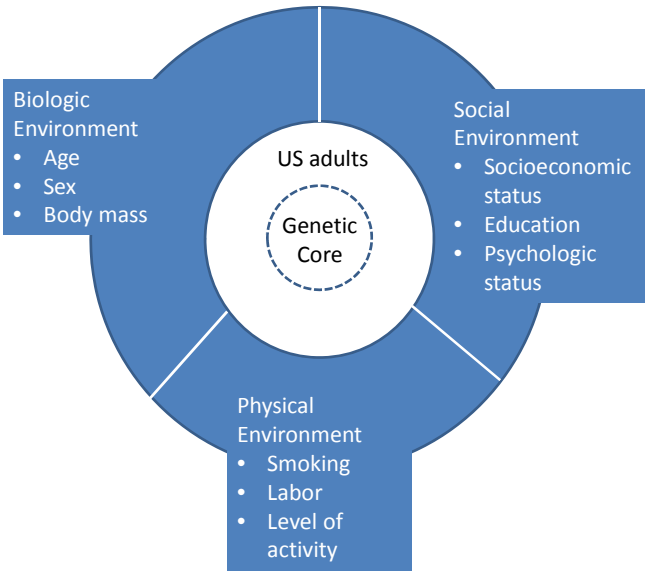

Figure 2.4. Wheel of causation when considering back pain and the numerous variables involved with back pain.

The wheel of causation as it relates to smoking and back pain informs the analytical model (Figure 2.5) used for this study, which involves biologic variables, mediating psychosocial variables, and outcome variables (Luginaah, Taylor, Elliott, & Eyles, 2002).

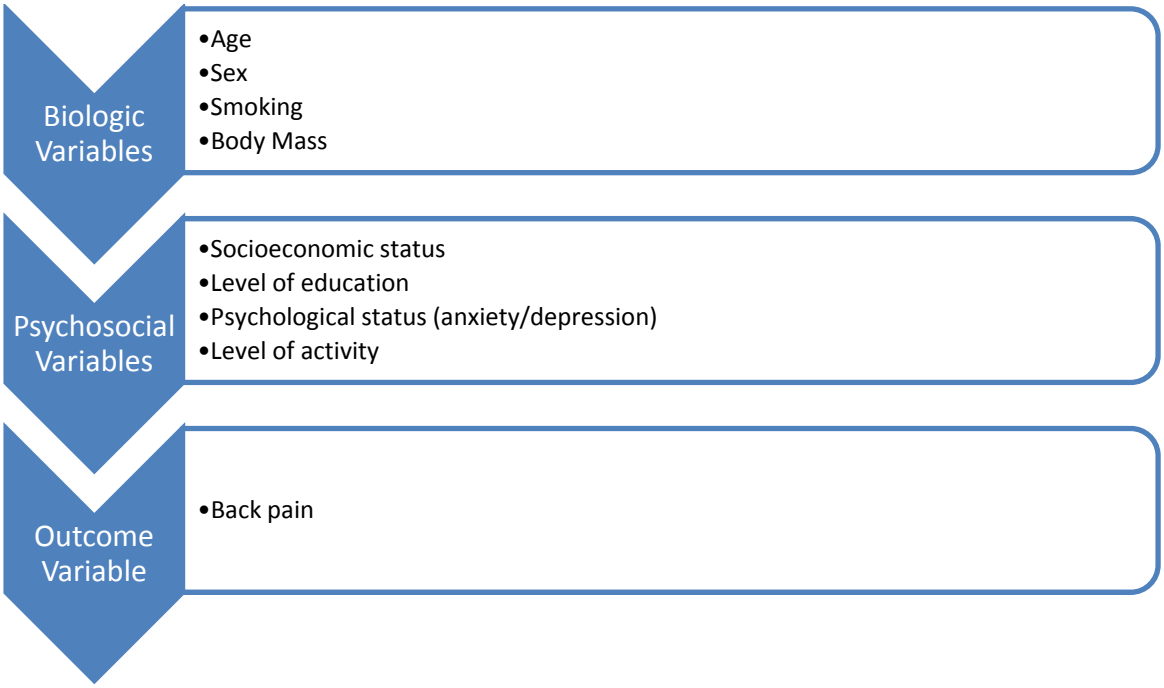

Figure 2.5. Analytical model informing the multiple variable analysis used in this research.

Literature Review Related to Key Variables and/or Concepts

The hardship that is placed upon individuals, families, communities, and nations is evident when reviewing the literature that pertains to smoking and back pain. Biological, psychological, and social problems surround the association of back pain and smoking, as can be seen when reviewing the literature from the classical epidemiological aspects of the populations affected, the frequency of disease, distribution of disease, disease determinants, disease control, and disease burden.

Populations

Disorders and pain syndromes of the musculoskeletal system are exceedingly common in most age groups and across cultures and sociodemographic groups (Woolf, Erwin, & March, 2012; Woolf, Vos, & March, 2010). Musculoskeletal disorders are

estimated to represent one-third of occupational diseases worldwide (Andersen, Mortensen, Hansen, & Burr, 2011). While these problems carry enormous morbidity, they are often not given serious consideration due to the low mortality associated with them (Woolf et al., 2010). In short, musculoskeletal problems affect nearly everyone but kill very few. Back pain, in particular, knows no geographic, cultural, or socioeconomic boundaries. It has been estimated that back pain affects 80% of the world's population at one time during each individual's life (Freburger et al., 2009; Waddell, 1987).

Smoking is a worldwide health behavior with a vast array of negative consequences. Smoking is reported in all countries, regardless of socioeconomic status (World Health Organization, 2013).

Disease Frequency

Back pain is one of the most common health problems (Evans, 2011) and is the most common chronic painful condition in Americans (Johannes et al., 2010). The three month prevalence of back pain in the adult U.S. population is estimated to be 17% (Strine & Hootman, 2007), affecting 34 million adults and the prevalence of back pain is on the rise (Wai, Rodriguez, Dagenais, & Hall, 2008). It has been estimated that approximately 22 million Americans suffer from back pain annually (Martin et al., 2009) and that the lifetime prevalence of back pain exceeds 70% (Rubin, 2007). One of the reasons for the high prevalence of back pain is that it is highly recurrent (Hoy, Brooks, Blyth, & Buchbinder, 2010).

The prevalence of smoking amongst working American adults (over the age of 18 years, n = 141,000,000) is 19.6%, or approximately 27,636,000 (2011).

Disease Distribution

Back pain is present worldwide (Kent & Keating, 2005) and is a leading cause of morbidity and lowered quality of life (Wai et al., 2008). Back pain is slightly more common in females (Wai et al., 2008). Back pain is common in both adults and children (Wai et al., 2008). Back pain is common in the working population. However, several reports document rapid increases in the prevalence of back pain in children and adolescents (Kent & Keating, 2005). Back pain is more common in older people, accounting for 81% of the health care visits for people 75 years of age or older in the United States during the year 2004 (Andersson, 2008).

The National Health Interview Survey has been studied for smoking behaviors among U.S. adults. Smoking is more common in Americans between the ages of 18 and 35 and those who work in the construction, food services, and mining industries (2011). It is known that 99% of the incidence of first tobacco use is in people under the age of 26 years, representing a greater burden of tobacco use on younger people (U.S. Department of Health and Human Services, 2012). However, the cumulative effect of long term tobacco use has a higher disease burden on older adults (Mokdad et al., 2004). There are some social disparities in cigarette use that affect the social burden associated with smoking. It is more typical for men to smoke as well as those who are of American Indian or Alaska Native heritage and it is noted that more Vietnamese and Korean Asian Americans are smoking than in the late 1990s. Cigarette smoking is also higher in homosexuals, people with 9 to 11 years of education, and the poor (U.S. Department of Health and Human Services Office of Disease Prevention and Health Promotion, 2010).

Disease Determinants

Previous studies have identified potential determinants of back pain, however full agreement is not present in the literature regarding the association of smoking to back pain (Shiri et al., 2010). Some studies have demonstrated a connection between smoking and others have not, suggesting the potential influence of confounders and/or effect modifiers (Alkherayf et al., 2010).

Covariates that have been studied in previous research are numerous. Strine and Hootman (Strine & Hootman, 2007) included a massive array of variables in their research on neck and back pain. Their variables were as follows: personal and socioeconomic variables (older age, female, white non-Hispanic, lower education, not working); comorbid conditions (asthma, allergy, chronic bronchitis, sinusitis, coronary heart disease, angina, heart attack, stroke, other heart diseases, hypertension, hypercholesterolemia, peptic or duodenal ulcer, inflammatory bowel disease, facial pain, jaw pain, temporomandibilar pain, migraine headache, severe headache, arthritis, cancer, diabetes, hypo- or hyperthyroidism, neuropathy, seizures; psychological symptoms (depressed, anxious, insomnia, daytime sleepiness, recurring pain, nervousness); health behaviors: (smoking, drinking, activity levels, BMI). The authors point out that depression symptoms, smoking behavior, heavy drinking, and obesity were significantly linked to back pain.

As impressive as the list of variables studied by Strine and Hootman (2007) may be, there was little rationale for including such a vast array. As such, there was no link between epidemiological theory and the selection of variables. One could argue that if

enough variables are included in a model with a large sample size that a statistically significant relationship can be found, but it may not be particularly meaningful, and that is a criticism of the Strine and Hootman paper. Another shortcoming of this paper is that multiple *t*-tests were performed but no adjustments were made during statistical analysis to account for performing multiple comparisons within the same sample. Finally, while the authors provide data from odds ratio calculations, the statistical methods used to derive the data were not clearly described. The proposed research will attempt to select and align variables with a plausible causation theory and report the statistical methods in a transparent manner.

In a large population based survey of Canadians, Alkherayf and colleagues investigated age, sex, BMI, activity, and education based upon previously published research (Deyo & Weinstein, 2001; Kopec, Sayre, & Esdaile, 2004; Kovacs et al., 2003; Kwon et al., 2006; Mikkonen et al., 2008) indicating that these variables had an effect on the prevalence of back pain in adults (Alkherayf et al., 2010). They found that age and sex were significant effect modifiers for the relationship between smoking and back pain and that BMI and level of education were significant confounders while level of activity was a marginally significant confounder. A major issue with this study was that the statistical analysis is not reproducible in the form that it is written. While indicating that logistic regression was used and that the Wald statistic was used to assess for significance, the authors do not reveal which variables were effect modifiers, nor did they report potential confounders.

Schmidt and colleagues in a highly cited study also found that level of education was associated with back pain in its level of disability (Schmidt et al., 2007) and other authors have reported similar findings (Y. C. Chou, Shih, Lin, Chen, & Liao, 2012; Hoy, Protani, De, & Buchbinder, 2010; Martin et al., 2009).

Tissue pathology need not be present for pain to be expressed and it is widely known that pain is both a bodily sensation and an emotional experience (Gatchel et al., 2007). If pain is chronic, it is a multifactorial process with many biopsychosocial interactions (Ditre et al., 2011). Interestingly, anxiety and depression are more common in chronic pain sufferers and smokers (Ditre et al., 2011). Emotional distress may increase a person's likelihood of experiencing pain, prolong the pain experience, or modulate pain intensity (Gatchel et al., 2007). People who experience chronic pain often feel abandoned by modern health care systems, shunned by their peers, and a burden to their families. Because pain can be chronic, its long-term effect on one's emotions should not be ignored.

Anxiety is one emotion most commonly associated with acute pain but it can also be present in people with chronic pain. People with pain worry about whether or not their pain will get worse, if their health will deteriorate, how much care will cost, if they will experience permanent disability and many other concerns (Gatchel et al., 2007). These concerns and the fear of worsening pain can lead to fear-avoidance behavior, which further complicates the pain experience because these behaviors create more disability (Rainville et al., 2011). Smokers who suffer from pain have greater levels of pain-related anxiety (Ditre et al., 2011).

Pain may also be associated with depression, with upwards of 50% of pain sufferers also experiencing one or more depressive disorders (Gatchel et al., 2007). While it is unknown if depression causes chronic pain or vice versa, it is known that these two problems coexist but not for all pain patients (Gatchel et al., 2007). Smoking is associated with increased psychological morbidity, including more severe depressive symptoms and a greater prevalence of suicidal ideation, in people who suffer from both chronic pain and depression (Ditre et al., 2011).

Disease Control

There are no cures available for the majority of musculoskeletal disorders. There are more than 200 treatments for back pain and no known effective prevention (Haldeman & Dagenais, 2008); thus, back pain is entirely out of control. Smoking likely has better science to support its control, as there are effective smoking cessation programs available and it has been shown that reducing smoking also reduces the prevalence of smoking-associated diseases (Mokdad et al., 2004). A recent study demonstrated that smokers who elected to quit smoking experienced significantly less severe low back pain than people who continued to smoke during back pain treatment (Behrend et al., 2012). However, this is preliminary evidence that needs confirmation.

The World Health Organization (WHO) has led a campaign for several years to ban tobacco products. While the organization can now claim that 2.3 billion people worldwide are “covered” by one of the tobacco control measures advocated by the WHO this represents just 10 percent of the world’s population (World Health Organization, 2013). Smoking and tobacco control has a long way to go.

Disease Burden

The lack of control of back pain and other musculoskeletal problems has resulted in unnecessary irreversible disability that places enormous suffering and financial hardship on individuals, families, and society (Woolf et al., 2012).

Human suffering. Back pain is the most common reason for Americans to have years lived with disability (Collaborators, 2013). Approximately 20% of back pain sufferers report significant restrictions to typical activities and 33% have pain for at least one year after an acute episode (R. Chou et al., 2007). For chronic back pain sufferers, pain is persistent, distressing, and can involve a declining sense of lifestyle and changes in personality (Snelgrove & Liossi, 2013). From an ethical standpoint, such suffering is profoundly harmful (Giordano & Schatman, 2008). For Americans under the age of 45 years, back pain is the most common cause of activity limitation (U.S. Department of Health and Human Services, 2000). Each person who has back pain also suffers from a lack of direction in how to best treat it, as more than 200 treatments are available for back pain and not all treatments work for each individual (Haldeman & Dagenais, 2008). Back pain often does not occur in isolation; studies have shown that anxiety and depression are commonly associated with musculoskeletal pain, especially back pain (Dunn, Green, Formolo, & Chicoine, 2011; Hoy, Brooks, et al., 2010; Magni, Marchetti, Moreschi, Merskey, & Luchini, 1993; Waddell, 1992).

Smoking accounts for enormous human suffering, represented by nearly 443,000 deaths each year in the United States (Centers for Disease Control and Prevention,

2010a). Worldwide, tobacco use is responsible for six million deaths annually (World Health Organization, 2013).

Financial hardship. From a financial perspective, back problems rank as the sixth most costly consumer health condition and the fourth most costly health condition for employers in the United States with direct costs of back pain estimated between $12.2 and $90.6 billion annually for the United States alone, representing approximately $45 to $335 per person each year (Dagenais, Caro, & Haldeman, 2008). People with back pain are high users of the health care system; back pain is the second most frequent reason for physician visits, the fifth most common reason for hospitalization, and the third most common reason for surgery (Praemer, Furner, Rice, & American Academy of Orthopaedic Surgeons., 1992). Indirect costs, such as lost work time and productivity, are five to six times the direct costs of back pain (Dagenais, Tricco, & Haldeman, 2010). As an example of the indirect costs and social burden of back pain, it was estimated that Americans lost 186.7 million work days due to back pain in the year 2004 (G. Andersson, 2008). It is estimated that two percent of the American work force is compensated annually for back pain (R. Chou et al., 2007). Summarily, the total annual cost of back pain (direct and indirect) is approximately $500 billion in the United States (Dagenais et al., 2010), which represents approximately four percent of the national gross domestic product (G. Andersson, 2008).

Smoking presents another financial burden to the U.S. economy. For the time period 2000 to 2004, the yearly costs associated with smoking were $97 billion in productivity losses and $96 billion in health-care services (U.S. Department of Health

and Human Services, 2012). In a separate investigation, the annual medical expenses and lost productivity associated with smoking were estimated to be $190 billion (Centers for Disease Control and Prevention, 2008). On a global scale, tobacco is said to cause more than 500 billion dollars in economic damage annually (World Health Organization, 2013).

Social burden. The social burden of back pain is undeniable. Back pain sufferers can be perceived as a burden upon society and have reported feeling burdensome to their families (Snelgrove & Liossi, 2013). Back pain affects all age groups (Balague et al., 2012). Back problems are not only painful, they are recurrent, posing a regular burden upon society, loss of productivity (Dagenais et al., 2008), impairment to essential activities of daily living (e.g., walking), and lower quality of life (G. Andersson, 2008). Back pain is a large burden on the health care system; it is estimated that 15 million U.S. primary care visits are used annually for back pain (Hart, Deyo, & Cherkin, 1995). Back pain does not merely affect individuals; when working adults are unable to work, must rely upon expensive treatments, and may be unable to work due to side effects from medications or surgery, the burden of back pain is born by families, communities, industry and governments (Hoy, Brooks, et al., 2010). Despite this enormous burden, there are no clearly identifiable treatments or set of treatments to quickly ameliorate pain and suffering, no clear risk factors (Wai et al., 2008), and no known primary prevention strategy (Balague et al., 2012). As pain is harmful and a social burden, there is a moral obligation of all involved in pain care to reduce pain amongst pain sufferers (Giordano & Schatman, 2008).

Smoking imposes the greatest preventable disease burden of any modifiable risk factor (Mokdad et al., 2004; World Health Organization, 2013). It is reported that either primary or secondary exposure to tobacco smoke is associated with 443,000 premature deaths for the five year period between 2000 and 2004 (2011). Further, it is estimated that 88 million Americans who elect not to smoke are exposed to secondhand smoke (Centers for Disease Control and Prevention, 2010b), representing an unfair imposition upon those who choose healthy behaviors.

