

2022

Predictors of Social and Psychological Functioning Among Participants in the COPDGene Cohort

Shandi Watts
Walden University

Follow this and additional works at: <https://scholarworks.waldenu.edu/dissertations>



Part of the [Psychology Commons](#)

This Dissertation is brought to you for free and open access by the Walden Dissertations and Doctoral Studies Collection at ScholarWorks. It has been accepted for inclusion in Walden Dissertations and Doctoral Studies by an authorized administrator of ScholarWorks. For more information, please contact ScholarWorks@waldenu.edu.

Walden University

College of Social and Behavioral Sciences

This is to certify that the doctoral dissertation by

Shandi Watts

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. William Tetu, Committee Chairperson, Psychology Faculty

Dr. Karen Gil, Committee Member, Psychology Faculty

Dr. Stephen Rice, University Reviewer, Psychology Faculty

Chief Academic Officer and Provost
Sue Subocz, Ph.D.

Walden University
2021

Abstract

Predictors of Social and Psychological Functioning Among Participants in the

COPDGene Cohort

by

Shandi Watts

MS, Walden University, 2015

BS, Capella University, 2013

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

In Research Psychology

Walden University

February 2022

Abstract

This study addressed the relationship among longitudinal smoking status, depression, anxiety, health-related quality of life, and psychological and social functioning due to airflow limitations among adult patients with COPD who are enrolled in the COPDGene Cohort Study. The purpose of this research was to address the gap in the literature concerning the relationship among these variables at two time points, 5 years apart. A cognitive approach was used suggesting that as disease progresses and physical ability becomes more limited, then depression and anxiety will increase, and quality of life will decrease. This was a quantitative, longitudinal, nonexperimental study using archived data from the COPDGene cohort. Research Questions 1 through 5 were designed to assess if there was a significant change in anxiety, depression, health-related physical and mental health quality of life and psychological and social functioning due to airflow limitations between the time points in four, separate smoking status groups using paired *t* tests; Research Question 6 was designed to assess the relationship among these variables using regression. The results showed, (a) significant change in anxiety scores for the Stable Former smokers'; (b) significant change in the PCS of health-related quality of life in the Current to Former smokers'; (c) significant change in psychological and social functioning due to airflow limitations for the Stable Former and the Former to Current smokers'; and (d) anxiety, depression, and health-related quality of life statistically significantly predicted social functioning due to airflow limitations. This study will lead to positive social change by bringing awareness to the challenges that COPD patients face.

Predictors of Social and Psychological Functioning Among Participants in the

COPDGene Cohort

by

Shandi Watts

MS, Walden University, 2015

BS, Capella University, 2013

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

In Research Psychology

Walden University

February 2022

Acknowledgments

I would like to thank my dissertation committee, Dr. Tetu, Dr. Rice, Dr. Gil, and Dr. Wowra, for their patience, encouragement, and mentorship. Two of the senior investigators in COPDGene, Dr. Regan and Dr. Make, for allowing me to be a part of something so big and teaching me so much along the way. Most of all, I want to thank my family, Nickolas, Brandon, Isabella, and Julie, for enduring my hours of homework and stresses along the way; you four have been my pillars of strength, and ultimate inspiration through this long process and I could not have gotten this far without your support.

Table of Contents

Contents	
List of Tables	vi
List of Figures	vii
Chapter 1: Introduction to the Study.....	1
Introduction.....	1
Background of the Study	2
Problem Statement.....	3
Purpose of the Study	5
Research Questions and Hypotheses	6
Framework	8
Nature of the Study.....	9
Definitions.....	10
Assumptions.....	13
Assumptions of the Statistical Analysis.....	13
Scope and Delimitations	14
Limitations	16
Significance.....	17
Summary	18
Chapter 2: Literature Review	20
Introduction.....	20
Literature Search Strategy.....	21

Theoretical Framework.....	22
Types of Disease.....	23
COPD.....	23
In Relation to Disease Type.....	24
Physiology.....	24
History of COPD.....	25
Causes.....	26
Genetics and Sex.....	27
Treatment.....	28
Smoking and Health Status.....	29
Anxiety and Depression.....	30
How Depression and Anxiety are Measured.....	31
Depression.....	32
Construct.....	32
Etiology.....	32
Diseases and Outcomes.....	33
COPD and Depression.....	35
Anxiety.....	36
Construct.....	36
Etiology.....	37
Diseases and Outcomes.....	37
COPD and Anxiety.....	39

HADS.....	40
History.....	40
Psychometric Properties.....	40
Measurements	41
SF-36.....	41
History.....	41
Psychometric Properties.....	42
How Quality of Life is Measured	42
Quality of Life and COPD	42
SGRQ.....	43
History.....	43
Psychometric Properties.....	44
Measurements	44
Impacts Domain: Disturbances in Social and Psychological Functioning	44
Methodology.....	45
Quantitative Survey Research.....	45
Longitudinal Research	46
Nonexperimental, Observational Research.....	47
Data Analysis.....	47
Paired <i>T</i> Tests.....	47
Multiple Regression.....	48
Conclusion	48

Chapter 3: Research Method.....	50
Purpose of the Study	50
Research Questions.....	50
Research Design.....	52
Sample.....	53
Instrumentation	54
Role of the Researcher	57
Data Analysis Plan.....	57
Threats to Validity	58
Security and Ethics	58
Data Coordinating Center	58
Physical Security-Computer Room.....	59
Database Security.....	59
Web Applications.....	59
Data Collection and Handling.....	60
Ethics and Participant Safety	60
Summary.....	61
Chapter 4: Results.....	62
Introduction.....	62
Data Collection	65
Data Entry and Cleaning.....	66
Data Analysis.....	66

Descriptive Statistics.....	66
Statistical Assumption	67
Analysis of Results	76
HADS.....	76
SF-36.....	80
SGRQ.....	84
Summary.....	89
Chapter 5.....	89
Purpose of the Study.....	89
Interpretation of Findings	90
Limitations of Study	92
Recommendations for Future Research.....	93
Implications for Positive Social Change.....	93
Summary.....	94
References.....	95

List of Tables

Table 1 <i>Descriptive Statistics</i>	67
Table 2 <i>Collinearity</i>	75
Table 3 <i>Casewise Diagnostics^a</i>	75
Table 4 <i>Changes in Mean Anxiety Scores between P2 and P3</i>	77
Table 5 <i>Changes in Mean Depression Scores between P2 and P3</i>	79
Table 6 <i>Changes in Mean PCS Scores between P2 and P3</i>	81
Table 7 <i>Changes in Mean MCS Scores between P2 and P3</i>	83
Table 8 <i>Changes in Mean Impact Scores between P2 and P3</i>	85
Table 9 <i>Regression Coefficients and Standard Errors</i>	88

List of Figures

Figure 1 <i>The Test of Normality Run Using the Shapiro-Wilk Test on the Anxiety Variable.....</i>	67
Figure 2 <i>Test of Normality Run Using the Shapiro-Wilk Test on the Depression Variable</i>	68
Figure 3 <i>Test of Normality Run Using the Shapiro-Wilk Test on the MCS Variable</i>	69
Figure 4 <i>Test of Normality Run Using the Shapiro-Wilk Test on the PCS Variable</i>	69
Figure 5 <i>Test of Normality Run Using the Shapiro-Wilk Test on the Psychological and Social Functioning Due to Airflow Limitations Variable</i>	70
Figure 6 <i>Partial Regression Plot: Anxiety and Impact Score</i>	71
Figure 7 <i>Partial Regression Plot: Depression and Impact Score.....</i>	71
Figure 8 <i>Partial Regression Plot: MCS and Impact Score.....</i>	72
Figure 9 <i>Partial Regression Plot: PCS and Impact Score.....</i>	72
Figure 10 <i>Partial Regression Plot: Smoking Status and Impact Score</i>	73
Figure 11 <i>Partial Regression Plot: Anxiety, Depression, MCS, PCS, Smoking Status, and Impact Score</i>	73
Figure 12 <i>Simple Scatter of Studentized Residual by Unstandardized Predicted Value</i>	74
Figure 13 <i>Normal Distribution</i>	76

Chapter 1: Introduction to the Study

Introduction

Patients with chronic obstructive pulmonary disease (COPD) report a decreased quality of life and psychological and physical distress (Mi Jang et al., 2018). The two most common comorbidities in COPD are anxiety and depression (Doyle et al., 2013). When comparing those without COPD and those with COPD, those with COPD showed depression was more prevalent (Hanania et al., 2011). It is estimated that one in four patients with COPD has persistent depressive symptoms (Yohannes et al., 2016).

COPD exacerbations are a regression, or “flare-up” of COPD symptoms (American Thoracic Society, 2018). Exacerbations of COPD are the third largest cause of emergency room visits (Pooler & Beech, 2014). It has been found that when these exacerbations occur, the patient has increased symptoms of anxiety and depression (Pooler & Beech, 2014). One of my goals for this study was to determine if those symptoms of anxiety and depression remain over time.

Anxiety and depression are two separate ailments but are often linked together. Depression and anxiety have a bidirectional relationship where depression may be both a cause and an effect of anxiety (Kinser et al., 2012). Anxiety involves repeated episodes of intense fear, terror, stress, or nervousness that is known to peak within minutes (Mayo Clinic, 2019). With depression comes feelings of sadness and can keep the sufferer from enjoying life (Mayo Clinic, 2020). High anxiety and depression will decrease a person’s overall quality of life.

The human brain will moderate the effects of stress to sustain optimal performance (Dhabhar, 2018). The regulatory functions that take place to try to maintain the ideal performance enhance the individual's responses to the stressors and the ability to manage negative effects (Epel, 2009). When these stressors persist without being relieved, the same regulatory functions begin to impair neural function and other regulatory systems; this wear can cause depression (Kinser & Lyon, 2014).

In this chapter, I will discuss the study's background, problem statement, and purpose. The research questions and hypotheses, theoretical framework, the nature of the study, definitions, assumptions, limitations, and delimitations of the study are included followed by the potential contributions and significance of this study.

Background of the Study

COPDGene is a multicenter, observational study that began in January 2008 (Regan et al., 2010). The study was designed to identify genetic factors associated with COPD. At its start, the study enrolled over 10,000 subjects across 21 sites located throughout the United States. Among the physical assessments, the participants were also administered self-reported questionnaires (COPDGene, 2019). As the study grew, so did the scope of the project. 5 years after the initial study began, the investigators called back those whom they enrolled in Phase 1 (P1) for another visit, this time adding to the battery of assessments to obtain more information in the Phase 2 (P2) of the study. Phase 3 (P3) began in 2018, a full decade after the initial start.

Problem Statement

The problem I addressed in this study was the gap in the literature that addresses the relationship among longitudinal smoking status, depression (measured with the Hospital Anxiety and Depression Scale [HADS]), anxiety (measured with the HADS), health-related quality of life (measured with the Short Form 36 [SF-36]) and psychological and social functioning due to airflow limitations (measured with the St. George's Respiratory Questionnaire [SGRQ]) among patients enrolled in the COPDGene Cohort Study.

COPD is a term used to describe progressive lung diseases such as chronic bronchitis, refractory asthma, and emphysema (COPD Foundation, 2018). COPD is incurable, but treatable. With a diagnosis of COPD, a patient may have symptoms of anxiety and depression and decreased health and quality of life. While these symptoms have been documented, estimates of the prevalence over time vary (Hill et al., 2008). Anxiety and depression are shown to correlate with pulmonary-specific COPD symptoms (Doyle et al., 2013). Regardless of if anxiety and depression being reported together or separate, they affect the patient's health-related quality of life and contribute to the physical disability and the financial burden of COPD (Hill et al., 2008).

It is important to determine if anxiety and depression increase over time in COPD patients; if these symptoms do become worse, this could further complicate the disease progression. It was found that there is a possible casual effect of depression on hospitalizations and COPD exacerbations (Xu et al., 2008). This suggests that depression can contribute to a COPD patient being more prone to experience exacerbations or being

required to enter hospitalization. Anxiety is shown to correlate with pulmonary-specific COPD symptoms. Anxiety is associated with higher levels of shortness of breath, fatigue, and frequency of COPD symptoms (Doyle et al., 2013). When the symptoms of anxiety and depression go unmanaged, there is a negative impact on functional capacity and quality of life in COPD patients (Lee et al., 2016). While there have been studies conducted that examine anxiety, depression, and quality of life in COPD, they focus on short-term differences. For example, Dowson et al. (2001) administered the HADS during Day 3 of hospitalization and Day 60 (discharge) of hospitalization. In this study, I looked at the difference over a 5-year span in the first 983 participants of the COPDGene cohort (COPDGene, 2019) with the first administration of HADS being between 2013 and 2014 in P2 and the second administration being between 2018 and 2019 in P3; HADS was not administered in P1. P. Mahesh et al. (2018) administered the SF-36 to COPD patients two times over the course of that 1 year; COPDGene coordinators administered the SF-36 in P2 and P3 with the time between being 5 years apart. With other studies having a focus on short-term differences, the longitudinal smoking status of the COPD patients is not taken into consideration (i.e., stable current smokers, current to former smokers, stable former smokers, and former to current smokers). I looked at the change between P2 and P3 in anxiety, depression, health-related quality of life and psychological and social functioning due to airflow limitations in these four smoking status groups.

There are studies that have determined a difference in overall quality of life based on specific treatments, but those studies were focused on the treatment itself instead of

the patient (von Leupoldt, Taube, Lehmann, Fritzsche, & Magnussen, 2011), (Cannon, et al., 2016), (Janssen, et al., 2010), (Bratås, Espnes, Rannestad, & Walstad, 2010). There are many types of COPD treatments; however, not every treatment will have the same affects for every patient; this study was aware that these patients have been living with the disease for multiple years and have experienced many treatment types. One portion in the SGRQ concerns the treatment the patient was receiving; not all subjects have received the same treatment.

Purpose of the Study

The purpose of this research was to address the gap in the literature concerning the relationship among longitudinal smoking status, depression, anxiety, health-related quality of life and psychological and social functioning due to airflow limitations among patients enrolled in the COPDGene Cohort Study. I measured depression and anxiety with the HADS; I measured health-related quality of life with the two components of the SF-36 (the physical health component score [PCS], and the mental health component score, [MCS]), and I measured psychological and social functioning due to airflow limitations with the SGRQ. I examined if there was a change over time in these variables with a paired samples *t* test and the relationship among the variables was assessed using multiple regression analysis.

In this quantitative, longitudinal, nonexperimental study, I used archived data from the COPDGene cohort (COPDGene, 2019) to: (a) identify if there was a change in anxiety and depression scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to

current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; (b) identify if there was a change in health-related quality of life scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; (c) identify if there was a change in psychological and social functioning due to airflow limitations in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; and (d) identify if there was a relationship at P3 between longitudinal smoking status, anxiety, depression, health-related quality of life, and physical and social functioning due to airflow limitations.

Research Questions and Hypotheses

Research Question 1 (RQ1): Is there a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups?

Alternative Hypothesis (H_11): There is a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups.

Null Hypothesis (H_01): There is no significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups.

Research Question 2 (RQ2): Is there a significant change in depression between P2 and P3 in the four longitudinal smoking status groups?

Alternative Hypothesis (H_12): There is a significant change in depression between P2 and P3 in the four longitudinal smoking status groups.

Null Hypothesis (H_02): There is no significant change in depression between P2 and P3 in the four longitudinal smoking status groups.

Research Question 3 (RQ3): Is there a significant change in PCS between P2 and P3 in the four longitudinal smoking status groups?

Alternative Hypothesis (H_13): There is a significant change in PCS between P2 and P3 in the four longitudinal smoking status groups.

Null Hypothesis (H_03): There is no significant change in PCS between P2 and P3 in the four longitudinal smoking status groups.

Research Question 4 (RQ4): Is there a significant change in MCS of health-related quality of life between P2 and P3 in the four longitudinal smoking status groups?

Alternative Hypothesis (H_14): There is a significant change in MCS between P2 and P3 in the four longitudinal smoking status groups.

Null Hypothesis (H_04): There is no significant change in MCS between P2 and P3 in the four longitudinal smoking status groups.

Research Question 5 (RQ5): Is there a significant change in psychological and social functioning due to airflow limitations (measured with the SGRQ) between P2 and P3 in the four longitudinal smoking status groups?

Alternative Hypothesis (H_15): There is a significant change in SGRQ between P2 and P3 in the four longitudinal smoking status groups.

Null Hypothesis (H_05): There is no significant change in SGRQ between P2 and P3 in the four longitudinal smoking status groups.

Research Question 6 (RQ6): Is there a relationship between longitudinal smoking status, anxiety, depression, health-related quality of life (PCS and MCS), and psychological and social functioning due to airflow limitations measured with the SGRQ at P3?

Alternative Hypothesis (H_16): There is a relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36 psychological and social functioning due to airflow limitations measured with the SGRQ.

Null Hypothesis (H_06): There is no relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36 psychological and social functioning due to airflow limitations measured with the SGRQ.

Framework

The problem was that there was a gap in the literature that addresses the relationship among longitudinal smoking status, depression, anxiety, health-related quality of life, and psychological and social functioning due to airflow limitations (measured with the SGRQ) among patients enrolled in the COPDGene Cohort Study is grounded in the literature because it examines people's self-reported anxiety, depression, quality of life, and disturbances in social and psychological functioning through the cognitive theory. Cognitive theory is the assumption that thoughts, opinions, or perceptions are determinants of emotions and behaviors (Clark et al., 1999). Anxiety and depression affect people's physical and emotional symptoms and physical and emotional

distress are thought to be associated with higher levels of anxiety and/or depression (Kinser et al., 2012). This suggests that as disease progresses and the person's physical ability becomes more limited, then depression and anxiety will increase, and quality of life will decrease.

Nature of the Study

I used a quantitative, longitudinal, nonexperimental design using archived data from COPDGene. I used paired *t* tests with alpha adjusted to 0.01 to address RQ1 through RQ5 and I used regression to address RQ6.

The problem that I examined in this study was original because, until now, there has not been another study conducted that has looked at difference in anxiety and depression and difference in quality of life over multiple years in adult COPD patients. Other studies use the HADS and/or the SF-36 on one visit to determine how the patient feels at that time. I looked at the relationship between anxiety and depression scores and the difference in quality of life over the course of 5 years in adult patients with COPD with the first administration of HADS and SF-36 being between 2013 and 2014 in P2 and the second administration being between 2018 and 2019 in P3.

I examined differences in symptoms ($n=983$) of anxiety and depression and overall quality of life in smokers (≥ 10 pack-years) in the COPDGene cohort who were grouped by a change in their longitudinal smoking status over a 5-year period were examined. (i.e., stable current smokers, current to former smokers, stable former smokers, and former to current smokers).

The problem was amenable to scientific study because investigators can determine how a person with COPD rates their anxiety, depression, quality of life, and disturbances in social and psychological functioning after living with COPD for a minimum of 5 years and if during those 5 years their smoking status changed. This will help medical providers better understand the importance of treating the secondary symptoms of COPD to help alleviate some of the primary symptoms.

Definitions

Anxiety: “Anxiety is an emotion characterized by feelings of tension, worried thoughts, and physical changes like increased blood pressure” (American Psychological Association, 2020). Anxiety can cause an individual to avoid certain situations and can lead to physical symptoms like dizziness, rapid heartbeat, and sweating (American Psychological Association, 2020)

Bodily Pain: This is defined as a generalized, unpleasant bodily sensation that causes mild to severe physical distress that usually results from a disorder, injury, or disease (Merriam-Webster Dictionary, 2020).

COPD: Chronic obstructive pulmonary disease is a disease of the lungs that is characterized by chronic obstruction of the airflow in the lung that will interfere with normal breathing and is not fully reversible (World Health Organization, 2020).

Current to Former Smokers: Those who were smokers at P2 but quit smoking before P3.

Depression: Depression makes people have a lack of pleasure or interest in regular activities; it can cause weight loss or weight gain, insomnia, or excessive

sleeping, inability to concentrate, and can give the sufferer feelings of worthlessness, excessive guilt, and thoughts of death or suicide (American Psychological Association, 2020)

Former to Current Smokers: Those who were not smokers at P2 but began smoking before P3.

HADS: Hospital Anxiety and Depression Scale was designed to provide a simple, reliable tool to screen anxiety and depression in patients (Snaith, 2003). This questionnaire has 14 items that the patient answers on a 4-point scale. Seven items relate to anxiety and the remaining seven relate to depression. The final tally of anxiety and depression will result in a numerical score that indicates the patient is either mildly, moderately, or severely affected by either anxiety, depression, or both (Stern, 2014).

Mental Health: Mental health is an individual's state of well-being in which they can realize their own abilities in coping with normal life stresses and are able and willing to contribute to their community (World Health Organization, 2018).

Physical Functioning: Activities considered crucial for sustaining independence, and those considered optional that are not required for independent living but may impact quality of life (Painter et al., 1999).

Role Activities: The customary or normal activity of an individual in a setting (Vocabulary.com, 2020).

