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Increasing Knowledge About Alpha-1 Antitrypsin Deficiency in the Chronic Obstructive Pulmonary Disease Population

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Walden University

College of Health Sciences

This is to certify that the doctoral study by

Maureen Barta

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.

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Walden University
2015

Abstract

Increasing Knowledge About AATD in the COPD Population

by

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MSN, Pacific Lutheran University, 1996

BSN, Pacific Lutheran University, 1992

Project Submitted in Fulfillment

Of the Requirements for the Degree of

Doctor of Nursing Practice

Walden University

November 2015

Abstract

The purpose of the project was to increase awareness about alpha-1 antitrypsin deficiency (AATD) in chronic obstructive pulmonary disease (COPD), particularly among those with a familial history of genetic factor AATD; an additional goal was to understand its relationship to COPD. COPD is the third leading cause of death in the United States, with more than half of COPD patients experiencing significant disabilities. Major causes for COPD include smoking, air pollution, secondary smoke, upper respiratory infections, hereditary factors, occupational factors, environmental factors, and socioeconomic factors. Genetic factors, however, also play a significant role in early onset COPD and in those who smoke and have the genetic factor related to COPD (AATD), symptoms are more severe and exacerbations more frequent. Undiagnosed AATD can result in under treatment and lack of planning for preventing COPD onset and exacerbation in these patients. COPD clients of a local pharmacy ($n = 31$) were invited to complete a Likert survey and given materials on COPD exacerbation prevention and information on AATD. Results indicated that 38.7% of respondents had early onset symptoms, positive family history, and no improvement in symptoms with smoking cessation. The results support that targeting those family members with COPD and providing information on genetic factors for this condition could decrease the frequency and severity of exacerbations. This is in keeping with the health belief model that guided this study in that a perceived risk for harm has the potential to improve the use of preventative health measures in individuals.

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Dedication

This project is dedicated to my father, Paul John Wentink Jr., whose life-long motto of “Perseverance Wins Success” has served me well.

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Gratitude is expressed to the faculty and staff at Walden University for guidance and assistance with the project. Special gratitude is extended to my Committee Chair, Dr. Cheryl Holly, and the committee members, Dr. Eric Anderson and Dr. Vincent Hall for having such patience and support throughout the process.

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Section 1: Nature of the Project
Increasing Knowledge About AATD in the COPD Population

The overall aim of this project was to increase knowledge and awareness regarding the genetic factor alpha -1 antitrypsin deficiency (AATD) and its relationship to increased severity of symptoms and more frequent exacerbations in those with chronic obstructive pulmonary disease (COPD), particularly among those with a familial history of COPD. COPD is a chronic disease targeted by Healthy People 2020 as it is the third leading cause of death in the United States (CDC, 2013). More than half of COPD patients have limitations in employment, family life, social life, and ability to engage in household chores (American Lung Association, 2011). Major causes for COPD include smoking, air pollution, secondary smoke, upper respiratory infections, hereditary factors, occupational factors, environmental factors, and socioeconomic factors (American Lung Association, 2011). Genetic factors also play a significant role in early onset COPD and in those who smoke with the genetic factors related to COPD (i.e., AATD), symptoms are more severe and exacerbations more frequent (Alpha-1 Foundation, 2013). Undiagnosed AATD can result in under-treatment and lack of planning for preventing COPD onset and exacerbation in these patients. Notably, almost 90% of COPD deaths are related to smoking habits, which is a preventable factor in COPD onset and exacerbation (CDC, 2012). Although smoking rates in adults have decreased from over 40% in the 1950s to just above 19% currently with cessation efforts, those who have a genetic predisposition such as AATD and who smoke continue to have a higher mortality rate.

AATD is a genetic disease that causes the body to not have the necessary lung protection to fight inflammation and irritants. This results in the early onset of COPD in afflicted individuals (American Thoracic Association, 2003). In 2003, the American Thoracic Association set a standard for genetic testing in adult onset asthma and newly diagnosed COPD clients for AATD. Identifying the presence of AATD within the COPD population allows for preventive health program development for the most effective interventions and teaching strategies for COPD prevention. The COPD population may be unaware of AATD as an influencing factor on the exacerbation and severity of this disease condition, resulting in under-treatment and lack of preventive planning.

Overview of Evidence-based Project

The purpose of the project was to increase knowledge and awareness regarding the genetic factor AATD in those patients diagnosed with COPD and to provide opportunities for genetic testing and information on self-care management strategies. The overall aim of this project was to improve quality of life and decrease exacerbations and severity of symptoms in the COPD population by increasing awareness of AATD within a sample of that population and providing access to strategies for preventing exacerbation for high risk individuals. The theoretical basis was the health belief model that asserts motivation to participate in health promotion practices is related to perceived susceptibility to a specific disease (Hodges & Videto, 2011).

There were both long term and short term goals for this increasing knowledge about AATD in the COPD population. The long term goals were, first, decreasing

exacerbation rates within the COPD population, and, secondly, improving the quality of life for those afflicted with COPD through the use of self-care management techniques. Short term goals for this project were increased awareness of potential genetic predisposition for COPD and increased understanding of strategies to minimize risk for COPD exacerbation within the sample population. Therefore, the specific objectives of the project were to (a) provide resources for AATD risk, testing, and diagnosis along with self-care management techniques for prevention of COPD exacerbation, and (b) survey residents who are diagnosed with COPD in a defined geographical area regarding AATD risk factors.

Significance and Relevance to Practice

The significance and relevance to practice for this project was related to a potential improvement in the frequency and severity of COPD symptom exacerbation for those who are at risk for genetic predisposition to COPD from AATD (CDC, 2013). Awareness of risk potential, according to the health belief model, provides for an increase in health promotion behaviors. Among the strategies that can reduce the risk of COPD exacerbation are smoking cessation and use of focused self-care management techniques. Although smoking prevention is the major focus in preventing COPD and smoking rates have decreased since 1954 to current day, COPD continues to rise and is now the third leading cause of death in the United States (CDC, 2013). However, Bourbeau (2003) evaluated 10 studies that showed a correlation with improved health and decreased use of medical resources for COPD clients who engaged in self-management that included relaxation strategies. It is an anticipated outcome of this project that combining an awareness of the relationship between AATD and COPD,

opportunities for genetic testing, and knowledge of self-care management strategies and resources, such as smoking and cessation and relaxation techniques, will result in COPD patients taking a more focused approach to attenuating onset and exacerbation of this condition.

In COPD prevention and exacerbation management, current measures focus on smoking cessation and prevention as the primary measure (CDC, 2013). The CDC (2013) approximates 20% of smokers will experience COPD. Statistics on other factors contributing to COPD such as genetic factors are unavailable except the estimated 4% with AATD (CDC, 2013). Without the incidence data to connect genetic factors to COPD exacerbation, smoking prevention becomes the only reasonable measure for focus in prevention that is statistically connected to smoking. Bridging this knowledge gap provides for alternate measures to decrease incidence and exacerbation of COPD such as targeting aggressive preventative measures to high risk families.