Issues with Prior Studies

There is great heterogeneity in the literature devoted to the association of back pain and other variables. One problem encountered is that authors do not report the methods used in their statistical analyses with any degree of regularity, a problem that is equally present in the reporting of results. This makes it very difficult to compare the results of studies and nearly impossible to pool data if one desired to conduct a meta-analysis. Authors' choices of statistical methods is an area where the perceived hard edge of science intersects with some ill-defined art of math. Over the years different authorship teams have looked at similar variables but used entirely different statistical analyses. Much of this relates to the quality of the research performed, and it has been noted that there is indeed a need for higher quality methods in this research domain (Shiri et al., 2010).

Another shortcoming of the literature reviewed is that the rationale for the selection of variables is often lacking. While some authors have strong methods, there appears to be no good reason for studying the variables, as authors do not make a case for

variable selection or link variables to a guiding theory in the vast majority of papers, a point that has been made previously (Green & Johnson, 2013b; Leboeuf-Yde, 1995).

The literature reviewed is not unlike epidemiological studies in other areas, as a lack of theoretical grounding in planning studies is common in epidemiology and a significant criticism of the discipline (Krieger, 1994, 2000; Krieger & Zierler, 1996; Weed, 2001).

Another matter, but perhaps one of progress, is that there are research designs available now that were only beginning to be developed 30 years ago and nearly impossible to conduct without the assistance of powerful computer programs that are now commonplace. Thus, while the use of regression coefficients in the 1970s may have been appropriate, more advanced methods are required in modern epidemiology to meet the standard for scientific reporting and acceptance for publication. This adds to the heterogeneity present in the literature.

Summary and Conclusions

While there have been epidemiological studies that have investigated the association of smoking with back pain, the present study fills several gaps in the literature. First, this study makes use of a recent NHIS data set. No other studies have used these data; the most recent use of NHIS for back pain was the year 2000. Thus, the present study provides a much-needed update on back pain prevalence in the U.S. adult population and provides new data on the association of back pain and smoking, as there are no current estimates of this association. Second, no studies to date have investigated the association between back pain and smoking in adult Americans using a multivariable

analysis that incorporates biopsychosocial variables selected and analyzed together specifically because these variables have borne out in previous studies to be independent and/or mediating variables for back pain.

Chapter 3: Research Method

Introduction

This quantitative cross-sectional study explored the association of self-reported smoking behavior and the presence of back pain in U.S. adults using data from the 2012 NHIS, a nationally representative cross sectional epidemiologic survey. Covariates involved in this relationship were included. To do this, I conducted a multivariable analysis. In this chapter, I describe the research design used and my rationale for selecting this design. Further, I discuss the methodology I used to conduct the study including the population studied, sampling procedures, data collection processes, and the data analysis plan. I then examine potential threats to validity and present the ethical procedures used in my preparation and conduct of this research.

Research Design and Rationale

The dependent variable was back pain. The independent variable was smoking. The covariates were age, sex, BMI, level of education, level of activity, race, depression, and anxiety.

This study used a cross-sectional design that made use of already available data (i.e., secondary analysis). The cross-sectional design gathers data on the exposure and outcome simultaneously, at one point in time, and retrospectively (Noordzij, Dekker, Zoccali, & Jager, 2009), and does so from a non-controlled environment. This design is ideal for investigating diseases with a high disease frequency (prevalence) (Friis & Sellers, 2009), such as back pain. Since it was my intention to provide quantification of

the measure of association between smoking, covariates, and back pain amongst a large sample of U.S. adults, this design was appropriate for my study.

Before deciding to use a cross-sectional design, I considered two other methodologies. The first was a cohort study. While this design provides more information about the exposure/outcome relationship, I chose not to use it out of pragmatic issues involving the amount of time and money that would be necessary to complete the study. The cohort design would have been the preferred alternative design, as it would have gathered exposure data from two groups of people without back pain and follow them forward in time and determine if those who had back pain later in time were more likely to be smokers. This would also have allowed me to calculate incidence (Mann, 2003), which could not be done with the cross-sectional design. I could have then made comparisons between groups to see if the exposure carried an increased risk of the outcome, and risk ratios could have been established for the association (Friis & Sellers, 2009). Like the cross-sectional design, the cohort study design would also have allowed for the simultaneous assessment of the level of association between multiple variables (Mann, 2003).

The case-control design is the other research design that I considered for this study. Like the cross-sectional study, the case-control design is retrospective. The case-control design matches a person with the outcome (back pain) with a person without the outcome; such matching usually includes the matching of age, sex, and other important variables. However, the case-control design would have been less well suited to my

research because it is best used for studying rare outcomes (Mann, 2003) and back pain is not a rare health condition.

I conducted this study entirely during my spare time and at my own expense. Therefore, my employer assigned no time for me to conduct this study, and I received no funding to cover the costs of this research. These posed significant restraints to the project, as the research had to be done over an extended period of time and with moderate resources available in-kind.

I excluded myself from data collection procedures. Because my study used a national health survey conducted annually by the U.S. Census Bureau, I could not affect how the data were collected. I modified (e.g., code, merge) and analyzed the data set, but only after data collection.

Methodology

Population

The target population was civilian non-institutionalized adults living in the United States (National Center for Health Statistics, 2013), comprised of approximately 309 million people (United States Census Bureau, 2010). Participants in this study were respondents to the 2012 version of the NHIS which was conducted by the Centers for Disease Control and Prevention and the National Center for Health Statistics in cooperation with the U.S. Census Bureau. Any adult American is eligible to participate in this survey, which has been conducted annually since 1957 (National Center for Health Statistics, 2013).

Sampling and Sampling Procedures

Data for the NHIS are collected by U.S. Census Bureau interviewers in face-to-face computer-assisted personal interviews with respondents throughout the course of each year. Respondents' answers to survey questions are entered into the computer by the interviewer. At times interviewers may conduct a telephone follow up interview to complete data collection. Quality control mechanisms are in place to ensure that appropriate response and completion rates are met. The NHIS survey excludes persons in long-term care institutions, correctional facilities, U.S. nationals living in foreign countries, and active-duty Armed Forces personnel (military dependents are included). No compensation or other incentives are provided to encourage respondents to participate in the NHIS (National Center for Health Statistics, 2013).

NHIS sampling employs a complex "multistage area probability design" that allows interviewers to obtain appropriate sampling of households and group living areas, such as college dormitories. The first stage of the sampling plan covers 428 primary sampling units from approximately 1,900 geographically defined primary sampling units across the 50 states and the District of Columbia. For the second stage, 8, 12, or 16 addresses are selected geographically or by using building permits issued in the primary sampling unit which contain an expected four addresses (National Center for Health Statistics, 2013).

For my study, I used two parts of the NHIS: the Sample Adult questionnaire, and the Person file. These two data sets are part of the Household Composition questionnaire, which is answered by any household member who is of legal adult age. This respondent

provides demographic and relationship information about all household members (National Center for Health Statistics, 2013). In the Sample Adult questionnaire, one adult (> 18 years of age) from the family is randomly selected as the "sample adult." Because the NHIS is structured to have an overrepresentation of older Black, Hispanic, or Asian respondents, sampling methods employ enhanced chances of selection for Black, Hispanic, or Asian persons aged 65 years or older. This sample adult provides personal responses to the questions in the Sample Adult questionnaire unless he or she is physically or mentally unable to do so. If this is the case, a proxy is used to obtain data for the sample adult: something which occurred in 468 cases for the 2012 NHIS (National Center for Health Statistics, 2013).

In 2012 the NHIS Household Composition included 42,366 households (77.6% response rate), representing 108,131 people in the Person file from 43,345 families. For the Sample Adult component there were 34,525 respondents (79.7% response rate) (National Center for Health Statistics, 2013).

Data Collection, Confidentiality, and Management Procedures

NHIS data sets are freely available on the Internet. The 2012 data release is available at http://www.cdc.gov/nchs/nhis/nhis_2012_data_release.htm. The data set is not password protected; once it is downloaded it may be opened with the appropriate statistical software package. All data in NHIS are deidentified; thus, one cannot identify any particular person by looking at the data. Data in the public use data set are not broken by geographic area. The data I used were stored on a personal computer hard drive that was backed up weekly.

Instrumentation and Operationalization of Constructs

This was a secondary analysis of a pre-existing data set. Thus, I used no previously published or new instruments or surveys in this research.

Data Analysis Plan

Data screening and verification. Once I downloaded and opened the zip files for NHIS 2012 using SPSS, I performed data screening to check for any data errors by running frequency counts for each categorical variable and descriptives for each continuous variable. I noted the values for each frequency count, and compared them to the values provided for each variable by NHIS in its variable frequencies file that accompanies each data set.

Data file merge. I merged the NHIS 2012 Sample Adult file (n = 34,525) with the NHIS 2012 Person file (n = 108,130) because the data needed for level of education and race were stored in the Person file. To do so, I prepared each file by transforming the pertinent string variables in the data set to a numeric variable so that eventually the exact cases were matched between the Sample Adult file and the Person file. I then merged string variables together using the order recommended in the NHIS readme file, that being household number (HHX), family number (FMX), and then person number (FPX). Once I created a "master" key variable by properly cross referencing these variables, I sorted the variables in ascending order. Then, I applied the "add variables" function of SPSS where the "match cases on key variables" function was used to merge the Sample Adult file with the Person file.

Data cleaning. I cleaned the data in SPSS version 21 (IBM Corp, Armonk, NY, USA). I removed NHIS variables that were not needed in the analysis to make the data set more manageable during data analysis. I recoded variables as new variables where appropriate; recoding is described in more detail in the "Variables" section, below.

Variables and Variable Recoding. The variables are constructed from the answers provided by the survey respondents. Variable descriptions are reported in detail in Table 3.1. Briefly, the dependent variable was back pain. Respondents were asked if they had back pain for an entire day or longer in the previous three months. They could answer "yes", "no", refuse to answer, or "don't know". I transformed the data into a dichotomous variable (yes or no), whereas "don't know", and refusal to answer were categorized as system missing. Responses of "don't know and refusal to answer totaled 20, which was less than 0.1% of the sample.

The primary independent variable was smoking. The variable used (SMKSTAT2) was a recode variable already recoded by NIHS. It was derived from the 2 parent questions, "Have you smoked at least 100 cigarettes in your ENTIRE LIFE?" (SMKEV) and "Do you NOW smoke cigarettes every day, some days or not at all?" (SMKNOW) I designated those who reported smoking at least 100 cigarettes in their lifetime as smokers or former smokers and those who did not as non-smokers. Differentiation between current smokers and former smokers was determined by the answer to the question, "Do you NOW smoke cigarettes every day, some days or not at all?" I considered respondents not smoking "now" as former smokers and the others as current smokers. Responses were recoded into 4 categories: current smoker (smoke daily or some days), former smoker,

never smoker and those that answered “smoker but current status unknown” or “unknown if ever smoked” I coded as system missing. There were 269 responses recoded as system missing, representing 0.8% of the sample.

Biological independent variables were age, sex, and body mass index. Age was recorded as “under 1 year”, “1-84 years”, or “85+ years”. No recodes were necessary for age. Sex was asked as, “Are you male or female?” No recodes were necessary for sex. Body mass index was calculated automatically by NHIS when the respondent provided information pertaining to height and weight. The BMI variable in NHIS was an automatic recode based upon the input of the height and weight variables using the formula weight/height squared. Height was recorded in inches and self-reported to the question of, “How tall are you without shoes?” Weight was recorded in pounds and self-reported to the question of, “How much do you weigh without shoes?” I recoded BMI so that unknown values were recoded to “system missing” and ran BMI as a continuous variable in the ANOVA for research question 4. For research question 5, I recoded BMI as a categorical variable for the logistic regression analysis, using the following categories: underweight was BMI < 18.5; healthy weight was BMI 18.5 to < 25; overweight was BMI ≥ 25 to < 30; obese was BMI ≥ 30.

Psychological variables included questions about symptoms pertaining to depression and anxiety. I considered respondents to have depressive symptoms if they answered affirmative to the dichotomous question, “Have you EVER been told by a doctor or other health professional that you had depression?” I recoded response options of “refused,” “not ascertained,” and “don’t know” were recoded into “system missing,”

which totaled 26 responses, or less than 0.1% of the sample. I operationally defined respondents to have symptoms of anxiety if they answered "yes" to the dichotomous question, "During the past 12 months have you frequently felt anxious, nervous, or worried?" I recoded response options of "refused", "not ascertained", and "don't know" into "system missing", which totaled 22 responses, or less than 0.1% of the total sample.

Social independent variables included level of education, level of physical activity, and race. I considered level of education a continuous variable determined by the question, "What is the HIGHEST level of school you have completed or the highest degree you have received? A value of 0 was assigned to the response of "never attended and/or kindergarten only". The range progressed to 21 being assigned to attaining doctoral level education. I recoded responses of "refused", "not ascertained", and "don't' know" into "system missing", which represented 153 responses, or 0.4% of the total sample.

I determined level of physical activity from variables reporting on the frequency of vigorous or light/moderate activity per week and the average number of minutes engaged in these activities per session. Physical activity variables were based upon the 2008 Physical Activity Guideline for Americans (United States Department of Health and Human Services, 2008); active persons were considered those that participated in light or moderate activities (exercise that caused light sweating or a slight to moderate increase in breathing or heart rate) for 150 minutes or more per week or vigorous activity (exercise that caused heavy sweating or large increases in breathing or heart rate) for 75 minutes or more per week during their leisure time, or a combination of these activities. Per the 2008

Physical Activity Guideline for Americans, vigorous physical activity was counted as double the amount of effort as light/moderate physical activity. I recoded physical activity into a continuous variable for the ANOVA, following the example set in several other studies that use large complex data sets for secondary data analysis for assessment of physical activity(Carlson, Fulton, Schoenborn, & Loustalot, 2010; Schoenborn & Stommel, 2011; Tucker, Welk, & Beyler, 2011). I created a new physical activity variable using the following formula as the basis for the recodes: [(VIGFREQW x VIGMIN) x 2] + [(MODFREQ x MODMIN)]. At the lowest end of the range of values for both vigorous and light/moderate physical activity per week, NHIS gave respondents the opportunity to state that they exercise less than once a week. Consideration was given as to whether respondents should be given "partial credit" for this activity or "no credit." To address this range, I created a variable that assigned partial credit = 0.5 for activity less than once a week as well as one that assigned partial credit = 0 for activity less than once a week and conducted preliminary analyses to see which variable provided the best data. For the logistic regression analysis in research question 5, I dichotomized the level of physical activity into those who met the recommended physical activity per week (150 minutes or more) and those who did not.

Race was a nominal variable categorized as Hispanic or non-Hispanic/race as per the Office of Management and Budget categories. "Other Race" and "Unspecified Multiple Race" were no longer available as separate race responses in 2012 NHIS and these response categories were treated as "system missing," and the race was imputed if

these were the only race responses. There were no responses categorized as system missing.

Table 3.1

Variable Description Table from National Health Interview Survey 2012

Variable	File Location, Question #	Data Scale	Response Options	Question	Code
OUTCOME (DEPENDENT) VARIABLE					
Back Pain	Sample Adult, ACN.310_00.000	Nominal	Yes, No, Refused, Don't know	The following questions are about pain you may have experienced in the PAST THREE MONTHS. Please refer to pain that LASTED A WHOLE DAY OR MORE. DURING THE PAST THREE MONTHS, did you have ... Low back pain?	PAINLB
EXPOSURE (INDEPENDENT) VARIABLE					
Smoking	Sample Adult, AHB.040_01.000	Nominal	Current every day smoker, Current some day smoker, Former smoker, Never smoke, Smoker but current status unknown, Unknown if ever smoked	This is a variable that is already recoded by NHIS. It is derived from the 2 parent questions, "Have you smoked at least 100 cigarettes in your ENTIRE LIFE?" (SMKEV) and "Do you NOW smoke cigarettes every day, some days or not at all?" (SMKNOW)	SMKSTAT2
BIOLOGICAL INDEPENDENT VARIABLES					
Sex	Sample Adult, HHC.110_00.000	Nominal	Yes, No, "Yes" for refused or did not know	Are you male or female?	SEX
Age	Sample Adult, HHC.420_00.000	Continuous	Years, Refused, Don't know	How old are you?	AGE
BMI	Sample Adult, AHB.200_02.000	Continuous		Calculated from self-report of height and weight	BMI
PSYCHOLOGICAL INDEPENDENT VARIABLES					
Depression	Sample Adult, ACN.121_00.150	Nominal	Yes, No, Refused, Don't know	Have you EVER been told by a doctor or other health professional that you had depression?	ADEPRSEV
Anxiety	Sample Adult, ACN.125_00.260	Nominal	Yes, No, Refused, Don't know	DURING THE PAST 12 MONTHS, have you Frequently felt anxious, nervous, or worried?	ANXNWYR
SOCIAL INDEPENDENT VARIABLES					
Education	Person, FSD.010_00.000	Continuous	Never attended/kindergarten only 1st grade 2nd grade 3rd grade 4th grade 5th grade 6th grade 7th grade 8th grade 9th grade 10th grade 11th grade 12th grade, no diploma	What is the HIGHEST level of school you have completed or the highest degree you have received?	EDUC

(continued)

			GED or equivalent High School Graduate Some college, no degree Associate degree: occupational, technical, or vocational program Associate degree: academic program Bachelor's degree; Master's degree; Professional School degree (eg: MD, JD); Doctoral degree (eg PhD, EdD) Child under 5 years old Refused Don't know		
Vigorous Physical Activity Frequency	Sample Adult, AHB.090_02.000	Continuous	Less than once per week, 1-28 times per week, Never, Unable to do vigorous activity, Refused, Not ascertained, Don't know	How often do you do VIGOROUS leisure-time physical activities for AT LEAST 10 MINUTES that cause HEAVY sweating or LARGE increases in breathing or heart rate?	VIGFREQW
Vigorous Physical Activity Duration	Sample Adult, AHB.100_02.000		10-720 minutes, Refused, Not ascertained, Don't know	About how long do you do these vigorous leisure-time physical activities each time?	VIGMIN
Light or Moderate Physical Activity Frequency	Sample Adult, AHB.110_02.000	Continuous	Less than once per week, 1-28 times per week, Never, Unable to do vigorous activity, Refused, Not ascertained, Don't know	How often do you do LIGHT OR MODERATE leisure-time physical activities for AT LEAST 10 MINUTES that cause ONLY LIGHT sweating or a SLIGHT to MODERATE increase in breathing or heart rate?	MODFREQW
Light or Moderate Physical Activity Duration	Sample Adult, AHB.120_02.000	Continuous	10-720 minutes, Refused, Not ascertained, Don't know	About how long do you do these light or moderate activities each time?	MODMIN
Hispanic	Person File, HHC.170_00.000	Categorical	Yes, No	Does person consider self Hispanic/Latino?	ORIGIN_I
Race	Person File, HHC.200_01.000	Categorical	Hispanic, Non-Hispanic White, Non-Hispanic Black, Non-Hispanic Asian, Non-Hispanic All other race groups	This is a recode variable from NHIS based upon a number of other questions about race and ethnicity. First, respondents self-identified as Hispanic or not, then other questions were asked and the recode was based on those other questions	HISCODI3

Research questions and hypotheses. The primary research objective was to examine the prevalence of back pain among smokers compared to former smokers and non-smokers. I considered the influence of confounders in the potential risk of back pain for smokers. The secondary objective of this study was to identify if certain covariates

were confounders for the potential relationship of smoking and back pain. I used the following research questions and associated hypotheses (H_0 = null hypothesis, H_1 = alternative hypothesis) in my study:

1. What is the association between back pain in current smokers, former smokers, and never smokers?

   H_0: There is no significant difference in the prevalence of back pain between current smokers, former smokers, and never smokers as measured by the National Health Interview Survey.