SF-36: Short Form Health Survey is a health-related quality of life measure that assesses eight health concepts: limits in physical function because of health complications; limits in social function because of physical or emotional difficulties;

limits in role activities because of physical health problems; bodily pain; general mental health; limits in role activities because of emotional problems; vitality; and general health perceptions (Gandek et al., 2004).

SGRQ: St. George's Respiratory Questionnaire was designed to help investigators learn more about how the patient's breathing is disturbing them and how it affects their quality of life (Jones et al., 1992). This questionnaire is scored in three separate domains: symptoms, activity, and impacts. For this study, the impacts domain will be analyzed. The impacts domain measures the disturbances in social and psychological functioning (Nagai et al., 2015). This section includes questions asking the participant: if their respiratory condition causes them problems, if they have ever had to stop or change their line of work because of their respiratory problems, if they get breathless when conducting everyday activities such as walking or talking; other effects such as: if the respiratory condition is embarrassing in public, a nuisance to family and friends, if the sufferer panics when they cannot catch their breath, if they feel they are not in control of their illness, they do not expect their condition to improve, if they feel frail or invalid because of their condition, and if everything seems to be too much of an effort; questions about their medication is asked: do they feel their medication is helping them, do they get embarrassed by using their medication in public, do they have unpleasant side effects from their medication, and does the medication interfere with their life; this section also asks how the illness affects their daily life, can they play sports or go out to do recreational activities, can they go shopping on their own, can they do housework, can

they move from their chair or bed; lastly, this section of the questionnaire asks if their illness stops them from doing things they enjoy doing.

Social Functioning: Interactions an individual has with their environment and the capability to accomplish their role within environments as work, social activities, and relationships (Bosc, 2000).

Stable Current Smokers: Those who were smokers at P2 and remained smokers at P3.

Stable Former Smokers: Those who were not smokers at P2 and remained non-smokers at P3.

Vitality: Vitality refers to the individual's physical strength, mental vigor, and the capacity for a meaningful existence (Dictionary.com, 2020).

Assumptions

I assumed that because the coordinators across all study sites administering the questionnaires have been trained, that they administered the questionnaires correctly. It was assumed that the data collected from the surveys is accurate and reliable. Each instrument is a self-report measure that relies on the participants to report how they feel. The participants' reports of symptoms could vary, depending upon their memory and evaluation of symptoms. I also assumed that the sample is reflective of the adult COPD population.

Assumptions of the Statistical Analysis

There was a total of 20 paired t tests to answer the first five research questions. Laerd Statistics (2018) lists the assumptions of the t test as: (a) there was one dependent

variable that was measured at the continuous level, (b) there was one independent variable that consists of two related groups, (c) there are no significant outliers in the differences between the related groups, and (d) the distribution of the differences of the dependent variable between the two related groups was normally distributed. The third and fourth assumptions were tested using SPSS with the third assumption tested using a boxplot and the fourth assumption with the Shapiro-Wilk test for normality.

I analyzed the sixth research question using multiple regression. Laerd Statistics (2018) lists the assumptions for multiple regression as: (a) the dependent variable was measured on a continuous scale, (b) there are two or more independent variables, (c) there was an independence of observations, (d) there was linear relationship between the dependent variable and each independent variable, (e) the data needs to show homoscedasticity, (f) the data must not show multicollinearity, (g) there should be no significant outliers, and (h) check that the residuals are normally distributed (Laerd Statistics, 2018). The first 2 assumptions were checked first. The third assumption was checked using the using the Durbin-Watson statistic, the fourth assumption was checked by creating scatterplots and partial regression plots, the fifth, sixth, and seventh assumptions were checked in SPSS Statistics, and the eighth assumption was checked with a histogram and a Normal P-P Plot.

Scope and Delimitations

In this study, I (a) identified if there was a change in anxiety and depression scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the

COPDGene cohort during the 5-year period between P2 and P3; (b) identified if there was a change in health-related quality of life scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; (c) identified if there was a change in psychological and social functioning due to airflow limitations in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; and (d) identified if there was a relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36 psychological, and social functioning due to airflow limitations measured with the SGRQ.

The participants were limited to those who were already enrolled in COPDGene. The data collection was delimited to adult respondents because the age for enrolling into COPDGene was 45-80 years old.

Due to aging, this population may be susceptible to memory or cognitive impairment (Small, 2002). It is possible that memory or cognitive impairment may have influenced the results of this study; however, it cannot be assumed that all the aging population has these mental health issues.

The data collection took place in face-to-face (F2F) interviews. A F2F survey is when an interviewer asks the respondent the questions on the survey and records the answers. One advantage to F2F data collection is the researchers' ability to explain the

questions to the participant. Unfortunately, the response rate with F2F is not as robust as online for those studies that are researching topics that are considered taboo or controversial (Liu & Wang, 2016). The nonresponse rate in F2F surveys is higher than in online surveys (Zhang et al., 2017). In some situations, the participant will simply answer the questions in a way that will make themselves sound better, will answer with how they think the researcher wants them to answer, or will refuse to answer the question at all. The reason the F2F method was used is because other datapoints were collected that are not being used in the present study that require the in-person contact; for example, a CT scan and blood draw was part of the research visit, among other procedures that cannot be completed online. Another reason for the F2F interview is because this aging population sample may not have the technology or knowledge to navigate an online survey.

This study will not look at the P1 data. During P1 the HADS questionnaire was not administered.

Limitations

COPDGene has an Ancillary Studies and Publications Committee that decides if an investigator is allowed access to COPDGene data and to ensure there is no overlapping of projects and publications within COPDGene. This committee meets twice a month on the first and third Thursdays. Once the data request is reviewed, the committee decides if the investigator is allowed access or if the request is deferred pending further information needed. This study has gone through all necessary reviews and was approved to move forward.

There are limitations to consider when using self-report questionnaires. Self-reported measures showed greater concordance for monthly compared to yearly metrics (Short et al., 2009). This means that the accuracy of the answers provided on the self-report measure decreases as the recall period increases. Also, the accuracy of self-report measures as quantitative measures has not been well documented (Tommelein et al., 2014).

Collection of HADS data ceased when the quarantine portion of the COVID-19 pandemic began in March 2020. COPDGene stopped in-person visits to protect both the subjects and staff. Some questionnaires were completed over the phone, but because the HADS is considered sensitive and could require medical follow-up, this measure was postponed until in-person visits began once more and medical care providers were back in their offices so the coordinators can inform them of any alarming scores.

Significance

Regardless of COPD being the fourth primary cause of death in the United States, there is a lack of awareness of the disease by the public (Sayiner et al., 2012). This lack of awareness spans in part, on the patients, as well as health care professionals. While the consensus shows that patients believe their providers are committed to help, there is a less favorable attitude relating to whether effective treatments exist (Sayiner et al., 2012). Not understanding if their disease can be treated in an effective way can cause a great deal of anxiety and depression among patients which, in turn, can lead to more disease complications (Xu et al., 2008).

Bridging the gap of knowledge about a long-term difference in anxiety, depression, and quality of life and how those relate to the patient's longitudinal smoking status and if a difference in anxiety, depression, and health-related quality of life relates to the patient's social and psychological functioning can aid healthcare professionals in determining a proper educational intervention to help these patients understand their disease and treatments better to prevent further complications to their disease by tracking and alleviating symptoms before they start. Tracking anxiety, depression, and quality of life long-term can present the healthcare provider with patterns of the patients' triggers to better treat them.

The findings of this research will lead to positive social change by bringing awareness to the healthcare providers of the challenges that COPD patients face in relation to current management and mental health issues concerning their disease.

Summary

COPD continues to be a leading cause of death in the United States (Sayiner et al., 2012). The comorbid conditions may increase the exacerbations a person experiences leading to further disease complications. If these comorbid conditions can be identified and treated, doctors may be able to slow-down disease progression.

Chapter 2 contains a review of the literature. COPD with depression, COPD with anxiety, COPD, and limits in physical function because of health complications, limits in social function because of physical or emotional difficulties, limits in role activities because of physical health problems, bodily pain, general mental health, limits in role activities because of emotional problems, vitality, and general health perceptions will be

discussed. A review of the brain's ability to moderate the effects of stress is presented.

The literature review includes studies on COPD, HADS, SF-36, SGRQ, and COPD treatments.

Chapter 2: Literature Review

Introduction

With a diagnosis of COPD, a patient may have feelings of anxiety and depression and a decreased quality of life. While these feelings have been documented, estimates of the prevalence over time vary considerably (Hill et al., 2008). Anxiety and depression are shown to correlate with pulmonary-specific COPD symptoms (Doyle et al., 2013). It is important to determine if anxiety and depression worsen over time in COPD patients; if worsening does occur, this could further complicate the disease progression. The purpose of this research was to address the gap in the literature concerning the relationship among longitudinal smoking status, depression, anxiety, health-related quality of life and psychological and social functioning due to airflow limitations, among participants enrolled in the COPDGene Cohort Study.

This chapter begins with a discussion of the strategies that I used to research the literature followed by a discussion about the theoretical model of the cognitive approach. I discuss the model's origins and assumptions along with recent research related to the model. In the third section, I describe the classifications of diseases and where COPD fits into those classifications. The fourth section contains a review of literature on COPD, its physiology, history, causes, symptoms, prevalence, treatment, and public awareness as well as how genetics and sex affect the disease. In the fifth section I describe smoking and its effects of health status. In the sixth section I give an overview of anxiety and depression and how these are measured. In the seventh section I describe depression's construct, etiology, and outcomes with the eighth section delving into COPD with

depression. The ninth section includes information about anxiety with descriptions of the construct, etiology, and outcomes with Section 10 containing information about COPD with anxiety. Sections 11 and 12 describe the HADS and SF-36 questionnaires that I used in this study. Section thirteen describes COPD and quality of life; section fourteen describes the SGRQ questionnaire that I used for this study.

Literature Search Strategy

I conducted a literature search using multiple resources and various databases, experts on COPD, anxiety, and depression, and a list of search terms. I searched databases such as MEDLINE, CINAHL Plus, Directory of Open Access Journals, Science Citation Index, Science Direct, ProQuest, and Psychology Databases Combined Search to locate peer-reviewed articles; I searched psychology, respiratory, and medical journals, magazines, and books from the Walden University library and other online, open-access sites to complete this review. I searched the websites American Thoracic Society, COPD Foundation, COPDGene Study, National Heart, Lung, and Blood Institute, National Institute of Mental Health, American Lung Association, the World Health Organization, Center for Disease Control, and the Mayo Clinic were consulted to complete this literature review. I limited search years for studies conducted on the topic were 2010 to 2020; when literature could not be found the search was extended back to 1990.

I used the following search terms and phrases: *cognitive framework, types of diseases, categories of diseases, COPD, COPDGene, COPD and anxiety, COPD and depression, depression, disease types and depression, anxiety, disease types and anxiety,*

disease types and quality of life, HADS, SF-36, COPD and SF-36, SGRQ, and COPD treatments.

Theoretical Framework

I used a cognitive approach for this study. A cognitive approach focuses on people's beliefs (Stenling et al., 2014). This approach suggests that behavioral, emotional, and physical symptoms result from cognitive irregularity (Kinser & Lyon, 2014); this means that depressed patients think differently than those who are not depressed.

Anxiety and depression are two separate ailments but are often linked together. Depression and anxiety have a bidirectional relationship where depression may be both a cause and an effect of anxiety (Kinser et al., 2012). Anxiety involves repeated episodes of intense fear, terror, stress, or nervousness that is known to peak within minutes (Mayo Clinic, 2020). With depression comes feelings of sadness and can keep the sufferer from enjoying life (Mayo Clinic, 2019).

Typically, the human brain will moderate the effects of stress to sustain optimal performance. The regulatory functions that take place to try to maintain the ideal functioning enhance the individual's responses to the stressors and the ability to manage negative effects (Epel, 2009). When these stressors persist without being relieved, the same regulatory functions begin to impair neural function and other regulatory systems; this wear can cause depression (Kinser & Lyon, 2014). It has been found that patients with moderate to severe COPD exhibited changes in their brain's grey matter density and white matter integrity resulting in cognitive deficits (Yin et al., 2019).

Types of Disease

According to International Classification of Diseases, 10th Revision (ICD-10), there are 20 disease classifications: a) certain infectious or parasitic diseases, b) neoplasms, c) diseases of the blood or blood-forming organs, d) diseases of the immune system, e) endocrine, nutritional, or metabolic diseases, f) mental, behavioral, or neurodevelopmental disorders, g) sleep-wake disorders, h) diseases of the nervous system, i) diseases of the visual system, j) diseases of the ear or mastoid process, k) diseases of the circulatory system, l) diseases of the respiratory system, m) diseases of the digestive system, n) diseases of the skin, o) diseases of the musculoskeletal system or connective tissue, p) diseases of the genitourinary system, q) conditions related to sexual health, r) diseases of pregnancy, childbirth, or the puerperium, s) certain conditions originating in the perinatal period, and t) developmental anomalies (World Health Organization, 2020). COPD would be classified into the diseases of the respiratory system category.

COPD

COPD is a term used to describe progressive lung diseases such as chronic bronchitis, refractory asthma, and emphysema (COPD Foundation, 2018). COPD can be treated, but it cannot be cured. It is the fourth leading cause of death in the United States with exasperations from the disease being the third largest cause of emergency room visits (Pooler & Beech, 2014).

In Relation to Disease Type

As mentioned, COPD is grouped into the diseases of the respiratory system category according to ICD-10. COPD is coded as: COPD, with acute lower respiratory infection, COPD, with acute exacerbation, COPD, unspecified, which includes COPD with asthma, chronic bronchitis, emphysema, or chronic obstructive asthma (Nicolacakis, 2015). Additional codes can be used to identify a patient's history of tobacco use, current tobacco use, and tobacco dependence (Nicolacakis, 2015). ICD-10 codes are used by physicians, nurses, and medical coders for a variety of purposes to include statistics and billing and claims reimbursement; they are also used to aid in surveillance and research activities (Center for Disease Control and Prevention, 2015).

Physiology

COPD is characterized by airflow obstruction that cannot be reversed and an inflammatory response in the lungs (O'Donnell et al., 2015). All cigarette smokers and those who are exposed to noxious particles and gases have inflammation in their lungs; those who develop COPD have an enhanced response to inhaling the toxic agents (MacNee, 2006). The enhanced responses can result in chronic bronchitis, emphysema, and bronchiolitis (MacNee, 2006).

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) developed a four-stage classification of COPD severity (Safka et al., 2017). Patients are categorized based on airflow limitation (FEV_1), symptoms (using modified Medical Resource Council dyspnea scale or COPD Assessment Test score), and exacerbation history (Safka

et al., 2017). When a patient is put into a category, there are guidelines for treatment to aid the medical practitioner in treating that specific category.

COPD is not a single disease but is more of a syndrome that is defined as a set of symptoms that commonly occur together and related to each other (Agustí & Celli, 2017). Symptoms of COPD include difficulty breathing, mucus production, wheezing, and a persistent cough (Mayo Clinic, 2020). COPD-affected lungs are like aging lungs; COPD has been viewed as an accelerated premature aging of the lungs (Cho et al., 2019). People with COPD will also likely experience exacerbations; these are episodes where their symptoms become worse than the usual day-to-day challenges and usually last for several days.

Divo et al. (2012) prospectively evaluated comorbidities and mortality risk in COPD patients. Twelve comorbidities were found to negatively influence survival. These are: pulmonary fibrosis, congestive heart failure, lung cancer, coronary artery disease, A. fibrillation, anxiety, breast cancer, esophageal cancer, liver cirrhosis, pancreatic cancer, gastric duodenal ulcer, and diabetes with neuropathy (Divo et al., 2012).

History of COPD

COPD dates to the 1600s. Theophile Bonet, a Swiss-born physician, performed more than 3,000 autopsies on his patients identifying those patients with emphysema had larger lungs than those who did not have the disease (Plank, 2018). During the 1800s, Dr. Rene Laënnec identified air pollution and genetic factors to be causes of COPD; he was the first to compare the relationship between chronic bronchitis and emphysema to COPD (Petty, 2006). In 1846, the spirometer was invented (Plank, 2018). The spirometer is an

essential tool used in the diagnosis of COPD; the spirometer measures FEV₁. The full list of components to diagnose COPD was developed in 1959, but the term COPD was not coined until the 1960s (Petty, 2006). In the 1970s, it was discovered that stopping smoking helps to slow the progress of COPD while smoking will further accelerate the disease (Plank, 2018).

Causes

COPD is caused by long-term exposure to lung irritants that damage the lungs and airways (American Lung Association, 2020). The most common irritant in the United States is cigarette smoke; there are more than 7,000 chemicals created when a cigarette burns; these toxins weaken lungs' defense against infections which causes swelling in the air tubes, destroying the air sacs which contributes to COPD (American Lung Association, 2020). Cigarettes are not the only contributing factor to developing COPD. Cigars, pipes, and other types of tobacco smoke can cause COPD, especially when inhaled (National Heart, Lung, and Blood Institute, 2019). Environmental factors can also play a role in disease development. Long-term exposure to air pollution, second-hand smoke, dust, chemicals, and fumes can also cause COPD (American Lung Association, 2020).

Since COPD is caused by long-term exposure to irritants, COPD is more common in adults than children; however, events taking place in one's youth can influence the likelihood of contracting the disease as an adult. Childhood maltreatment was found to be a risk factor for COPD in adulthood because those who were mistreated as a child are more likely to smoke cigarettes and less likely to quit smoking (Shields et al., 2016). In

one study, it was found that those who lived with a smoker throughout their childhood had a 31% higher mortality from COPD compared to those who did not live with a smoker throughout childhood (Nogrady, 2018).

Genetics and Sex

More recent studies have found that a genetic condition called alpha-1 antitrypsin deficiency may play a role in causing COPD (National Heart, Lung, and Blood Institute, 2019). People with this condition have low blood levels of alpha-1 antitrypsin (AAT) in their liver; this low level of AAT can lead to lung damage (National Heart, Lung, and Blood Institute, 2019).

The differences between females and males with COPD are a result of sex-specific differences and gender-specific differences (Raghavan et al., 2017).

Epidemiological data indicate that the prevalence of COPD among females is now comparable to males; the number of female COPD cases continues to climb (Tsiligianni et al., 2017). Females are at an increased risk of small airway disease while males are more vulnerable to an emphysematous phenotype (Jenkins et al., 2017). Compared to men, women with COPD tend to be of a lower socioeconomic class, are younger, are more vulnerable to developing COPD, and experience a more rapid progression of the disease (Jenkins et al., 2017). Epidemiological trends are showing that mortality rates have been decreasing more quickly among men with COPD than among women (Raghavan et al., 2017).

Treatment

The key goal when selecting a treatment for COPD patients is to relieve the patient's current symptoms to reduce the burden on daily activities, to lower the risk of future exacerbations, and to prevent disease progression (Ding et al., 2017). Physicians take factors such as exacerbation history, lung function, and presenting symptoms into consideration when determining an appropriate treatment choice for COPD (Roche et al., 2019). Disease conditions vary in COPD patients so care interventions are tailored according to the disease-related factors (Ridwan et al., 2019). During the last 20 years, there has been a significant increase in the number of pharmacologic agents available for COPD patients (Roche et al., 2019). To date, the only treatment known to alter the course of COPD is long term, supplemental oxygen (Plank, 2018).

Prescribed, inhaled medication is the most common treatment method for COPD (Rogliani et al., 2017). The level of adherence to the prescribed medications is low which has a negative influence on outcomes (Rogliani et al., 2017). Factors to take into consideration before prescribing these inhaled medications are, the age and cognitive status of the patient, manual dexterity and strength, and the ability to coordinate the inhaler (Rogliani et al., 2017). On occasion, COPD patients are prescribed a different type of inhaled medication if the physician finds the first medication is not working for the patient. The switch between different types of inhalers can cause confusion for the patient as they may have to utilize different techniques to use them. This confusion will often cause the patient to use the medication incorrectly, or not at all should they become frustrated (Bosnic-Anticevich et al., 2017).

Another common treatment method used alone, or along with the prescribed, inhaled medication is breathing exercises. Diaphragmatic breathing, yoga breathing, breathing gymnastics, and singing are common treatments that have shown to improve a COPD patients' pulmonary function (Lu et al., 2020). Like the inhaled medications, the physician cannot be sure the patient is performing the exercises correctly.

With the physical ailments of COPD being at the forefront, there is a significant amount of unmet mental health needs among current and former smokers whether they have COPD or not with one in five people having symptoms that are un-medicated (Norwood, 2006). Having untreated mental health challenges jeopardizes patients' ability to utilize COPD treatment. Depression and anxiety are associated with higher levels of shortness of breath, fatigue, and frequency of COPD symptoms (Doyle et al., 2013).

There is low awareness about COPD (Sayiner et al., 2012). Although COPD is regarded as a chronic inflammatory lung disease, the disease mechanism is still unknown (Cho et al., 2019). There is documented data showing low knowledge about the disease, beliefs about COPD, treatment satisfaction, and attitudes concerning care. Better patient, and physician education are needed.