Implications for Social Change in Practice

For this project, the social change implications were related to an increased quality of life for the COPD client. Disabilities in lifestyle, employment, and self-care present a major concern for early onset COPD clients. Planning for exacerbation prevention for this population requires a holistic view that can encourage self-management and maintain productivity. Keeping this population productive longer can affect individual lives and enhance the community with capable, independent members as well as contributing to an overall decrease in healthcare cost and utilization.

Project Questions

The questions guiding this project were the following:

- What are the risk factors for AATD present within a geographically defined COPD population?
- What are the opinions of the target population in changing lifestyle to prevent COPD exacerbation?
- How does the presence of risk factors for AATD within the COPD population of the Key Peninsula in Washington State compare with those reported by the CDC?

Evidence-Based Significance of the Project

In 2003, the American Thoracic Society published a document describing standards for diagnosis and management of AATD. The recommendations at that time included testing new COPD and asthma diagnosed clients for genetic factors (American Thoracic Society, 2003). In 2005, Campos, Wanner, Zhang and Sandhaus estimated only 5% of AATD cases had been diagnosed up to 2003. The CDC (2013) continues to estimate only 3% to 5% of COPD is related to AATD, yet testing has not become standard. In 2012, Blue Cross and Blue Shield of Montana set out a standard for testing for AATD that includes testing for clients with clinical presentation or familial history. Recommendations continue to be made for testing; however, incidence rates continue to be only estimates. The lack of awareness of genetic factors for COPD and ability to obtain free AATD testing is a major barrier that can be remedied with public exposure to this issue.

In the United Kingdom, AATD is listed as an autosomal recessive genetic disorder with an incidence second to cystic fibrosis and is number 11 on the list of 23 genetic disorders related to chronic disease (Genetic Alliance, 2013). However, on the CDC (2013) site, although COPD is the third highest cause of hospitalization in this country (CDC, 2013), AATD is not mentioned nor is any other potential genetic connection to COPD except as a ruled out neonatal testing disorder. Therefore, the need for practice change in genetic testing is evident even though the change is slow despite recommendations by the American Thoracic Society (2003).

The anticipated outcomes for testing according to the American Thoracic Society standard for AATD can be seen on both the individual client basis and population health improvements (Carpenter et al., 2007). Acknowledging genetic factors allows multiple interventions for self-care management that can improve individual health and future family health instead of focusing on only the smoking issue. Additionally, healthcare facilities and the community can begin to provide environmental protection for AATD affected persons, including air quality and prevention of upper respiratory diseases. In addition, having increased tools available for COPD prevention can decrease exacerbation rates and improve economic outcomes. Maintaining quality of life and decreasing disability rates for this group can impact the healthcare cost.

Recognition of genetic factors in illness and how to use these factors in ethical ways pose issues for both stakeholders and end-users. Decreasing smoking rates and respiratory illness is part of promoting healthy living (Healthy People 2020, 2012). Improving knowledge and access and promoting health are key factors. Providing interventions for COPD management that address genetic, environmental, and healthy

habits, rather than just a single factor etiology, can serve to promote a healthy lifestyle. Education for providers on testing and treatment availability can provide options for individual clients and population based care.

Awareness and education programs for providers to identify at risk clients are necessary to have stakeholders use testing standards and target high risk individuals for preventative programs. Identification of AATD prevalence within the COPD population is a required first step for the healthcare providers to recognize genetic factors as a valid risk.

Definition of Terms

There are multiple genetic factors that can affect the respiratory system including AATD, immune deficiency syndrome, and cystic fibrosis (CDC, 2013). For the purpose of this project the genetic factor was AATD. COPD, for this study, included emphysema, chronic bronchitis and adult onset asthma.

For COPD, causation can be from both internal and external factors for the susceptible host. Smoking or exposure to smoke is an external contributing factor for 80 to 90% of those with COPD (CDC, 2012). Other external factors include environmental and occupational exposure, allergies, and respiratory infection (CDC, 2012). Alpha-1 antitrypsin is a protein that protects white blood cells from damaging normal tissue such as lung tissue and circulates in the blood stream. In persons affected with the genetic disorder AATD, the protein is deficient or lacking causing lung tissue to become damaged when white cells are present in the lungs for various reasons such as bronchitis, pneumonia, allergies, and irritants (American Thoracic Association, 2003). This

becomes an internal factor that is a genetic with a sequence of lung tissue damage in response to irritants or illness. (see Appendix A).

Assumptions and Limitations

The assumptions underlying the project were, first, that genetics interact with the environment and irritants, affecting the health of an individual (Janz & Becker, 1984). Second is that the individual has psychosocial interactions and interpersonal relationships that affect decision making (Janz & Becker, 1984). Using the health belief model, it is assumed that increased awareness of risk for disease can improve the potential for self-care management in the COPD population (Deavenport, Modeste, Marshak, & Neish, 2010). It is important to understand and clarify assumptions to minimize researcher bias in any significant study that may include personal views of participants or researchers (Terry, 2012).

Limitations are noted in fiscal and time limitations along with procedural difficulties. Barriers for procedural issues include an undefined sample size and potential for limited response. A significant barrier is clients not wanting to hear potential health risks or that the information is not useful (Bernhardt & Pyeritz, 2011). Another factor in low testing rates is the lack of knowledge of availability of free testing for clients by both clients and providers (Berhardt & Pyeritz, 2011). To combat this, increased awareness of the potential benefits must be made clear to the public with education. Individuals must be assured that testing is confidential and genetic discrimination must be legally managed. Time-line limitations become significant with sample participation, as potential respondents may not return surveys in a timely manner.

In summary, awareness of genetic factors allows clients multiple interventions for healthcare management that can improve individual health and future family health instead of focusing on only the smoking issue. Public facilities and communities can begin to provide environmental protection for AATD affected persons including air quality, irritant-free home heating systems, and prevention of upper respiratory diseases. Having increased tools available for COPD prevention can decrease exacerbation rates and improve economic outcomes. Maintaining quality of life and decreasing disability rates for this group can impact the healthcare cost. Targeting those individuals with high risk for COPD early in life can delay or prevent onset and decrease severity.

Section 2: Review of Literature and Theoretical and Conceptual Framework

Review of Scholarly Evidence

The problem presented for this DNP project was that the COPD population may be unaware of AATD as an influencing factor on the exacerbation and severity of this disease condition, resulting in under-treatment and lack of preventive planning. The purpose of the project, therefore, was to increase knowledge and awareness regarding the genetic factor AATD) in those patients diagnosed with COPD and to provide opportunities for genetic testing and information on self-care management strategies.

An assumption of this project was that the COPD population is unaware of AATD as an influencing factor on the exacerbation and severity of this disease condition resulting in under-treatment and lack of preventive planning. A review of the literature was conducted using both the Walden University Library and the worldwide web (www). Google, Scholar, CINAHL, and MEDLINE/PubMed were searched for English language. Key search terms included *AATD, Alpha-1 Antitrypsin Deficiency, genetic factors, COPD, obstructive lung disease, prevalence, and self-care*. Results of the search were limited to current evidence-based studies. Many prevalence studies were international. In the United States, however, only estimates were found for prevalence. Although these research articles are important to the topic of AATD, little relevance was found to the main purpose of this study. Foundational research was found in earlier articles from the American Thoracic Society in 2003 and has been referred to in research throughout the years. Peer review articles were found in relation to COPD prevention but little on increasing awareness of AATD within the COPD population.