   H_1: The prevalence of back pain is greater in current smokers than in former smokers and never smokers.

2. What is the association between back pain and the non-modifiable individual factor of sex and race?

   H_0: There is no significant difference in the prevalence of back pain between sex and racial groups.

   H_1: The prevalence of back pain is significantly different between sexes and races.

3. What is the association between back pain and the non-modifiable individual factors of age?

   H_0: There is no significant difference in the prevalence of back pain across ages.

   H_1: The prevalence of back pain is significantly different for certain ages.

4. What is the association between back pain and modifiable individual factors, including BMI, level of education, and level of physical activity?

   H_0: There is no significant relationship between back pain and BMI, education, or level of activity.

   H_1: There is a relationship between back pain and BMI, education, and/or level of activity.

5. Do smoking, age, sex, BMI, level of education, level of physical activity, race, depression, or anxiety predict back pain?

   H_0: There is no statistical model that predicts back pain using the variables under study.

   H_1: A combination of variables predicts the occurrence of back pain.

Rationale for covariate inclusion. Previous studies demonstrated that age, sex, body mass index, depression, anxiety, level of education, level of physical activity, and race were related to smoking and/or pain or specifically back pain. Adjustments for these variables were included in my statistical analysis. Researchers have found that increasing age was a risk factor for low back pain (Andersson, 2008; Hoy, Brooks, Blyth, & Buchbinder, 2010) and identified age as an effect modifier for the relationship between smoking and back pain in a large nationally representative sample of Canadians (Alkherayf et al., 2010). Sex was determined to be an effect modifying variable in one previous study (Alkherayf et al., 2010), although a recent systematic review did not find sex to be a determinant for back pain (Hoy, Brooks, et al., 2010). Body mass index was

noted to be a confounder in the association between smoking and back pain (Alkherayf et al., 2010).

In previous research, pain was found to be a trigger for smoking, even though smoking may not be analgesic (Dhingra et al., 2013). Low back pain was shown to be predictive of future cigarette smoking behavior and smoking was predictive of mid back pain in the future, demonstrating a bi-directional relationship between these variables (Gill, Davis, Smith, & Straker, 2013). Depressive symptoms served as a mediating variable for the relationship between pain and smoking (Goesling, Brummett, & Hassett, 2012; Strine & Hootman, 2007). Symptoms of anxiety were common in people with pain and in smokers (Ditre et al., 2011) and highly prevalent in back pain sufferers (Linton, 2000). The exclusion of psychological variables in previous studies was a criticism of previous works, as such variables may be confounders when studying the relationship between smoking and pain (Ditre et al., 2011). Thus, symptoms of depression and anxiety were included in the present study.

Level of education was noted to be directly related to pain and cigarette smoking (Ditre et al., 2011) and was a significant confounder when studying smoking and back pain in one large study (Alkherayf et al., 2010). Schmidt and colleagues in a highly cited study also found that level of education was associated with back pain in its level of disability (Schmidt et al., 2007) and other authors have reported similar findings (Y. C. Chou et al., 2012; Hoy, Protani, et al., 2010; Martin et al., 2009).

Level of physical activity was previously reported as a significant confounder (Alkherayf et al., 2010). This may be because back pain is more bothersome for people at

the extremes of the spectrum of physical activity. A study done in the Netherlands showed that both a sedentary lifestyle and strenuous activities were associated with a greater risk of chronic low back pain (Heneweer, Vanhees, & Picavet, 2009). One systematic review demonstrated that because physical activity was difficult to measure due the myriad types of activities that people engage in that it could only be said that back pain was related to the nature and intensity of the activity (Heneweer, Staes, Aufdemkampe, van Rijn, & Vanhees, 2011).

Studies investigating race and back pain have shown that Asians and Pacific Islanders had a lower frequency of back pain than other races (Knox, Orchowski, & Owens, 2012; Waterman, Belmont, & Schoenfeld, 2012). Blacks were shown to have the highest prevalence of back pain (Knox et al., 2012). A recent study looking at spine pain showed similar results, with Asians having the lowest prevalence of neck and back pain and blacks having the highest (Perruccio, Gandhi, Rampersaud, & Arthritis Program, 2013).

Complex samples analysis plan. Because the NHIS utilizes a complex sample design that oversamples black persons, Hispanic, and Asian persons, as well as adults of these races who are 65 years-of-age or older, SPSS Complex Samples Analysis was used. To prepare for this, I created a complex samples plan with the variable "Pseudo-stratum for public use file variance estimation [STRAT_P]" as the strata, "Pseudo-PSU for public use file variance estimation [PSU-P]" as the cluster, and "Weight-Final Annual [WTFA_SA]" as the sample weight.

Verification of assumptions for statistical tests. Multicollinearity between independent variables was assessed by Pearson correlation coefficients (Katz, 2011) and further described in Chapter 4.

Sample size estimate/power analysis. Using G*Power 3.1.9.2 for Windows (Universität Düsseldorf, Germany) (Faul, Erdfelder, Lang, & Buchner, 2007), I performed a power analysis to determine the minimal number of subjects necessary to identify an effect between variables for the logistic regression. An *a priori* method of power analysis was used (Cohen, 1988, pp. 24-26). I selected a medium effect size for Pr(Y=1) H0 = 0.5, with alpha set at 0.05 (two-tailed) and power of 0.95, based upon the methods of Faul and colleagues (Faul, Erdfelder, Buchner, & Lang, 2009). I conservatively estimated the odds ratio for the power analysis as OR = 1.3, based upon a review of outcomes of previously published studies (Green & Johnson, 2013a) and also upon estimates of one-month and one-year prevalence of low back pain in smokers and non-smokers from a meta-analysis (Shiri et al., 2010). Given these parameters, the estimated number of subjects required for the logistic regression analysis was 863 (Fig 3.1). Increasing the power above 0.95 to 0.99 yielded an estimated sample size of 1040. Given that the NHIS 2012 data set contained more than 30,000 respondents, the minimum number of subjects necessary to determine an effect between variables was easily obtained.

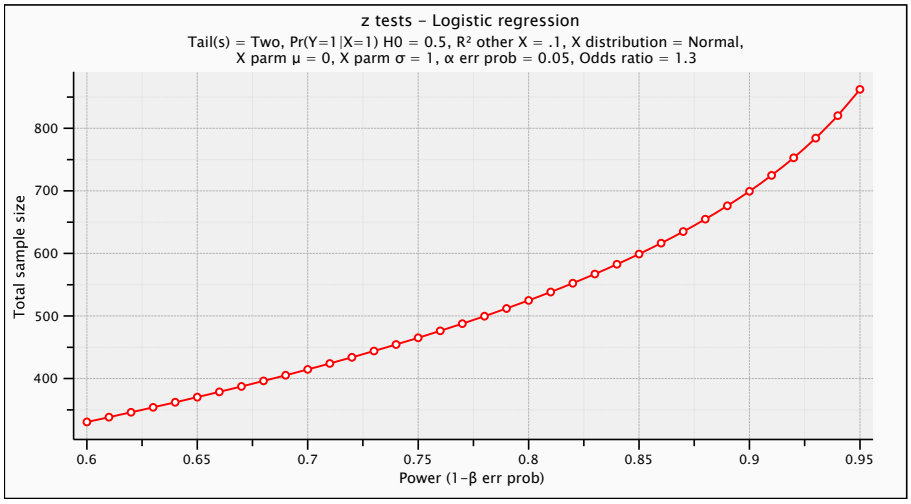

Figure 3.1. Power analysis plot for determining minimum sample size.

Data analysis. I analyzed data in SPSS version 21 (IBM Corp, Armonk, NY, USA) software program using Complex Samples Analysis. Unadjusted and adjusted odds ratios and 95% confidence intervals were calculated with α set as $P < .05$. The statistical tests conducted for each research question are shown in Table 3.2. I conducted statistical analyses for each research question in steps building up to a full logistic regression model. For research question one I employed chi square to assess for association between the categorical variables of smoking status and presence of back pain. I also used chi square in research question two to assess for association between the categorical variables of sex, race, and back pain. I used a *t*-test for research question three to assess for a difference in the mean age (continuous variable) of those with and without back pain (categorical variable). For research question four, I conducted a one-way analysis of variance to assess for a difference in the mean BMI, education, or level of activity (continuous variables) of those with and without back pain (categorical variable). Finally,

in research question five, I entered all of the independent variables, including anxiety and depression, into a logistic regression analysis to predict back pain.

Table 3.2

Statistical Analyses Conducted per Research Question and Corresponding Null Hypothesis

Research Question	Null Hypothesis	Statistical Procedure
What is the association between back pain in smokers, non-smokers, and former smokers?	There is no significant difference in the prevalence of back pain between current smokers, former smokers and never smokers, as measured by the National Health Interview Survey.	chi-square
What is the association between back pain and the non-modifiable individual factors of sex and race?	There is no significant difference in the prevalence of back pain between sex and racial groups.	chi-square
What is the association between back pain and the non-modifiable individual factor of age?	There is no significant difference in the prevalence of back pain across ages.	*t*-test
What is the association between back pain and modifiable individual factors, including BMI, level of education, and level of physical activity?	There is no significant relationship between back pain and BMI, education, or level of activity.	one-way analysis of variance
Do smoking, age, sex, BMI, level of education, level of physical activity, race, depression, or anxiety predict back pain?	There is no statistical model that predicts back pain using the variables under study.	multiple logistic regression

Threats to Validity

Unknown effect modifiers or confounders posed a threat to the internal validity of this study. Self-report also posed a threat to internal validity, as respondents may have been inaccurate in their knowledge of medical terms or symptoms, which could have lead to under- or over-reporting. Further, NHIS methods were based upon recall for up to 12 months, which introduced a level of inaccuracy in reporting data, compared to being able to ask questions about the respondents' current health status.

External validity could be compromised in the level of importance of the back pain experienced. Since NHIS questions did not quantify the severity of the back pain or to what degree the respondent's function was affected by back pain, I can only calculate the prevalence of back pain and not its individual or social implications. External validity may be affected by operationally defining depression as respondents who stated that they had depression versus using a diagnosis of depressive disorder. Similarly, the operational definition for anxiety was those who had frequently felt anxious, nervous, or worried in the previous 12 months and was not a diagnosis of anxiety provided by a licensed health care provider. Due to inherent limitations in the research design it was impossible to determine if smoking was causal for low back pain since there was no control group, the design was retrospective, and the study was observational in nature (Schoenbach & Rosamond, 2000). Thus, causal generalizations from any cross-sectional study were avoided (Mann, 2003). Further, this was a sample of adult Americans and results may not be generalizable to people under the age of 18 years or to those in other countries.

Ethical Procedures

Prior to initiating this research, I completed training in ethical research conduct, the protection of data sets, and ethical considerations of using human subjects via the Collaborative Institutional Training Initiative, The National Institutes of Health Office of Extramural Research, and the Office of Research Integrity of the Department of Health and Human Services.

All data used from the NHIS data set were anonymous and therefore no individuals in the Sample Adult data or families in Household data were identified. As

NHIS data were openly accessible on the Internet, no permissions were necessary to use the data for secondary data analysis. Per the user agreement with NHIS (National Center for Health Statistics, 2013), I used the data in the data files solely for statistical reporting and analysis.

There were no human participants in this secondary data analysis, but the research protocol was reviewed and approved prior to the commencement of data collection by the Walden University institutional review board November 4, 2014 (Appendix A).

I had no potential conflicts of interest to declare, as this research was done as an educational exercise for the doctorate in philosophy, conducted on my own time, with my own resources, and at my own expense. The only incentive underpinning this research was to complete this project in fulfillment of the dissertation requirement for the aforementioned doctorate.

Summary

Summarily, this research endeavor is a quantitative cross-sectional study that assessed the association of smoking and other biopsychosocial variables with the presence of back pain in a large nationally representative sample of U.S. non-institutionalized adults using a publicly accessible data set, the 2012 NHIS. Multivariable analysis is used and includes the calculation of prevalence as well as unadjusted and adjusted odds ratios to determine measures of association between the dependent variable of back pain and the independent variable of smoking, taking into account several potential biopsychosocial covariates. Chapter 4 presents the results of the data analysis, including descriptive data of the respondents, prevalence of smoking and back pain in the

sample, results of the multivariable analysis, appropriate data tables and significant odds ratios found between variables.

Chapter 4: Results

Introduction

This quantitative cross-sectional study explored the association of self-reported smoking behavior and the presence of back pain in U.S. adults using data from the 2012 NHIS. This study also included covariates involved in this relationship. I used several statistical tests to identify associations between variables, and ultimately culminated the study with a multivariable logistic regression analysis to create a model that would attempt to predict the probability of back pain. The dependent variable was back pain. The independent variable was smoking. The covariates were age, sex, BMI, level of education, level of activity, race, depression, and anxiety. My primary research objective was to examine the prevalence of back pain among smokers compared to former smokers and non-smokers. My secondary objective for this study was to identify if certain covariates are confounders for the potential relationship of smoking and back pain. I used the following research questions and associated hypotheses (H_0 = null hypothesis, H_1 = alternative hypothesis) in this study:

1. What is the association between back pain in current smokers, former smokers, and never smokers?

   H_0: There is no significant difference in the prevalence of back pain between current smokers, former smokers, and never smokers, as measured by the NHIS.

   H_1: The prevalence of back pain is greater in current smokers than in former smokers and never smokers.

2. What is the association between back pain and the non-modifiable individual factor of sex and race?

   H_0: There is no significant difference in the prevalence of back pain between sex and racial groups.

   H_1: The prevalence of back pain is significantly different between sexes and races.

3. What is the association between back pain and the non-modifiable individual factor of age?

   H_0: There is no significant difference in the prevalence of back pain across ages.

   H_1: The prevalence of back pain is significantly different for certain ages.

4. What is the association between back pain and modifiable individual factors, including BMI, level of education, and level of physical activity?

   H_0: There is no significant relationship between back pain and BMI, education, or level of activity.

   H_1: There is a relationship between back pain and BMI, education, and/or level of activity.

5. Do smoking, age, sex, BMI, level of education, level of physical activity, race, depression, or anxiety predict back pain?

   H_0: There is no statistical model that predicts back pain using the variables under study.

   H_1: A combination of variables predicts the occurrence of back pain.

This chapter will present a description of the sample and its representativeness of the population through univariate statistics of the study variables with weighting to the population of the United States. Next, the chapter outlines the assumptions that were necessary for me to perform the various statistical tests and presents the analyses I performed to assess for compliance with these assumptions. The chapter concludes with a presentation of the results of the statistical analyses for each research question and corresponding hypothesis.

Descriptive and Univariate Analyses

The merged data set consisted of the Sample Adult and Person Files from NHIS 2012 and included 108,131 cases. Of these, 34,525 were over the age of 18 years and all adults were included in the analysis. Back pain was prevalent in 29% of adults and smoking was prevalent in 19%. Of the entire adult sample, most people had never smoked, there were slightly more females, and the majority were white. Most respondents had not been told that they had depression and did not feel anxious or worried. The average respondent was approximately 46 years of age, had a BMI considered overweight, did not meet the physical activity recommendations of 150 minutes per week, and had a high school education with some college but no degree. An overview of the study demographics and primary variables are presented in Table 4.1.

Table 4.1.

Demographics of Study Sample (n = 34,525) and Primary Variables

Demographics			
		Frequency	Percent
Sex	Male	15273	44.2
	Female	19252	55.8
Race	Hispanic	5859	17
	Non-Hispanic white	20842	60.4
	Non-Hispanic black	5282	15.3
	Non-Hispanic Asian	2168	6.3
	Non-Hispanic other	374	1.1
Back Pain	No back pain	24427	70.8
	Back pain	10078	29.2
	System missing	20	0.1
Smoking	Current smoker	6436	18.6
	Former smoker	7584	22
	Never smoker	20236	58.6
	System missing	269	0.8

	Range	Minimum	Maximum	SD
Age	67	18	85+	18.2

Univariate statistics for categorical variables for all adult respondents are presented in Table 4.2.

Table 4.2.