Smoking and Health Status

Habitual cigarette smoking is associated with an increased risk of numerous adverse health effects including, but not limited to, lung disease, cardiovascular disease, and premature death (Surgeon General, 2014). Over the past 50 years, smoking has decreased, however, 15.5% of the population are current smokers (Jamal et al., 2018). The major component in cigarette smoke is nicotine, which is highly addictive causing

the user to become dependent (Picciotto & Kenny, 2020). Nicotine dependence is a concept used to classify a group of symptoms including, heavy consumption of tobacco products, compulsive use of tobacco products, building a tolerance to the products, and withdrawal if a product is not used in a period of time (Frandsen et al., 2017).

Smoking cessation benefits smokers of every age (Surgeon General, 2014). Efforts to increase smoking cessation include education about the risks of smoking, behavioral modifications, nicotine replacement, and antidepressant medications (Schauer et al., 2016). A survey administered in 2015 showed that two thirds of those who were active smokers were interested in quitting with just over half having received advice of how to quit from a medical health professional (Babb et al., 2017). Fewer than one third of smokers who attempted to quit used cessation treatments that were proven to work, and fewer than one tenth of smokers were successful in quitting (Babb et al., 2017).

Anxiety and Depression

There is a link between anxiety and depression. The body of research on anxiety creation has results suggesting that depressed individuals tend to generate primarily dependent interpersonal stress (Uliaszek et al., 2012). Life stresses and anxieties is characterized in two ways: interdependent/dependent and interpersonal/non-interpersonal (Uliaszek et al., 2012). Independent stress refers to events that are beyond one's control. Dependent stress occurs because of the individual's own actions. Interpersonal stress is difficulties with family, peers, or significant others, whereas non-interpersonal stress refers to occupational, educational, and health problems (Uliaszek et al., 2012). It is suggested that a person who is depressed will also assume more stress and anxiety. The

anxiety a COPD patient experiences in relation to their disease could be either independent or dependent depending on how they contracted the disease; and could also be categorized as non-interpersonal because of their health challenges or interpersonal because those health challenges could affect their occupational choices and relationships with others.

There is also reason to believe that the effects can work in reverse: when a person is anxious it can cause depression. There is a bidirectional relationship between anxiety and depression; specifically, that in addition to the triggering effects of anxiety or depression, depressed individuals are more likely to experience subsequent stressful events that will cause anxiety (Brown & Rosellini, 2011).

Cognitive theories suggest that mental representations formed by humans play a central role in guiding their behavior (Franken, 2007). If a person becomes stressed, they may have trained themselves to become depressed because of the stress or vice versa.

How Depression and Anxiety are Measured

To measure anxiety and depression, clinicians will use either an interview, self-report measures, or both. Subjective tests, such as an interview, allows for more data to be collected that may not have been planned (Evans, 2018). One of the most used methods of data collection in the social sciences is the qualitative, semi-structured interview (Bradford & Cullen, 2012). These types of interviews allow the researcher to investigate subjective viewpoints and gather more in-depth explanations of a person's experience (Flick, 2009). This form of data collection allows for a more free-flowing conversation to happen between the interviewer and interviewee which can result in

voluntary information to be offered that was not asked. This type of data collection is also susceptible to biases that can influence the outcome of the assessment.

A self-report measure can also be administered to determine a person's anxiety and depression levels. These measures do not have the biases associated with them as subjective techniques do, however, they also do not capture the breadth of information that some situations need to make an informed diagnosis (Cucato et al., 2013). These psychiatric rating scales were developed more than four decades ago and serve to assign a numerical value to the complex range of behaviors, feelings, and affects (Anderson et al., 2002). There is a plethora of validated, self-report measures to choose from depending on what the clinician seeks to measure.

Depression

Construct

Depression is a common illness and the leading cause of disability worldwide (World Health Organization, 2018). There are more than three hundred million people affected by this illness across the globe (World Health Organization, 2018). Depression is something that most people feel at some point in their life. It can be described as feeling, sad, down in the dumps, or feeling blue. "True clinical depression is a mood disorder in which feelings of sadness, loss, anger, or frustration interfere with everyday life for weeks or longer" (A.D.A.M. Medical Encyclopedia, 2012).

Etiology

It is suggested that depression begins within a person's emotional inertia in adolescence. Adolescence is a period marked by significant psychosocial, biological, and

cognitive changes that lead to increases in emotionality, greater sensitivity to social interactions, and greater reward seeking and associated risk taking (Kuppens et al., 2012). During this time, a person's executive, and self-regulatory skills to cope with these changes mature more gradually and are not fully developed. Emotional inertia is how people respond to and regulate emotional events and is reflected in the patterns that their emotions change over time (Kuppens et al., 2012). Emotional inertia reflects that one's current emotional state is predictable from one's prior emotional state. High emotional inertia means that a person's emotional changes show a high degree of drive, with current emotional states being influenced by previous emotional states. On the other hand, low emotional inertia is the opposite, indicating that current emotional states are less predictable from previous emotional states (Kuppens et al., 2012).

Diseases and Outcomes

Major depression is more likely to occur in males; unipolar depression is more likely to occur in females (Albert, 2015). Depression is also more prevalent in young women than men, which introduces age as another biological factor (Albert, 2015).

Many patients who suffer from mood disorders such as depression never seek treatment. There has been a modest increase over the past decade in which there has been an increase in public awareness of the availability of effective treatments and there is less shame associated with mood disorders (Butcher et al., 2013). Depression can be treated with medications, psychotherapy, and electroconvulsive therapy.

There are several antidepressant medications available today. "Antidepressants are generally categorized by how they affect the naturally occurring chemicals in your

brain to change your mood” (Mayo Foundation for Medical Education and Research, 2012). Among these are: Selective serotonin reuptake inhibitors (SSRIs), serotonin and norepinephrine reuptake inhibitors (SNRIs), norepinephrine and dopamine reuptake inhibitors (NDRIs), atypical antidepressants, tricyclic antidepressants, and monoamine oxidase inhibitors (MAOIs).

In addition to medications (or in lieu of) psychotherapy may be used to treat depression. Cognitive-behavioral therapy (CBT) is “based on altering dysfunctional thoughts and cognitive distortions” (Butcher et al., 2013, p. G-4). CBT is based on the idea that thoughts cause feelings and behaviors, not external things, like people, situations, and events (National Association of Cognitive-Behavioral Therapists, 2010). One benefit of this type of therapy is that a person can change how they think about a given situation and make themselves feel better about it even if the situation does not change. In CBT, the individual controls how they feel. CBT centers on the assumption that most emotional and behavioral reactions are learned. Therefore, the goal of therapy is to help clients unlearn their unwanted reactions and to learn a new way of reacting.

Psychodynamic therapy is a “psychological treatment that focuses on individual personality dynamics, usually from a psychodynamic or psycho-dynamically derived perspective” (Butcher et al., 2013, p. G-16). “The goals of psychodynamic therapy are a client’s self-awareness and understanding of the influence of the past on present behavior” (Haggerty, 2012). A psychodynamic approach aids the client in re-visiting past unresolved issues from dysfunctional relationships that manifest symptoms in present situations.

Electroconvulsive therapy (ECT) is the third option to treat depression. In ECT, electrical currents are passed through the brain. This procedure is thought to affect levels of neurotransmitters in the brain (Mayo Foundation for Medical Education and Research, 2012).

The availability of effective depression treatment over the past decade has increased, but the number of people seeking treatment has not increased as much as one would hope. “On average, people living with depression go for nearly a decade before receiving treatment, and less than one-third of people who seek help receive minimally adequate care” (Mental Health America, 2012). About 5% of Americans ages twelve and older are depressed, and 80% say the illness interferes at some level with daily functioning (Fiore, 2008).

COPD and Depression

Major depressive disorder (MDD) is a mood disorder that causes persistent feelings of sadness and despair; it affects how you think, feel, behave, and can lead to a variety of physical and emotional issues (Mayo Clinic, 2019). MDD has a prevalence of 17% in the United States making it the most common psychiatric disorder (Heinzman et al., 2019).

Patients with COPD often report psychological distress and a decreased quality of life. When comparing those without COPD and those with COPD, those with COPD showed depression was more prevailing (Hanania et al., 2011). It is estimated that one in four patients with COPD has persistent depressive symptoms (Yohannes et al., 2016).

Disease symptoms and quality of life were shown to be more important determinants of depression than clinical and biologic measures (Hanania et al., 2011).

Iyer et al. (2019) compared the frequency depressive symptoms and the use of anxiolytic-hypnotics or anti-depressants in smokers with and without COPD, along with identification of characteristics associated with having un-medicated symptoms. It was found that depressive symptoms were more frequent in severe to very severe COPD patients (Iyer et al., 2019). Those with severe to very severe COPD had the highest use of anxiolytic-hypnotics, while there was no significant difference between smokers with and without COPD and antidepressant use.

Xu et al. (2008) studied the effect of depression on the risk of hospitalizations and COPD exacerbations. Anxiety and depression are comorbid, modifiable conditions in COPD. It was found that there is a causal effect of depression on hospitalizations and COPD exacerbations (Xu et al., 2008). This suggests that depression can contribute to a COPD patient having exacerbations or needing to be hospitalized.

Anxiety

Construct

Anxiety disorders are the most prevalent psychiatric disorders (Bandelow et al., 2017). While it is normal for the average person to occasionally have anxiety, those with an anxiety disorder will experience the anxiety more frequently, intensely, and may have a persistent worry or fear about everyday occurrences (Mayo Clinic, 2020). Some examples of an anxiety disorder include, phobias, separation anxiety, and generalized anxiety among others (Bandelow et al., 2017). Anxiety is often underrecognized and

undertreated in primary care and can sometimes be a result of a medical condition (Mayo Clinic, 2020).

Etiology

What causes anxiety disorders is not completely understood. The etiology of anxiety disorders is multifactorial with contributions from environmental and genetic influences. Those who have family members who suffer from an anxiety disorder are more likely to have an anxiety disorder themselves (Ohi et al., 2020). However, in the elderly population, there is greater prominence placed on the biochemical alterations that occur with aging and illness and neurodegenerative changes (Connor & Blazer, 2007). Other conditions that lead to an anxiety disorder could be a side effect caused by a medication a person takes to treat a medical condition (Felman, 2018). Anxiety can also be a response from being diagnosed with a medical problem such as heart disease, diabetes, respiratory disorders, or drug/alcohol withdrawal (Mayo Clinic, 2020).

Diseases and Outcomes

According to the U.S. Department of Health and Human Services (2014) there are five major types of anxiety disorders. Generalized Anxiety Disorder is characterized as chronic anxiety or tension even when there is nothing to provoke it; Obsessive-Compulsive Disorder is branded by repeated, unwanted thoughts or obsessions, and/or repetitive behavior; Panic Disorders are characterized by unexpected, repeated episodes of intense anxiety that is accompanied by physical symptoms; Post-Traumatic Stress Disorder is developed after exposure to a terrifying event; and Social Phobia is

characterized by an devastating anxiety and excessive self-consciousness in social situations (U.S. Department of Health and Human Services, 2014).

Treatments for anxiety are usually implemented in an out-patient setting; exceptions to this that would require hospitalization include a parent's desire for suicide, unresponsiveness to treatments, or comorbidities such as major depression or personality disorders (Bandelow et al., 2017). There is substantial undertreatment of anxiety disorders. In a European study, only 20.6% of participants with an anxiety disorder sought professional help. Of those participants who contacted health care services, 23.2% received no treatment at all, 19.6% received only psychological treatment, 30.8% received only drug treatment, and 26.5% were treated both with drugs and psychotherapy (Alonso & Lepine, 2007).

Pharmacologic treatments are the first choice for clinicians when treating an anxiety disorder (Slee et al., 2019). Duloxetine, venlafaxine, escitalopram Mirtazapine, sertraline, fluoxetine, buspirone, and agomelatine have been found to be efficacious with good acceptability (Slee et al., 2019). There has been strong evidence that over-the-counter supplements such as St. John's Wort, Saffron, and Zinc can also help with anxiety disorders (Sarris & Mischoulon, 2017).

Like depression, anxiety can also be treated with psychotherapy. Behavioral interventions to include learning coping mechanisms when confronted by the extreme emotion has shown to help anxiety sufferers (Asaloo et al., 2015). Other treatments such as animal assisted therapies are also showing strong health benefits to those with both anxiety and depression disorders. Being engaged with an animal enhances oxytocin,

serotonin, dopamine, phenylethylamine, neuropeptides, and prolactin to create a sense of serenity (Creagan et al., 2015).

COPD and Anxiety

An anxiety disorder is more than the occasional anxious feeling. A person with an anxiety disorder experiences anxiety that does not go away and can get worse over time (National Institute of Mental Health, 2018). The symptoms of anxiety can interfere with a person's relationships, and daily activities.

Doyle et al. (2013) examined the association of anxiety with COPD to determine how much disease functional capacity and severity alter the association. Anxiety is shown to correlate with pulmonary-specific COPD symptoms. Anxiety is associated with higher levels of shortness of breath, fatigue, and frequency of COPD symptoms (Doyle et al., 2013).

Anxiety and depression among COPD patients are commonly reported together (Hill et al., 2008). Regardless of these symptoms being reported together or separately they affect the patient's health-related quality of life and contribute to the physical disability and financial burden of COPD. According to one study, self-management education was common among COPD patients, however, this does not include education on how to manage anxiety or depression; this same study found that because health care providers tend to have their own scopes regarding what to educate the COPD patients on, psychological well-being are often ignored (Siltanen et al., 2020). It is vital to provide better treatment and recognition of depression and anxiety among those who suffer from COPD (Pooler & Beech, 2014).

HADS

History

The HADS was created more than 30 years ago by Snaith and Zigmond to measure depression and anxiety in a general patient medical population (Stern, 2014). HADS has a focus on the non-physical symptoms associated with anxiety and depression, however it does not include all the diagnostic criteria to diagnose depression according to the Diagnostic and Statistical Manual of Mental Disorders Fourth/Fifth Edition or all those that are required by the Health and Work Development Unit National Depression and Long-Term Sickness Absence Screening Audit (Stern, 2014).

Psychometric Properties

The HADS has a two-factor structure measuring anxiety and symptoms of depression. HADS has shown to be a valid instrument to measure psychological distress in older people (Djukanovic et al., 2017). Internal consistency was found to be good in both factors and was measured with ordinal and traditional alpha (Djukanovic et al., 2017). Likewise, HADS has been found to be a valid instrument in COPD patients. In one study, the HADS psychometric properties were assessed by comparing to that of another health questionnaire; it was found that both questionnaires can be recommended as screening tools for psychological distress in COPD patients (Bratås et al., 2014). The HADS demonstrates excellent internal consistency (HADS-A = 0.83; HADS-D = 0.82) and test-retest reliability (HADS-A = 0.89; HADS-D = 0.92) (Paine, et al., 2019).

Measurements

There are seven questions for anxiety and seven questions for depression interspersed within the questionnaire but scored separately. It takes the average person around 2-5 minutes to complete (Snaith, 2003). The scoring scale for both anxiety and depression are as follows: <7 indicates non-cases; 8-10 mild anxiety or depression; 11-14 moderate anxiety or depression; and 15-21 severe anxiety or depression (Stern, 2014).

Depression (HADS-D)

The seven items in this subscale focus on the inability to feel pleasure. The respondent rates each item on a four-point scale from 0-3 (absent to extreme presence) (Rehabilitation Measures Database, 2012).

Anxiety (HADS-A)

The seven items in this subscale focus on generalized anxiety. Just as with the HADS-D, the respondent rates each item on a four-point scale from 0-3 (absent to extreme presence) (Rehabilitation Measures Database, 2012).

SF-36

History

The SF-36 was developed by RAND Health Care to help explain differences in patient outcomes concerning quality of life (RAND Health Care, 2020). Optum Incorporated, Health Assessment Lab, The Medical Outcomes Trust, and QualityMetric Incorporated all hold copyrights and trademarks for the SF-36; these organizations, together, have developed policies for granting permissions to use the SF-36 form (Lins & Carvalho, 2016).

Psychometric Properties

The SF-36 is often used as a measure to assess other scales against and is increasingly reported in scientific literature (Lins & Carvalho, 2016; Stavem et al., 1999). In COPD patients, SF-36 internal consistency reliability was high ($\alpha = 0.76-0.90$ for all scales except the role-emotional scale, $\alpha = 0.66$); test-retest reliability also was high, with Spearman's $\rho = 0.59-0.88$ for the eight scales, only with the role-emotional and social functioning scales below 0.75 (Stavem et al., 1999).

How Quality of Life is Measured

The SF-36 is a health-related quality of life measure that assesses eight health concepts: limits in physical function because of health complications; limits in social function because of physical or emotional difficulties; limits in role activities because of physical health problems; bodily pain; general mental health; limits in role activities because of emotional problems; vitality; and general health perceptions (Gandek et al., 2004). These eight health concepts form two, larger summary measures, physical and mental health (Sharma et al., 2019). The two groups, physical health and mental health are the basis of analysis of health-related quality of life. Each domain has a score of 0 to 100 with higher scores indicating a better health-related quality of life (Ozalevli et al., 2008). The measured constructs are not specific to an age which enables individual patient assessment with comparisons taking place longitudinally (Limsuwat et al., 2014).

Quality of Life and COPD

The World Health Organization defines quality of life (QOL) as:

an individual's perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards, and concerns. It is a broad ranging concept affected in a complex way by the person's physical health, psychological state, personal beliefs, social relationships, and their relationship to salient features of their environment (World Health Organization, 2020).

Quality of life (QOL) is impaired by COPD and continues to deteriorate with the increasing progression of the disease (Zamzam et al., 2013). One in four adults with COPD report that they are not able to work and those who are employed report that their disease causes them to miss work; half of those with COPD say that their activities are limited due to the disease with one third of those affected saying they have trouble walking and climbing stairs (Centers for Disease Control and Prevention, 2020). Exacerbation of dyspnea, a longer duration of the illness, oxygen use therapy, and comorbidities were found to negatively affect the QOL of COPD patients (Rosińczuk et al., 2018). Sharma et al. (2019), found that those with COPD have a low health-related quality of life according to the SF-36 questionnaire.

SGRQ

History

To fill the need of a self-report questionnaire measuring health in chronic airflow limitation, Dr. Paul Jones and colleagues created the SGRQ (Jones et al., 1992). The SGRQ is a 50-item questionnaire designed to help health care professionals evaluate how

breathing problems affect the patient and which aspects of the illness cause the patient the most problems (American Thoracic Society, 2020).

Psychometric Properties

Psychometric testing has demonstrated its repeatability, reliability, and validity (American Thoracic Society, 2020). The intraclass correlations were 0.795 to 0.900 for reproducibility; there were significant correlations between total score and the presence of sputum, cough, and wheeze as well as significant correlations between symptom, activity, and impact domains and other measures of disease (Jones & Forde, 2009).

Measurements

The SGRQ has sixteen sections that are broken up into two parts; part one covers the patients' recollection of their symptoms over the preceding year, part two addresses the patient's current state (Jones & Forde, 2009). In all, three domain scores are calculated: symptoms, activity, impacts, and a fourth, total score is also calculated (Jones & Forde, 2009). The self-reported scoring system ranges from 0 to 100 with higher scores indicating more limitations (Wilke et al., 2012).

Impacts Domain: Disturbances in Social and Psychological Functioning

There are three domains in the SGRQ: symptoms, activity, and impacts. The impacts domain of the SGRQ covers employment, being in control of health, panic, the need for medication and its side effects, and expectations for health and disturbance of daily life (Wilson et al., 1996).

There are seven questions in the impact domain. The total score provides assessment of a person who has airflow limitation's psychological and social functioning.

Psychological Functioning is a person's ability to achieve their goals within their environment; it includes an individual's emotion, behavior, social skills, and overall mental health (Preedy & Watson, 2010). Social functioning is an individual's ability to fulfill their role within their work environments, social activities, and their personal relationships (Bosc, 2000).

The impact of COPD on mental and social status has been demonstrated in multiple studies (Franssen et al., 2018). Due to the progressive and chronic nature of the disease, patients are not only limited in their physical abilities, but often show reduced social and psychological functioning (von Leupoldt et al., 2012).

Each of the three domains of the SGRQ have been found to be reliable as stand-alone assessments. The person reliability resulting from Rasch model analysis showed internal consistency among all items within the same domain, which is like the Cronbach's coefficient Alpha (Lo et al., 2015). A value greater than 0.7 indicates good internal consistency or model fit; the reliability coefficient for the impact domain is 0.81 (Lo et al., 2015).

Methodology

This was a quantitative, longitudinal, nonexperimental study using archived data from the COPDGene cohort (COPDGene, 2019).

Quantitative Survey Research

Quantitative research is when numerical data are collected and analyzed. This data is used to construct tables and graphs which can show averages and patterns. Quantitative data can be used to confirm or reject a hypothesis by examining

relationships between variables (McLeod, 2019). Quantitative data can be understood with statistical analysis; the quantitative approach is viewed as scientifically objective, and rational (Carr, 1994).

There are many types of data collection strategies in quantitative research, however in the present study the type of data collection is limited to survey research. The goal of a survey is to obtain reliable, valid, unbiased data from the representative sample of respondents with as little error as possible (McColl et al., 2001). It is suggested to stay with measures that have already been standardized and proven valid and reliable; the cost of using a poor measure may be greater than any benefits attained (DeVellis, 2017).