Specific Literature

Facilitators for the change to increase testing for genetic factors includes agencies such as the Alpha-1 Foundation (2013) which provides free testing for at risk individuals and families. The barriers include lack of awareness of genetic predisposition and potential cost for testing along with the correlation of smoking as the major contributor to COPD exacerbation (CDC, 2012). Johnson, Campbell, Bowers, and Nichol (2007) asserted that the COPD patient has a stigma that holds the patient responsible for causing their illness, which affects healthcare quality. This is a bias that affects the ability of healthcare providers to look for other factors related to COPD etiology, regardless of whether the client ever even smoked.

Smoking prevention is considered to be a cost effective method of controlling the rise of COPD (Tsevat, 1992). Studies have been done showing a variety of genetic factors that contribute to the rapid COPD progression (Sanford, Weir, & Pare, 1997). Concentrating the efforts of smoking prevention for those at risk for rapid COPD progression can improve outcomes of patient lives. Campos, et al., (2005) discussed the trends and barriers in diagnosis of AATD that have not improved despite recommendation by the American Thoracic Association to test newly diagnosed COPD clients (American Thoracic Association, 2003). In 2013, Stoller and Brantly again discussed the challenges of diagnosis with an estimated 10% of American AATD patients diagnosed. Estimates from studies such as Spinola (2009) provided information about multiple ethnic groups affected by AATD. Moreover, Bourdeau (2010) compared the prevalence to that of cystic fibrosis rates.

In 2005, Lindberg, Bjerg-Backlund, Ronmark, Larsson, and Lundback conducted a study that documented a significant family correlation in both obstructive airway disease and chronic obstructive airway disease along with a greater than expected result for clients who have never with disease. The research question for the awareness of risk for AATD within the COPD population can easily be seen as a gap in current, available data that requires addressing.

Theoretical Framework

The health belief model provided the theoretical framework for this project. The health belief model originated in the U.S. Public Health Service social psychology department. The focus of the model was low rates of participation in preventive health programs by the populations.

The HBM centers on the client perspective of the risk for a specific disease and the severity of the disease (Hodges & Videto, 2011). The likelihood of change for preventative action is an equation of the perceived benefits minus the perceived barriers for the preventative action (Nursing Theories, 2013). Modifying factors are involved and include demographics, socio-psychological issues, and cues to action such as family illness, provider suggestion, media, or social suggestion (Hodges & Videto, 2011). For the health belief model, the disease must be perceived as a significant risk factor for the person and have available modifying factors that the person can change. Resources may need to be moved from competing areas of perceived risk for the individual to meet the change (see Appendix B).

In nursing, the use of HBM is seen in health promotion. For example, Deavenport et al. (2010) studied the lack of mammogram screening in low-income

Hispanic women. They used questions for the study that related to HBM concepts. The perceived risk for breast cancer needed to outweigh the other issues for the women in the population and adequate modification factors needed to be present. Screening for breast cancer needed to have high enough risk to require resource use from competing risks present. Janz and Becker (1984) presented a review of 29 studies using HBM with perceived barriers and perceived susceptibility both having strong significance for action. Kaufert, Rabkin, Syrotuik, Boyoko, and Shane (1986) also showed correlation with general health concerns and perceived vulnerability with smoking in a health education program. The application of this model for smoking cessation, breast cancer screening, and other preventative health programs provides a basis for use of this model for a program that is preventative and coupled with genetic considerations (see Appendix B). For AATD management and COPD prevention, many of the health promotion activities are not high resource utilizers but lifestyle changes. Understanding genetic components can increase the perceived risk and promote behavioral change without requiring high resource demands (Bourbeau, 2010).

Program Planning for AATD Prevalence in COPD

Program planning for AATD prevalence within a COPD population was based on client perception of the disease process and factors contributing to that process. Not every COPD client is going to be positive for genetic factors. However, the process of beginning to look at contributing factors and potential for minimizing respiratory symptoms can begin with the questions about contributing factors and genetics for this population. The question of what the client sees as a contributing factor to the disease realizes that potential. Changes in perceived risk for disease can change potential for

preventative action (Hodges & Videto, 2011). Hodges and Videto (2011) asserted that health education program designs that alter the client or population perception of susceptibility to a disease can be effective for promoting self-care and preventive care actions. The program for increasing knowledge of AATD within the COPD population was an assessment of the population views and an educational project that was specifically designed to alter the client, population, and healthcare provider perception of the risk factors and susceptibility to COPD by identifying risk of genetic factors as significant in the onset and exacerbation of COPD.

Section 3: Methodology

Project Design and Methods

This was an exploratory project using survey methods with a volunteer sample. This method was chosen due to the ease of participation for the sample population. Likert-like questions were used to gather information about risk factors and symptom exacerbation related to disease process (see Appendix C).

Population and Sampling

The target population for this assessment is COPD patients using a specified local pharmacy in Washington State. Convenience sampling was used. The sample population was a voluntary group of clients from among the 250 to 300 clients renewing COPD medications of inhalers in the specific local pharmacy. Although AATD rates are estimated at 4% of the COPD population in the United States (CDC, 2013), European studies show rates as high as 1 in 25 in entire populations (Campos, et al., 2005). In Kitsap County, Washington State, the project site shows European heritage up to 28% (USA.com, 2013), making this a potential "hot spot" of COPD influenced by AATD.

Assessing this population for demographics, care management strategies, and history along with providing testing information for AATD testing in the survey can guide the change in preventative programs for COPD. The COPD patients from the pharmacy can voluntarily return the survey and make their own appointments for testing for AATD. Risk factors for AATD may be identified from survey results. This sample population would be generalizable to the area to design preventative programs for COPD and smoking cessation.

Recruitment and Distribution of the Survey

The COPD survey was brought to the pharmacy where pharmacy clients refill medication for COPD and asthma on a routine basis monthly. The pharmacist offered the survey to the client at the medication refill time and described participation.

Data were collected using survey methods. The surveys were distributed and collected over a month's time. Data were collected using survey methods. The survey included demographic data using descriptor choices and disease history as well as needs assessment information using Likert-like questions. The survey was completed by the participant and returned to me via the self-addressed stamped envelope provided. The needs assessment for this population specifically focused on the value the population places on a variety of contributing factors and willingness to participate in prevention interventions for those factors including smoking, environment, respiratory infections, and genetics (see Appendix D).

Protection of Human Subjects

Informed consent was provided to each respondent, and voluntary participation information was included with the survey brochure. The regulations of Walden University in the conduct of human subject protection were followed throughout the conduct of this project. Anonymity of the respondents was maintained. No names or other personal identification were solicited. The IRB representative was identified for clients and the number for access was included (612-312-1210). Completed surveys were kept in a locked file drawer in my office for a period determined by the Walden University IRB.

Data Analysis

Demographic data were calculated for percentages. Likert-like survey questions have mean results for willingness to change and AATD knowledge. Central tendency data for risk factors was performed as appropriate to data. Data analysis was done for the quantitative data to compare risk rates for AATD in the sample population to the estimated AATD rates presented by the CDC. Correlation was done using the onset age and number of family members with symptoms.