Univariate Statistics for Categorical Variables for All Adults in Sample and Population Estimates

Variable	Population Size Estimate	% of Total	Standard Error	95% Confidence Interval		Unweighted Count
				Lower	Upper	
Back Pain						
Back pain	65823057	28.0	965737	63922579	67723534	10078
No back pain	168937180	72.0	1844349	165307679	172566680	24427
Total	234760237	100.0	2291291	230251198	239269275	34505
Smoking						
Smoker	42098139	18.1	813046	40498141	43698136	6436
Former smoker	51621850	22.2	865475	49918678	53325021	7584
Never smoker	139327445	59.8	1617264	136144825	142510064	20236
Total	233047434	100.0	2287231	228546384	237548483	34256
Sex						
Male	113070897	48.1	1471455	110175215	115966578	15273
Female	121849773	51.9	1395324	119103909	124595636	19252
Total	234920670	100.0	2292625	230409005	239432334	34525
Depression						
Depressed	33060907	14.1	665993	31750296	34371517	5370
Not depressed	201715693	85.9	2040206	197700765	205730620	29129
Total	234776600	100.0	2293196	230263811	239289388	34499
Anxious						
Anxious	44997520	19.2	810173	43403176	46591863	6943
Not anxious	189780355	80.8	1942556	185957592	193603117	27560
Total	234777875	100.0	2292510	230266436	239289313	34503
Race						
Hispanic	34946432	14.9	816213	33340203	36552660	5859
Non-Hispanic White	157787860	67.2	2071449	153711448	161864271	20842
Non-Hispanic Black	27885414	11.9	626598	26652329	29118498	5282
Non-Hispanic Asian	12415030	5.3	424711	11579238	13250821	2168
Non-Hispanic All other race groups	1885934	0.8	198313	1495671	2276196	374
Total	234920670	100.0	2292625	230409005	239432334	34525

Univariate statistics for continuous variables are presented in Table 4.3.

Table 4.3

Univariate Statistics for Continuous Variables for All Adults in Sample and Population Estimates

Variable	Mean	SE	95% CI		Population Size	Unweighted Count	Skewness		Kurtosis	
			Lower	Upper			Stat	SE	Stat	SE
Age	46.6	.15	46.3	46.9	234920670	34525	.21	.01	-.94	.03
BMI	27.7	.05	27.6	27.8	225493641	33170	1.25	.01	2.80	.03
Education	15.0	.03	14.9	15.0	233868064	34372	-1.26	.01	2.57	03
Physical activity high	346	5.4	336	357	233591911	34357	12.75	.01	423	03
Physical activity low	344	5.4	333	355	233763472	34376	12.74	.01	423	03

SE = standard error; 95% CI = 95% confidence interval

Representativeness of the Study Sample

The target population for this study was civilian non-institutionalized adults living in the United States (National Center for Health Statistics, 2013), a population comprised of approximately 309 million people (United States Census Bureau, 2010). I gathered data from respondents to the 2012 version of the NHIS. For this sample, any non-institutionalized, non-military, and non-incarcerated adult American was eligible to participate in the survey (National Center for Health Statistics, 2013). NHIS sampling

employs a complex multistage area probability design that allows interviewers to obtain appropriate sampling. The data are considered to be independent samples done at random and are collected in a fashion that is reputed to be representative of the national population (National Center for Health Statistics, 2013). Because the NHIS systematically oversamples some ethnic and racial groups, I performed statistical analyses weighted to the population in order to provide results most closely representing the population.

Assessment for Statistical Assumptions

The four statistical tests I used for this study were chi-square, independent samples *t*-test, ANOVA, and logistic regression analysis. Each test has its own set of assumptions, presented here with a description of how I assessed the data for each relevant variable to see if they met the assumptions prior to running the statistical analyses for each research question.

Chi-square test for independence. There are few assumptions made when using the chi-square statistic and these are generally less restrictive than those assumptions made when using parametric tests, such as *t*-test and ANOVA. The chi-square statistic assumes that the test variables are drawn from a random sample and independent observations (Pallant, 2010). Chi-square also assumes that the frequency counts represent individual data counts (as opposed to percent or another summary statistic) and that each case can only be assigned to one category in the contingency table (Portney & Watkins, 2009). It is also a requirement that there be at least 5 or more frequency counts per cell in

the contingency table (Munro, 2005). The data for the chi-square analyses met all of the above assumptions.

Independent samples *t*-test. For the *t*-test there is a grouping variable and there are test variable(s). The *t*-test assumes that the test variables are a continuous scale, from a random sample, and independent observations. The *t*-test also assumes that the test variables are normally distributed and that the variability of scores for each of the groups is similar. A variety of methods may be used to assess for normality. Commonly, skewness, kurtosis, and visual examination of a histogram plotting the distribution of data are used. I employed these methods in this study. Inspection of the histogram involves looking for a symmetrical bell shaped curve where the mean is at the center of the curve. If the curve is shifted to the left or right, it is skewed (Munro, 2005). Tests for skewness may also be performed on variables; values should be between -1 and +1 (Munro, 2005). If the distribution is not significantly skewed, then kurtosis may be assessed. Kurtosis is an indication of the height of the curve where the value should be 0 if the variable is distributed over a bell shaped curve. A large positive number indicates that the peak is very high and a large negative number indicates that there is no or a small peak, that the curve is relatively flat (Munro, 2005).

For the continuous variable in this *t*-test (age), the histogram (Figure 4.1) showed a reasonably bell shaped curve and skewness of .21 (SE = .013), indicating it was acceptably distributed. The kurtosis was -.95 (SE = 0.26), indicating that the curve was relatively flat but was not outside of bounds for consideration as relatively normal in distribution. Also noted is a spike in the 85+ years of age category. This was inevitable,

as the data reported in NHIS are not broken down in smaller increments for age older than 85 years. If this was a small sample, then there may be some concern over the kurtosis. However, since there are more than 34,000 independent, random observations in the sample, the *t*-test run in SPSS is rather robust and should be able to provide a reliable value for this variable (Pallant, 2010).

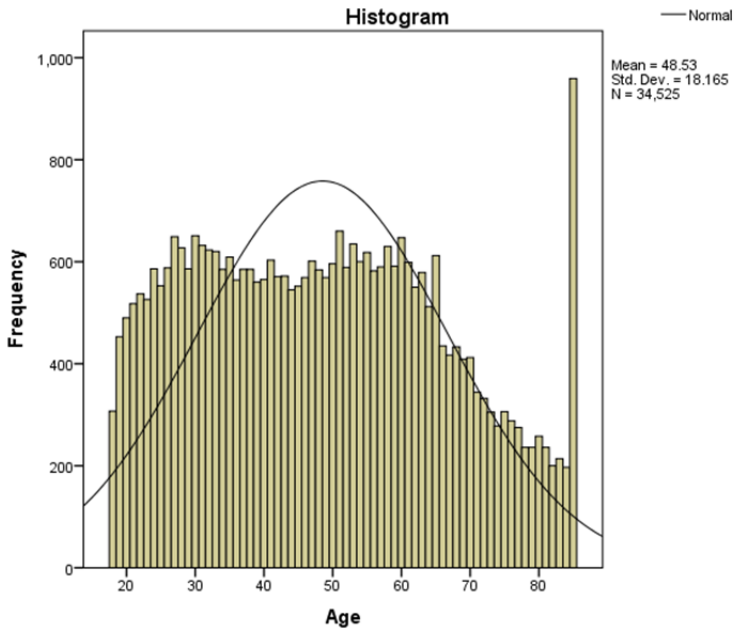

Figure 4.1. Histogram of distribution of age.

ANOVA. ANOVA makes the same assumptions as the *t*-test. The continuous variables in the ANOVA were BMI, level of education, and level of physical activity. BMI and level of physical activity were positively skewed with large kurtosis values (Table 4.3). Education had a negative skew (-1.3) with a tall kurtosis (2.6). Thus, all variables had levels of skewness or kurtosis considered unacceptable (Table 4.3) for conducting ANOVA in a small sample (Munro, 2005). Some authors opine that such diversions from normality are easily managed by the robust ANOVA provided in SPSS,

especially with a large sample, as there is adequate power in the sample (Pallant, 2010; Riffenburgh, 2012). However, another recommended approach is to transform the skewed variables (Munro, 2005). For the ANOVA in research question four, I transformed BMI and level of physical activity logarithmically and I transformed level of education using the reflect and logarithm procedure in SPSS in an effort to provide a more normal distribution for each variable (Pallant, 2010). Following transformation, skewness and kurtosis were markedly improved and considered adequate for this ANOVA with a large sample. Values are presented in Table 4.4.

Table 4.4

Transformed Variables for ANOVA

		Transformed Variables		
		Education Transformed	BMI Transformed	Physical Activity Transformed
N	Valid	34372	33170	22810
	Missing	153	1355	11715
Mean		.81	1.43	2.42
Median		.84	1.42	2.48
Mode		.90	1.41	1.78
Skewness		-.61	.48	-.28
Std. Error of Skewness		.01	.01	.02
Kurtosis		1.4	.40	.16
Std. Error of Kurtosis		.03	.03	.03

Multiple logistic regression. The use of multiple logistic regression to predict the probability of an outcome makes several assumptions. While a normal distribution of scores for the independent variables is not a requisite and equal variance for the

dependent variable is not a requisite, as in parametric analyses, logistic regression does assume that the same probability is maintained across independent variables. This is usually accounted for in a random sample (Peng, Lee, & Ingersoll, 2002).

Logistic regression relies upon an adequate sample size to manage the statistical analysis of many variables (Pallant, 2010). This data set, with more than 34,000 cases far exceeded the estimated sample size of 1,040 needed for power of 0.99, even when considering that the final model included 9 independent variables.

Logistic regression uses a dichotomous dependent variable. It is assumed that the dichotomous variables are coded correctly to represent a true dichotomous categorization where the difference between the choices is no more than 1 (Katz, 2011). The dependent variable was correctly coded. I ascertained all categorical variables for appropriate convention where each increment was a value of one.

It is also necessary to discover if any of the independent variables have a high degree of association to one another, or demonstrate multicollinearity (Katz, 2011). One recommended method for assessing for multicollinearity is to use collinearity diagnostics for a typical multiple linear regression (Katz, 2011; Pallant, 2010). While the main output from such an analysis is of little value, the collinearity statistics inform if there is interaction amongst the independent variables by producing tolerance values (Pallant, 2010). A tolerance value less than .25 demonstrates that a variable has high correlation with other independent variables in the model and may need to be managed, accordingly (Katz, 2011). Values of .10 or less are considered problematic (Pallant, 2010). Variance inflation factors are also provided in the assessment for collinearity. Variance inflation

factors are equal to 1/tolerance and values greater than 10 are concerning (UCLA Statistical Consulting Group, 2015). The collinearity assessment for the independent variables that I performed in this study revealed no tolerance values of concern (Table 4.5).

Table 4.5

Assessment of Collinearity amongst Independent Variables

Coefficients[a]

	Collinearity Statistics	
	Tolerance	VIF
Smoking Status	.946	1.057
Sex	.958	1.044
Age	.978	1.023
Race/ethnicity	.964	1.037
Physical Activity	.968	1.034
Depression	.800	1.251
Anxiety	.804	1.244
Education	.937	1.067
BMI	.988	1.012

a. Dependent Variable: Low Back Pain. VIF = variance inflation factor.

Another method for assessing for interaction between independent variables is to use SPSS Complex Samples Analysis to run bivariate logistic regression on the independent variables to assess for significant relationships. Based upon logical potential relationships, I performed the following bivariate logistic regressions: BMI/ level of physical activity; anxious/depression; smoker/BMI; smoker/level of physical activity. No

significant interaction was observed between the pairs of independent variables, confirming the outcome of the assessment of multicollinearity described above.

Results

Research question 1. In research question one I asked, "What is the association between back pain in smokers, non-smokers, and former smokers?" The null hypothesis was that there is no significant difference in the prevalence of back pain between current smokers, non-smokers, and former smokers. Back pain was present in 9,995 of the 34,241 cases. The chi-square test for independence using weighting for population estimates showed a significant association between back pain and smoking status, X^2 (2, 599, n = 34, 241) = 546.3, $p < .001$, population estimate = 232,918,356. Back pain was estimated to be present in 36.9% (95% CI, 35.3-38.4) of smokers, 33.1% (95% CI, 31.8-34.4) of former smokers, and 23.5% (95% CI, 22.7-24.3) of never-smokers. With none of the 95% confidence intervals overlapping, a significant association between smoking status and back pain was demonstrated. The null hypothesis that there was no difference in the prevalence of back pain between current smokers, former smokers, and never smokers was therefore rejected.

Research question 2. Research question two investigated the association between back pain and the non-modifiable individual factors of sex and race. The null hypothesis was that there is no significant difference in the prevalence of back pain between sexes or races. Back pain was present in 10,078 of the unweighted 34,505 cases.

The chi-square test for independence using weighting for population estimates showed a significant association between back pain and sex, X^2 (1, 300, n = 34,505) =

81.3, $p < .001$, population estimate =234,760,237. Back pain was estimated to occur in 25.8% (95% CI, 24.9-26.6) of males and 30.1% (95% CI, 29.3-31.0) of females. None of the 95% confidence intervals overlapped, indicating a significant association between female sex and back pain. The null hypothesis that there was no difference in the prevalence of back pain between sexes was therefore rejected.

The chi-square test for independence using weighting for population estimates was performed and showed a significant association between back pain and race, X^2 (4, 1,185, n = 34,505) = 136, $p < .001$, population estimate = 234,760,237. Back pain was estimated to occur in 25.6% (95% CI, 24.2-27.0) of Hispanics, 29.6% (95% CI, 28.8-30.4) of non-Hispanic whites, 25.4% (95% CI, 24.0-26.9) of non-Hispanic blacks, 19.2% (95% CI, 17.2-21.5) of non-Hispanic Asians, and 36.1% (95% CI, 29.8-42.8) of those in all other groups. There was some overlap of the 95% confidence intervals, indicating that a distinct association of race with back pain with race was not entirely clear. The null hypothesis was therefore accepted for the association of race with back pain.

Research question 3. Research question three investigated the association between back pain and the non-modifiable individual factor of age using an independent samples *t*-test. The null hypothesis was that there is no difference in the mean age between those who had back pain for at least an entire day in the prior three months and those who did not. The independent samples *t*-test with weighting for population estimates showed that the mean age for those with back pain was significantly higher (M = 49.0 years; SE = .24; 95% CI: 48.6-49.5) than for respondents without back pain (M = 45.7 years; SE = .18; 95% CI: 45.4-46.1), $t(300) = 200.6$, $p < .001$, $d = .72$. I calculated

the magnitude of the differences in the means (mean difference = 3.33) using eta squared ($t^2/t^2 + (n1 + n2 – 2)$ and found it to be 0.72, which was a large effect (Cohen, 1988). Thus, the null hypothesis that there was no difference in the mean age of those who had back pain and those who did not was rejected.

Research question 4. Research question four investigated the association between back pain and the modifiable individual factors of BMI, level of education, and level of activity. The null hypothesis was that there is no significant relationship between back pain and the mean values for BMI, level of education, or level of physical activity. The significance values for all three independent variables were $p < .001$ for the Levene's test for homogeneity of variance; thus the robust tests of equality of means were used (Welch and Brown-Forsythe tests yielding identical results).

The one-way ANOVA using the transformed variables showed that the mean BMI for those with back pain ($M = 1.47$; $SD = .01$; 95% CI: 1.45-1.45) was higher than for those without back pain ($M = 1.45$; $SD = .09$; 95% CI: 1.43-1.43), $F(1,16490) = 336$, $p < .001$, $d = .01$. The mean level of education for those with back pain ($M = .83$; $SD = .21$; 95% CI: .83 -.84) was lower than for those without back pain ($M = .80$; $SD = .22$; 95% CI: .79 - .80), $F(1,19708) = 216.7$, $p < .001$, $d = .006$. The mean level of physical activity for those with back pain ($M = 2.36$; $SD = .54$; 95% CI: 2.35 – 2.38) was lower than for those without back pain ($M = 2.44$; $SD = .50$; 95% CI: 2.43 – 2.45), $F(1,10272) = 95.1$, $p < .001$, $d = .004$. The effect size (d) for all analyses was calculated using the formula for eta squared: d = sum of squares between groups/total sum of squares (Pallant, 2010) from the analysis of variance table (Table 4.6). Thus, the null hypothesis that there was no

difference in the mean BMI, level of education, and level of physical activity between those who had back pain and those who did not was rejected.

Table 4.6

Analysis of Variance for Education, Body Mass Index, and Physical Activity for those with and without Back Pain

		Sum of Squares	df	Mean Square	F	Sig.
Education Reflect	Between Groups	9.50	1	9.50	207.33	.000
	Within Groups	1574.67	34353	.05		
	Total	1584.17	34354			
BMI Log	Between Groups	3.31	1	3.31	167.00	.000
	Within Groups	684.42	34503	.02		
	Total	687.73	34504			
Physical Activity Log	Between Groups	26.93	1	26.93	102.09	.000
	Within Groups	6014.78	22800	.26		
	Total	6041.71	22801			

df = degrees of freedom

Research question 5. Research question five investigated the ability of smoking, age, sex, BMI, level of education, level of physical activity, race, depression, or anxiety to predict back pain using multiple logistic regression. The null hypothesis was that there was no statistical model that predicted back pain using these independent variables.

One method of performing logistic regression is to use a stepwise or statistical technique. This is sometimes done as a hypothesis building model but is not preferred because independent variables are selected for the model entirely based upon statistical criteria without input from the researcher who is familiar with the nature and scope of the research question. However, this technique may be preferred as a screening procedure

and I used it here to assess how the independent variables interacted with one another in predicting the dependent variable and to see which independent variables contributed the most to the model. The full model containing all nine predictors was statistically significant, X^2 (15, n = 32,839) = 3199, $p < .001$, and each variable except non-Hispanic black ($p = .29$) and non-Hispanic all other ($p = .06$) contributed significantly to the predictive ability of the model ($p < .001$ for Wald chi square for all significant variables). As shown in Table 4.7, anxiousness, depression, and smoking status had the highest odds of being associated with back pain.