Longitudinal Research

Longitudinal research is when participants are followed over prolonged periods of time to gather continuous or repeated data (Caruana et al., 2015). Longitudinal research is beneficial for evaluating the relationship between risk factors and the development and advancement of disease; because data are collected for specific individuals within a predefined group, statistical testing may be used to analyze change over time for the whole group, or for specific individuals (Caruana et al., 2015).

There are both advantages and disadvantages to longitudinal research. An advantage is this type of research allows for repeated observations of the same individual over time, meaning any changes in the dependent variable cannot be contributed to differences between individuals; another advantage is being able to keep track of variables in real time (Caruana et al., 2015). Disadvantages are the longitudinal studies are time consuming and require a significant number of resources to be effective (Wang

et al., 2017). Longitudinal studies also face the risk of attrition, especially in those populations that are aging or have high mortality rates (Coggon et al., 2003).

Longitudinal research is observational (Caruana et al., 2015).

Nonexperimental, Observational Research

Nonexperimental studies are observational and retrospective (Thomson & Panacek, 2007). These types of studies examine activities that have already occurred making any manipulation of the independent variable not possible. The dependent variable will have already occurred before data collection takes place (Simon & Goes, 2013).

Data Analysis

Paired *T* Tests

The *t* test is used to determine whether two group means are different and if that difference is statistically significant (Allen, 2017). Significance is the probability of observing an event or result by chance alone; the lower that probability, the more statistically significant that observed result is (Allen, 2017). A significance level (p-value) of .05 is used most, which means, the likelihood of the observed result must be less than 5% (1 in 20) to be considered statistically significant; in some cases, the significance level is set to 1% or 10%. In a paired sample *t* test, each subject is measured twice, which results in pairs of observation.

There are two competing hypotheses with the paired sample *t* test, the null hypothesis, and the alternate hypothesis. The null hypothesis assumes that the mean difference between the paired samples is zero, meaning, all observable differences are

explained by random variation (Aczel et al., 2018). The alternative hypothesis assumes that the mean difference between the paired samples is not equal to zero.

Statistics Solution (2020) lists four main assumptions for the paired sample t test: a) the dependent variable is continuous, b) the observations are independent of one another, c) the dependent variable should be normally distributed, and d) the dependent variable should not contain any outliers.

Multiple Regression

Multiple regression is used to calculate the value of a variable founded on the value of two or more other variables; it uses several explanatory variables to predict the outcome of a response variable (Laerd Statistics, 2018).

Laerd Statistics lists the assumptions for multiple regression as: a) the dependent variable is measured on a continuous scale, b) there are two or more independent variables, c) there is an independence of observations, d) there is linear relationship between the dependent variable and each independent variable, e) the data needs to show homoscedasticity, f) the data must not show multicollinearity, g) there should be no significant outliers, and h) check that the residuals are normally distributed.

Conclusion

Studies have shown that anxiety and depression can complicate the disease progression of COPD (Doyle et al., 2013). There has not been a study that has shown how anxiety and depression progress over time in the adult COPD population. Determining if there is a difference in anxiety and depression can help doctors develop better treatments. Correlating the scores of the HADS, the SF-36, and the SGRQ in adult

COPD patients, doctors will be able to get a full picture of the patient's mental health and quality of life while they are living with the disease. These findings can help doctors determine better treatment plans so their COPD patients can live more productive, happier lives.

Chapter 3: Research Method

Purpose of the Study

The purpose of this research was to address the gap in the literature concerning the relationship among longitudinal smoking status, depression, anxiety, health-related quality of life and psychological and social functioning due to airflow limitations among participants enrolled in the COPDGene Cohort Study. I measured depression and anxiety with the HADS, health-related quality of life with the two components of the SF-36 (PCS and MCS), and psychological and social functioning due to airflow limitations with the impact domain of the SGRQ. I examined if there was a change over time in these variables with a paired samples *t* test and the relationship among the variables was assessed using multiple regression analysis. In this chapter I describe the methods and procedures I used in assessing data including research questions, research design, population, instrumentation used, role of the researcher, and the data analysis plan. Finally, this chapter discusses issues of trustworthiness.

Research Questions

RQ1: Is there a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups?

H_1 : There is a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups.

H_0 : There is no significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups.

RQ2: Is there a significant change in depression between P2 and P3 in the four longitudinal smoking status groups?

*H*₁₂: There is a significant change in depression between P2 and P3 in the four longitudinal smoking status groups.

*H*₀₂: There is no significant change in depression between P2 and P3 in the four longitudinal smoking status groups.

RQ3: Is there a significant change in PCS between P2 and P3 in the four longitudinal smoking status groups?

*H*₁₃: There is a significant change in PCS between P2 and P3 in the four longitudinal smoking status groups.

*H*₀₃: There is no significant change in PCS between P2 and P3 in the four longitudinal smoking status groups.

RQ4: Is there a significant change in MCS between P2 and P3 in the four longitudinal smoking status groups?

*H*₁₄: There is a significant change in MCS between P2 and P3 in the four longitudinal smoking status groups.

*H*₀₄: There is no significant change in MCS between P2 and P3 in the four longitudinal smoking status groups.

RQ5: Is there a significant change in psychological and social functioning due to airflow limitations (measured with the SGRQ) between P2 and P3 in the four longitudinal smoking status groups?

H₁₅: There is a significant change in SGRQ between P2 and P3 in the four longitudinal smoking status groups.

H₀₅: There is no significant change in SGRQ between P2 and P3 in the four longitudinal smoking status groups.

RQ6: Is there a relationship between longitudinal smoking status, anxiety, depression, health-related quality of life (PCS and MCS), and psychological and social functioning due to airflow limitations measured with the SGRQ at P3?

H₁₆: There is a relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36 psychological and social functioning due to airflow limitations measured with the SGRQ.

H₀₆: There is no relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36 psychological and social functioning due to airflow limitations measured with the SGRQ.

Research Design

My study was designed as quantitative, longitudinal, nonexperimental study using archived data from the COPDGene cohort (COPDGene, 2019). My study: a) identified if there was a change in anxiety and depression scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; b) identified if there was a change in health-related quality of life scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current

smokers) in the COPDGene cohort during the 5-year period between P2 and P3; c) identified if there was a change in psychological and social functioning due to airflow limitations scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; and d) identified if there was a relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36 psychological, and psychological and social functioning due to airflow limitations measured with the SGRQ.

Sample

The COPDGene cohort consists of current and former smokers, enrolled between the ages of 45 and 80 years, with a minimum 10 pack-year smoking history, as well as a small group of similar aged non-smokers. Subjects with COPD or evidence of decreased lung function are classified by GOLD stage. Subjects whose lung function is normal are assigned as controls.

The COPDGene data collection takes place at 20 clinical centers across the United States. Those centers are: Ann Arbor VA Medical Center, Baylor College of Medicine, Brigham and Women's Hospital, Columbia University Medical Center, Duke University Medical Center, Reliant Medical Center, Lundquist Institute for Biomedical Innovation at Harbor, Minnesota Health Partners, Johns Hopkins University, Morehouse School of Medicine, Minneapolis VA Medical Center, National Jewish Health, University of Pittsburgh, Temple University, UTHSC at San Antonio, University of

Alabama, Birmingham, University of Iowa, University of Michigan, University of Minnesota, and University of California, San Diego.

I conducted a power analysis on the regression analysis question (RQ6: Is there a relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36, psychological and social functioning due to airflow limitations measured with the SGRQ?) which had one DV and five IV using a medium effect size ($d = .50$), and an alpha of .05. Result showed that a total sample of 54 participants will achieve a power of 0.8. My study had a total of 983 participants that was analyzed.

Instrumentation

I used the results of the HADS, SF-36, and the impact domain of the SGRQ. I used the HADS to answer RQ1 and RQ2. I used SF-36 to answer RQ3 and RQ4. I used the SGRQ to answer RQ5. I used all the measures to answer RQ6.

HADS

The HADS has a two-factor structure measuring anxiety and symptoms of depression. HADS has shown to be a valid instrument to measure psychological distress in older people (Djukanovic et al., 2017). Internal consistency was found to be good in both factors and was measured with ordinal and traditional alpha (Djukanovic et al., 2017). Likewise, HADS has been found to be a valid instrument in COPD patients. In one study, the HADS psychometric properties were assessed by comparing to that of another health questionnaire; it was found that both questionnaires can be recommended as screening tools for psychological distress in COPD patients (Bratås et al., 2014). Paine

et al., (2019) states the HADS demonstrates excellent internal consistency (HADS-A = 0.83; HADS-D = 0.82) and test-retest reliability (HADS-A = 0.89; HADS-D = 0.92).

There are seven questions for anxiety and seven questions for depression interspersed within the questionnaire but scored separately. It takes the average person around 2 to 5 minutes to complete (Snaith, 2003). The scoring scale for both anxiety and depression are as follows: <7 indicates noncases; 8 to 10 mild anxiety or depression; 11 to 14 moderate anxiety or depression; and 15 to 21 severe anxiety or depression (Stern, 2014).

The seven items in the depression subscale (HADS-D) focus on the inability to feel pleasure. The respondent rates each item on a 4-point scale from 0 to 3 (absent to extreme presence; Rehabilitation Measures Database, 2012).

The seven items in the anxiety subscale (HADS-A) focus on generalized anxiety. Just as with the HADS-D, the respondent rates each item on a 4-point scale from 0 to 3 (absent to extreme presence; Rehabilitation Measures Database, 2012).

SF-36

In COPD patients, SF-36 internal consistency reliability was high ($\alpha = 0.76-0.90$ for all scales except the role-emotional scale, $\alpha = 0.66$); test-retest reliability also was high, with Spearman's $\rho = 0.59-0.88$ for the eight scales, only with the role-emotional and social functioning scales below 0.75 (Stavem et al., 1999).

The SF-36 is a health quality of life measure that assesses eight health concepts: limits in physical function because of health complications, limits in social function because of physical or emotional difficulties, limits in role activities because of physical

health problems, bodily pain, general mental health, limits in role activities because of emotional problems, vitality, and general health perceptions (Gandek et al., 2004). These eight health concepts form two, larger summary measures, physical and mental health (Sharma et al., 2019). The two groups, physical health and mental health are the basis of analysis of health-related quality of life. Each domain has a score of 0 to 100 with higher scores indicating a better health-related quality of life (Ozalevli et al., 2008).

SGRQ

Psychometric testing has demonstrated the SGRQ's repeatability, reliability, and validity (American Thoracic Society, 2020). The intraclass correlations were 0.795 to 0.900 for reproducibility, 0.88 for the impacts section which will be used in this study; there were significant correlations between total score and the presence of sputum, cough and wheeze as well as significant correlations between symptom, activity, and impact domains and other measures of disease activity (Jones & Forde, 2009).

The SGRQ has 16 sections that are broken up into two parts; part one covers the patients' recollection of their symptoms over the preceding year, part two addresses the patient's current state (Jones & Forde, 2009). In all, three domain scores are calculated: symptoms, activity, impacts, and a fourth total score is calculated (Jones & Forde, 2009). The self-reported scoring system ranges from 0 to 100 with higher scores indicating more limitations (Wilke et al., 2012).

This questionnaire is scored in three separate domains: symptoms, activity, and impacts. For this study, I analyzed the impacts domain. The impacts domain has seven questions that measure the disturbances in social and psychological functioning (Nagai et

al., 2015). This section includes questions asking the participant to rate their respiratory condition, it asks about the participants cough and shortness of breath, other effects that the respiratory disease may have on the participant, how the respiratory problems affect daily life, questions about treatment and medications, and what activities are prevented from the respiratory problems.

Role of the Researcher

I am the COPDGene Research Program Administrator. I have collected some of the data at the study site in Denver, Colorado and I oversee the data collection across all twenty sites throughout the United States.

Data Analysis Plan

To answer RQ1 (Is there a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups?), RQ2 (Is there a significant change in depression between P2 and P3 in the four longitudinal smoking status groups?), RQ3 (Is there a significant change in PCS between P2 and P3 in the four longitudinal smoking status groups?), RQ4 (Is there a significant change in MCS between P2 and P3 in the four longitudinal smoking status groups?), and RQ5 (Is there a significant change in psychological and social functioning due to airflow limitations [measured with the SGRQ] between P2 and P3 in the four longitudinal smoking status groups?), I used paired *t* tests to determine if there was a significant change between the P2 and P3 scores. Although they are a priori there are a lot of them, so the alpha level was adjusted to 0.01. The final question, RQ6 (Is there a relationship between longitudinal smoking status, anxiety, depression, health-related quality of life [PCS and MCS], and psychological and

social functioning due to airflow limitations measured with the SGRQ?), I answered using regression analysis to assess whether the IV affect the DV.

Threats to Validity

Validity is whether the test measures what is intended to be measured. The questionnaires I chose are appropriate for this study's needs; however, how the tests were administered could come into question. Multiple coordinators across all study sites were trained on how to correctly administer the questionnaires; I assumed that the coordinators followed their training and administered the questionnaires correctly. Each instrument is a self-report measure that relies on the participants to report how they feel. The participants' reports of symptoms could vary, depending upon their memory and evaluation of symptoms. Some of the participants were administered the questionnaires in-person before the COVID-19 pandemic, some over the phone during the COVID-19 self-quarantine, and some in-person during the COVID-19 self-quarantine. The COVID-19 pandemic could have affected the answers given concerning their anxiety, depression, quality of life and disturbances in social and psychological functioning.

Security and Ethics

Data Coordinating Center

COPDGene has a Data Coordinating Center (DCC) at National Jewish Health. The DCC maintains the confidentiality of all the protected health information collected under the protocol using physical security, database security, and web applications security.

Physical Security-Computer Room

National Jewish Health maintains servers in their computer room, a secure environment whose access is restricted to essential personnel. This server room has dedicated power, cooling, lighting, and an environmental monitoring system.

Database Security

The database is stored on a dedicated server with access only granted to essential personnel with each group of access users having their own permissible actions. The database has daily backups with transaction logs maintained and run multiple times daily. Patient data are encrypted by the backup process to further ensure security.

Web Applications

REDCap has been utilized as the electronic data capturing (EDC) system for P3. It is a secure, web-based application designed to support data capture for research studies. REDCap is maintained by the REDCap Consortium which is comprised of over two thousand institutional partners (Vanderbilt, 2018). EDC has been shown to improve quality, reduce effort, and has allowed faster access to data (Donovan, 2007). EDC has been shown to be more time-effective allowing for review and analysis of data in real-time (Walther et al., 2011). Edit checks within the system allow for fewer mistakes. For strict detection parameters, automatic checks were built into the system at the start of the study to consistently check all data throughout the study based on the specifications set (Prokscha, 2012). These edit checks are more reliable and the discrepancy is automatically logged.

Data Collection and Handling

Each clinical center involved in the study has obtained approval of the COPDGene Study protocol from their local Institutional Review Board (IRB). For each study visit, the participant signs an informed consent document. All information collected from the subjects has been given a subject ID that consists of a three-letter center code, letters, and numbers.

During P2 data were collected on paper forms at the clinical centers then faxed directly into the DCC servers. During P3, REDCap has been utilized as the EDC system.

Ethics and Participant Safety

As mentioned, the data collection takes place at 20 clinical centers. Each of these centers has their own, dedicated IRB that reviews the protocol to ensure the protection of the rights and welfare of the participants. Each site is required to submit the protocol and subsequent amendments to their IRB before they can begin study procedures. The protocol is re-reviewed yearly at each clinical site to ensure the continued compliance.

COPDGene has an Ancillary Studies and Publications Committee that decides if an investigator is allowed access to their data and to ensure there is no overlapping of projects and publications within COPDGene. This committee meets twice a month on the first and third Thursdays. Once the data request is reviewed, the committee decides if the investigator is allowed access or if the request is deferred pending further information needed.

COPDGene has been appointed an Observational Safety and Monitoring Board (OSMB) by the Director of the National Heart, Lung and Blood Institute (NHLBI) to

oversee data and participant safety. The COPDGene OSMB meets a minimum of once a year. Each OSMB report includes recruitment, performance, safety, and follow-up data along with a list of reportable adverse events.

Summary

In this chapter I discussed the essential components of the research methods used in this study and procedures used in assessing data including research questions. I also discussed research design, population, instrumentation used, role of the researcher, and the data analysis plan, security, and trustworthiness.

Chapter 4: Results

Introduction

The purpose of this research was to address the gap in the literature concerning the relationship among longitudinal smoking status, depression, anxiety, health-related quality of life and psychological and social functioning due to airflow limitations measured with the SGRQ among patients enrolled in the COPDGene Cohort Study. I addressed the gap in the literature by answering my research questions.

RQ1: Is there a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups?

H_1 1: There is a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups.

H_0 1: There is no significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups.

RQ2: Is there a significant change in depression between P2 and P3 in the four longitudinal smoking status groups?

H_1 2: There is a significant change in depression between P2 and P3 in the four longitudinal smoking status groups.

H_0 2: There is no significant change in depression between P2 and P3 in the four longitudinal smoking status groups.

RQ3: Is there a significant change in PCS between P2 and P3 in the four longitudinal smoking status groups?

*H*₁₃: There is a significant change in PCS between P2 and P3 in the four longitudinal smoking status groups.

*H*₀₃: There is no significant change in PCS between P2 and P3 in the four longitudinal smoking status groups.

RQ4: Is there a significant change in MCS of health-related quality of life between P2 and P3 in the four longitudinal smoking status groups?

*H*₁₄: There is a significant change in MCS between P2 and P3 in the four longitudinal smoking status groups.

*H*₀₄: There is no significant change in MCS between P2 and P3 in the four longitudinal smoking status groups.

RQ5: Is there a significant change in psychological and social functioning due to airflow limitations (measured with the SGRQ) between P2 and P3 in the four longitudinal smoking status groups?

*H*₁₅: There is a significant change in SGRQ between P2 and P3 in the four longitudinal smoking status groups.

*H*₀₅: There is no significant change in SGRQ between P2 and P3 in the four longitudinal smoking status groups.

RQ6: Is there a relationship between longitudinal smoking status, anxiety, depression, health-related quality of life (PCS and MCS), and psychological and social functioning due to airflow limitations measured with the SGRQ at P3?

*H*₁₆: There is a relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-

36, and psychological and social functioning due to airflow limitations measured with the SGRQ.

H₀₆: There is no relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36, and psychological and social functioning due to airflow limitations measured with the SGRQ.

I completed four goals in this study. In this study I identified a) if there was a change in anxiety and depression scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; b) if there was a change in health-related quality of life scores in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; c) if there was a change in psychological and social functioning due to airflow limitations in the four groups based on longitudinal smoking status (stable current smokers, stable former smokers, current to former smokers, and former to current smokers) in the COPDGene cohort during the 5-year period between P2 and P3; and d) if there was a relationship at P3 between longitudinal smoking status, anxiety, depression, health-related quality of life, and physical and social functioning due to airflow limitations.

In this chapter, I include information concerning the data collection, data cleaning, the statistical assumptions, and the findings of six research questions which

included data from P2 and P3 of the COPDGene cohort. I analyzed data in the HADS questionnaire, the SF-36 questionnaire, and the impacts domain of the SGRQ.

Data Collection

The COPDGene data collection takes place at 20 clinical centers beginning in 2013 and is currently ongoing. The centers where the data collection takes place are: Ann Arbor VA Medical Center, Baylor College of Medicine, Brigham and Women's Hospital, Columbia University Medical Center, Duke University Medical Center, Reliant Medical Center, Lundquist Institute for Biomedical Innovation at Harbor, Minnesota Health Partners, Johns Hopkins University, Morehouse School of Medicine, Minneapolis VA Medical Center, National Jewish Health, University of Pittsburgh, Temple University, UTHSC at San Antonio, University of Alabama, Birmingham, University of Iowa, University of Michigan, University of Minnesota, and University of California, San Diego.

Each clinical center involved in the study had obtained approval of the COPDGene Study protocol from their local IRB. For each study visit, the participant signed an informed consent document.

All coordinators who collect the data undergo training to ensure data are collected in the same manner across all the sites and upholds the data collections standards of COPDGene. All of the coordinators are trained using the same techniques; all training is overseen by me and senior investigators of COPDGene.

During P2 data were collected on paper forms at the clinical centers then faxed directly into the DCC servers. During P3, REDCap has been utilized as the EDC. The data collection took place in F2F interviews using validated surveys.

Data Entry and Cleaning

REDCap is the research EDC system for P3 data entry. It is a secure, web-based application designed to support data capture for research studies. REDCap is maintained by the REDCap Consortium which is composed of over two thousand institutional partners.

Data entry for the study is critical for good quality. Good quality data will allow accurate conclusions to be drawn from the project. The DCC performed quality checks on all data and queried clinical centers about inconsistent or out of range data entry. REDCap also has functions to do many in-time queries, which queried out of range data at the time of data entry.