Summary

A project that focuses on identifying high risk for AATD within a COPD population can provide increased resources for decreasing the COPD onset and exacerbation rates. The results of the project needed to be disseminated in order to promote the use of increased resources and to promote holistic preventative plans for preventing COPD onset and exacerbation. Dissemination of information must include the target population and the health care policy makers and providers (Hampton, 2011b). The target population had the opportunity to be informed of project results and resources at a post conference for participants that was open to the community following data analysis. Information about the project may have direct effects on the quality of life for the participants and community. The project poster was submitted to Sigma Theta Tau (see Appendix E) and research brief manuscript to the CDC Chronic Disease Prevention. Reaching the nursing professionals with data to address genetic factors in health prevention issues would provide options for health care planning in COPD and other chronic diseases. An additional presentation was to be made to the partnering agency

stakeholders. The purpose was for planning healthcare resource management and marketing for the COPD population in the community.

Result dissemination was viewed with ethical considerations. Confidentiality and anonymity were maintained for the participants in result reporting. Ethical practice, also, dictates that the participants have an opportunity to receive follow-up care for potential stress or physical results from the study. Resources were provided for follow-up in the post conference. Regardless of study results, the potential for quality of life improvements with self-care education and follow-up can provide continued progress toward meeting the goals of Healthy People 2020.

Section 4: Findings, Discussion, and Implications

Summary of Findings

Participation rates for the survey were calculated on the responses to the invitation to participate. Each variable in the survey was calculated for distribution, central tendency, and dispersion. All invited clients received the educational and survey materials. Data were not available on whether the clients reviewed the materials. There were 33 respondents to the survey. Two respondents were disqualified related to age outside of survey criteria; therefore, the sample size for this study was 31 participants who met the 95% confidence interval for non-medical research, and was large enough to meet the tenets of the central limits theorem regarding a normal distribution of the sample (Dinov, Christou, & Sanchez, 2008).

Gender of the sample was split equally within the respondents with one respondent not answering the gender question. Of the 31 respondents eligible for participation, the majority were aged 41 to 60 years (48.3%), followed the 61 to 65 years (45.2%). Female respondents were younger than male respondents (see Appendix D). The participants reported onset of symptoms earlier than 40 years in 45.2% of responses. Onset of symptoms between 41 and 60 was reported by 38.7%. Symptom onset between 61 to 65 years was reported by only 16% of the respondents. Symptom onset for 60 years and less was 84%. Only 22.6% of the respondents reported no other family members with symptoms. Smoking made symptoms worse for 80.6% of the respondents while 48.3% found other irritants to exacerbate symptom. These include respiratory infection (45.2%), allergies (48.3%), and pollution (29%).

The survey asked the participants to report what made their symptoms improve. Medication improved 93.5% of the respondents. Only 13% of respondents reported that clean air improved symptoms. One respondent reported that nothing improved symptoms. Smoking cessation improved 58% of the participants' symptoms. Of the respondents with onset at 60 years and earlier and a family history, 52.2% reported no improvement in symptoms with smoking cessation. This group comprised 38.7% of all respondents to the survey. The survey showed close to 68% of the respondents wished to know if they had a genetic predisposition for COPD with 55% willing to change behaviors if they knew about a genetic predisposition.

Correlation was conducted using Pearson's r value for age of onset as the independent value and family history as the dependent value. Calculations were done by assigning Likert values to the variables. The female respondent r value of 0.3384 showed a moderately positive correlation ($p = 0.2173$) but was not statistically significant. The male respondent value of $r = .6488$ showed a high positive correlation and statistical significance within the variables ($p = 0.0089$). The entire respondent value of $r = 0.5272$ represents a moderate positive correlation that is statistically significant between early onset symptoms and family history ($p = 0.0023$).

Discussion and Implications

The implications were based around the trends noted in the responses of the returned surveys. Positive correlation between early onset symptoms and positive family history of COPD suggests a higher percentage of genetic involvement than the 4% estimated by the CDC, 2013. In this small convenience sample, the percentage of those with COPD and family history was 77.4%. Rates for actual risk for genetic

involvement were not found in the CDC statistics for U.S. populations. Testing for genetic factors as recommended by the American Thoracic Society (2003) may prove helpful in minimizing COPD frequency and severity. Positive and significant correlation of early onset symptoms and positive family history, as found in this project, can provide the opportunity to target high risk families for preventative programs that can begin early in life and focus on promotion of respiratory health, including pulmonary toilet, and prevention of high risk habits.

Genetic diseases affect an estimated 12 million Americans, yet according to a survey of 1,000 people conducted by the Genetic Disease Foundation-, while two-thirds of those surveyed were willing to and saw the benefits of undergoing testing, close to 80 % had never talked to their physician about genetic screening—an inconsistency that can have serious implications on a person's overall health and the health of their immediate family members. Given the results of this project, that 45% of respondents met the criteria for genetic testing for COPD, it is important to realize that in fact, almost every human trait and disease has a genetic component, and there are more than 6,000 known genetic disorders, from widely recognized conditions like Down syndrome, spina-bifida and sickle cell anemia, to lesser-known conditions like Tay-Sachs disease and Fabry disease (Genetic Alliance. 2013).

Genetic diseases are caused by abnormalities in a person's DNA and can range from a small mutation in a single gene to the addition or subtraction of an entire chromosome or set of chromosomes. These abnormalities can disrupt the normal function of a vital system, such as the immune system or the nervous system, or prevent normal development of organs, bones, or skin, significantly increasing a person's chance

of developing a particular disease. The addition of both environmental and lifestyle factors to the genetic abnormalities has a significant impact on the development of disease in specific groups of individuals.

The results of the study support the use of the health belief model for improving outcomes in patients with COPD and risk factors for AATD. This is evident by the survey results of willingness to change health behaviors with knowledge of genetic risk. The knowledge of risk for AATD can provide an increased perceived risk for the COPD client and other family members. The model supports that the perceived risk for disease improves the potential for participation in healthy behaviors. The survey results indicate that using the health belief model to develop COPD prevention and management programs, especially for those at risk for AATD, would be successful.

This project provided a survey that supports the results of studies that have done AATD testing such as Spinola (2009) and Bourbeau (2010). Although it did not provide actual AATD incidence rates, the study provided information that there is a higher population at risk for AAT with COPD than the CDC (2013) rates indicated. The expanded knowledge of risk for early COPD onset and exacerbation related to family members with COPD is knowledge that is useful to program development and target populations.

Strengths and Limitations

The primary focus of the project was to increase knowledge for AATD risk within the sample population. All of the sample population received information about prevention for exacerbation of COPD and risk for genetic predisposition. It was

undeterminable whether the sample population read the supplied material. However, the information was made accessible to this population.

The next limitation for this project was found in the response rate to the survey of COPD clients at the local pharmacy who were invited to participate. In the 6-week period, only 35 of the 75 surveys were distributed to clients. Self-reporting can have limited reliability in surveys (Terry, 2012). Therefore, in order to promote accurate answers, smoking status was not asked in the survey. Trends were visible in the responses of the participants. The survey collection occurred from March 01, 2015 to April 10, 2015. Two additional responses were received but ineligible to age.