Table 4.7

Stepwise Forward Logistic Regression Odds of United States Adults Having Back Pain[a]

Variables in the Equation

	B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
							Lower	Upper
Never Smoker			160.40	2	.000			
Former Smoker	.25	.03	60.84	1	.000	**1.29**	1.21	1.38
Current Smoker	.40	.03	139.86	1	.000	**1.50**	1.40	1.60
Age	.01	.00	107.84	1	.000	1.01	1.01	1.01
Female sex	.11	.03	18.54	1	.000	**1.12**	1.07	1.18
BMI Obese			144.90	3	.000			
BMI Underweight	-.51	.10	24.02	1	.000	**0.60**	0.50	0.74
BMI Healthy weight	-.38	.03	131.05	1	.000	**0.69**	0.64	0.73
BMI Overweight	-.25	.03	62.25	1	.000	**0.78**	0.73	0.83
Physical Activity Meeting/exceeding	-.17	.03	39.72	1	.000	**0.84**	0.80	0.89
Education	-.02	.00	28.88	1	.000	**0.98**	0.97	0.99
Depression	.64	.04	325.29	1	.000	**1.90**	1.78	2.04
Anxious	.96	.03	896.67	1	.000	**2.62**	2.46	2.79
Hispanic			24.24	4	.000			
Non-Hispanic White	.10	.04	6.73	1	.009	**1.11**	1.02	1.19
Non-Hispanic Black	.03	.05	.30	1	.586	1.03	0.94	1.12
Non-Hispanic Asian	-.15	.07	5.17	1	.023	**0.86**	0.75	0.98
Non-Hispanic other	.18	.12	2.15	1	.142	1.20	0.94	1.5
Constant	-1.26	.08	276.73	1	.000	0.28		

Significant odds ratios (OR) are in bold face. 95% CI indicates 95% confidence interval.
[a]Respondents who answered "yes" to the question, "The following questions are about pain you may have experienced in the past three months. Please refer to pain that lasted a whole day or more. During the past three months, did you have low back pain?"

For the forward stepwise logistic regression, the independent variable with the greatest contribution to the model was anxiousness, followed by depression, age, and smoking (Table 4.8). Including 7 independent variables (anxiousness, depression, age, smoking status, BMI, physical activity, education) provided a correct classification of back pain or no back pain 73.3% of the time. The further addition of sex and race decreased the accuracy of the model.

Table 4.8

Step Summary for Forward Stepwise Logistic Regression Odds of United States Adults Having Back Pain[a]

Step Summary[a,b]

Step	Improvement			Model			Correct Class	Variable
	Chi-square	df	Sig.	Chi-square	df	Sig.	%	
1	2018.33	1	.000	2018.33	1	.000	71.8%	IN: Anxiousness
2	477.77	1	.000	2496.10	2	.000	72.9%	IN: Depression
3	219.52	1	.000	2715.62	3	.000	72.7%	IN: Age
4	183.00	2	.000	2898.62	5	.000	73.0%	IN: Smoking
5	177.22	3	.000	3075.84	8	.000	73.1%	IN: BMI
6	55.02	1	.000	3130.86	9	.000	73.2%	IN: Physical Activity
7	24.42	1	.000	3155.29	10	.000	73.3%	IN: Education
8	18.95	1	.000	3174.24	11	.000	73.2%	IN: Sex
9	24.72	4	.000	3198.95	15	.000	73.2%	IN: Race

a. No more variables can be deleted from or added to the current model.

b. End block: 1

Direct logistic regression, sometimes also referred to as forced logistic regression, is a preferred technique (Pallant, 2010; Tabachnick & Fidell, 2007). Direct logistic

regression enters all variables into the model at the same time as if each independent variable is entered into the logistic regression equation last (Tabachnick & Fidell, 2007). No variables are left out of the model. Thus, this is the preferred method when performing theory-driven regression, as it allows the researcher to determine if a variable is included in the model. This is particularly important in health care where the dichotomous outcome is related to a health variable involving morbidity or mortality. Thus, with direct regression, independent variables are not left out of the model based upon statistical criteria alone. I therefore also ran the data as a direct logistic regression. Un-adjusted odds ratios for each independent variable for adults with back pain are presented in Table 4.9.

Table 4.9

Prevalence and Unadjusted Odds Ratios for Independent Variables for Adults with Back Pain[a].

Variable	Percentage (95% CI)	Odds Ratio (95% CI)
Anxiousness		
Anxious	34.5 (33.1-35.8)	3.4 (3.2-3.7)
Not Anxious	65.5 (64.2-66.9)	Referent
Depression		
Depression	25.5 (24.4-26.6)	3.2 (3.0-3.4)
No Depression	74.5 (73.4-75.6)	Referent
Smoking		
Current Smoker	36.9 (35.3-38.4)	1.9 (1.8-2.0)
Former Smoker	33.1 (31.8-34.4)	1.6 (1.5-1.7)
Never Smoker	23.5 (22.7-24.3)	Referent
Body Mass Index		
Underweight	1.6 (1.3-2.0)	1.1 (.8-1.4)
Healthy Weight	30.6 (29.4-31.8)	Referent
Overweight	33.3 (32.1-34.4)	1.1 (1.0-1.2)
Obese	34.6 (33.3-35.9)	1.6 (1.5-1.7)
Physical Activity		
Not meeting recommendation	57.7 (56.4-59.1)	1.4 (1.3-1.5)
Meeting recommendation	42.3 (40.9-43.6)	Referent
Sex		
Female	30.1 (29.3-31.0)	1.2 (1.2-1.3)
Male	25.8 (24.9-26.6)	Referent
Race		
Non-Hispanic White	29.6 (28.8-30.4)	1.2 (1.1-1.3)
Non-Hispanic Black	25.4 (24.0-26.9)	1.0 (0.9-1.1)
Non-Hispanic Asian	19.2 (17.2-21.5)	0.7 (0.6-0.8)
Non-Hispanic Other	36.1 (29.8-42.8)	1.6 (1.2-2.2)
Hispanic	25.6 (24.2-27.0)	Referent
Education	N/A	1.0 (1.0-1.0)
Age	N/A	1.0 (1.0-1.0)

[a]Respondents who answered "yes" to the question, "The following questions are about pain you may have experienced in the past three months. Please refer to pain that lasted a whole day or more. During the past three months, did you have low back pain?"

The full direct logistic regression model containing all nine predictors was statistically significant, X^2 (15, n = 32,839) = 1475, $p < .001$, and each variable contributed significantly to the predictive ability of the model ($p < .001$ for Wald chi square for all independent variables), particularly anxiousness, depression, and smoking

(Table 4.10). The model was estimated to a population size of 223,048,717 adults, explained between 8.5% (Cox and Snell R square) and 12.2% (Nagelkerke R square) of the variance in back pain status, and correctly classified 73.3% of the cases. Thus, the null hypothesis that there was no model to predict back pain was rejected.

Table 4.10

Forced Entry Logistic Regression Likelihood of United States Adults Having Back Pain[a]

	Comparison	B	SE	Wald Chi Square	df	p	OR[b]	95% CI	
								Lower	Upper
Anxious	Anxious vs. Not Anxious	-.95	.04	426	1	.000	**2.58**	2.37	2.82
Depression	Depression vs. No Depression	-.60	.04	186	1	.000	**1.81**	1.67	1.98
Smoking Status	Smoker vs. Never Smoker	.45	.04	115	2	.000	**1.57**	1.44	1.71
	Former Smoker vs. Never Smoker	.28	.04	-	-	-	**1.32**	1.22	1.43
Body Mass Index	Underweight vs. Healthy Weight	-.49	.14	85	3	.000	0.87	0.65	1.15
	Overweight vs. Healthy Weight	-.25	.04	-	-	-	**1.10**	1.02	1.20
	Obese vs. Healthy Weight	-.35	.04	-	-	-	**1.41**	1.30	1.53
Physical Activity	Not Meeting vs. Meeting Exercise Recommendations	.14	.04	15.7	1	.000	**1.16**	1.08	1.24
Sex	Male vs. Female	-.12	.03	12.9	1	.000	**1.13**	1.06	1.20
Race	Non-Hispanic White vs. Hispanic	-.27	.14	23.6	4	.000	**1.13**	1.03	1.24
	Non-Hispanic Black vs. Hispanic	-.15	.14	-	-	-	0.99	0.89	1.11
	Non-Hispanic Asian vs. Hispanic	-.28	.15	-	-	-	0.86	0.73	1.02
	Non-Hispanic Other vs. Hispanic	-.42	.16	-	-	-	1.32	0.99	1.74
Education	Unit of change = 1	-.02	.01	13.3	1	.000	**0.98**	0.97	0.99
Age	Unit of change = 1	.01	.00	61.4	1	.000	1.01	1.01	1.01
Intercept		.42	.17	31.0	1	.000			

Significant odds ratios (OR) are in bold face. 95% CI indicates 95% confidence interval.
[a]Respondents who answered "yes" to the question, "The following questions are about pain you may have experienced in the past three months. Please refer to pain that lasted a whole day or more. During the past three months, did you have low back pain?"
[b]Adjusted by anxiousness, depression, smoking status, body mass index, physical activity, sex, race, education, and age.

A one unit change in feeling anxious or worried increased the odds of having back pain for a whole day or longer in the previous three months by more than two and half times. A one unit change in being diagnosed with depression increased the odds of having back pain for a whole day or longer in the previous three months by nearly two times. A one unit change in current smoking status increased the odds of back pain by one and a half times and a one unit change in former smoking status increased the odds of back pain by one and a third times. A one unit change in overweight increased the odds of back pain 10% and a one unit change in obesity increased the odds of back pain nearly one and a half times. Males were 13% as likely to have back pain than females and non-Hispanic Whites were 13% as likely than Hispanics to have back pain. The odds ratio of 0.98 for level of education was just less than one with 95% confidence intervals hovering around 1, suggesting for every one unit increase in education respondents were 2% less likely to have back pain, but this should be interpreted with caution due to the confidence intervals. Age had no significant association with back pain in this model.

Summary

This study explored for associations between the dependent variable, those who reported back pain, and several independent variables. Smokers were more likely than former smokers and never smokers to have back pain. Former smokers were also more likely to have back pain than never-smokers. Thus, I rejected the null hypothesis for research question one. Females were more likely than males to have back pain. Differences in back pain prevalence based upon race were not statistically significant. I

therefore rejected the null hypothesis for research question two for sex but accepted it for race. Those people with back pain had a higher mean age than those who did not have back pain. I therefore rejected the null hypothesis. The mean BMI was higher in those with back pain while the mean level of education and level of physical activity was lower in those with back pain. The null hypothesis for research question four was therefore rejected. Research question five investigated the ability of smoking, age, sex, BMI, level of education, level of physical activity, race, depression, or anxiety to predict back pain. A logistic regression model was produced that could explain some of the variance between those who have back pain and those who do not and several significant associations were found between the independent variables and the dependent variable. The null hypothesis was therefore rejected.

Chapter 5 will provide an interpretation of the research findings and place the findings in context of what is currently known about back pain and its association with the various independent variables. Limitations to the current research, alternative interpretations, and potential implications for social change and application of this research will also be presented in Chapter 5.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

This quantitative cross-sectional study explored the association of smoking and a number of other independent variables with the prevalence of back pain in U.S. adults using a complex samples analysis, which provided results weighted to the U.S. population. I drew data from the 2012 NHIS and conducted this research to better understand the relationships between smoking and back pain on a population level because current measurements of association for the U.S. population are unavailable.

I performed several analyses, culminating with a multiple logistic regression analysis to predict back pain. Using a chi square test, I found smokers to be more likely than former smokers and never smokers to have back pain. Former smokers were also more likely to have back pain than never-smokers. I thus rejected the null hypothesis for research question one. Using the chi square test, I found that females were more likely than males to have back pain. However, differences in back pain prevalence based upon race were not statistically significant. I thus rejected the null hypothesis for research question two for sex but accepted it for race. Using an independent samples *t*-test, I found that those with back pain had a higher mean age and I rejected the null hypothesis for research question three. Using a one-way analysis of variance, I found that the mean BMI was higher in those with back pain while the mean level of education and level of physical activity was lower in those with back pain. I therefore rejected the null hypothesis for question four. Finally, the logistic regression model was accurate at classifying approximately 73% of cases and could explain 8.5% and 12.2% of the

variance between those who have back pain and those who do not. I found several significant associations between the independent variables and the dependent variable, the most significant being anxiousness, depression, and smoking. I therefore rejected the null hypothesis for research question five.

This chapter provides interpretation of the research findings and places the findings in context of what is currently known about back pain and its association with the various independent variables. In this chapter I also discuss limitations to the current research, alternative interpretations, and potential implications for social change and application of this research.

Interpretation of the Findings

Research question 1. This question assessed for association between smoking status and back pain. I found that current smokers have a higher prevalence of back pain at 36.9% (95% CI, 35.3-38.4) than never smokers at 23.5% (95% CI, 22.7-24.3) and that former smokers have a higher prevalence of back pain at 33.1% (95% CI, 31.8-34.4) than never smokers. All results were statistically significant.

Sir Austin Bradford Hill proposed nine criteria for causation including strength of association, consistency of association between studies, temporality (outcome follows exposure), biological gradient, specificity of the association, plausibility, coherence with other data, experiments that support the hypothesis, and analogy to other exposure and outcome associations (Hill, 1965). Research question one may support the concept of temporality. The survey was conducted after all respondents had a smoking status and back pain had been ascertained for just the previous three months. It therefore suggests

that smoking preceded the back pain. However, it is possible that the back pain could have preceded smoking in some cases. The survey question for back pain asked if the respondent had experienced back pain for an entire day in the previous three months. For smoking, the survey asked, "Do you NOW smoke cigarettes every day, some days or not at all?" Respondents not smoking "now" were considered former smokers and the others as current smokers. It is possible that some of the respondents had back pain prior to being smokers in this sequence of events and the suggestion that the results of data analysis support temporality should be interpreted with some degree of caution. Second, the data show a biological gradient. Smokers had a higher prevalence of back pain than former smokers and both of these groups had a higher prevalence of back pain than never smokers. There are several plausible theories pertaining to causal mechanisms between smoking and back pain which I reviewed in Chapter 2.

The findings from research question one are parsimonious with studies done elsewhere. There are few studies that look at the bivariate relationship between smoking and back pain, as most authors attempt to investigate the effects of covariates through the use of regression. However, some data are available for comparison. In Canada, 23.3% of daily smokers had chronic low back pain, 17.2% of occasional smokers had low back pain, and 15.7% of non-smokers had back pain (Alkherayf & Agbi, 2009). Data from the current study support this trend but the prevalence of back pain amongst Americans is alarmingly higher with 36.9% of current smokers, 33.1% of former smokers, and 23.5% of never smokers having back pain, respectively. Mohseni-Banpei et al. (2011) investigated the prevalence of back pain in Iranian surgeons and found that 13.5% of the

sample smoked and 63% of them had back pain (X^2 = 7.93; OR = 3.03 (95%CI 1.36-6.74); p = .005).

Similarly, in an earlier study by Deyo and Bass (1989), among those who ever smoked, the prevalence of low back pain was 10.9%, versus 9.6% among those who never smoked. In this study, the one-year prevalence of back pain increased as the greatest amount of smoking was considered, showing a 9.6% prevalence for non-smokers versus 25.1% for smokers who inhaled more than three packs per day. They also noted that back pain prevalence increased with increased pack years.

It is also important to recognize that because this is a cross-sectional study it is impossible to know the direction of association of smoking and back pain. It is biologically plausible that smoking may cause back pain but also that back pain may lead people to increase their consumption of cigarettes. Another possibility is that people who have back pain coincidentally smoke more cigarettes than people who do not and the association is not biological.

Research question 2. This question investigated the association between back pain and the non-modifiable individual factors of sex and race. For sex, back pain was estimated to occur in 25.8% (95% CI, 24.9-26.6) of males and 30.1% (95% CI, 29.3-31.0) of females, a statistically significant relationship. These data support the findings of others (Taylor, Goode, George, & Cook, 2014). It is not known why there is a higher prevalence of back pain in females but this relationship does appear to be consistent across studies (Taylor et al., 2014).

For race, back pain was estimated to occur in 25.6% (95% CI, 24.2-27.0) of Hispanics, 29.6% (95% CI, 28.8-30.4) of non-Hispanic whites, 25.4% (95% CI, 24.0-26.9) of non-Hispanic blacks, 19.2% (95% CI, 17.2-21.5) of non-Hispanic Asians, and 36.1% (95% CI, 29.8-42.8) of those in all other groups. While the chi square result was significant, showing that one or more relationships in race may be associated with back pain, there was significant overlap between some of the 95% confidence intervals. For example, the 95% confidence intervals for Hispanics and non-Hispanic Blacks were nearly identical. Further, the 95% confidence intervals for non-Hispanic Whites and all other groups had significant overlap. Thus, based upon the chi square analysis, race failed to show an association with the prevalence of back pain. The association of race with back pain prevalence has been studied many times and fails to produce consistent results. This may be due to researchers studying different parameters of pain. For example, Carey et al. studied back pain severity and prevalence and found lower prevalence in Latinos but higher pain scores in Blacks (Carey et al., 2010). The results of my study support the hypothesis that back pain is an equal burden across racial groups (G. Andersson, 2008), and that perhaps studying ethnicity or culture may be a more accurate method of assessing for differences in this disease burden amongst people of different backgrounds.

Research question 3. Age and its relationship to back pain was the focus of research question three. The *t*-test showed that the mean age for those with back pain was significantly higher ($M = 49.0$ years; $SE = .24$; 95% CI: 48.6-49.5) than for those without back pain ($M = 45.7$ years; $SE = .18$; 95% CI: 45.4-46.1), $t(300) = 200.6$, $p < .001$, $d = .72$. When one considers the cumulative load on the body over years, it makes sense that

those who are older may have more back pain prevalence. It is widely known that back pain is highly recurrent (Hoy, Brooks, et al., 2010) and one would expect a higher prevalence of back pain with time. My study's findings pertaining to age and back pain are similar to other publications (G. Andersson, 2008; Taylor et al., 2014). However, the difference in means was only 3.3 years. While this difference may have been statistically significant, the actual difference is not very large from a pragmatic point of view.

Research question 4. Research question 4 investigated the association between back pain and the modifiable individual factors of BMI, level of education, and level of activity. The raw data for the independent variables were highly skewed and had marked kurtosis. I therefore transformed them to be eligible for use in a one-way analysis of variance.