Data Analysis

I analyzed RQ1, RQ2, RQ3, RQ4, and RQ5 with paired t tests. Although they are a priori there are a lot of them, so I adjusted the alpha level to 0.01 as mentioned in the data analysis plan in Chapter 3. In cases where $p = 0.01$, however, it was taken as $p < 0.01$. I answered RQ6 using regression analysis to assess whether the IV affect the DV.

Descriptive Statistics

Table 1*Descriptive Statistics*

	Sex		Race		Age		
	Male	Female	White	Black or African American	Mean	Minimum	Maximum
Stable Current	142	136	149	129	60.20	49.70	77.70
Current to Former	36	39	47	28	61.6	50.60	79.00
Stable Former	291	307	520	78	67.90	50.6	87.4
Former to Current	15	17	19	13	60.70	50.5	76.4
Total	484	499	735	248			

Statistical Assumption

For paired sample *t* tests, there are four assumptions; all four assumptions have been met. a) The dependent variable was measured on a continuous scale; b) the independent variable should consist of two categorical, related groups; c) there should be no significant outliers in smaller sample sizes; and d) “the distribution of the differences in the dependent variable between the two related groups should be approximately normally distributed” (Laerd Statistics, 2018). I ran Tests of Normality using the Shapiro-Wilk test.

Figure 1

The Test of Normality Run Using the Shapiro-Wilk Test on the Anxiety Variable

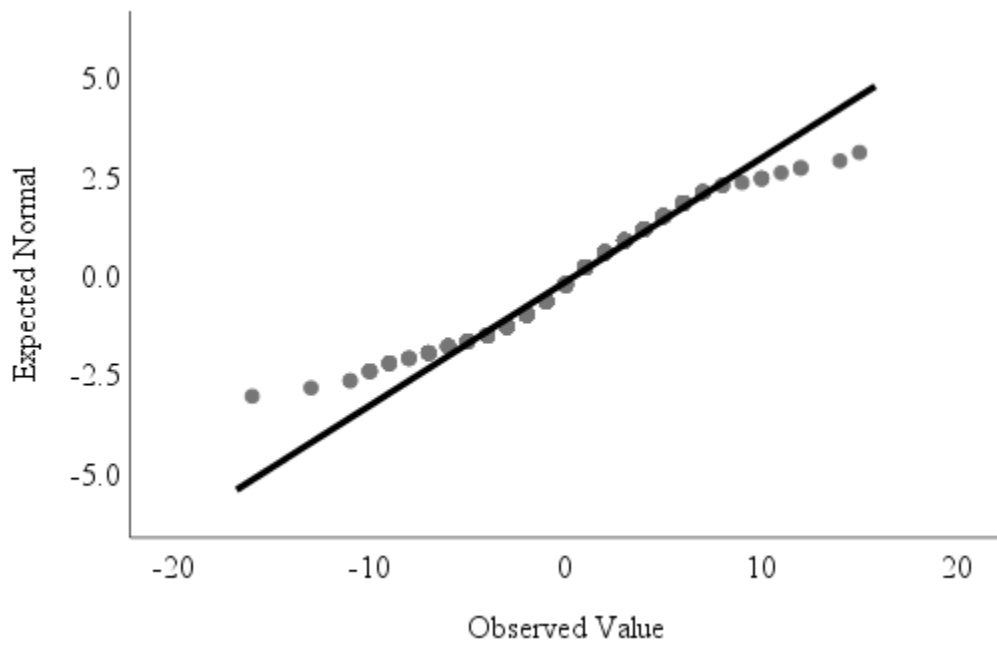


Figure 2

Test of Normality Run Using the Shapiro-Wilk Test on the Depression Variable

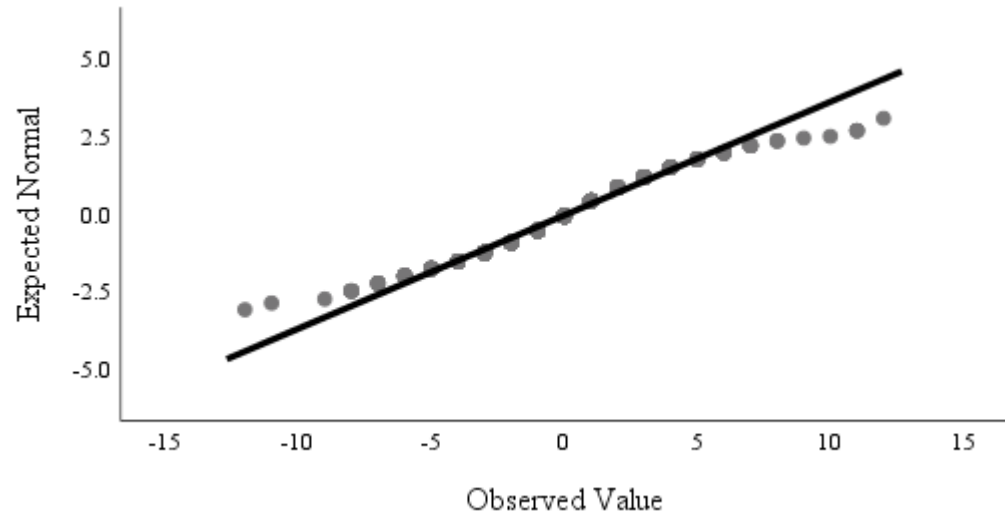
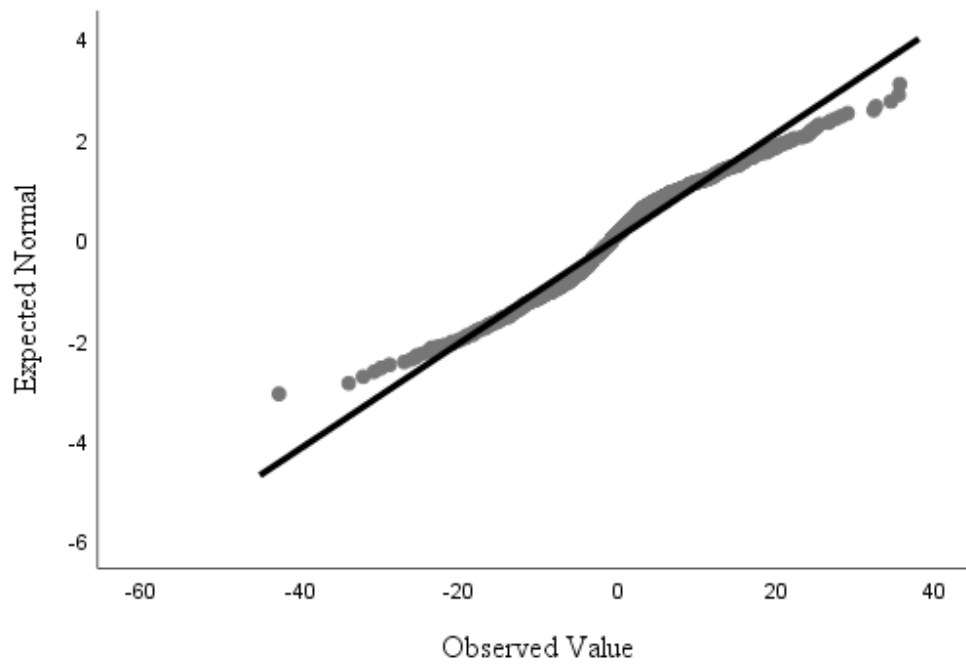


Figure 3

Test of Normality Run Using the Shapiro-Wilk Test on the MCS Variable

**Figure 4**

Test of Normality Run Using the Shapiro-Wilk Test on the PCS Variable

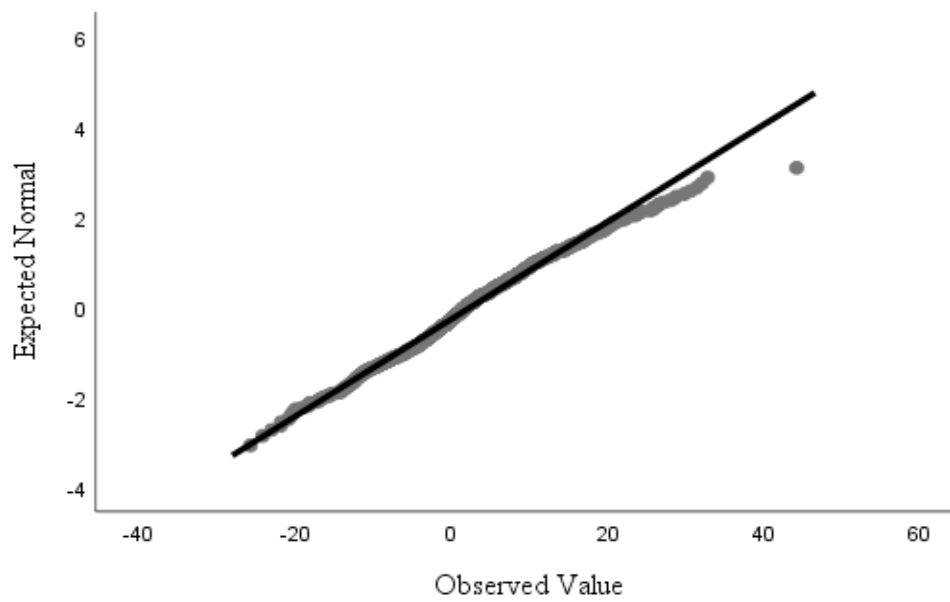
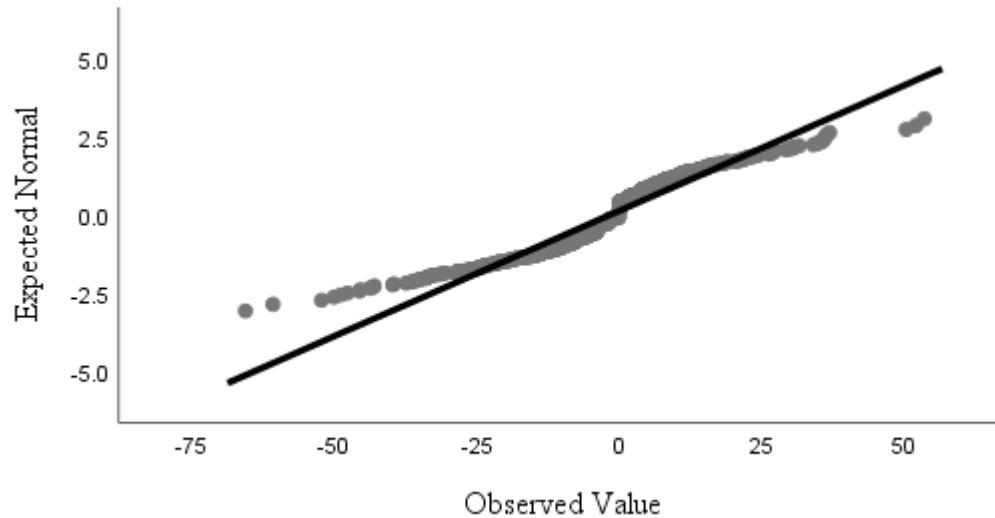


Figure 5

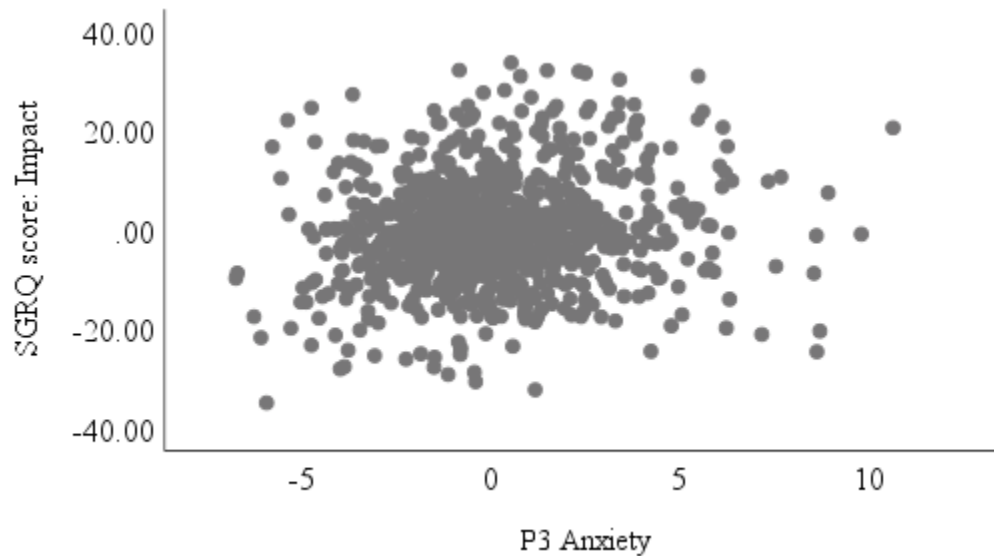
Test of Normality Run Using the Shapiro-Wilk Test on the Psychological and Social Functioning Due to Airflow Limitations Variable



The multiple regression analysis assumptions have been met. Those assumptions are: a) the dependent variable was measured on a continuous scale; b) the independent variables are either continuous or categorical; c) there was independence of residuals assessed by Durbin-Watson statistic of 2.03 meaning that the observations are not related; d) there was a linear relationship between the dependent variable and each independent variable and the dependent variable and the independent variables collectively.

Figure 6

Partial Regression Plot: Anxiety and Impact Score

**Figure 7**

Partial Regression Plot: Depression and Impact Score

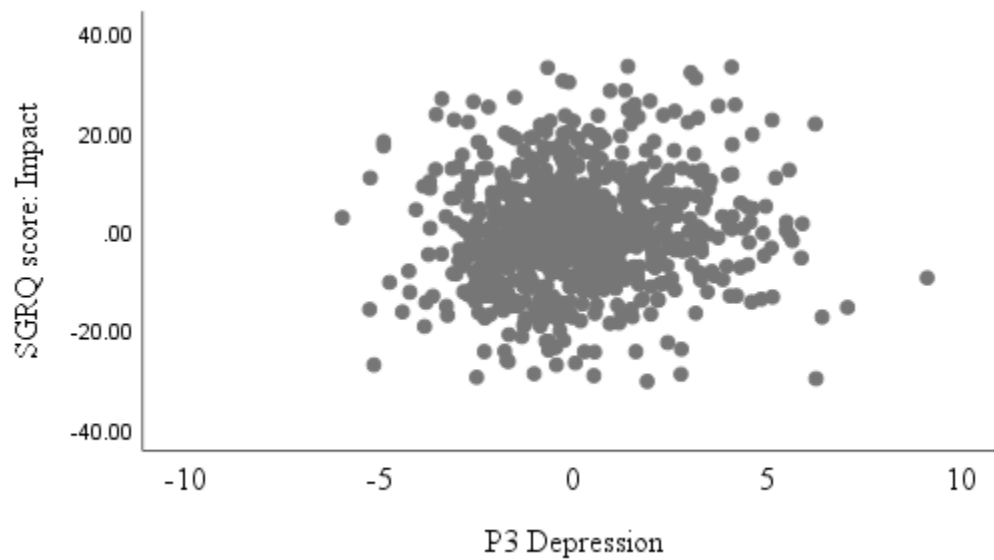
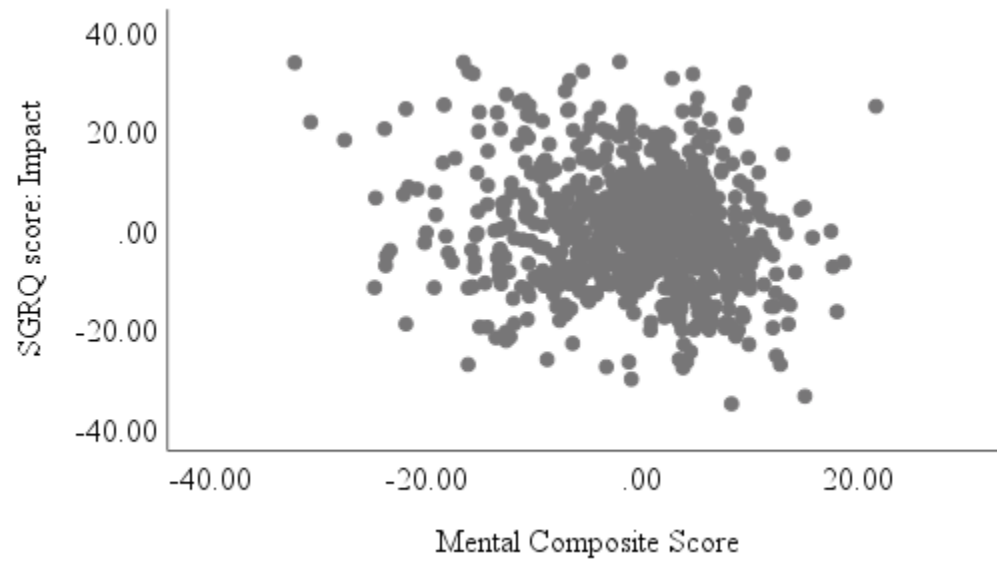


Figure 8

Partial Regression Plot: MCS and Impact Score

**Figure 9**

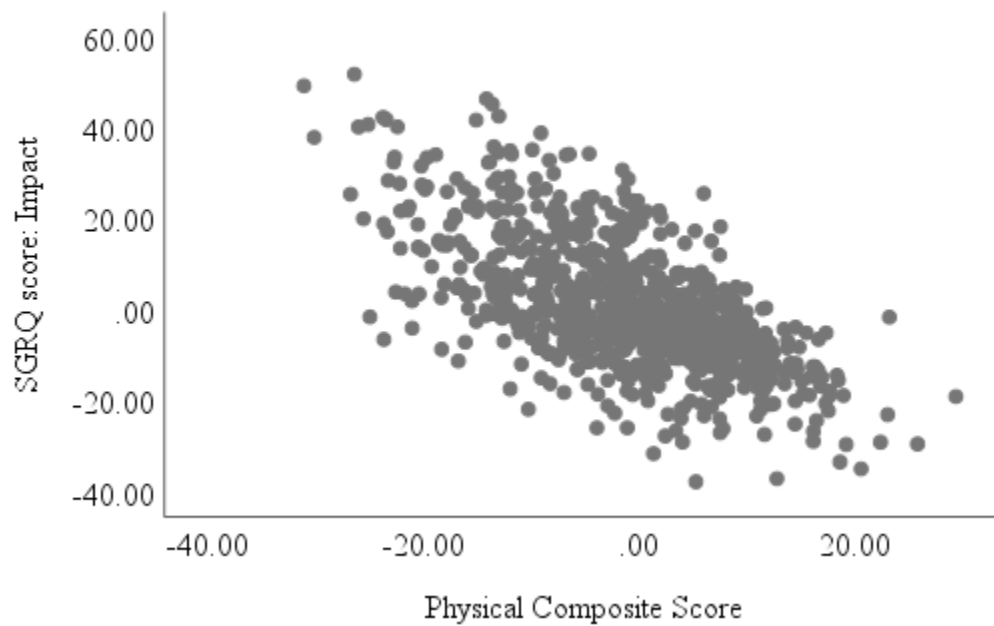


Figure 10

Partial Regression Plot: Smoking Status and Impact Score

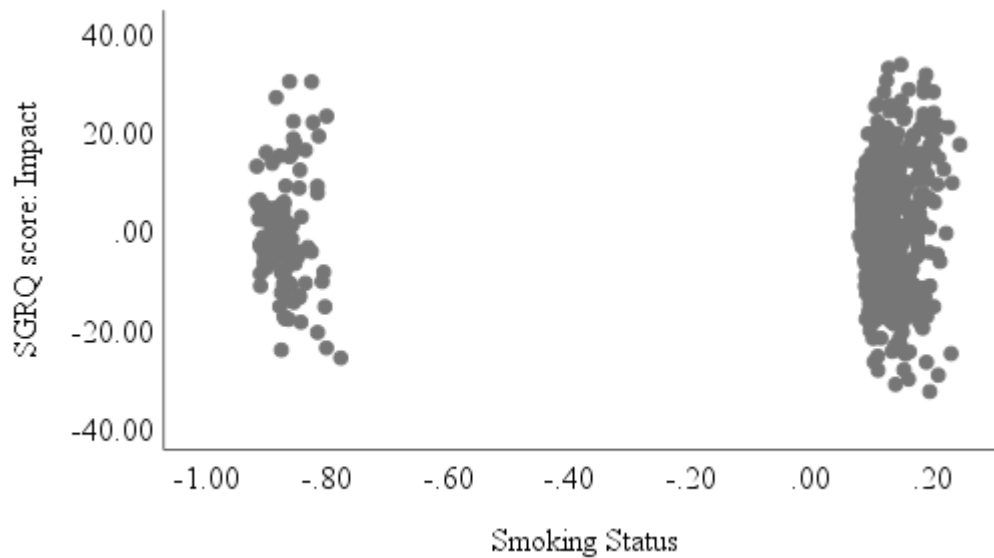
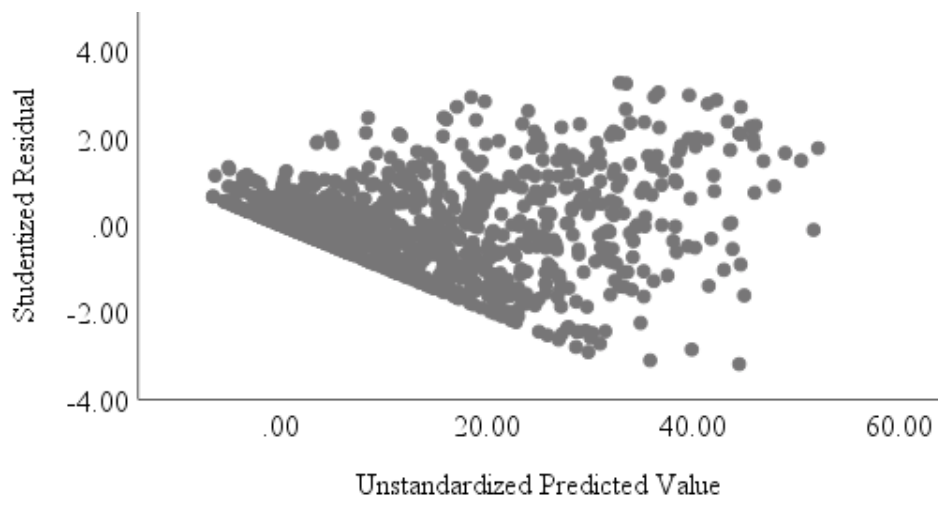


Figure 11

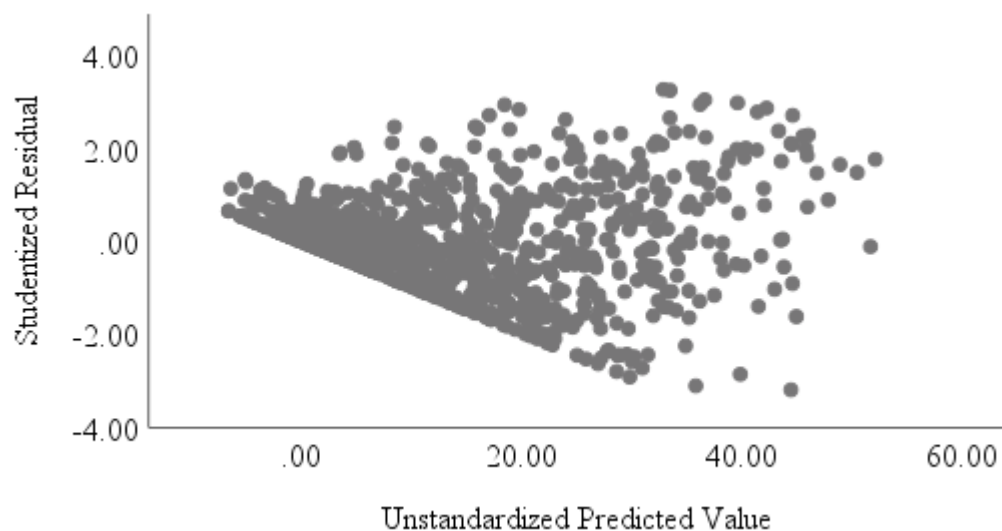
Partial Regression Plot: Anxiety, Depression, MCS, PCS, Smoking Status, and Impact Score



5) there was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values.