Although the survey responses did not provide information for specifically AATD, it provided information that suggests familial or genetic factors in the respondents. More than 70% of respondents had indicators of both early onset and positive family history that would qualify for AATD testing. Further testing would be required to determine the nature of the trends. These could be related to genetic factors, familial life style factors, or environmental factors.

The survey information provides a basis for further research on the prevalence of genetic predisposition for COPD. The responses of the participants also provided data that supports that this knowledge may improve the quality of life for this population by targeting health prevention programs to those families at risk for COPD. Johnson et al. (2007) asserted that there is a stigma of patient blame that affects healthcare of this population, and Berger, Kapella, and Larson (2011) attributed this increasing trend of patient blame for COPD to smoking. Dispelling the blame for COPD from the patients

may improve healthcare responsiveness along with social interaction and self-care management.

Evaluation and Analysis of Self

As a practitioner in healthcare, the DNP process has increased my own need to improve assessment skills of individuals and populations. This was especially important to me as the Affordable Care Act (2010) promotes universal health insurance coverage and population health to improve health equity and outcomes. There is a trend to move away from illness focused care toward population health and preventative healthcare services. The ability to provide evidence-based care requires a deeper level of assessment in order to provide care that truly minimizes and prevents illness. Care that stems from limited assessment and unfounded practices can no longer provide what is required to improve the national health dilemma. As a scholar, it has become important to me to provide research and evidence-based studies that can improve individual and population based outcomes. The work is not easy, but having completed a DNP education, this work is manageable. For nursing to survive the healthcare system as it evolves, evidence-based knowledge must be part of the professional