The mean BMI for those with back pain (M = 1.47; SD =.01; 95% CI: 1.45-1.45) was higher than for those without back pain (M = 1.45; SD =.087; 95% CI: 1.427-1.429), $F(1,16490) = 336$, $p < .001$, $d = .01$. While the difference is statistically significant, it is not large. One explanation for the small difference in means is that logarithmic transformation was performed on the data, which effectively shrunk the range of values for BMI. Another explanation could be that there is just not a large difference in BMI between the groups. BMI has been studied by other researchers. For example, a Canadian study found that BMI was a confounder for the association between smoking and back pain (Alkherayf et al., 2010). By itself, BMI has not been shown to have a strong association with back pain in numerous studies (Taylor et al., 2014). It seems plausible that an increasing body mass would be related to an increase in back pain, as the spine

and body frame must carry the weight of the individual. However, it may be that the body is able to cope with this excess mass up to a threshold that is not easily teased out using a continuous variable.

The mean level of education for those with back pain (M =.83; SD =.21; 95% CI: .83 - .84) was lower than for those without back pain (M = .80; SD =.22; 95% CI: .79 - .80), $F(1,19708) = 216.7$, $p < .001$, $d = .006$. I used a reflect and logarithm transformation on these data. Because of the data transformation, it is difficult to say what the real difference is between the level of education in the back pain versus the no back pain groups. Level of education has been studied by other authors and it has historically been shown that lower levels of education are associated with more back pain prevalence (Bergenudd & Nilsson, 1988; Carr & Moffett, 2005; Dionne et al., 1995; Reisbord & Greenland, 1985; Stewart Williams et al., 2015). The findings of my study support those in the literature. There are many hypotheses as to why this relationship exists, but there is no overwhelming or compelling evidence to support any particular theory.

The mean level of physical activity for those with back pain (M = 2.36; SD = .54; 95% CI: 2.35 – 2.38) was lower than for those without back pain (M = 2.44; SD = .50; 95% CI: 2.43 – 2.45), $F(1,10272) = 95.1$, $p < .001$, $d = .004$. Once again, these data should be interpreted with some degree of caution since a logarithmic transformation was performed on the independent variable. The transformation makes it very difficult to say what the actual mean number of minutes per week of physical activity were for each group. What can be seen is that the difference between the means for physical activity is relatively large. This relationship supports the hypothesis that those who do more

physical activity may have less back pain. An alternate explanation could be that those who have back pain are more fearful that physical activity could worsen their symptoms (Marchand et al., 2015). Another interpretation is that the mean for each group could be so skewed in the direction of no physical activity that any difference between the groups is meaningless for pragmatic purposes. There is also the possibility that those who have back pain are more likely to occupy positions at the far ends of the activity spectrum and either do no activity or engage in a great deal of vigorous activity, as has been suggested by Heneweer et al (2009). As tempting as Heneweer and colleagues' U-shaped hypothesis is, it seems to lack application to the participants in the current study where more than 55% of Americans did not meet the recommended physical activity guideline of 150 minutes per week of basic exercise.

Research question 5. Once I completed the analyses in the previous research questions, I employed multiple logistic regression analysis to create a statistical model using all of the study variables that would be predictive of back pain and elicit odds ratios for the independent variables. Initial bivariate logistic regression showed some significant associations and odds ratios for some variables, such as anxiousness, depression, and smoking status. As with most multiple logistic regression analyses, the magnitude of the odds ratios tended to decline when more variables were added to the model. For example, the odds ratio for those with back pain was 3.4 for those who self-reported feeling anxious or worried in the 12 months leading up to the survey compared to those who did not report feeling anxious or worried. When I entered all of the variables into the

regression model, the odds ratio for being anxious or worried was 2.6 after being adjusted for the effects of the other independent variables.

While psychological factors have been identified as being significantly related to pain and smoking (Ditre et al., 2011; Gatchel et al., 2007), the magnitude of their effect on back pain is a nascent area of investigation. It is often assumed that acute and severe pain is most associated with anxiety and that chronic pain may be more associated with depression. However, it is possible for a person in any type of pain to have both psychological comorbidities (Ditre et al., 2011). The present study not only supports the multiple psychologic comorbidities assertion, it shows that the variables with the greatest magnitude of association with back pain were psychological variables. Once I adjusted for the other variables, those who were anxious or worried were 2.6 times as likely to have back pain than those who were not anxious or worried and people with depression were nearly twice as likely to have back pain than those who did not have depression. This is further evidence that the use of biopsychosocial theory for epidemiological investigations of back pain (Green & Johnson, 2013b) is relevant and appropriate. The surprising magnitude of association of anxiousness and depression with back pain is a significant finding of my current study.

These findings pertaining to the psychological connection with back pain are in congruence with those reported elsewhere in recent literature but not often investigated in earlier research. Matsudaira et al (2015) recently reported on psychological risk factors associated with chronic low back pain, to include anxiety (OR = 2.89; 95%CI: 0.97–8.57). Christensen et al (2015) found that those with mental distress had a higher

prevalence of back pain. Hung, Liu, and Fu (Hung, Liu, & Fu, 2015)recently demonstrated that depression was the most important factor in predicting disability associated with low back pain.

As in the bivariate chi square analysis from research question one and the bivariate logistic regression, smoking status continued to have a significant association with back pain once all other variables were added in the multiple logistic regression. The directional and dose response nature of this relationship continued, with current smokers having an odds ratio higher than former smokers and never smokers and former smokers having an odds ratio higher than never smokers but lower than current smokers. Other authors have looked at this relationship but not all have found a directional relationship with a biological gradient (Shiri et al., 2010). Given the large sample size, the random and independent nature of the sampling, the weighting of the analysis to population estimates, and adjusting for the effects of the other variables, the magnitude of association, direction, and biological gradient from the current study are significant findings of my study.

When looking at year 2002 data from the study that inspired the present research, Americans who were daily smokers were more likely to have back pain than those who were not (adjusted OR = 1.3; 95% CI, 1.2-1.4). When reviewing all epidemiologic studies up until February 2009, Shiri et al (2010) found a variety of odds ratios pertaining to smoking and back pain and the results of the my study fit within these ranges. Shiri found an increased prevalence of back pain in the previous month among smokers (pooled OR = 1.3, 95% CI, 1.2-1.4), more pain in the past 12 months (OR = 1.3, 95% CI,

1.3-1.4), seeking care for low back pain (OR = 1.5, 95% CI, 1.4-1.6), chronic low back pain (OR = 1.8, 95% CI, 1.3-2.5) and disabling low back pain (OR = 2.1, 95% CI, 1.1-4.1). Similarly, Caragee et al (2006) investigated 200 subjects, 100 without lumbar pain and 100 with low back pain. Those with were more likely to be smokers. After combining chronic nonlumbar pain, smoking, and abnormal psychological findings and adjusting for age and sex, an abnormal psychometric profile and smoking correctly identified 72 of 118 (61%) serious low back pain events (OR = 3.97; 95% CI, 2.19-7.22).

Once I recoded BMI from a continuous variable to a categorical variable in alignment with current classifications for healthy and unhealthy BMI, a significant association between BMI and back pain emerged. People who were obese were 1.4 times as likely to have back pain than those who were healthy weight. Those who were overweight were 1.1 times as likely to have back pain than those who were healthy weight. Underweight did not show a significant relationship with back pain. Thus, I concluded that those who are obese are more likely to have back pain than those who are healthy weight and those who are overweight are more likely to have back pain than those who are healthy weight but no comparison between other BMI categories can be made from these data. Also, since underweight had no significant relationship, it is impossible to state if there is a biological gradient between BMI and back pain.

When assessing the association of physical activity to back pain and using physical activity as a continuous variable in the one-way analysis of variance the difference in the means was large. However, the data were skewed to such a degree that I had to transform them and then it became very difficult, if not impossible, to find out

what the real means were for each group (back pain vs no back pain). For the logistic regression I recoded physical activity independent variable into meeting or not meeting the physical activity recommendation of 150 or more minutes of physical activity. A significant difference in back pain prevalence was evident immediately in bivariate logistic regression and this relationship continued in the multiple logistic regression. Over 50% of U.S. adults failed to meet the physical activity recommendation. When reviewing the adjusted odds ratios those who did not meet the physical activity recommendation were 1.2 times as likely as those who did meet the recommendation to have back pain. This does suggest that being active is potentially protective to back pain. However, for those who are extremely active, this may not be the case and I am unable to make a conclusion in that regard.

In the logistic regression model, the association of race with back pain remained unimpressive, as it was in the chi square for research question two. The only significant odds ratio in the model was between Hispanics and non-Hispanic Whites, but it was low at 1.1. The interpretation provided for the results of this variable in research question two is applicable here.

More females than males tended to have lower back pain in the chi square performed for research question two and the relationship persisted in the multiple logistic regression after controlling for other variables. Females were 1.2 times as likely as males to have back pain.

Level of education was left as a continuous variable for the logistic regression since a normal distribution is not required for this analysis and there were no *a priori*

relevant categories in to which I could break it down. As in the one-way analysis of variance, higher levels of education demonstrated a potentially protective effect on back pain, consistent with previous literature. However the magnitude of association was very small and once rounded it could be argued there was no magnitude of association when controlling for the other variables. Dionne et al (1995) found that subjects with less than 12 years of education were more often smokers than those with more than 12 years of education and had more back pain. Thus, education may be a confounder for smoking.

Once I entered age into the multiple logistic regression, there was no significant magnitude of association with back pain when controlling for the other variables. This was not surprising since the difference in the mean age between those who had back pain and those who did not have back pain was only 3.3 years when the *t*-test was performed in research question two.

The impact of anxiousness, depression, and smoking on correctly classifying cases of back pain was more obvious when I reviewed the data from the forward stepwise logistic regression analysis. By the fourth step, little was added to the value of the model with each added independent variable. This means that anxiousness, smoking, age, and depression were able to correctly classify 73% of cases of back pain. Conversely, there must be other variables that have a significant effect on the back pain experience that were not investigated in this study.

Limitations of the Study

There are a number of limitations to this study. This study is limited by the survey research data collection and the data are based on self-report and influenced by recall

bias. The data are only as valid as the answers provided by the respondents at the time of the survey. One of the inherent limitations to the study is the poor definition afforded by the survey creators to the term "back pain." Back pain can exist anywhere in the spine from the base of the neck to the gluteal folds but without a clear definition of the term it is possible that this lack of clarity could have influenced the prevalence of back pain in the sample and therefore the estimated population prevalence of back pain from the complex samples analysis.

The NHIS is limited to non-institutionalized and non-military adults. Considering the psychological and physical demands upon these populations, it is possible that the estimates provided from this research under-report the prevalence of back pain and the magnitudes of association of some of the independent variables with back pain. For example, war fighters in combat likely have higher levels of anxiety or depression than the average U.S. adult. Thus, the magnitude of association of anxiousness or depression with back pain may be higher for military members. Adults who are institutionalized for psychological health diagnoses may also have different magnitudes of association and different prevalence of back pain. Thus, the results of the present study may not be applicable to these other populations.

While the results of research question one showed a higher back pain prevalence for smokers over former smokers over never smokers it may be a relationship that is bidirectional. People may have more back pain if they smoke more or they may smoke more because they have back pain. Smoking may also be serving as a marker for other

comorbid factors, such as depression or anxiety. Thus those that have psychological comorbidities may have a higher smoking prevalence and back pain.

Another consideration for this research question is the choice of one-way analysis of variance for the statistical analysis. In hindsight, multiple *t*-tests with correction for multiple comparisons would have been a more appropriate choice for this question because the categorical dependent variable of back pain had only two categories. While one-way analysis of variance can produce the same information, it is usually reserved for dependent variables with three or more categories (Portney & Watkins, 2009). Unfortunately, since there were only two categories for the dependent variable, post-hoc comparisons between and within groups were unable to be utilized.

The analyses from this research created a logistic regression model from which it may be possible to predict back pain in some people. There are limitations to extending these findings. First, the model has not been validated on a different sample and it should not be generalized beyond the context of the 2012 NHIS data set. Second, the model does not correctly classify 27% of cases, leaving substantial room for error. Finally, the model is able to account for only 8.5% to 12.2% of the variance.

This is a cross-sectional research design. The strength of this design is in providing prevalence data. The design is unable to imply causation of back pain from smoking or any of the other independent variables included in this study since there is no control group, the design is retrospective, and the study is observational in nature (Schoenbach & Rosamond, 2000). Further, since the cross-sectional design observes prevalence at one point in time, it is unable to account for allostatic load where

development of disease is potentially more cumulative. The allostatic load concept is popular in life course disease epidemiologic models (Liu, Jones, & Glymour, 2010) and may have application when assessing associations between back pain and biopsychosocial variables.

There are other associations that may affect the outcome in this study. It is theoretically possible that people in physically demanding jobs may smoke more. Therefore, such physical demands could confound the association between smoking and back pain. Occupation was not included as an independent variable in the present study and may add to the predictive nature of the logistic regression. Investigators of future regressions may consider including this variable and assessing for collinearity to find out if it is a confounder.

It has also been suggested that smokers have poorer mental health than nonsmokers and that smoking may serve as a proxy for psychological variables that are strongly related to back pain (Shiri et al., 2010). While this may be the case in some studies, these appeared to have minimal association with each other when assessed for association using two different methods. Another consideration is that individuals with sporadic cigarette consumption may not have been classified as current smokers using the NHIS question, “Have you ever smoked 100 cigarettes?” These individuals also would have not been classified as former smokers, misclassifying them as never smokers. While the number of such misclassifications would have been very small in such a large sample, it is theoretically possible that correctly including them in the current smoker category may increase the odds ratio for current smokers and back pain.

Recommendations

While this study is unable to establish a causal link between mental health comorbidities and back pain or smoking and back pain, the data from this study support the need for further investigation into behavioral health interventions for some patients with back pain. Perhaps this research will help provide some guidance as to which patients would potentially benefit most from such psychological interventions and which may benefit most from more physical interventions, such as exercise and body-based therapies.

Future studies may benefit from a more thorough inclusion of variables that may aid in increasing the predictive nature of the multiple logistic regression. Based upon the large odds ratios revealed from anxiousness and depression, larger representation from the psychological aspect of the biopsychosocial model may be telling. Particularly, with regard to the NHIS, including other psychological variables and variables querying about effects of psychological conditions on activities of daily living may provide further information about the mind-back pain relationship. It is known that back pain sufferers often develop fear-avoidance behaviors for various activities of daily living (Leeuw et al., 2007) and the degree to which anxiety, depression, or other coexisting psychological comorbidities may affect the fear-avoidance relationship in back pain sufferers is an area ripe for further investigation. This recommendation comes with one note of caution in that some of the psychological variables changed from the 2012 NHIS to 2013 and beyond; new variables would have to be located and it would be difficult to combine data sets from years that span this change.

It has also been shown in some studies that levels of income are related to back pain (G. Andersson, 2008). Future work should include the relevant variables from NHIS and may aid in better defining the factors that are predictive for back pain. Likewise, inclusion of occupations in future work, to the degree possible while doing a secondary data analysis from NHIS, should be considered in order to identify any confounders or covariates.

Finally, as was mentioned earlier, the results of the present research present just one statistical model that is not entirely accurate for predicting back pain. If other variables can be found that enhance this predictive ability, it will be necessary to validate the model on a set of data different than the 2012 NHIS. One method of doing this would be to validate the model on 2011 or earlier NHIS data, as the variables are the same as 2012. In order to validate the data on 2013 and later NHIS, the depression and anxiousness/worried variables are different. Because of this, it may be required to create a new model using half of one of those years and then validate the model on the other half of the sample. Since the data set is very large and also has a lot of power, this may be a reasonable recommendation. Otherwise, a new model could be produced in NHIS 2013 and then validated on NHIS 2014 and/or later data sets.

Implications for Social Change

At the outset of this research it was postulated that if a measure of association was detected between smoking exposure and the outcome of back pain that it could inform future back pain prevention research. The findings of this study did show that smokers and former smokers were more likely to have back pain than never smokers and that the

association has a biological gradient. Thus, from this very large sample containing significant statistical power, it is reasonable to suggest that studies investigating the effect of therapy for back pain should potentially include interventions for smoking cessation. One recent cohort study has done this and found that therapeutic outcomes for back pain were significantly better for those who quit smoking compared to those people who refused to quit (Behrend et al., 2012). Moreover, this research demonstrates that the inclusion of variables related to the psychological condition of research subjects is imperative in back pain research. Ignoring or inadvertently not including appropriate psychological variables in retrospective or prospective observational or experimental studies of back pain leaves out significant influences on the back pain experience.

Back pain and smoking are two highly prevalent problems that have a staggering negative effect on the health of the public. Currently, there is no cure for back pain. The present research demonstrates that smoking status and psyschological variables have significant relationships to back pain. In that smoking and some psychological variables are modifiable, using the data and conclusions from this research may help in reducing the immense morbidity, social, and economic burdens associated with back pain in U.S. adults.

Conclusion

This quantitative cross-sectional study of a large, representative sample of non-institutionalized, non-military U.S. adults was examined using a complex samples analysis that provides prevalence data weighted to U.S. population estimates. I found that those people who self-reported being anxious or worried, had been told by a health care

provider that they had depression, or who were current of former smokers were significantly more likely to have back pain than people who did not self-identify with these categories. Those people who were obese and those who failed to meet recommended levels of physical activity were also more likely to have back pain than their reference categories. Sex and race had a small association with back pain in that females were slightly more likely than males to have back pain and non-Hispanic whites were slightly more likely than Hispanics to have back pain. Level of education and age were not significantly associated with back pain.

References

Ackerman, W. E., 3rd, & Ahmad, M. (2007). Effect of cigarette smoking on serum hydrocodone levels in chronic pain patients. *Journal of the Arkansas Medical Society, 104*(1), 19-21.

Akmal, M., Kesani, A., Anand, B., Singh, A., Wiseman, M., & Goodship, A. (2004). Effect of nicotine on spinal disc cells: a cellular mechanism for disc degeneration. *Spine (Phila Pa 1976), 29*(5), 568-575.

Alkherayf, F., & Agbi, C. (2009). Cigarette smoking and chronic low back pain in the adult population. *Clinical and Investigative Medicine. Medecine Clinique et Experimentale, 32*(5), E360-367.