Figure 12

Simple Scatter of Studentized Residual by Unstandardized Predicted Value



6) The collinearity statistics showed each variable has a Tolerance greater than 0.1.

Table 2*Collinearity*

	Tolerance
Anxiety	0.494
Depression	0.433
MCS	0.527
PCS	0.755
Smoking Status	0.992

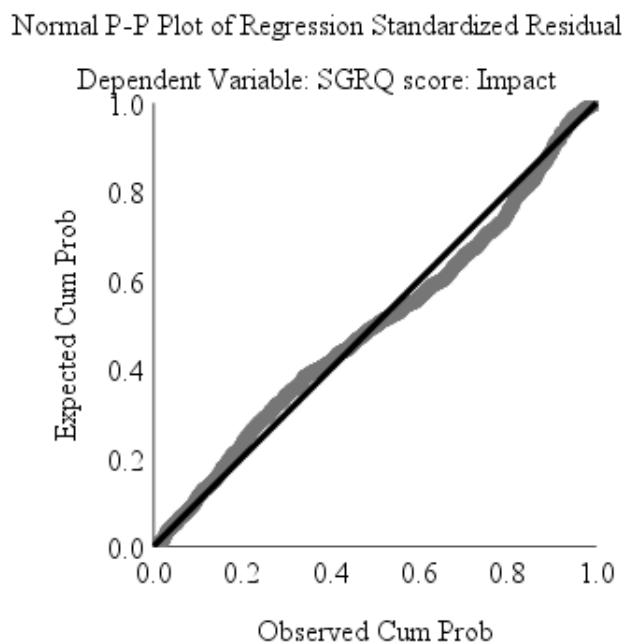
7) there were five significant outliers, however given the large sample size these were left in the analysis.

Table 3*Casewise Diagnostics^a*

Case Number	Std. Residual	SGRQ score: Impact	Predicted Value	Residual
2	-3.207	12.05	44.9122	-32.8622
7	-3.061	4.15	35.5132	-31.3633
9	3.051	67.53	36.2706	31.25938
17	3.186	66.13	33.4825	32.64753
48	3.255	66.59	33.2386	33.35143

a. Dependent Variable: SGRQ score: Impact

8) the residuals were normally distributed (Laerd Statistics, 2018).

Figure 13*Normal Distribution***Analysis of Results****HADS**

This questionnaire has fourteen items that the patients answered on a four-point scale. Seven items relate to anxiety and the remaining seven relate to depression. The final tally of anxiety and depression results in a numerical score that indicates the patient is either mildly, moderately, or severely affected by either anxiety, depression, or both (Stern, 2014). The scoring scale for both anxiety and depression are as follows: <7 indicates non-cases; 8-10 mild anxiety or depression; 11-14 moderate anxiety or depression; and 15-21 severe anxiety or depression (Stern, 2014). For any participant that scored fifteen or above, the participant and the participant's primary care physician was notified so the participant could undergo further evaluation.

I used Paired t tests to determine if there was a significant change between the P2 and P3 scores for anxiety and depression in the four groups.

RQ1: Is there a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups?

H_1 : There is a significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups.

H_0 : There is no significant change in anxiety between P2 and P3 in the four longitudinal smoking status groups.

Table 4

Changes in Mean Anxiety Scores between P2 and P3

	P2	P3	Change	t value	p value	Significance	Effect Size
Stable Current	5.10	4.61	-0.49	2.11	0.04	no	0.13
Current to Former	5.15	4.73	-0.42	1.23	0.23	no	0.13
Stable Former	3.97	3.29	-0.68	5.93	0.01	yes	0.21
Former to Current	5.09	5.09	0.00	0.00	1.00	no	0.00

Stable Current Smokers

This group is defined as those who were smokers at P2 and remained smokers through P3.

The mean anxiety scores in the Stable Current Smokers group at P2 ($M = 5.10$; $SD = 3.77$) was not significantly different from P3 ($M = 4.61$; $SD = 3.95$), $t(276) = 2.11$, $p = 0.04$, $d = 0.13$

Current to Former Smokers

This group is defined as those who were smokers at P2 but quit smoking before P3.

The mean anxiety scores in the Current to Former Smokers group at P2 ($M = 5.15$; $SD = 3.04$) was not significantly different from P3 ($M = 4.73$; $SD = 3.46$), $t(74) = 1.23$, $p = 0.23$, $d = 0.13$

Stable Former Smokers

This group is defined as those who were not smokers at P2 and remained non-smokers at P3.

The mean anxiety scores in the Stable Former Smokers group at P2 ($M = 3.97$; $SD = 3.16$) was significantly different from P3 ($M = 3.29$; $SD = 3.27$), $t(597) = 5.93$, $p < 0.01$, $d = 0.21$

Former to Current Smokers

This group is defined as those who were not smokers at P2 but began smoking before P3.

The mean anxiety scores in the Former to Current Smokers group at P2 ($M = 5.09$; $SD = 3.67$) was not significantly different from P3 ($M = 5.09$; $SD = 3.93$), $t(31) = 0.00$, $p = 1.00$, $d = 0.00$

RQ2: Is there a significant change in depression between P2 and P3 in the four longitudinal smoking status groups?

H_{12} : There is a significant change in depression between P2 and P3 in the four longitudinal smoking status groups.

H_{02} : There is no significant change in depression between P2 and P3 in the four longitudinal smoking status groups.

Table 5

Changes in Mean Depression Scores between P2 and P3

	P2	P3	Change	t value	p value	Significance	Effect Size
Stable Current	3.74	3.84	0.10	-0.53	0.60	no	0.03
Current to Former	3.61	3.73	0.12	-0.36	0.72	no	0.04
Stable Former	2.87	2.68	-0.19	1.92	0.06	no	0.07
Former to Current	4.31	4.31	0.00	0.00	1.00	no	0.00

Stable Current Smokers

This group is defined as those who were smokers at P2 and remained smokers at P3.

The mean depression scores in the Stable Current Smokers group at P2 ($M = 3.74$; $SD = 3.30$) was not significantly different from P3 ($M = 3.84$; $SD = 3.48$), $t(276) = -0.53$, $p = 0.60$, $d = 0.03$

Current to Former

This group is defined as those who were smokers at P2 but quit smoking before P3.

The mean depression scores in the Current to Former Smokers group at P2 ($M = 3.61$; $SD = 2.51$) was not significantly different from P3 ($M = 3.73$; $SD = 3.00$), $t(74) = -0.36$, $p = 0.72$, $d = 0.04$

Stable Former

This group is defined as those who were not smokers at P2 and remained non-smokers at P3.

The mean depression scores in the Stable Former Smokers group at P2 ($M = 2.87$; $SD = 2.77$) was not significantly different from P3 ($M = 2.68$; $SD = 2.63$), $t(597) = 1.92$, $p = 0.06$, $d = 0.07$

Former to Current

This group is defined as those who were not smokers at P2 but began smoking before P3.

The mean depression scores in the Former to Current Smokers group at P2 ($M = 4.31$; $SD = 3.42$) was not significantly different from P3 ($M = 4.31$; $SD = 3.97$), $t(31) = 0.00$, $p = 1.00$, $d = 0.00$

SF-36

The SF-36 is a health-related quality of life measure that assesses eight health concepts: limits in physical function because of health complications; limits in social function because of physical or emotional difficulties; limits in role activities because of physical health problems; bodily pain; general mental health; limits in role activities because of emotional problems; vitality; and general health perceptions (Gandek et al., 2004). These eight health concepts form two, larger summary measures, physical and mental health (Sharma et al., 2019). The two groups, physical health and mental health are the basis of analysis of health-related quality of life. Each domain has a score of 0 to 100 with higher scores indicating a better health-related quality of life (Ozalevli et al.,

2008). The measured constructs are not specific to an age which enables individual patient assessment with comparisons taking place longitudinally (Limsuwat et al., 2014).

I used paired t tests to determine if there was a significant change between the P2 and P3 scores for the PCS and MCS in the four groups.

RQ3: Is there a significant change in PCS of health-related quality of life between P2 and P3 in the four longitudinal smoking status groups?

H_{13} : There is a significant change in PCS between P2 and P3 in the four longitudinal smoking status groups.

H_{03} : There is no significant change in PCS between P2 and P3 in the four longitudinal smoking status groups.

Table 6

Changes in Mean PCS Scores between P2 and P3

	P2	P3	Change	t value	p value	Significance	Effect Size
Stable Current	44.86	43.68	-1.18	2.18	0.03	no	0.11
Current to Former	45.49	42.83	-2.66	2.68	0.01	yes	0.25
Stable Former	45.31	44.73	-0.58	1.78	0.08	no	0.05
Former to Current	42.42	41.06	-1.36	0.69	0.49	no	0.11

Stable Current

This group is defined as those who were smokers at P2 and remained smokers at P3.

The mean PCS scores in the Stable Current Smokers group at P2 ($M = 44.86$; $SD = 10.21$) was not significantly different from P3 ($M = 43.68$; $SD = 10.85$), $t(277) = 2.18$, $p = 0.03$, $d = 0.11$

Current to Former

This group is defined as those who were smokers at P2 but quit smoking before P3.

The mean PCS scores in the Current to Former Smokers group at P2 ($M = 45.49$; $SD = 10.50$) was significantly different from P3 ($M = 42.83$; $SD = 11.13$), $t(74) = 2.68$, $p < 0.01$, $d = 0.25$

Stable Former

This group is defined as those who were not smokers at P2 and remained non-smokers at P3.

The mean PCS scores in the Stable Former Smokers group at P2 ($M = 45.31$; $SD = 10.87$) was not significantly different from P3 ($M = 44.73$; $SD = 10.70$), $t(597) = 1.78$, $p = 0.08$, $d = 0.05$

Former to Current

This group is defined as those who were not smokers at P2 but began smoking before P3.

The mean PCS scores in the Former to Current Smokers group at P2 ($M = 42.42$; $SD = 12.54$) was not significantly different from P3 ($M = 41.06$; $SD = 12.17$), $t(31) = 0.69$, $p = 0.49$, $d = 0.11$

RQ4: Is there a significant change in MCS of health-related quality of life between P2 and P3 in the four longitudinal smoking status groups?

H_{14} : There is a significant change in MCS between P2 and P3 in the four longitudinal smoking status groups.

H_{04} : There is no significant change in MCS between P2 and P3 in the four longitudinal smoking status groups.

Table 7

Changes in Mean MCS Scores between P2 and P3

	P2	P3	Change	t value	p value	Significance	Effect Size
Stable Current	50.13	50.24	0.11	-0.17	0.87	no	0.01
Current to Former	51.37	50.99	-0.38	0.32	0.75	no	0.04
Stable Former	54.94	54.90	-0.04	0.09	0.93	no	0.00
Former to Current	48.56	48.72	0.16	-0.08	0.94	no	0.01

Stable Current

This group is defined as those who were smokers at P2 and remained smokers at P3.

The mean MCS scores in the Stable Current Smokers group at P2 (M = 50.13; SD = 10.72) was not significantly different from P3 (M = 50.24; SD = 10.83), $t(277) = -0.17$, $p = 0.87$, $d = 0.01$

Current to Former

This group is defined as those who were smokers at P2 but quit smoking before P3.

The mean MCS scores in the Current to Former Smokers group at P2 (M = 51.37; SD = 9.01) was not significantly different from P3 (M = 50.99; SD = 10.92), $t(74) = 0.32$, $p = 0.75$, $d = 0.04$

Stable Former

This group is defined as those who were not smokers at P2 and remained non-smokers at P3.

The mean MCS scores in the Stable Former Smokers group at P2 ($M = 54.94$; $SD = 8.30$) was not significantly different from P3 ($M = 54.90$; $SD = 9.24$), $t(597) = 0.09$, $p = 0.93$, $d = 0.00$

Former to Current

This group is defined as those who were not smokers at P2 but began smoking before P3.

The mean MCS scores in the Former to Current Smokers group at P2 ($M = 48.56$; $SD = 9.70$) was not significantly different from P3 ($M = 48.72$; $SD = 12.58$), $t(31) = -0.08$, $p = 0.94$, $d = 0.01$

SGRQ

SGRQ was designed to help investigators learn more about how the patient's breathing is disturbing them and how it affects their quality of life (Jones et al., 1992). This questionnaire is scored in three separate domains: symptoms, activity, and impacts. For this study, the impacts domain will be analyzed. The impacts domain measures the disturbances in social and psychological functioning (Nagai et al., 2015). This section includes questions asking the participant: if their respiratory condition causes them problems, if they have ever had to stop or change their line of work because of their respiratory problems, if they get breathless when conducting everyday activities such as walking or talking; other effects such as: if the respiratory condition is embarrassing in public, a nuisance to family and friends, if the sufferer panics when they cannot catch

their breath, if they feel they are not in control of their illness, they do not expect their condition to improve, if they feel frail or invalid because of their condition, and if everything seems to be too much of an effort; questions about their medication is asked: do they feel their medication is helping them, do they get embarrassed by using their medication in public, do they have unpleasant side effects from their medication, and does the medication interfere with their life; this section also asks how the illness affects their daily life, can they play sports or go out to do recreational activities, can they go shopping on their own, can they do housework, can they move from their chair or bed; finally, this section asks if their illness stops them from doing things they enjoy doing.

I used paired t tests to determine if there was a significant change between the P2 and P3 scores for the psychological and social functioning due to airflow limitations measured with the SGRQ in the four groups.

RQ5: Is there a significant change in psychological and social functioning due to airflow limitations (measured with the SGRQ) between P2 and P3 in the four longitudinal smoking status groups?

H_{15} : There is a significant change in SGRQ between P2 and P3 in the four longitudinal smoking status groups.

H_{05} : There is no significant change in SGRQ between P2 and P3 in the four longitudinal smoking status groups.

Table 8

Changes in Mean Impact Scores between P2 and P3

	P2	P3	Change	t value	p value	Significance	Effect Size
--	----	----	--------	------------	------------	--------------	----------------

Stable Current	13.65	15.74	2.09	-2.25	0.03	no	0.12
Current to Former	16.64	17.80	1.16	-0.62	0.54	no	0.06
Stable Former	11.28	12.89	1.61	-3.75	0.01	yes	0.10
Former to Current	9.65	16.86	7.21	-2.90	0.01	yes	0.42

Stable Current

This group is defined as those who were smokers at P2 and remained smokers at P3.

The mean impact scores in the Stable Current Smokers group at P2 (M = 13.65; SD = 16.82) was not significantly different from P3 (M = 15.74; SD = 18.53), $t(277) = -2.25$, $p = 0.03$, $d = 0.12$

Current to Former

This group is defined as those who were smokers at P2 but quit smoking before P3.

The mean impact scores in the Current to Former Smokers group at P2 (M = 16.64; SD = 17.91) was not significantly different from P3 (M = 17.80; SD = 18.68), $t(74) = -0.62$, $p = 0.54$, $d = 0.06$

Stable Former

This group is defined as those who were not smokers at P2 and remained non-smokers at P3.

The mean impact scores in the Stable Former Smokers group at P2 (M = 11.28; SD = 15.19) was significantly different from P3 (M = 12.89; SD = 15.76), $t(597) = -3.75$, $p < 0.01$, $d = 0.10$

Former to Current

This group is defined as those who were not smokers at P2 but began smoking before P3.

The mean impact scores in the Former to Current Smokers group at P2 ($M = 9.65$; $SD = 13.54$) was significantly different from P3 ($M = 16.86$; $SD = 19.87$), $t(31) = -2.90$, $p < 0.01$, $d = 0.42$

RQ6: Is there a relationship between longitudinal smoking status, anxiety, depression, health-related quality of life (PCS and MCS), and psychological and social functioning due to airflow limitations measured with the SGRQ at P3?

H_{16} : There is a relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36, and psychological and social functioning due to airflow limitations measured with the SGRQ.

H_{06} : There is no relationship between longitudinal smoking status, anxiety, depression, physical component score of the SF-36, mental component score of the SF-36, and psychological and social functioning due to airflow limitations measured with the SGRQ.

I ran a multiple regression analysis to determine if there was a relationship between longitudinal smoking status, anxiety, depression, health-related quality of life (PCS and MCS), and psychological and social functioning due to airflow limitations measured with the SGRQ at P3. I only analyzed those who were in the stable smoking group and the stable former smoking groups. I made this decision because a) these two groups are the largest, and b) the participants were not asked when they quit smoking,

i.e., the person could have reported being a former smoker at P3 with the cessation date being anywhere between 5 years before or one day before the study visit. The stable smoking group and the stable former smoking groups all have at least 5 years of steady smoking/non-smoking.

Anxiety, depression, and health-related quality of life (PCS and MCS) statistically significantly predicted psychological and social functioning due to airflow limitations measured with the SGRQ at P3, $F(5, 974) = 290.704, p < .001, R^2 = .774$. The R^2 (.774) indicated approximately 77% of the variation in the dependent variable (psychological and social functioning due to airflow limitations) was accounted for by the set of predictors (Anxiety, depression, health-related quality of life (PCS and MCS), and smoking status). In the final model, anxiety ($t = 4.35, p = .01$), depression ($t = 2.72, p = .01$) and health-related quality of life (PCS ($t = -26.4, p = .01$) and MCS ($t = -5.72, p = .01$)) provided significant contribution. The longitudinal smoking status did not statistically significantly predict psychological and social functioning due to airflow limitations measured with the SGRQ at P3.

Table 9

Regression Coefficients and Standard Errors

	B	SE B	β	t	p	95% CI for B	
						LL	UL
Mental Composite Score	-0.26	0.05	-0.16	-5.72	0.01	-0.35	-0.17
Physical Composite Score	-0.93	0.04	-0.62	-26.40	0.01	-1.00	-0.87
Smoking Status	0.65	1.01	0.01	0.65	0.52	-1.70	2.60
Anxiety	0.59	0.14	0.13	4.35	0.01	0.16	0.73
Depression	0.47	0.17	0.08	2.72	0.01	0.13	0.81

Summary

I found there was a significant change in anxiety scores for the Stable Former smokers' group. I found there was a significant change in the PCS of health-related quality of life in the Current to Former smokers' group. I found there was a significant change in the psychological and social functioning due to airflow limitations score for the Stable Former smokers' and the Former to Current smokers' groups. I also found that anxiety, depression, and health-related quality of life (PCS and MCS) statistically significantly predicted social functioning due to airflow limitations measured with the SGRQ at P3; smoking status did not statistically significantly predict social functioning due to airflow limitations measured with the SGRQ at P3.