obligations. The leadership role of the DNP can provide this knowledge and move nursing forward as a recognized profession. For me, having close to 40 years of practice as a nurse, it is important to provide future nurses with the knowledge and skills that can continue to provide improved outcomes and quality of life for the individual as well as the community. I have seen how this specific project working with the COPD population has the potential to improve quality of life at multiple levels.

Summary and Conclusions

The project for increasing knowledge for AATD within the COPD population accomplished two objectives. First, it provided information about the potential relationship between genetic factors and COPD, which could result in the prevention of exacerbation for COPD clients. Secondly, it provided information on the risk factors for AATD found in the sample population. The results of the survey in this project indicate familial trends for COPD clients consistent with AATD testing qualifications, but did not provide specific genetic indicators. The results support that testing for predisposition to COPD is a factor that could decrease the frequency and severity of COPD exacerbation. The continuing increasing national burden of COPD demands that healthcare providers do more for the at risk population than provide advice for smoking cessation. Positive health promotion, including genetic testing and lifestyle support, could begin to decrease the individual and national cost of this disabling disease.

Section 5: Scholarly Product

A research brief was written for submission for publication with the CDC Chronic Disease Prevention. This was done with the assistance of Dr. Cheryl Holly as coauthor.

A poster was also constructed that will be on display at the local pharmacy for the community to view. This is being done to improve the awareness of AATD and COPD prevention. The poster will be on view where the participants of the survey may view it.

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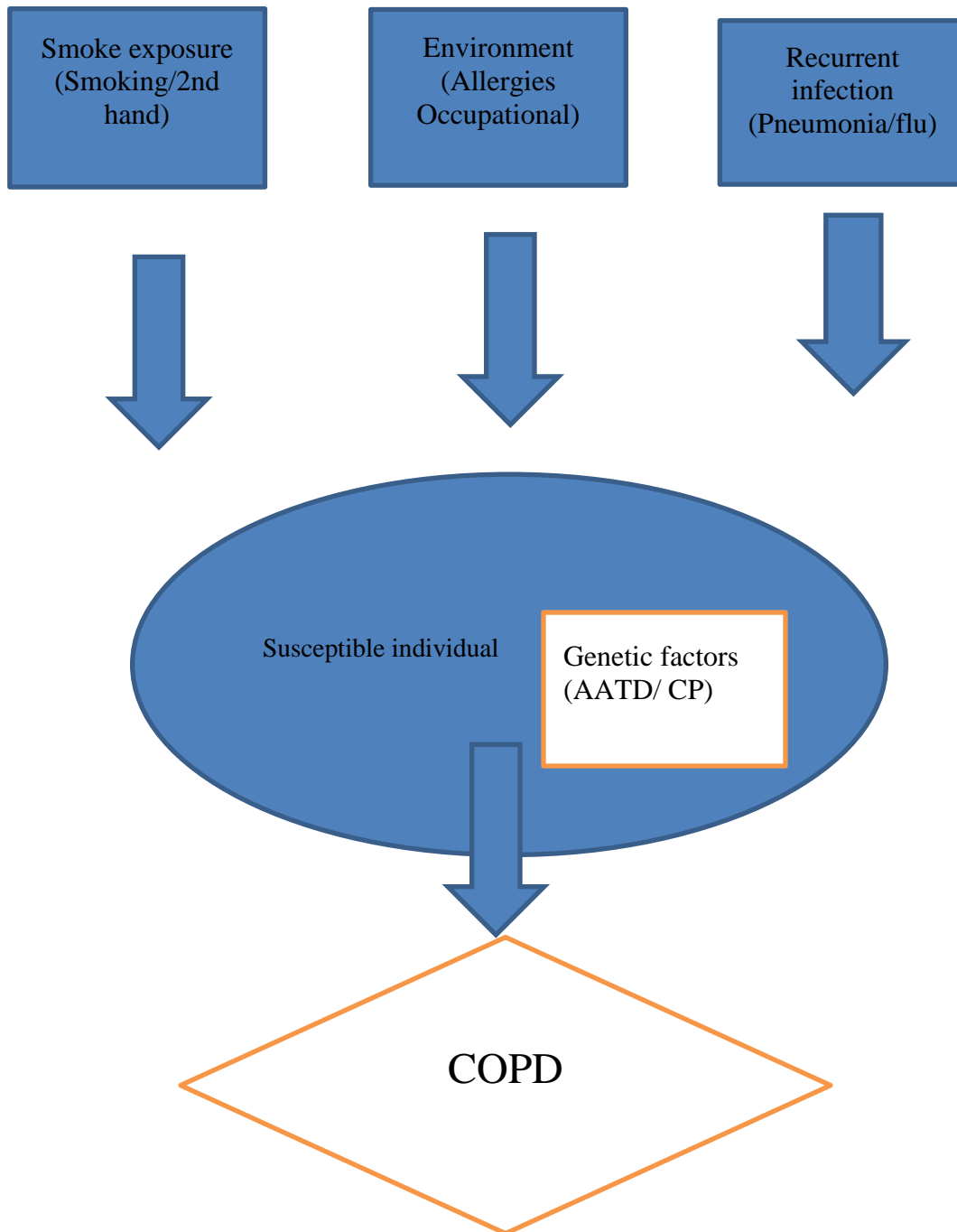
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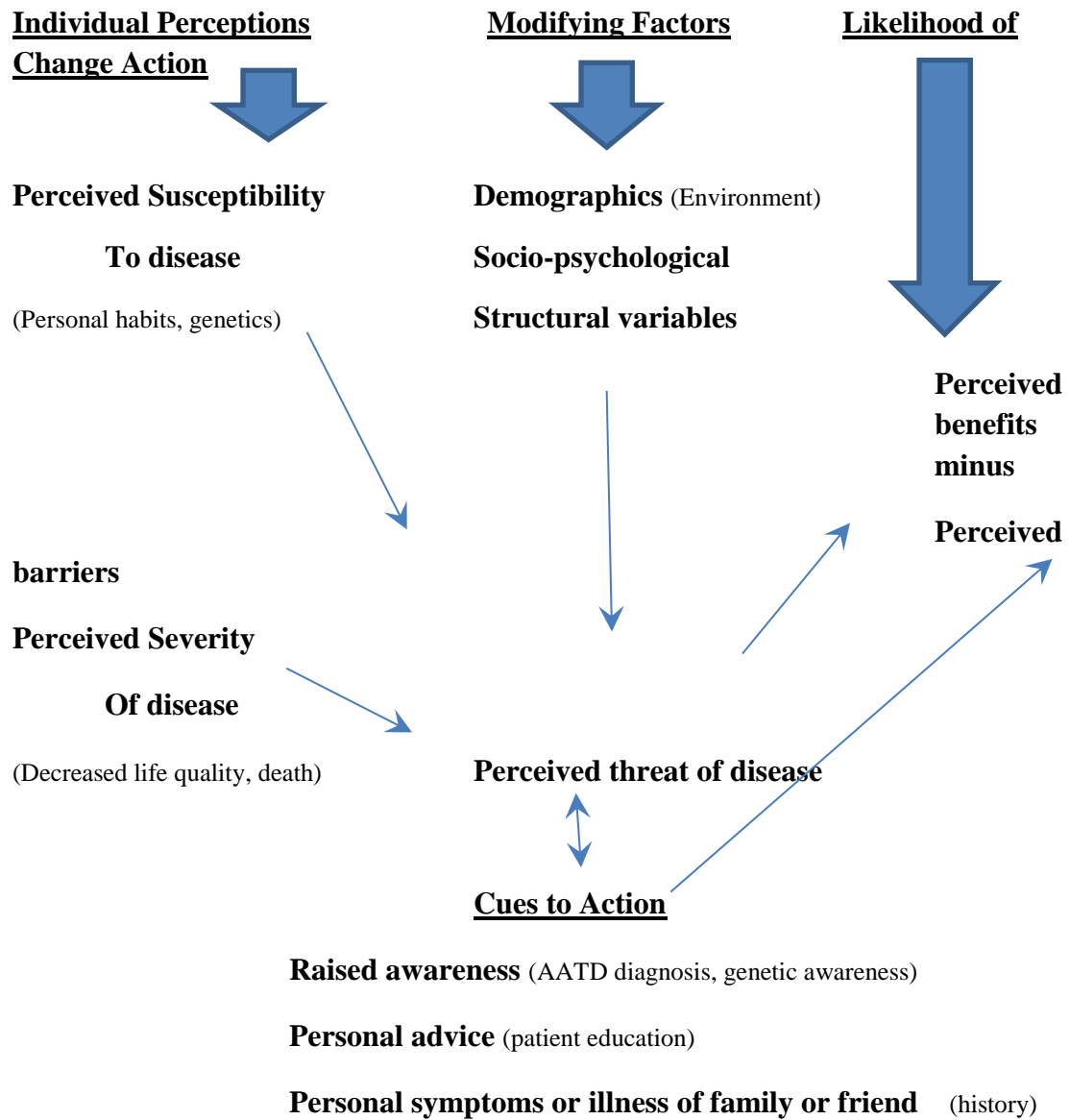
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Appendix A: COPD Web of Causation
COPD Web of Causation