Alkherayf, F., Wai, E. K., Tsai, E. C., & Agbi, C. (2010). Daily smoking and lower back pain in adult Canadians: the Canadian Community Health Survey. *Journal of pain research, 3*, 155-160. doi: 10.2147/JPR.S11031

Alonso, Y. (2004). The biopsychosocial model in medical research: the evolution of the health concept over the last two decades. *Patient Education and Counseling, 53*(2), 239-244. doi: 10.1016/S0738-3991(03)00146-0

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders : DSM-5* (5th ed.). Washington, D.C.: American Psychiatric Association.

Andersen, L. L., Mortensen, O. S., Hansen, J. V., & Burr, H. (2011). A prospective cohort study on severe pain as a risk factor for long-term sickness absence in blue- and white-collar workers. *Occupational and Environmental Medicine, 68*(8), 590-592. doi: 10.1136/oem.2010.056259

Andersson, Gunnar. (2008). *The burden of musculoskeletal diseases in the United States : prevalence, societal, and economic cost*. Rosemont, IL: American Academy of Orthopaedic Surgeons.

Andersson, H., Ejlertsson, G., & Leden, I. (1998). Widespread musculoskeletal chronic pain associated with smoking. An epidemiological study in a general rural population. *Scandinavian Journal of Rehabilitation Medicine, 30*(3), 185-191.

Baker, E. A., Metzler, M. M., & Galea, S. (2005). Addressing social determinants of health inequities: learning from doing. *American Journal of Public Health, 95*(4), 553-555. doi: 95/4/553 [pii]10.2105/AJPH.2005.061812

Balague, F., Mannion, A. F., Pellise, F., & Cedraschi, C. (2012). Non-specific low back pain. *Lancet, 379*(9814), 482-491. doi: 10.1016/S0140-6736(11)60610-7

Behrend, C., Prasarn, M., Coyne, E., Horodyski, M., Wright, J., & Rechtine, G. R. (2012). Smoking Cessation Related to Improved Patient-Reported Pain Scores Following Spinal Care. *Journal of Bone and Joint Surgery, 94*(23), 2161-2166. doi: 10.2106/JBJS.K.01598

Bejia, I., Younes, M., Jamila, H. B., Khalfallah, T., Ben Salem, K., Touzi, M., . . . Bergaoui, N. (2005). Prevalence and factors associated to low back pain among hospital staff. *Joint Bone Spine, 72*(3), 254-259. doi: 10.1016/j.jbspin.2004.06.001

Bergenudd, H., & Nilsson, B. (1988). Back pain in middle age; occupational workload and psychologic factors: an epidemiologic survey. *Spine (Phila Pa 1976), 13*(1), 58-60.

Bergman, S. (2007). Management of musculoskeletal pain. *Best Pract Res Clin Rheumatol, 21*(1), 153-166. doi: 10.1016/j.berh.2006.10.001

Bertalanffy, Ludwig von. (1972). The history and status of general systems theory. *The Academy of Management Journal, 15*(4), 407-426.

Borrell-Carrio, F., Suchman, A. L., & Epstein, R. M. (2004). The biopsychosocial model 25 years later: principles, practice, and scientific inquiry. *Ann Fam Med, 2*(6), 576-582. doi: 10.1370/afm.245

Carey, T. S., Freburger, J. K., Holmes, G. M., Jackman, A., Knauer, S., Wallace, A., & Darter, J. (2010). Race, care seeking, and utilization for chronic back and neck pain: population perspectives. *J Pain, 11*(4), 343-350. doi: 10.1016/j.jpain.2009.08.003

Carlson, S. A., Fulton, J. E., Schoenborn, C. A., & Loustalot, F. (2010). Trend and prevalence estimates based on the 2008 Physical Activity Guidelines for Americans. *American Journal of Preventive Medicine, 39*(4), 305-313. doi: 10.1016/j.amepre.2010.06.006

Carr, J. L., & Moffett, J. A. (2005). The impact of social deprivation on chronic back pain outcomes. *Chronic Illn, 1*(2), 121-129.

Carragee, E., Alamin, T., Cheng, I., Franklin, T., & Hurwitz, E. (2006). Does minor trauma cause serious low back illness? *Spine (Phila Pa 1976), 31*(25), 2942-2949. doi: 10.1097/01.brs.0000248429.10963.13

Cassel, J. (1964). Social Science Theory as a Source of Hypotheses in Epidemiological Research. *American Journal of Public Health and the Nations Health, 54*, 1482-1488.

Cassel, J. (1974). An epidemiological perspective of psychosocial factors in disease etiology. *American Journal of Public Health, 64*(11), 1040-1043.

Cassel, J. (1975). The contribution of the social environment to host resistance. *American Journal of Epidemiology, 104*(2), 107-123.

Centers for Disease Control and Prevention. (2008). Cigarette smoking among adults--United States, 2007. *MMWR. Morbidity and Mortality Weekly Report, 57*(45), 1221-1226.

Centers for Disease Control and Prevention. (2010a). *Health United States, 2009*. Atlanta: Centers for Disease Control and Prevention, National Center for Health Statistics.

Centers for Disease Control and Prevention. (2010b). Vital signs: nonsmokers' exposure to secondhand smoke --- United States, 1999-2008. *MMWR. Morbidity and Mortality Weekly Report, 59*(35), 1141-1146.

Centers for Disease Control and Prevention. (2011). Current cigarette smoking prevalence among working adults--United States, 2004-2010. *MMWR. Morbidity and Mortality Weekly Report, 60*(38), 1305-1309. doi: mm6038a2 [pii]

Chou, R., Qaseem, A., Snow, V., Casey, D., Cross, J. T., Jr., Shekelle, P., & Owens, D. K. (2007). Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. *Annals of Internal Medicine, 147*(7), 478-491. doi: 147/7/478 [pii]

Chou, Y. C., Shih, C. C., Lin, J. G., Chen, T. L., & Liao, C. C. (2012). Low back pain associated with sociodemographic factors, lifestyle and osteoporosis: A population-based study. *J Rehabil Med*. doi: 10.2340/16501977-1070

Christensen, J., Fisker, A., Mortensen, E. L., Olsen, L. R., Mortensen, O. S., Hartvigsen, J., & Langberg, H. (2015). Comparison of mental distress in patients with low back pain and a population-based control group measured by Symptoms Check List - A case-referent study. *Scand J Public Health, 43*(6), 638-647. doi: 10.1177/1403494815581697

Cohen. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, N.J.: L. Erlbaum Associates.

Cohen, Jules. (2000). Obituary: George L. Engel, MD. *Journal of the American Medical Association, 283*(21), 2857. doi: 10.1001/jama.283.21.2857

Collaborators, U. S. Burden of Disease. (2013). The state of US health, 1990-2010: burden of diseases, injuries, and risk factors. *JAMA, 310*(6), 591-608. doi: 10.1001/jama.2013.13805

Covic, T., Adamson, B., Spencer, D., & Howe, G. (2003). A biopsychosocial model of pain and depression in rheumatoid arthritis: a 12-month longitudinal study. *Rheumatology, 42*(11), 1287-1294. doi: 10.1093/rheumatology/keg369

Cramer, Gregory D., Darby, Susan A., & Cramer, Gregory D. (2014). *Clinical anatomy of the spine, spinal cord, and ANS* (3rd ed.). St. Louis: Elsevier.

Dagenais, S., Caro, J., & Haldeman, S. (2008). A systematic review of low back pain cost of illness studies in the United States and internationally. *Spine J, 8*(1), 8-20. doi: S1529-9430(07)00898-4 [pii] 10.1016/j.spinee.2007.10.005

Dagenais, S., Tricco, A. C., & Haldeman, S. (2010). Synthesis of recommendations for the assessment and management of low back pain from recent clinical practice guidelines. *Spine J, 10*(6), 514-529. doi: S1529-9430(10)00288-3 [pii] 10.1016/j.spinee.2010.03.032

Deyo, R. A., & Bass, J. E. (1989). Lifestyle and low-back pain. The influence of smoking and obesity. *Spine (Phila Pa 1976), 14*(5), 501-506.

Deyo, R. A., & Weinstein, J. N. (2001). Low back pain. *New England Journal of Medicine, 344*(5), 363-370. doi: 10.1056/NEJM200102013440508

Dhingra, L. K., Homel, P., Grossman, B., Chen, J., Scharaga, E., Calamita, S., . . . Portenoy, R. (2013). Ecological Momentary Assessment of Smoking Behavior in Persistent Pain Patients. *Clinical Journal of Pain*. doi: 10.1097/AJP.0b013e31829821c7

Dionne, C., Koepsell, T. D., Von Korff, M., Deyo, R. A., Barlow, W. I., & Checkoway, H. (1995). Formal education and back-related disability. In search of an explanation. *Spine (Phila Pa 1976), 20*(24), 2721-2730.

Ditre, J. W., Brandon, T. H., Zale, E. L., & Meagher, M. M. (2011). Pain, nicotine, and smoking: research findings and mechanistic considerations. *Psychological Bulletin, 137*(6), 1065-1093. doi: 10.1037/a0025544

Division of Health Interview Statistics. (2013). *2012 National Health Interview Survey (NHIS) Public Use Data Release: NHIS Survey Description*. Hyattsville: National Center for Health Statistics. Retrieved April 5, 2014 from: ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Dataset_Documentation/NHIS/2012/srvydesc.pdf.

Dumville, J. C., Petherick, E. S., & Cullum, N. (2008). When will I see you again? The fate of research findings from international wound care conferences*. *Int Wound J, 5*(1), 26-33. doi: IWJ343 [pii] 10.1111/j.1742-481X.2007.00343.x

Dunn, A. S., Green, B. N., Formolo, L. R., & Chicoine, D. (2011). Retrospective case series of clinical outcomes associated with chiropractic management for veterans with low back pain. *Journal of Rehabilitation Research and Development, 48*(8), 927-934.

Emmons, Karen M. (2000). Health behaviors in a social context. In Lisa F. Berkman & Ichiro Kawachi (Eds.), *Social Epidemiology* (pp. 242-266). Oxford: Oxford University Press.

Engel, G. L. (1977). The need for a new medical model: a challenge for biomedicine. *Science, 196*(4286), 129-136.

Evans, Marion Willard. (2011). The public health impact of musculoskeletal conditions. In Michael T. Haneline & William C. Meeker (Eds.), *Introduction to Public Health for Chiropractors* (pp. 241-249). Sudbury: Jones and Bartlett Publishers.

Faul, F., Erdfelder, E., Buchner, A., & Lang, A. G. (2009). Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. *Behav Res Methods, 41*(4), 1149-1160. doi: 10.3758/BRM.41.4.1149

Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods, 39*(2), 175-191.

Fos, Peter J. (2011). *Epidemiology foundations : the science of public health* (1st ed.). San Francisco: Jossey-Bass.

Freburger, J. K., Holmes, G. M., Agans, R. P., Jackman, A. M., Darter, J. D., Wallace, A. S., . . . Carey, T. S. (2009). The rising prevalence of chronic low back pain. *Archives of Internal Medicine, 169*(3), 251-258. doi: 10.1001/archinternmed.2008.543

Friis, Robert H., & Sellers, Thomas A. (2009). *Epidemiology for public health practice* (4th ed.). Sudbury, Mass.: Jones and Bartlett Publishers.

Gatchel, R. J., Peng, Y. B., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The biopsychosocial approach to chronic pain: scientific advances and future directions. *Psychological Bulletin, 133*(4), 581-624. doi: 10.1037/0033-2909.133.4.581

Gill, D. K., Davis, M. C., Smith, A. J., & Straker, L. M. (2013). Bidirectional relationships between cigarette use and spinal pain in adolescents accounting for psychosocial functioning. *Br J Health Psychol*. doi: 10.1111/bjhp.12039

Giordano, J., & Schatman, M. E. (2008). A crisis in chronic pain care: an ethical analysis. Part two: proposed structure and function of an ethics of pain medicine. *Pain Physician, 11*(5), 589-595.

Goesling, J., Brummett, C. M., & Hassett, A. L. (2012). Cigarette smoking and pain: depressive symptoms mediate smoking-related pain symptoms. *Pain, 153*(8), 1749-1754. doi: 10.1016/j.pain.2012.05.014

Goldberg, M. S., Scott, S. C., & Mayo, N. E. (2000). A review of the association between cigarette smoking and the development of nonspecific back pain and related outcomes. *Spine, 25*(8), 995-1014.

Goodman, C, & Anise, A. (2006). *What is known about the effectiveness of economic instruments to reduce consumption of foods high in saturated fats and other energy-dense foods for preventing and treating obesity?* . Copenhagen: WHO Regional Office for Europe. Accessed September 1, 2013 from http://www.euro.who.int/document/e88909.pdf.

Gordon, J. E. (1954). Epidemiology in modern perspective. *Proceedings of the Royal Society of Medicine, 47*(7), 564-570.

Green, B.N., & Johnson, C.D. (2013a). *Do people who smoke have more back pain?* Boston: Accepted presentation, 140th Annual Meeting and Exposition of the American Public Health Association, Nov 2-6, 2013. Available from: https://apha.confex.com/apha/141am/webprogram/Paper280334.html.

Green, B.N., & Johnson, C.D. (2013b). Establishing a theoretical basis for research in musculoskeletal epidemiology: a proposal for the use of biopsychosocial theory in investigations of back pain and smoking. *J Chiropr Humanit, 20*(1), 1-8. doi: 10.1016/j.echu.2013.10.004

Haldeman, S., & Dagenais, S. (2008). A supermarket approach to the evidence-informed management of chronic low back pain. *Spine J, 8*(1), 1-7. doi: S1529-9430(07)00902-3 [pii] 10.1016/j.spinee.2007.10.009

Hancock, M. J., Maher, C. G., Laslett, M., Hay, E., & Koes, B. (2011). Discussion paper: what happened to the 'bio' in the bio-psycho-social model of low back pain? *European Spine Journal, 20*(12), 2105-2110. doi: 10.1007/s00586-011-1886-3

Hart, L. G., Deyo, R. A., & Cherkin, D. C. (1995). Physician office visits for low back pain. Frequency, clinical evaluation, and treatment patterns from a U.S. national survey. *Spine (Phila Pa 1976), 20*(1), 11-19.

Heneweer, H., Staes, F., Aufdemkampe, G., van Rijn, M., & Vanhees, L. (2011). Physical activity and low back pain: a systematic review of recent literature. *European Spine Journal, 20*(6), 826-845. doi: 10.1007/s00586-010-1680-7

Heneweer, H., Vanhees, L., & Picavet, H. S. (2009). Physical activity and low back pain: a U-shaped relation? *Pain, 143*(1-2), 21-25. doi: 10.1016/j.pain.2008.12.033

Hill, A. B. (1965). The Environment and Disease: Association or Causation? *Proceedings of the Royal Society of Medicine, 58*, 295-300.

Holmberg, S. A., & Thelin, A. G. (2010). Predictors of sick leave owing to neck or low back pain: a 12-year longitudinal cohort study in a rural male population. *Annals of Agricultural and Environmental Medicine, 17*(2), 251-257.

Hoy, D., Brooks, P., Blyth, F., & Buchbinder, R. (2010). The epidemiology of low back pain. *Best Pract Res Clin Rheumatol, 24*(6), 769-781. doi: S1521-6942(10)00088-4 [pii] 10.1016/j.berh.2010.10.002

Hoy, D., Protani, M., De, R., & Buchbinder, R. (2010). The epidemiology of neck pain. *Best practice & research. Clinical rheumatology, 24*(6), 783-792. doi: 10.1016/j.berh.2011.01.019

Hung, C. I., Liu, C. Y., & Fu, T. S. (2015). Depression: An important factor associated with disability among patients with chronic low back pain. *International Journal of Psychiatry in Medicine, 49*(3), 187-198. doi: 10.1177/0091217415573937

International Association for the Study of Pain. (2011). *Pain Terms: A Current List with Definitions and Notes on Usage*. Washington, DC: International Association for the Study of Pain. Downloaded February 3, 2013 from http://www.iasp-pain.org/AM/Template.cfm?Section=Classification_of_Chronic_Pain&Template=/CM/ContentDisplay.cfm&ContentID=16283.

Jacob, Gary. (2003). Biopsychosocial perspective on low back pain: patient provider communications. *Journal of Minimally Invasive Spinal Techniques, 3*, 27-35.

Janwantanakul, P., Pensri, P., Jiamjarasrangsi, W., & Sinsongsook, T. (2009). Associations between prevalence of self-reported musculoskeletal symptoms of the spine and biopsychosocial factors among office workers. *J Occup Health, 51*(2), 114-122.

Joffe, M., Gambhir, M., Chadeau-Hyam, M., & Vineis, P. (2012). Causal diagrams in systems epidemiology. *Emerg Themes Epidemiol, 9*(1), 1. doi: 10.1186/1742-7622-9-1

Johannes, C. B., Le, T. K., Zhou, X., Johnston, J. A., & Dworkin, R. H. (2010). The prevalence of chronic pain in United States adults: results of an Internet-based survey. *J Pain, 11*(11), 1230-1239. doi: S1526-5900(10)00601-2 [pii] 10.1016/j.jpain.2010.07.002

Johnson, C., & Green, B. N. (2009). Public health, wellness, prevention, and health promotion: considering the role of chiropractic and determinants of health. *Journal of Manipulative and Physiological Therapeutics, 32*(6), 405-412. doi: 10.1016/j.jmpt.2009.07.001

Jull, G., & Sterling, M. (2009). Bring back the biopsychosocial model for neck pain disorders. *Manual therapy, 14*(2), 117-118. doi: 10.1016/j.math.2009.01.004

Katz, Mitchell H. (2011). *Multivariable analysis : a practical guide for clinicians and public health researchers* (Third Edition. ed.). Cambridge: Cambridge University Press.