Chapter 5

Purpose of the Study

The purpose of this research was to address the gap in the literature that concerns the lack of knowledge about long-term change in anxiety and depression scores and the scores of the SF-36, if a change in the patient's smoking status between P2 and P3 relates to a change in their depression, anxiety, and SF-36 scores over the course of 5-years, and if a change in anxiety, depression, and SF-36 scores relates to the patient's perception about their individual treatment efficacy. In this chapter, I will describe the key findings, the interpretation of findings, the limitations of the study, recommendations for future research, and implications for positive social change.

The key findings in this study are: There was a significant change in anxiety scores for the Stable Former smokers' group. There was a significant change in the PCS

of health-related quality of life in the Current to Former smokers' group. There was a significant change in the psychological and social functioning due to airflow limitations score for the Stable Former smokers' and the Former to Current smokers' groups. Anxiety, depression, and health-related quality of life (PCS and MCS) statistically significantly predicted social functioning due to airflow limitations measured with the SGRQ at P3; smoking status did not statistically significantly predict social functioning due to airflow limitations measured with the SGRQ at P3.

Interpretation of Findings

During my extensive review of existing literature, I established there has not been another study conducted that has looked at difference in anxiety and depression and difference in health-related quality of life over multiple years in adult COPD patients. It is important to determine if anxiety and depression increase over time in COPD patients; if these symptoms do become worse, this could further complicate the disease progression. Anxiety is shown to correlate with pulmonary-specific COPD symptoms. Anxiety is associated with higher levels of shortness of breath, fatigue, and frequency of COPD symptoms (Doyle et al., 2013). When the symptoms of anxiety and depression go unmanaged, there is a negative impact on functional capacity and quality of life in COPD patients (Lee et al., 2016). In the present study there was a significant decrease in anxiety scores for the Stable Former smokers' group between P2 and P3. This suggests that as time progresses, those who reported being former smokers at P2 and remained former smokers at P3 have lower levels of anxiety in P3 than they did in P2.

The Current to Former smokers' group reported a statistically significant decrease in the PCS of health-related quality of life. This suggests the participants in this group have had their physical health decrease between P2 and P3.

The impacts domain of the SGRQ measures the disturbances in social and psychological functioning (Nagai et al., 2015). There was a significant increase in the impacts domain score for the Stable Former smokers' group and the Former to Current smokers' group. This increase indicates the participants in these groups are experiencing significantly more limitations in P3 than they were in P2.

Anxiety, depression, and health-related quality of life (PCS and MCS) statistically significantly predicted social functioning due to airflow limitations measured with the SGRQ at P3; smoking status did not statistically significantly predict social functioning due to airflow limitations measured with the SGRQ at P3. This suggests that a person's health-related quality of life (PCS and MCS), level of anxiety, and level of depression has an impact on their psychological and social functioning due to airflow limitations.

The cognitive approach that I used in this study suggests that behavioral, emotional, and physical symptoms result from cognitive irregularity (Kinser & Lyon, 2014); this means that depressed patients think differently than those who are not depressed. This suggests that as disease progresses and the person's physical ability becomes more limited, then depression and anxiety will increase, and quality of life will decrease. While the findings of the first five research questions of this research project do not support this theory, the final question does. In the final question I discovered that a person's health-related quality of life (PCS and MCS), level of anxiety, and level of

depression has an impact on their psychological and social functioning due to airflow limitations.

Limitations of Study

COPDGene has an Ancillary Studies and Publications Committee that decides if an investigator is allowed access to their data and to ensure there is no overlapping of projects and publications within COPDGene. This committee meets twice a month on the first and third Thursdays. Once the data request is reviewed, the committee decides if the investigator is allowed access or if the request is deferred pending further information needed. Access to the data were granted.

There are limitations to consider when using self-report questionnaires. Self-reported measures showed greater concordance for monthly compared to yearly metrics (Short et al., 2009). This means that the accuracy of the answers provided on the self-report measure decreases as the recall period increases. The accuracy of self-report measures as quantitative measures has not been well documented (Tommelein et al., 2014).

Collection of HADS data ceased when the quarantine portion of the COVID-19 pandemic began in March 2020. COPDGene stopped in-person visits to protect both the subjects and staff. Some questionnaires were completed over the phone, but because the HADS is considered sensitive and could require medical follow-up, this measure was postponed until in-person visits begin once more and medical care providers are back in their offices so the coordinators can inform them of any alarming scores.

Recommendations for Future Research

During a review of findings from the current study I found areas which could further contribute to the analysis of longitudinal smoking status, depression, anxiety, health-related quality of life, and psychological and social functioning due to airflow limitations among adult patients with COPD who are enrolled in the COPDGene Cohort Study. Suggestions include: Recommend future research to look at numbers before the COVID-19 pandemic and after the pandemic to determine if the pandemic had an effect on participants; find other cohorts who have utilized the same measures and compare scores among different cohorts; take a closer look at the GOLD stage of the participants as a way to categorize instead of longitudinal smoking status change; once Phase 4 (P4) of COPDGene is underway, look at comparison of scores across P2, P3, and P4, for a total of 10 years between the first and last visits.

Implications for Positive Social Change

Considerable implications for social change exist based on the findings from this study. First is that the findings of the current study add to the body of knowledge about the relationship among longitudinal smoking status, depression, anxiety, health-related quality of life, and psychological and social functioning due to airflow limitations among adult patients with COPD who are enrolled in the COPDGene Cohort Study. The findings of this research will lead to positive social change by bringing awareness to the healthcare providers of the challenges that COPD patients face in relation to current management and mental health issues concerning their disease.

Summary

In this chapter I discussed, the interpretation of the findings on the current study, the limitations of this study, recommendations for future research in relation to this study, and the implications of this study on positive social change. There was a significant decrease in the mean anxiety scores for the Stable Former group, there was a significant decrease in the mean PCS scores for the Current to Former group, and both the Stable Former group and the Former to Current group had a significant increase in the mean score for psychological and social functions due to airflow limitations. A person's health-related quality of life (PCS and MCS), level of anxiety, and level of depression has an impact on their psychological and social functioning due to airflow limitations.

References

- Agustí, A., & Celli, B. (2017). Natural history of COPD: gaps and opportunities. *ERJ Open Research*, *3*(4), 1–11. <http://dx.doi.org/10.1183/23120541.00117-2017>
- A.D.A.M. Medical Encyclopedia. (2012). *Major depression*.
<http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001941/>
- Aczel, B., Palfi, B., Szollosi, A., Kovacs, M., Szecsi, B., Szecsi, P., . . . Wagenmakers, E. (2018). Quantifying support for the null hypothesis in psychology: An empirical investigation. *Advances in Methods and Practices in Psychological Science*, *1*(3), 357–366. <http://dx.doi.org/10.1177/2515245918773742>
- Albert, P. (2015). Why is depression more prevalent in women? *Journal of Psychiatry and Neuroscience*, *40*(4), 219–221. <http://dx.doi.org/10.1503/jpn.150205>
- Allen, M. (2017). *The sage encyclopedia of communication research methods (Vols. 1–4)*. SAGE Publications, Inc.
- Alonso, J., & Lepine, J. (2007). Overview of key data from the European study of the epidemiology of mental disorders (ESEMeD). *Journal of Clinical Psychiatry*, *68*(2), 3–9.
- American Lung Association. (2020). *COPD causes and risk factors*.
<https://www.lung.org/lung-health-diseases/lung-disease-lookup/copd/what-causes-copd>

American Psychological Association. (2020). *Anxiety*.

<https://www.apa.org/topics/anxiety/>

American Psychological Association. (2020). *Depression*.

<https://www.apa.org/topics/depression/index>

American Thoracic Society. (2018). *Exacerbation of COPD*.

<https://www.thoracic.org/patients/patient-resources/resources/copd-exacerbation-ecopd.pdf>

American Thoracic Society. (2020). *St. George's Respiratory Questionnaire (SGRQ)*.

<https://www.thoracic.org/members/assemblies/assemblies/srn/questionnaires/sgrq.php>

Anderson, J., Michalak, E., & Lam, R. (2002). Depression in primary care: Tools for screening, diagnosis, and measuring response to treatment. *BC Medical Journal*, *44*(8), 415–419.

Asalgoo, S., Jahromi, G., Meftahi, G., & Sahraei, H. (2015). Posttraumatic stress disorder (PTSD): Mechanisms and possible treatments. *Neurophysiology*, *47*(6), 482–489.

<http://dx.doi.org/10.1007/s11062-016-9559-9>

Babb, S., Malarcher, A., Schauer, G., Asman, K., & Jamal, A. (2017). Quitting smoking among adults. *Weekly*, *65*(52), 1457–1464.

Bandelow, B., Michaelis, S., & Wedekind, D. (2017). Treatment of anxiety disorders.

Dialogues in Clinical Neuroscience, *19*(2), 93–107.

- Bosc, M. (2000). Assessment of social functioning in depression. *Comprehensive Psychiatry*, 41(1), 63–69. [http://dx.doi.org/10.1016/S0010-440X\(00\)90133-0](http://dx.doi.org/10.1016/S0010-440X(00)90133-0)
- Bosnic-Anticevich, S., Chrystyn, H., Costello, R., Dolovich, M., Fletcher, M., Lavorini, F., . . . Price, D. (2017). The use of multiple respiratory inhalers requiring different inhalation techniques has an adverse effect on COPD outcomes. *International Journal of Chronic Obstructive Pulmonary Disease*, 12, 59–71. <http://dx.doi.org/10.2147/COPD.S117196>
- Bradford, S., & Cullen, F. (2012). *Research and research methods for youth practitioners*. Routledge.
- Bratås, O., Espnes, G., Rannestad, T., & Walstad, R. (2010). Pulmonary rehabilitation reduces depression and enhances health-related quality of life in COPD patients - especially in patients with mild or moderate disease. *Chronic Respiratory Disease*, 7(4), 229–237. <http://dx.doi.org/10.1177/1479972310374343>
- Bratås, O., Grønning, K., & Forbord, T. (2014). Psychometric properties of the Hospital Anxiety and Depression Scale and The General Health Questionnaire-20 in COPD inpatients. *Scandinavian Journal of Caring Sciences*, 28(2), 413–420. <http://dx.doi.org/10.1111/scs.12054>
- Brown, T. A., & Rosellini, A. J. (2011). The direct and interactive effects of neuroticism and life stress on the severity and longitudinal course of depressive symptoms. *Journal Of Abnormal Psychology*, 120(4), 844–856. <http://dx.doi.org/10.1037/a0023035>

- Butcher, J. N., Mineka, S., & Hooley, J. M. (2013). *Abnormal psychology (15th ed)*. Pearson Education, Inc.
- Cannon, D., Buys, N., Sriram, K., Sharma, S., Morris, N., & Sun, J. (2016). The effects of chronic obstructive pulmonary disease self-management interventions on improvement of quality of life in COPD patients: A meta-analysis. *Respiratory Medicine*, *121*, 81–90. <http://dx.doi.org/10.1016/j.rmed.2016.11.005>
- Carr, L. (1994). The strengths and weaknesses of quantitative and qualitative research: what method for nursing? *Journal of advanced nursing*, *20*(4), 716–721. <http://dx.doi.org/10.1046/j.1365-2648.1994.20040716.x>
- Caruana, E., Roman, M., Hernández-Sánchez, J., & Solli, P. (2015). Longitudinal studies. *Journal of Thoracic Disease*, *7*(11), E537–E540.
- Centers for Disease Control and Prevention. (2015). *International classification of diseases, (ICD-10-CM/PCS) transition - background*. https://www.cdc.gov/nchs/icd/icd10cm_pcs_background.htm
- Centers for Disease Control and Prevention. (2020). *Living with COPD*. <https://www.cdc.gov/features/copd/index.html>
- Cho, W., Lee, C., & Kim, L. (2019). COPD as a disease of immunosenescence. *Yonsei Medical Journal*, *60*(5), 407–413. <http://dx.doi.org/10.3349/ymj.2019.60.5.407>
- Clark, D., Beck, A., & Alford, B. (1999). *Scientific foundations of cognitive theory and therapy of depression*. John Wiley & Sons, Inc.

- Coggon, D., Rose, G., & Barker, D. (2003). *Epidemiology for the uninitiated (fourth edition)*. MBJ Publishing Group.
- Connor, K., & Blazer, D. (2007). Anxiety disorders. In J. Birren, *Encyclopedia of Gerontology (second edition)* (pp. 84–89). Elsevier Inc.
- COPD Foundation. (2018). *What is COPD?* <https://www.copdfoundation.org/What-is-COPD/Understanding-COPD/What-is-COPD.aspx>
- COPDGene. (2019). *Research*. <http://www.copdgene.org/research>
- Creagan, E., Bauer, B., Thomley, B., & Borg, J. (2015). Animal-assisted therapy at Mayo Clinic: The time is now. *Complementary Therapies in Clinical Practice, 21*, 101–104. <http://dx.doi.org/10.1016/j.ctcp.2015.03.002>
- Cucato, G., Zerati, A., Chehuen, M., Ritti-Dias, R., Saez, G., Ragazzo, L., & Wolosker, N. (2013). Comparison between subjective and objective methods to assess functional capacity during clinical treatment in patients with intermittent claudication. *Einstein, 11(4)*, 495–499.
- DeVellis, R. (2017). *Scale development: Theory and applications (fourth edition)*. Sage Publications.
- Dhabhar, F. (2018). The short-term stress response – Mother nature’s mechanism for enhancing protection and performance under conditions of threat, challenge, and opportunity. *Frontiers in Neuroendocrinology, 49*, 175–192. <http://dx.doi.org/10.1016/j.yfrne.2018.03.004>

- Dictionary.com. (2020). *Vitality*. <https://www.dictionary.com/browse/vitality>
- Ding, B., Small, M., & Homgren, U. (2017). a cross-sectional survey of current treatment and symptom burden of patients with COPD consulting for routine care according to gOLD 2014 classifications. *International Journal of COPD*, *12*, 1527–1537. <http://dx.doi.org/10.2147/COPD.S133793>
- Divo, M., Cote, C., de Torres, J., Casanova, C., Marin, J., Pinto-Plata, V., . . . Celli, B. (2012). Comorbidities and risk of mortality in patients with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine*, *186*(2), 155–161. <http://dx.doi.org/10.1164/rccm.201201-0034OC>
- Djukanovic, I., Carlsson, J., & Årestedt, K. (2017). Is the Hospital Anxiety and Depression Scale (HADS) a valid measure in a general population 65-80 years old? A psychometric evaluation study. *Health and Quality of Life Outcomes*, *15*(1), 1–10. <http://dx.doi.org/10.1186/s12955-017-0759-9>
- Donovan, H. (2007). EHR & EDC: Tomorrow's technology today. *Applied Clinical Trials*, *16*(2), 54–62.
- Dowson, C., Laing, R., Barraclough, R., Town, I., & Mulder, R. (2001). The use of the Hospital Anxiety and Depression Scale (HADS) in patients with chronic obstructive pulmonary disease: a pilot study. *New Zealand Medical Journal*, *114*(1141), 447–449.

- Doyle, T., Palmer, S., Johnson, J., Babyak, M., Smith, P., Mabe, S., . . . Blumenthal, J. (2013). Association of anxiety and depression with pulmonary-specific symptoms in chronic obstructive pulmonary disease. *International Journal of Psychiatric Medicine, 45*(2), 189–202. <http://dx.doi.org/10.2190/PM.45.2.g>
- Epel, E. (2009). Psychological and metabolic stress: A recipe for accelerated cellular aging? *Hormones (Athens), 8*(1), 7–22. <http://dx.doi.org/10.14310/horm.2002.1217>
- Evans, C. (2018). *Analysing semi-structured interviews using thematic analysis: Exploring voluntary civic participation among adults*. <https://pdfs.semanticscholar.org/e925/d9c92780a1d56af25fc01c8f2cdbf14d6c6e.pdf>
- Felman, A. (2018). *What causes anxiety?* <https://www.medicalnewstoday.com/articles/323456>
- Fiore, K. (2008, September 12). *Depression Stalks Five Percent of U.S. Population*. <http://www.medpagetoday.com/Psychiatry/Depression/10901>
- Flick, U. (2009). *An introduction to qualitative research*. SAGE Publishing.
- Frandsen, M., Thorpe, M., Shiffman, S., & Ferguson, S. (2017). A clinical overview of nicotine dependence and withdrawal. In F. Hall, J. Young, & A. Der-Avakian, *Negative affective states and cognitive impairments in nicotine dependence* (pp. 205–215). Elsevier Inc.

- Franken, R. E. (2007). *Human motivation (6th edition)*. Wadsworth Cengage Learning.
- Franssen, F., Smid, D., Deeg, D., Huisman, M., Poppelaars, J., Wouters, E., & Spruit, M. (2018). The physical, mental, and social impact of COPD in a population-based sample: results from the Longitudinal Aging Study Amsterdam. *npj Prim Care Resp Med*, *28(1)*, 1–6. <http://dx.doi.org/10.1038/s41533-018-0097-3>
- Gandek, B., Sinclair, S., Kosinski, M., & Ware, J. (2004). Psychometric evaluation of the SF-36® health survey in medicare managed care. *Health Care Financing Review*, *24(4)*, 5–25.
- Haggerty, M. J. (2012). *Psychodynamic Therapy*.
<http://psychcentral.com/lib/2006/psychodynamic-therapy/>
- Hanania, N., Müllerova, N., Locantore, N., Vestbo, J., Watkins, M., Wouters, E., . . . Sharafkhaneh, A. (2011). Determinants of depression in the ECLIPSE chronic obstructive pulmonary disease cohort. *American Journal of Respiratory and Critical Care Medicine*, *183(5)*, 604–611. <http://dx.doi.org/10.1164/rccm.201003-0472OC>
- Heinzman, J., Hoth, K., Cho, M., Sakornsakolpat, P., Regan, E., Make, B., . . . Shinozaki, G. (2019). GWAS and systems biology analysis of depressive symptoms among smokers from the COPDGene cohort. *Journal of Affective Disorders*, *243*, 16–22.
<http://dx.doi.org/10.1016/j.jad.2018.09.003>

- Hill, K., Geist, R., Goldstein, R., & Lacasse, Y. (2008). Anxiety and depression in end-stage COPD. *European Respiratory Journal*, *31*(3), 667–677.
<http://dx.doi.org/10.1183/09031936.00125707>
- Iyer, A., Holm, K., Bhatt, S., Kim, V., Kinney, G., Wamboldt, F., . . . Hoth, K. (2019). Symptoms of anxiety and depression and use of anxiolytic-hypnotics and antidepressants in current and former smokers with and without COPD - A cross sectional analysis of the COPDGene cohort. *Journal of Psychosomatic Research*, *118*, 18–26. <http://dx.doi.org/10.1016/j.jpsychores.2019.01.002>
- Jamal, A., Phillips, E., Gentzke, A., Homa, D., Babb, S., King, B., & Neff, L. (2018). Current cigarette smoking among adults . *Weekly*, *67*(2), 53–59.
- Janssen, D., Spruit, M., Leue, C., Gijzen, C., Hameleers, H., Schols, J., . . . On behalf of the Ciro network. (2010). Symptoms of anxiety and depression in COPD patients entering pulmonary rehabilitation. *Chronic Respiratory Disease*, *7*(3), 147–157.
<http://dx.doi.org/10.1177/1479972310369285>
- Jenkins, C., Chapman, K., Donohue, J., Roche, N., Tsiligianni, I., & Han, M. (2017). Improving the management of COPD in women. *Chest*, *151*(3), 686–696.
<http://dx.doi.org/10.1016/j.chest.2016.10.031>
- Jones, P., & Forde, Y. (2009). *St. George's Respiratory Questionnaire Manual*. London, England.

- Jones, P., Quirk, F., Baveystock, C., & Littlejohns, P. (1992). A self-complete measure of health status for chronic airflow limitation. The St. George's Respiratory Questionnaire. *The American Review of Respiratory Disease*, *145*(6), 1321–1327. <http://dx.doi.org/10.1164/ajrccm/145.6.1321>
- Kinser, P., & Lyon, D. (2014). A conceptual framework of stress vulnerability, depression, and health outcomes in women: Potential uses in research on complementary therapies for depression. *Brain and Behavior*, *4*(5), 665–674. <http://dx.doi.org/10.1002/brb3.249>
- Kinser, P., Goehler, L., & Taylor, A. (2012). How might yoga help depression? A neurobiological perspective. *EXPLORE (NY)*, *8*(2), 118–126. <http://dx.doi.org/10.1016/j.explore.2011.12.005>
- Kuppens, P., Sheeber, L. B., Yap, M. H., Whittle, S., Simmons, J. G., & Allen, N. B. (2012). Emotional inertia prospectively predicts the onset of depressive disorder in adolescence. *Emotion*, *12*(2), 283–289. <http://dx.doi.org/10.1037/a0025046>
- Laerd Statistics. (2018). *Independent t-test for two samples*. <https://statistics.laerd.com/premium/spss/pstt/paired-samples-t-test-in-spss-7.php>
- Laerd Statistics. (2018). *Multiple regression*. <https://statistics.laerd.com/spss-tutorials/multiple-regression-using-spss-statistics.php>
- Lee, J., Fan, V., Borson, S., Pike, K., Jarrett, M., & Nguyen, H. (2016). A structural equation modeling analysis of the relationships between symptoms and physical

performance in patients with COPD. *The American Journal of Respiratory and Critical Care Medicine*, 193, A2899.