Appendix B: Alpha-1 Antitrypsin Deficiency within COPD Using Health Belief Model



Appendix C: Tools

Prevalence of Alpha-1 Antitrypsin Deficiency (AATD) within the COPD Population of Key Peninsula (Genetic Predisposition) Survey**Part 1 Demographics: (Please circle your answer)**

1. What is your sex? a. Male b. Female
2. What is your age? a. 18-40 b. 41-60 c. 61-65
3. At what age did your symptoms begin? a. 18-40 b. 41-60 c. 61-65 d. Other__
4. How many members of your family also have symptoms of COPD? a. 0 b. 1-3
c. 4 or more

Part 2 Contributing Factors: (Please circle all that apply)

1. What makes your symptoms worsen?
 - a. Respiratory infections
 - b. Allergies
 - c. Pollution
 - d. Smoke
 - e. Other_____
2. What makes your symptoms improve?
 - a. Medication
 - b. Clean air
 - c. Smoking cessation
 - d. Relaxation
 - e. none
 - f. Other_____
3. Would you want to know if you had genetic predisposition to COPD?
 - a. Yes b. No
4. Would awareness of predisposing genetic factors for COPD change your Life style?
 - a. Yes
 - b. No

Appendix D: Figures

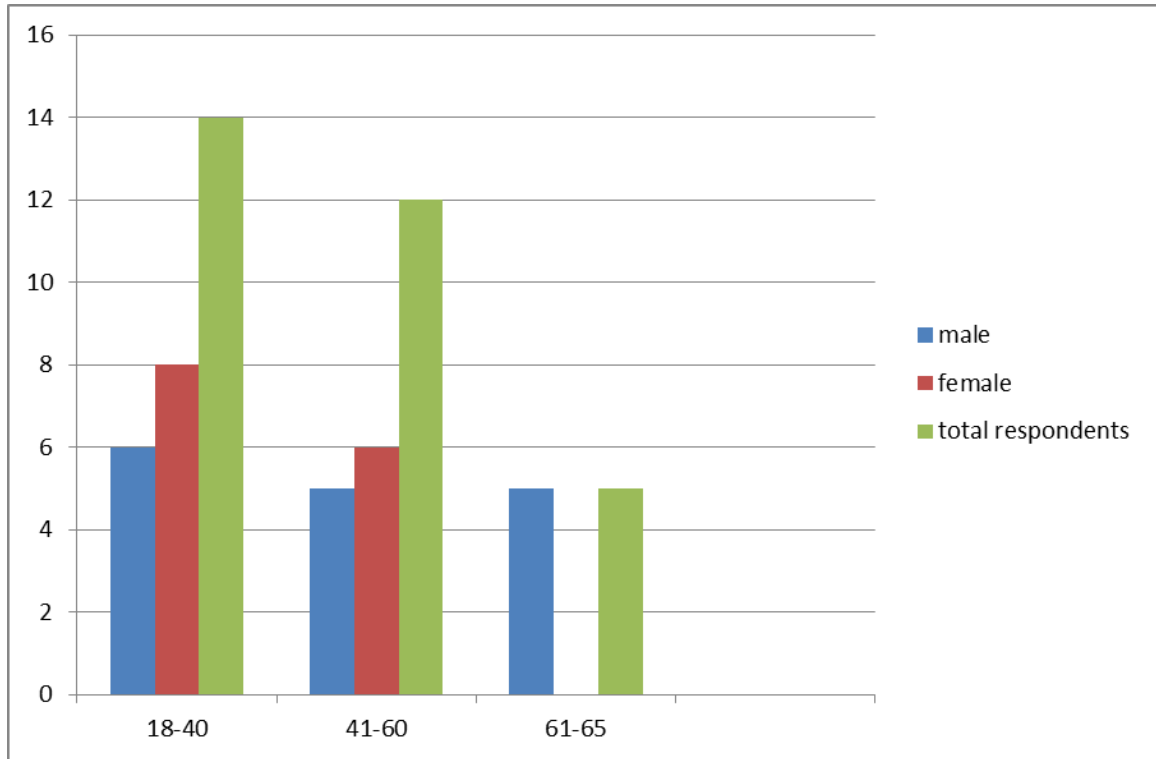


Figure 1. Age symptoms started

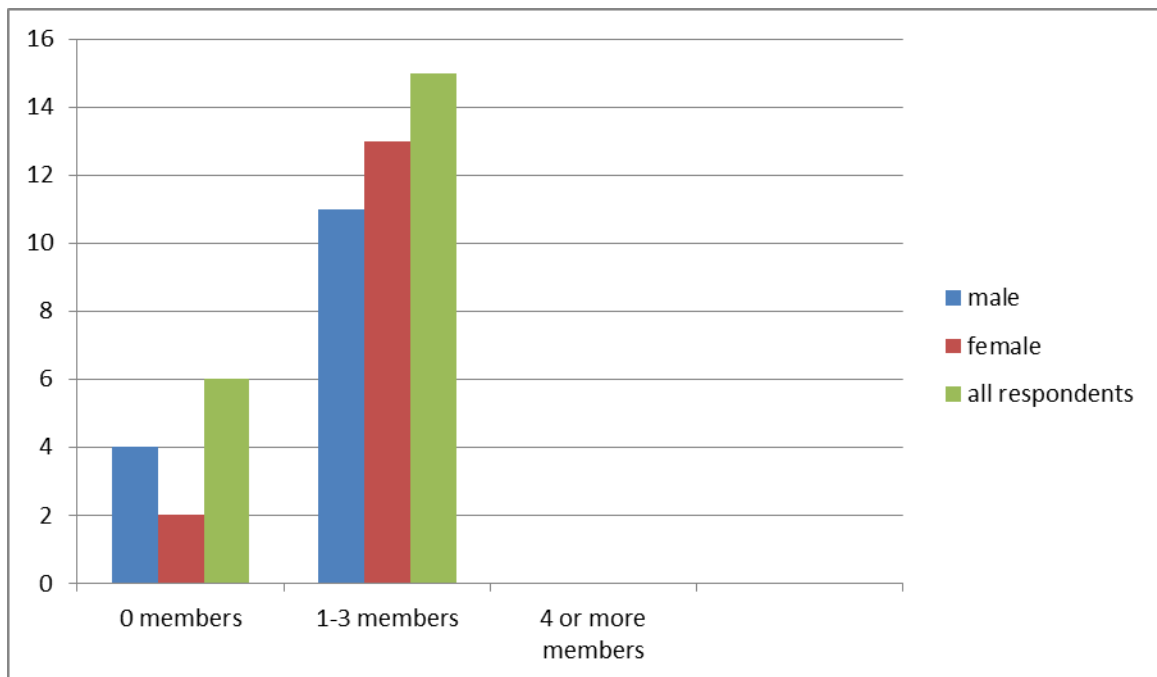


Figure 2. Number of family members with COPD

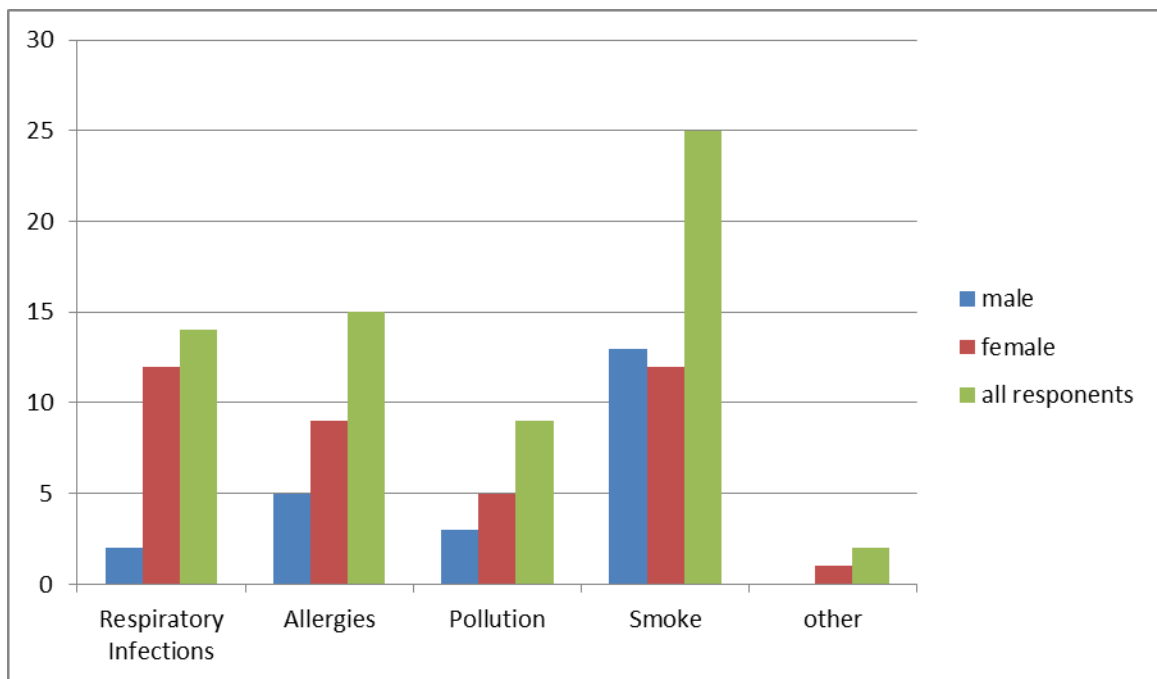


Figure 3. What makes symptoms worse?