Kent, P. M., & Keating, J. L. (2005). The epidemiology of low back pain in primary care. *Chiropractic & Osteopathy, 13*, 13. doi: 10.1186/1746-1340-13-13

Kikuchi, S. (2008). New concept for backache: biopsychosocial pain syndrome. *European Spine Journal, 17 Suppl 4*, 421-427. doi: 10.1007/s00586-008-0747-1

Knox, J. B., Orchowski, J. R., & Owens, B. (2012). Racial differences in the incidence of acute low back pain in United States military service members. *Spine (Phila Pa 1976), 37*(19), 1688-1692. doi: 10.1097/BRS.0b013e318255a07b

Kopec, J. A., Sayre, E. C., & Esdaile, J. M. (2004). Predictors of back pain in a general population cohort. *Spine (Phila Pa 1976), 29*(1), 70-77; discussion 77-78. doi: 10.1097/01.BRS.0000103942.81227.7F

Kovacs, F. M., Gestoso, M., Gil del Real, M. T., Lopez, J., Mufraggi, N., & Mendez, J. I. (2003). Risk factors for non-specific low back pain in schoolchildren and their parents: a population based study. *Pain, 103*(3), 259-268. doi: S0304395902004542 [pii]

Krieger, N. (1994). Epidemiology and the web of causation: has anyone seen the spider? *Social Science and Medicine, 39*(7), 887-903.

Krieger, N. (2000). Epidemiology and social sciences: towards a critical reengagement in the 21st century. *Epidemiologic Reviews, 22*(1), 155-163.

Krieger, N. (2001). Theories for social epidemiology in the 21st century: an ecosocial perspective. *International Journal of Epidemiology, 30*(4), 668-677.

Krieger, N. (2011). *Epidemiology and the people's health : theory and context*. New York: Oxford University Press.

Krieger, N., & Zierler, S. (1996). What explains the public's health?--A call for epidemiologic theory. *Epidemiology, 7*(1), 107-109.

Kwon, M. A., Shim, W. S., Kim, M. H., Gwak, M. S., Hahm, T. S., Kim, G. S., . . . Kim, T. H. (2006). A correlation between low back pain and associated factors: a study involving 772 patients who had undergone general physical examination. *Journal of Korean Medical Science, 21*(6), 1086-1091. doi: 2006121086 [pii]

Leboeuf-Yde, C. (1995). Does smoking cause low back pain? A review of the epidemiologic literature for causality. *Journal of Manipulative and Physiological Therapeutics, 18*(4), 237-243.

Leeuw, M., Goossens, M. E., Linton, S. J., Crombez, G., Boersma, K., & Vlaeyen, J. W. (2007). The fear-avoidance model of musculoskeletal pain: current state of scientific evidence. *Journal of Behavioral Medicine, 30*(1), 77-94. doi: 10.1007/s10865-006-9085-0

Lindau, S. T., Laumann, E. O., Levinson, W., & Waite, L. J. (2003). Synthesis of scientific disciplines in pursuit of health: the Interactive Biopsychosocial Model. *Perspectives in Biology and Medicine, 46*(3 Suppl), S74-86.

Linton, S. J. (2000). A review of psychological risk factors in back and neck pain. *Spine (Phila Pa 1976), 25*(9), 1148-1156.

Liu, S., Jones, R. N., & Glymour, M. M. (2010). Implications of Lifecourse Epidemiology for Research on Determinants of Adult Disease. *Public Health Reviews, 32*(2), 489-511.

Luginaah, I. N., Taylor, S. M., Elliott, S. J., & Eyles, J. D. (2002). Community reappraisal of the perceived health effects of a petroleum refinery. *Social Science and Medicine, 55*(1), 47-61.

Magni, G., Marchetti, M., Moreschi, C., Merskey, H., & Luchini, S. R. (1993). Chronic musculoskeletal pain and depressive symptoms in the National Health and Nutrition Examination. I. Epidemiologic follow-up study. *Pain, 53*(2), 163-168.

Malmgren, Helge. (2005). The theoretical basis of the biopsychosocial model. In Peter White (Ed.), *Biopsychosocial medicine* (pp. 21-35). Oxford: Oxford University Press.

Mann, C. J. (2003). Observational research methods. Research design II: cohort, cross sectional, and case-control studies. *Emerg Med J, 20*(1), 54-60.

Marchand, G. H., Myhre, K., Leivseth, G., Sandvik, L., Lau, B., Bautz-Holter, E., & Roe, C. (2015). Change in pain, disability and influence of fear-avoidance in a work-focused intervention on neck and back pain: a randomized controlled trial. *BMC Musculoskelet Disord, 16*(1), 94. doi: 10.1186/s12891-015-0553-y

Martin, B. I., Turner, J. A., Mirza, S. K., Lee, M. J., Comstock, B. A., & Deyo, R. A. (2009). Trends in health care expenditures, utilization, and health status among US adults with spine problems, 1997-2006. *Spine (Phila Pa 1976), 34*(19), 2077-2084. doi: 10.1097/BRS.0b013e3181b1fad1

Matsudaira, K., Kawaguchi, M., Isomura, T., Inuzuka, K., Koga, T., Miyoshi, K., & Konishi, H. (2015). Assessment of psychosocial risk factors for the development of non-specific chronic disabling low back pain in Japanese workers-findings from the Japan epidemiological research of Occupation-related Back pain (JOB) study. *Industrial Health*. doi: 10.2486/indhealth.2014-0260

Mausner, Judith S., & Bahn, Anita K. (1974). *Epidemiology; an introductory text*. Philadelphia,: Saunders.

Mikkonen, P., Leino-Arjas, P., Remes, J., Zitting, P., Taimela, S., & Karppinen, J. (2008). Is smoking a risk factor for low back pain in adolescents? A prospective cohort study. *Spine (Phila Pa 1976), 33*(5), 527-532. doi: 10.1097/BRS.0b013e3181657d3c

00007632-200803010-00014 [pii]

Mitchell, T., O'Sullivan, P. B., Smith, A., Burnett, A. F., Straker, L., Thornton, J., & Rudd, C. J. (2009). Biopsychosocial factors are associated with low back pain in female nursing students: a cross-sectional study. *International Journal of Nursing Studies, 46*(5), 678-688. doi: 10.1016/j.ijnurstu.2008.11.004

Mogil, J. S., Sternberg, W. F., Marek, P., Sadowski, B., Belknap, J. K., & Liebeskind, J. C. (1996). The genetics of pain and pain inhibition. *Proceedings of the National Academy of Sciences of the United States of America, 93*(7), 3048-3055.

Mohseni-Bandpei, M. A., Ahmad-Shirvani, M., Golbabaei, N., Behtash, H., Shahinfar, Z., & Fernandez-de-las-Penas, C. (2011). Prevalence and risk factors associated with low back pain in Iranian surgeons. *Journal of Manipulative and Physiological Therapeutics, 34*(6), 362-370. doi: 10.1016/j.jmpt.2011.05.010

Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. *JAMA, 291*(10), 1238-1245. doi: 10.1001/jama.291.10.1238291/10/1238 [pii]

Munro, Barbara Hazard. (2005). *Statistical methods for health care research* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.

National Center for Health Statistics. (2013). *Data File Documentation, National Health Interview Survey, 2012 (machine readable data file and documentation)*. Hyattsville [MD]: National Center for Health Statistics, Centers for Disease Control and Prevention.

Noordzij, M., Dekker, F. W., Zoccali, C., & Jager, K. J. (2009). Study designs in clinical research. *Nephron. Clinical practice, 113*(3), c218-221. doi: 10.1159/000235610

Novack, D. H., Cameron, O., Epel, E., Ader, R., Waldstein, S. R., Levenstein, S., . . . Wainer, A. R. (2007). Psychosomatic medicine: the scientific foundation of the biopsychosocial model. *Acad Psychiatry, 31*(5), 388-401. doi: 10.1176/appi.ap.31.5.388

Pallant, Julie. (2010). *SPSS Survival Manual : a Step by Step Guide to Data Aanalysis Using the SPSS Program* (4th ed.). Maidenhead, Berkshire. U.K: Open University Press.

Palmer, K. T., Harris, C. E., Griffin, M. J., Bennett, J., Reading, I., Sampson, M., & Coggon, D. (2008). Case-control study of low-back pain referred for magnetic resonance imaging, with special focus on whole-body vibration. *Scandinavian Journal of Work, Environment and Health, 34*(5), 364-373.

Peng, Chao-Ying Joanne, Lee, Kuk Lida, & Ingersoll, Gary M. (2002). An introduction to logistic regression analysis and reporting. *Journal of Educational Research, 96*, 3-14.

Perruccio, A. V., Gandhi, R., Rampersaud, Y. R., & Arthritis Program, University Health Network. (2013). Heterogeneity in health status and the influence of patient characteristics across patients seeking musculoskeletal orthopaedic care - a cross-sectional study. *BMC Musculoskelet Disord, 14*, 83. doi: 10.1186/1471-2474-14-83

Portney, Leslie Gross, & Watkins, Mary P. (2009). *Foundations of clinical research : applications to practice* (3rd ed.). Upper Saddle River, N.J.: Pearson/Prentice Hall.

Praemer, Allan, Furner, Sylvia, Rice, Dorothy P., & American Academy of Orthopaedic Surgeons. (1992). *Musculoskeletal conditions in the United States* (1st ed.). Park Ridge, Ill.: American Academy of Orthopaedic Surgeons.

Puustinen, R., Leiman, M., & Viljanen, A. M. (2003). Medicine and the humanities--theoretical and methodological issues. *Med Humanit, 29*(2), 77-80.

Rainville, J., Smeets, R. J., Bendix, T., Tveito, T. H., Poiraudeau, S., & Indahl, A. J. (2011). Fear-avoidance beliefs and pain avoidance in low back pain--translating research into clinical practice. *Spine J, 11*(9), 895-903. doi: 10.1016/j.spinee.2011.08.006

Reisbord, L. S., & Greenland, S. (1985). Factors associated with self-reported back-pain prevalence: a population-based study. *Journal of Chronic Diseases, 38*(8), 691-702.

Riffenburgh, R. H. (2012). *Statistics in medicine* (Third edition. ed.). Amsterdam: Elsevier/AP.

Rockett, Ian R.H. (2009). Descriptive epidemiology for public health professionals - part 2. *Sudanese Journal of Public Health, 4*(3), 354-359.

Rubin, D. I. (2007). Epidemiology and risk factors for spine pain. *Neurologic Clinics, 25*(2), 353-371. doi: 10.1016/j.ncl.2007.01.004

Runyan, C. W. (2003). Introduction: back to the future--revisiting Haddon's conceptualization of injury epidemiology and prevention. *Epidemiologic Reviews, 25*, 60-64.

Scherer, R. W., Langenberg, P., & von Elm, E. (2007). Full publication of results initially presented in abstracts. *Cochrane Database Syst Rev*(2), MR000005. doi: 10.1002/14651858.MR000005.pub3

Schmidt, C. O., Raspe, H., Pfingsten, M., Hasenbring, M., Basler, H. D., Eich, W., & Kohlmann, T. (2007). Back pain in the German adult population: prevalence, severity, and sociodemographic correlates in a multiregional survey. *Spine (Phila Pa 1976), 32*(18), 2005-2011. doi: 10.1097/BRS.0b013e318133fad800007632-200708150-00014 [pii]

Schoenbach, Victor J., & Rosamond, Wayne D. (2000). *Understanding the Fundamentals of Epidemiology - An Evolving Test*. Chapel Hill: the authors. Retreieved December 23, 2012 from http://www.epidemiolog.net/evolving/TableOfContents.htm.

Schoenborn, C. A., & Stommel, M. (2011). Adherence to the 2008 adult physical activity guidelines and mortality risk. *American Journal of Preventive Medicine, 40*(5), 514-521. doi: 10.1016/j.amepre.2010.12.029

Shiri, R., Karppinen, J., Leino-Arjas, P., Solovieva, S., & Viikari-Juntura, E. (2010). The association between smoking and low back pain: a meta-analysis. *The American journal of medicine, 123*(1), 87 e87-35. doi: 10.1016/j.amjmed.2009.05.028

Shorter, Edward. (2005). The history of the biopsychosocial approach in medicine: before and after Engel. In Peter White (Ed.), *Biopsychosocial Medicine: An Integrated Approach to Understanding Illness* (pp. 1-19). Oxford: Oxford University Press.

Snelgrove, S., & Liossi, C. (2013). Living with chronic low back pain: a metasynthesis of qualitative research. *Chronic Illn*. doi: 10.1177/1742395313476901

Stewart Williams, J., Ng, N., Peltzer, K., Yawson, A., Biritwum, R., Maximova, T., . . . Chatterji, S. (2015). Risk Factors and Disability Associated with Low Back Pain in Older Adults in Low- and Middle-Income Countries. Results from the WHO Study on Global AGEing and Adult Health (SAGE). *PLoS ONE, 10*(6), e0127880. doi: 10.1371/journal.pone.0127880

Strine, T. W., & Hootman, J. M. (2007). US national prevalence and correlates of low back and neck pain among adults. *Arthritis and Rheumatism, 57*(4), 656-665. doi: 10.1002/art.22684

Swisher, S. N. (1980). The biopsychosocial model: its future for the internist. *Psychosomatic Medicine, 42*(1 Suppl), 113-121.

Tabachnick, Barbara G., & Fidell, Linda S. (2007). *Using Multivariate Statistics* (5th ed.). Boston: Pearson Education.

Taylor, J. B., Goode, A. P., George, S. Z., & Cook, C. E. (2014). Incidence and risk factors for first-time incident low back pain: a systematic review and meta-analysis. *Spine J, 14*(10), 2299-2319. doi: 10.1016/j.spinee.2014.01.026

Tucker, J. M., Welk, G. J., & Beyler, N. K. (2011). Physical activity in U.S.: adults compliance with the Physical Activity Guidelines for Americans. *American Journal of Preventive Medicine, 40*(4), 454-461. doi: 10.1016/j.amepre.2010.12.016

Turk, D. C., & Okifuji, A. (2002). Psychological factors in chronic pain: evolution and revolution. *Journal of Consulting and Clinical Psychology, 70*(3), 678-690.

U.S. Department of Health and Human Services. (2000). *Healthy People 2010*. McLean [VA]: International Medical Publishing.

U.S. Department of Health and Human Services. (2012). *Preventing Tobacco Use Among Youth and Young Adults: A Report of the Surgeon General*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention, and Health Promotion, Office on Smoking and Health, downloaded January 29, 2012 from http://www.cdc.gov/tobacco.

U.S. Department of Health and Human Services Office of Disease Prevention and Health Promotion. (2010). *Healthy People 2020*. Washington, DC: The Department, downloaded June 8, 2012 from http://www.healthypeople.gov/2020/topicsobjectives2020/default.aspx.

UCLA Statistical Consulting Group. (2015). Chapter 2 - Regression diagnostics *SPSS Web Books: Regression with SPSS*. Los Angeles: University of California Los Angeles. Retrieved July 24, 2015 from http://www.ats.ucla.edu/stat/spss/webbooks/reg/chapter2/spssreg2.htm.

United States Census Bureau. (2010). *Profile of General Population and Housing Characteristics: 2010*. Washington, DC United States Census Bureau. Retrieved December 16, 2012 from http://factfinder2.census.gov/faces/nav/jsf/pages/index.xhtml.

United States Department of Health and Human Services. (2008). *2008 Physical Activity Guideline for Americans*. Washington, DC: United States Department of Health and Human Services. Retrieved June 20, 2015 from http://www.health.gov/paguidelines/pdf/paguide.pdf.

Urdang, Esther. (2002). *Human behavior in the social environment : interweaving the inner and outer worlds*. New York: Haworth Social Work Practice Press, p. 65.

Von Korff, M., Dworkin, S. F., & Le Resche, L. (1990). Graded chronic pain status: an epidemiologic evaluation. *Pain, 40*(3), 279-291.

Waddell, G. (1987). 1987 Volvo award in clinical sciences. A new clinical model for the treatment of low-back pain. *Spine (Phila Pa 1976), 12*(7), 632-644.

Waddell, G. (1992). Biopsychosocial analysis of low back pain. *Baillieres Clinical Rheumatology, 6*(3), 523-557.

Wai, E. K., Rodriguez, S., Dagenais, S., & Hall, H. (2008). Evidence-informed management of chronic low back pain with physical activity, smoking cessation, and weight loss. *Spine J, 8*(1), 195-202. doi: S1529-9430(07)00917-5 [pii]10.1016/j.spinee.2007.10.024

Waterman, B. R., Belmont, P. J., Jr., & Schoenfeld, A. J. (2012). Low back pain in the United States: incidence and risk factors for presentation in the emergency setting. *Spine J, 12*(1), 63-70. doi: 10.1016/j.spinee.2011.09.002

Weed, D. L. (2001). Theory and practice in epidemiology. *Annals of the New York Academy of Sciences, 954*, 52-62.

Weiner, B. K. (2008). Spine update: the biopsychosocial model and spine care. *Spine (Phila Pa 1976), 33*(2), 219-223. doi: 10.1097/BRS.0b013e3181604572

Wilson, L. M., Avila Tang, E., Chander, G., Hutton, H. E., Odelola, O. A., Elf, J. L., . . . Apelberg, B. J. (2012). Impact of tobacco control interventions on smoking initiation, cessation, and prevalence: a systematic review. *J Environ Public Health, 2012*, 961724. doi: 10.1155/2012/961724

Woolf, A. D., Erwin, J., & March, L. (2012). The need to address the burden of musculoskeletal conditions. *Best Pract Res Clin Rheumatol, 26*(2), 183-224. doi: S1521-6942(12)00036-8 [pii]10.1016/j.berh.2012.03.005

Woolf, A. D., Vos, T., & March, L. (2010). How to measure the impact of musculoskeletal conditions. *Best practice & research. Clinical rheumatology, 24*(6), 723-732. doi: 10.1016/j.berh.2010.11.002

World Health Organization. (2013). *WHO Report on the Global Tobacco Epidemic, 2013*. Geneva: World Health Organization. Retrieved January 20, 2014 from http://apps.who.int/iris/bitstream/10665/85380/1/9789241505871_eng.pdf?ua=1.

Young, E. E., Lariviere, W. R., & Belfer, I. (2011). Genetic basis of pain variability: recent advances. *Journal of Medical Genetics*. doi: 10.1136/jmedgenet-2011-100386