Limsuwat, C., McClellan, R., Amiri, H., & Nugent, K. (2014). Pulmonary rehabilitation improves only some domains of health-related quality of life measured by the Short Form-36 questionnaire. *Annals of Thoracic Medicine*, 9(3), 144–148.

Lins, L., & Carvalho, F. (2016). SF-36 total score as a single measure of health-related quality of life: Scoping review. *SAGE Open Medicine*, 4, 1–12.

<http://dx.doi.org/10.1177/2050312116671725>

Liu, M., & Wang, Y. (2016). Comparison of face-to-face and web surveys on the topic of homosexual rights. *Journal of Homosexuality*, 63(6), 17.

<http://dx.doi.org/10.1080/00918369.2015.1112587>

Lo, C., Liang, W., Hang, L., Wu, T., Chang, Y., & Chang, C. (2015). A psychometric assessment of the St. George's respiratory questionnaire in patients with COPD using rasch model analysis. *Health and Quality of Life Outcomes*, 13(1), 131.

<http://dx.doi.org/10.1186/s12955-015-0320-7>

Lu, Y., Li, P., Li, N., Wang, Z., Li, J., Liu, X., & Wu, W. (2020). Effects of home-based breathing exercises in subjects with COPD. *Respiratory Care*, 65(3), 377–387.

<http://dx.doi.org/10.4187/respcare.07121>

MacNee, W. (2006). Pathology, pathogenesis, and pathophysiology. *BMJ*, 332(7551),

1202–1204. <http://dx.doi.org/10.1136/bmj.332.7551.1202>

- Mahesh, P., Gunathunga, M., Jayasinghe, S., & Arnold, S. (2018). Quality of life and its associated factors among patients with COPD before and after an exacerbation in Sri Lanka. *Journal of the College of Community Physicians of Sri Lanka*, 24(2), 66. <http://dx.doi.org/10.4038/jccpsl.v24i2.8146>
- Mayo Clinic. (2019). *Atypical depression*. <https://www.mayoclinic.org/diseases-conditions/atypical-depression/symptoms-causes/syc-20369747>
- Mayo Clinic. (2020). *Anxiety disorders*. <https://www.mayoclinic.org/diseases-conditions/anxiety/symptoms-causes/syc-20350961>
- Mayo Clinic. (2020). *COPD*. <https://www.mayoclinic.org/diseases-conditions/copd/symptoms-causes/syc-20353679>
- Mayo Foundation for Medical Education and Research. (2012). *Depression (major depression)*. <http://www.mayoclinic.com/health/depression/DS00175/DSECTION=treatments-and-drugs>
- McColl, E., Jacoby, A., Thomas, L., Soutter, J., Bamford, C., Steen, N., . . . Bond, J. (2001). Design and use of questionnaires: A review of best practice applicable to surveys of health service staff and patients. *Health Technology Assessment*, 5(31), Core Research. <http://dx.doi.org/10.3310/hta5310>
- McLeod, S. (2019). *Qualitative vs. quantitative research*. <https://www.simplypsychology.org/qualitative-quantitative.html>

Mental Health America. (2012). *Ranking America's mental health: An analysis of depression across the States*. <http://www.nmha.org/go/state-ranking>

Merriam-Webster Dictionary. (2020). *Pain*. <https://www.merriam-webster.com/dictionary/pain#:~:text=>

Mi Jang, S., Uk Kim, K., Jung Na, H., Eun Song, S., Hee Lee, S., Lee, H., . . . Park, H. (2018). Depression is a major determinant of both disease-specific and generic health-related quality of life in people with severe COPD. *Chronic Respiratory Disease, 16*, 1–8. <http://dx.doi.org/10.1177/1479972318775422>

Nagai, K., Makita, H., Suzuki, M., Shimizu, K., Konno, S., Ito, Y., & Nishimura, M. (2015). Differential changes in quality of life components over 5 years in chronic obstructive pulmonary disease patients. *International Journal of Chronic Obstructive Pulmonary Disease, 10*, 745–757. <http://dx.doi.org/10.2147/COPD.S77586>

National Association of Cognitive-Behavioral Therapists. (2010). *Cognitive-Behavioral Therapy*. <http://www.nacbt.org/whatiscbt.htm>

National Heart, Lung, and Blood Institute. (2019). *COPD*. <https://www.nhlbi.nih.gov/health-topics/copd>

National Institute of Mental Health. (2018). *Anxiety disorders*. <https://www.nimh.nih.gov/health/topics/anxiety-disorders/index.shtml>

- Nicolacakis, K. (2015). ICD-10-CM coding for pulmonary medicine american thoracic society clinical practice committee webinar. (pp. 1–58). American Thoracic Society.
- Nogrady, B. (2018). Increased COPD mortality with secondhand smoke exposure in childhood. *American Journal of Preventative Medicine*, *55*(3), 345–352.
- Norwood, R. (2006). Prevalence and impact of depression in chronic obstructive pulmonary disease patients. *Current Opinion in Pulmonary Medicine*, *12*(2), 113–117. <http://dx.doi.org/10.1097/01.mcp.0000208450.50231.c6>
- O'Donnell, D., Webb, K., & Neder, J. (2015). Lung hyperinflation in COPD: applying physiology to clinical practice. *COPD Research and Practice*, *1*(1), 4. <http://dx.doi.org/10.1186/s40749-015-0008-8>
- Ohi, K., Otowa, T., Shimada, M., Sasaki, T., & Tanii, H. (2020). Shared genetic etiology between anxiety disorders and psychiatric and related intermediate phenotypes. *Psychological Medicine*, *50*(4), 692–704. <http://dx.doi.org/10.1017/S003329171900059X>
- Ozalevli, S., Karaali, H., Cankurtaran, F., Kilinc, O., & Akkoçlu, A. (2008). Comparison of Short Form-36 Health Survey and Nottingham Health Profile in moderate to severe patients with COPD. *Journal of Evaluation in Clinical Practice*, *14*(4), 493–499. <http://dx.doi.org/10.1111/j.1365-2753.2007.00904.x>

- Paine, N., Bacon, S., Bourbeau, J., Tan, W., Laboie, K., for the CanCOLD Study Investigators, . . . the CanCOLD Collaborative Research Group. (2019). Psychological distress is related to poor health behaviors in COPD and non-COPD patients: Evidence from the CanCOLD study. *Respiratory Medicine*, 146, 1–9.
- Painter, P., Stewart, A., & Carey, S. (1999). Physical functioning: definitions, measurement, and expectations. *Advances in Renal Replacement Therapy*, 6(2), 110–123. [http://dx.doi.org/10.1016/S1073-4449\(99\)70028-2](http://dx.doi.org/10.1016/S1073-4449(99)70028-2)
- Petty, T. (2006). The history of COPD. *International Journal of Chronic Obstructive Pulmonary Disease*, 1(1), 3–14. <http://dx.doi.org/10.2147/copd.2006.1.1.3>
- Picciotto, M., & Kenny, P. (2020). Mechanisms of nicotine addiction. *Cold Springs Harbor Perspectives in Medicine*, 10(6), a039610. <http://dx.doi.org/10.1101/cshperspect.a039610>
- Plank, J. (2018). *The history of COPD*. <https://copd.net/living/history/>
- Pooler, A., & Beech, R. (2014). Examining the relationship between anxiety and depression and exacerbations of COPD which result in hospital admission: A systematic review. *International Journal of COPD*, 9, 315–330. <http://dx.doi.org/10.2147/COPD.S53255>
- Preedy, V., & Watson, R. (2010). *Handbook of Disease Burdens and Quality of Life Measures*. Springer.

- Prokscha, S. (2012). *Practical guide to clinical data management (3rd edition)*. Taylor & Francis Group, LLC.
- Raghavan, D., Varkey, A., & Bartter, T. (2017). Chronic obstructive pulmonary disease: the impact of gender. *Current Opinion in Pulmonary Medicine*, 23(2), 117–123.
<http://dx.doi.org/10.1097/MCP.0000000000000353>
- RAND Health Care. (2020). *36-item short form survey (SF-36)*.
https://www.rand.org/health-care/surveys_tools/mos/36-item-short-form.html
- Regan, E., Hokanson, J., Murphy, J., Make, B., Lynch, D., Beaty, T., . . . Crapo, J. (2010). Genetic epidemiology of COPD (COPDGene) study design. *COPD*, 7(1), 32–43. <http://dx.doi.org/10.3109/15412550903499522>
- Rehabilitation Measures Database. (2012). *Hospital anxiety and depression scale*.
<https://www.sralab.org/rehabilitation-measures/hospital-anxiety-and-depression-scale>
- Ridwan, E., Hadi, H., Wu, Y., & Tsai, P. (2019). Effects of transitional care on hospital readmission and mortality rate in subjects with COPD: a systematic review and meta-analysis. *Respiratory Care*, 64(9), 1146–1156.
<http://dx.doi.org/10.4187/respcare.06959>
- Roche, N., Antoniadis, A., Hess, D., Li, P., Kelkel, E., Leroy, S., . . . COLIBRI COPD Research Group. (2019). Are there specific clinical characteristics associated with

physician's treatment choices in COPD? *Respiratory Research*, 20(1), 1–11.

<http://dx.doi.org/10.1186/s12931-019-1156-1>

Rogliani, P., Calzetta, L., Coppola, A., Cavalli, F., Ora, J., Puxeddu, E., . . . Cazzola, M.

(2017). Optimizing drug delivery in COPD The role of inhaler devices.

Respiratory Medicine, 124, 6–14. <http://dx.doi.org/10.1016/j.rmed.2017.01.006>

Rogliani, P., Ora, J., Puxeddu, E., Matera, M., & Cazzola, M. (2017). Adherence to

COPD treatment: Myth and reality. *Respiratory Medicine*, 129, 117–123.

<http://dx.doi.org/10.1016/j.rmed.2017.06.007>

Rosińczuk, J., Przyszlak, M., & Uchmanowicz, I. (2018). Sociodemographic and clinical

factors affecting the quality of life of patients with chronic obstructive pulmonary

disease. *International Journal of Chronic Obstructive Pulmonary Disease*, 13,

2869–2882. <http://dx.doi.org/10.2147/COPD.S165714>

Safka, K., Wald, J., Wang, H., & McIvor, L. (2017). GOLD stage and treatment in

COPD: A 500 patient point prevalence study. *Chronic Obstructive Pulmonary*

Diseases, 4(1), 45–55. <http://dx.doi.org/10.15326/jcopdf.4.1.2016.0126>

Sarris, J., & Mischoulon, D. (2017). Treatments for comorbid anxiety and mood

disorders. In D. Camfield, E. McIntyre, & J. Sarris, *Evidence-based herbal and nutritional treatments for anxiety in psychiatric disorders* (pp. 103–119).

Springer.

- Sayiner, A., Alzaabi, A., Obeidat, N., Nejjari, C., Beji, M., & Uzaslan, E. (2012). Attitudes and beliefs about COPD: Data from the BREATHE study. *Respiratory Medicine*, *106*(S2), S60–S74. [http://dx.doi.org/10.1016/S0954-6111\(12\)70015-X](http://dx.doi.org/10.1016/S0954-6111(12)70015-X)
- Schauer, G., Wheaton, A., Malarcher, A., & Croft, J. (2016). Health-care provider screening and advice for smoking cessation among smokers with and without COPD: 2009-2010 national adult tobacco survey. *Chest*, *149*(3), 676–684.
- Sharma, K., Jain, A., Goyal, V., Goel, N., Takhar, R., Singh, V., . . . Samaria, J. (2019). Depression and physical activity impairment in COPD subjects. *Journal of Clinical and Diagnostic Research*, *13*(7), 16–22. <http://dx.doi.org/10.7860/JCDR/2019/41581.13008>
- Shields, M., Hovdestad, W., Gilbert, C., & Tonmyr, L. (2016). Childhood maltreatment as a risk factor for COPD: Findings from a population-based survey of Canadian adults. *International Journal of COPD*, *11*, 2641–2650. <http://dx.doi.org/10.2147/COPD.S107549>
- Short, M., Goetzel, R., Pei, X., Tabrizi, M., Ozminkowski, R., Gibson, T., . . . Wilson, M. (2009). How accurate are self-reports? An analysis of self-reported healthcare utilization and absence when compared to administrative data. *Journal of Occupational and Environmental Medicine*, *51*(7), 786–796.
- Siltanen, H., Aine, T., Huhtala, H., Kaunonen, M., Vasankari, T., & Paavilainen, E. (2020). Psychosocial issues need more attention in COPD self-management

education. *Scandinavian Journal of Primary Health Care*, 38(1), 47–55.

<http://dx.doi.org/10.1080/02813432.2020.1717087>

Simon, M., & Goes, J. (2013). Ex post facto research. In *Dissertation and scholarly research: Recipes for success* (pp. 1–2). Dissertation Success LLC.

Slee, A., Nazareth, I., Bondaroneck, P., Liu, Y., Cheng, Z., & Freemantle, N. (2019).

Pharmacological treatments for generalised anxiety disorder: A systemic review and network meta-analysis. *The Lancet*, 393(10173), 768–777.

[http://dx.doi.org/10.1016/S0140-6736\(18\)31793-8](http://dx.doi.org/10.1016/S0140-6736(18)31793-8)

Small, G. (2002). What we need to know about age related memory loss. *BMJ*,

324(7352), 1502–1505. <http://dx.doi.org/10.1136/bmj.324.7352.1502>

Snaith, R. (2003). The hospital anxiety and depression scale. *Health and Quality of Life*

Outcomes, 1(29), 1–4.

Statistics Solutions. (2020). *Paired sample t-test*.

<https://www.statisticssolutions.com/manova-analysis-paired-sample-t-test/>

Stavem, K., Erikssen, J., & Boe, J. (1999). Performance of a short lung-specific health

status measure in outpatients with chronic obstructive pulmonary disease.

Respiratory Medicine, 93(7), 467–475. <http://dx.doi.org/10.1016/S0954->

[6111\(99\)90089-6](http://dx.doi.org/10.1016/S0954-6111(99)90089-6)

Stenling, A., Hassmén, P., & Holmström, S. (2014). Implicit beliefs of ability, approach-

avoidance goals and cognitive anxiety among team sport athletes. *European*

Journal of Sport Science, 14(7), 720–729.

<http://dx.doi.org/10.1080/17461391.2014.901419>

Stern, A. (2014). The hospital anxiety and depression scale. *Occupational Medicine*, 64(5), 393–394. <http://dx.doi.org/10.1093/occmed/kqu024>

Surgeon General. (2014). *The health consequences of smoking-50 years of progress*. Rockville, MD: US Department of Health and Human Services.

Thomson, C., & Panacek, E. (2007). Research study designs: Non-experimental. *Air Medical Journal*, 26(1), 18–22.

Tommelein, E., Mehuys, E., Van Tongelen, I., Brusselle, G., & Boussery, K. (2014). Accuracy of the medication adherence report scale (MARS-5) as a quantitative measure of adherence to inhalation medication in patients with COPD. *Annals of Pharmacotherapy*, 48(5), 589–595. <http://dx.doi.org/10.1177/1060028014522982>

Tsiligianni, I., Rodriguez, M., Lisspers, K., LeeTan, T., & Infantino, A. (2017). Call to action: Improving primary care for women with COPD. *Primary Care Respiratory Medicine*, 27(1), 1–6. <http://dx.doi.org/10.1038/s41533-017-0013-2>

U.S. Department of Health and Human Services. (2014). *What are the five major types of anxiety disorders?* <https://www.hhs.gov/answers/mental-health-and-substance-abuse/what-are-the-five-major-types-of-anxiety-disorders/index.html>

Uliaszek, A. A., Zinbarg, R. E., Mineka, S., Craske, M. G., Griffith, J. W., Sutton, J. M., . . . Hammen, C. (2012). A longitudinal examination of stress generation in

depressive and anxiety disorders. *Journal Of Abnormal Psychology*, *121(1)*, 4–15.

<http://dx.doi.org/10.1037/a0025835>

Vanderbilt. (2018). *REDCap about*. <https://www.project-redcap.org/about/>

Vocabulary.com. (2020). *Role*. <https://www.vocabulary.com/dictionary/role>

von Leupoldt, A., Fritzsche, A., Trueba, A., Meuret, A., & Ritz, T. (2012). Behavioral medicine approaches to chronic obstructive pulmonary disease. *Annals of Behavioral Medicine*, *44(1)*, 52–65. <http://dx.doi.org/10.1007/s12160-012-9348-7>

von Leupoldt, A., Taube, K., Lehmann, K., Fritzsche, A., & Magnussen, H. (2011). The Impact of Anxiety and Depression on Outcomes of Pulmonary Rehabilitation in Patients With COPD. *CHEST*, *140(3)*, 730–736.

<http://dx.doi.org/10.1378/chest.10-2917>

Walther, B., Hossin, S., Townend, J., Abernethy, N., Parker, D., & Jefferies, D. (2011). Comparison of electronic data capture (EDC) with the standard data capture method for clinical trial data. *Plos ONE*, *6(9)*, 1–11.

<http://dx.doi.org/10.1371/journal.pone.0025348>

Wang, M., Beal, D., Chan, D., Newman, D., Vancouver, J., & Vandenberg, R. (2017). Longitudinal research: A panel discussion on conceptual issues, research design, and statistical techniques. *Work, Aging and Retirement*, *3(1)*, 1–24.

<http://dx.doi.org/10.1093/workar/waw033>

- Wilke, S., Janssen, D., Wouters, E., Schols, J., Franssen, F., & Spruit, M. (2012). Correlations between disease-specific and generic health status questionnaires in patients with advanced COPD: A one year observational study. *Health and Quality of Life Outcomes*, *10*(1), 1–12. <http://dx.doi.org/10.1186/1477-7525-10-98>
- Wilson, C., Jones, P., O'Leary, C., Cole, W., & Wilson, R. (1996). Validation of the St. George's Respiratory Questionnaire in bronchiectasis. *American Journal of Respiratory and Critical Care Medicine*, *156*(2), 536–541. <http://dx.doi.org/10.1164/ajrccm.156.2.9607083>
- World Health Organization. (2018). *Depression*. <https://www.who.int/news-room/fact-sheets/detail/depression>
- World Health Organization. (2018, March 30). *Mental health: strengthening our response*. <https://www.who.int/news-room/fact-sheets/detail/mental-health-strengthening-our-response>
- World Health Organization. (2020). *COPD: Definition*. <https://www.who.int/respiratory/copd/definition/en/#:~:text=>
- World Health Organization. (2020). *ICD-10-CM official guidelines for coding and reporting*. https://www.cdc.gov/nchs/data/icd/10cmguidelines-FY2020_final.pdf
- World Health Organization. (2020). *WHOQOL: Measuring quality of life*. <https://www.who.int/healthinfo/survey/whoqol-qualityoflife/en/>

- Xu, W., Collet, J., Shapiro, S., Lin, Y., Yang, T., Platt, R., . . . Bourbeau, J. (2008). Independent effect of depression and anxiety on chronic obstructive pulmonary disease exacerbations and hospitalizations. *American Journal of Respiratory and Critical Care Medicine*, *178*(9), 913–920. <http://dx.doi.org/10.1164/rccm.200804-619OC>
- Yin, M., Wang, H., Hu, X., Li, X., Fei, G., & Yu, Y. (2019). Patterns of brain structural alteration in COPD with different levels of pulmonary function impairment and its association with cognitive deficits. *BMC Pulmonary Medicine*, *19*(1), 1–10. <http://dx.doi.org/10.1186/s12890-019-0955-y>
- Yohannes, A., Müllerová, H., Hanania, N., Lavoie, K., Tal-Singer, R., Vestbo, J., . . . Wouters, E. (2016). Long-term course of depression trajectories in patients with COPD. *CHEST*, *149*(4), 916–926. <http://dx.doi.org/10.1016/j.chest.2015.10.081>
- Zamzam, M., Azab, N., Wahsh, R., Ragab, A., & Allam, E. (2013). Quality of life in COPD patients. *Egyptian Journal of Chest Diseases and Tuberculosis*, *61*(4), 281–289. <http://dx.doi.org/10.1016/j.ejcdt.2012.08.012>
- Zhang, X., Kuchinke, L., Woud, M., Velten, J., & Margraf, J. (2017). Survey method matters: Online/offline questionnaires and face-to-face or telephone interviews differ. *Computers In Human Behavior*, *71*, 172–180. <http://dx.doi.org/10.1016/j.chb.2017.02.006>