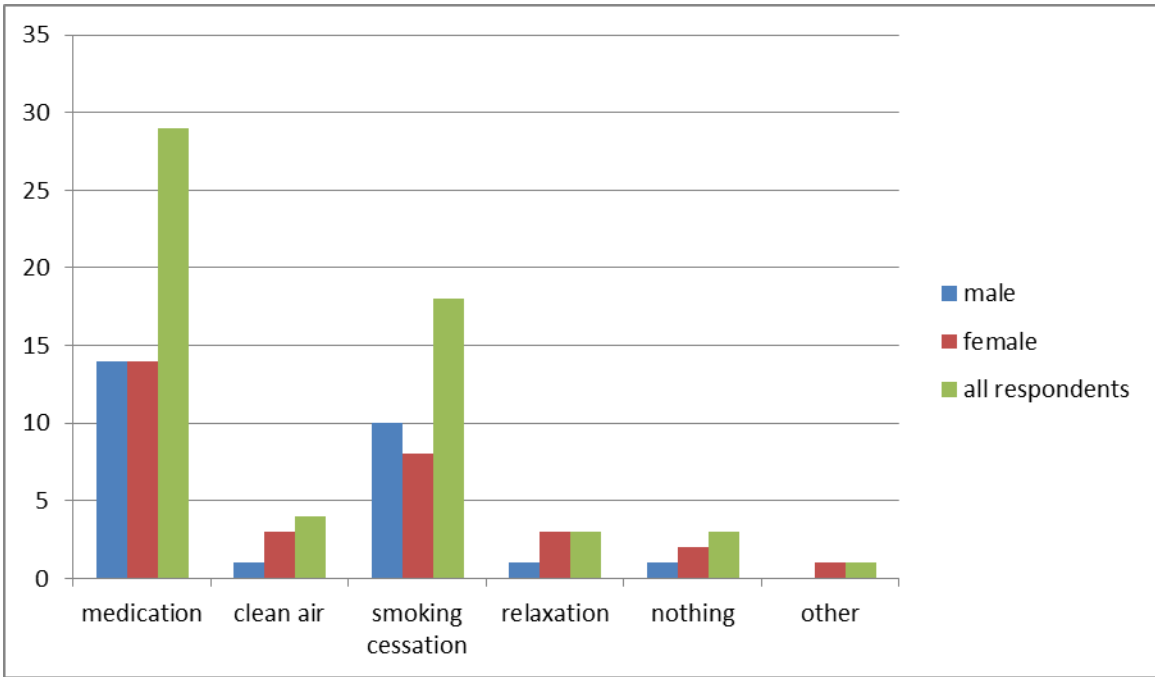


Figure 4. What makes symptoms improve?

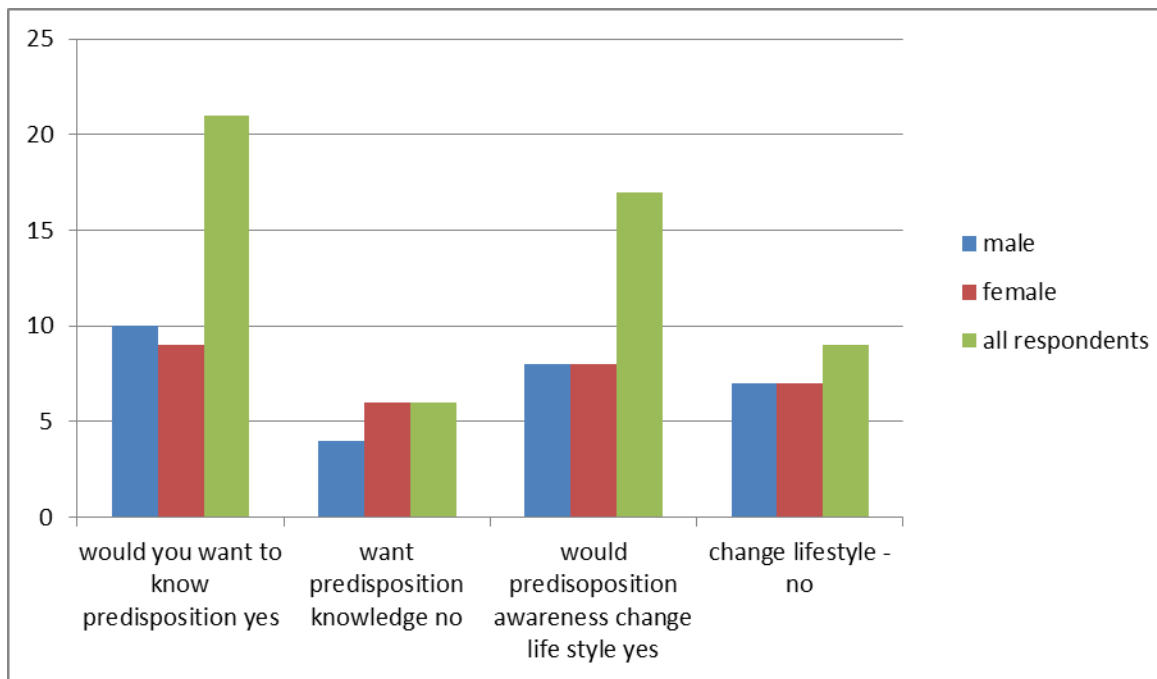


Figure 5. Genetic pre-disposition and willingness to change lifestyle

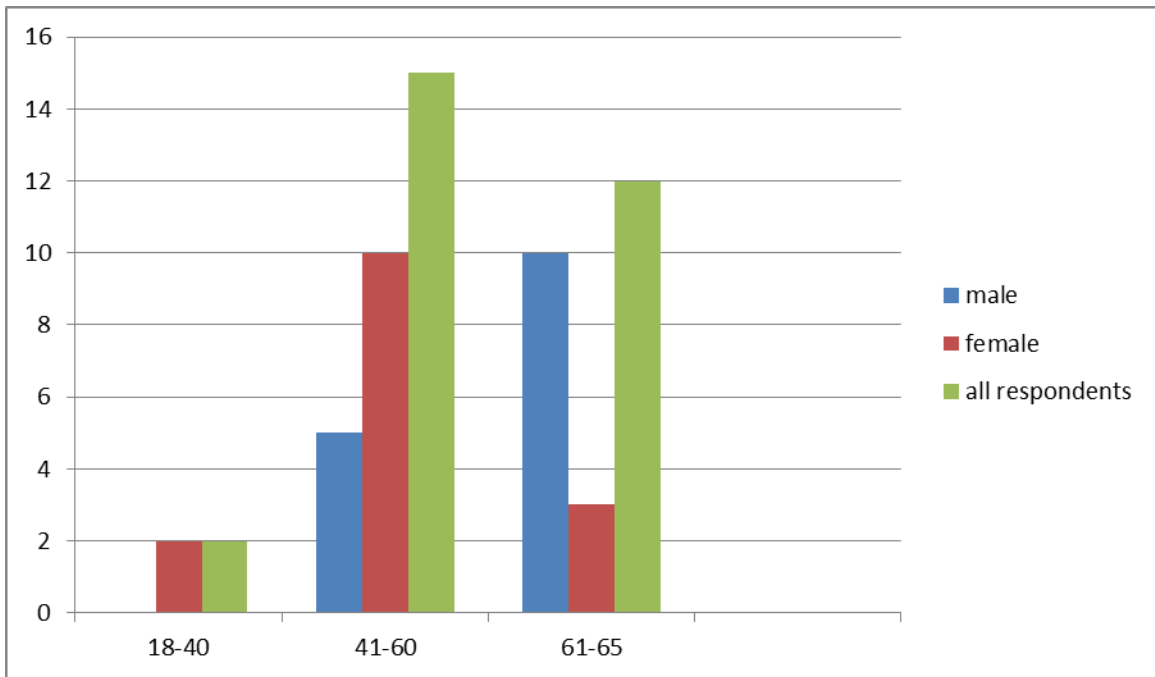


Figure 6. Age of participants

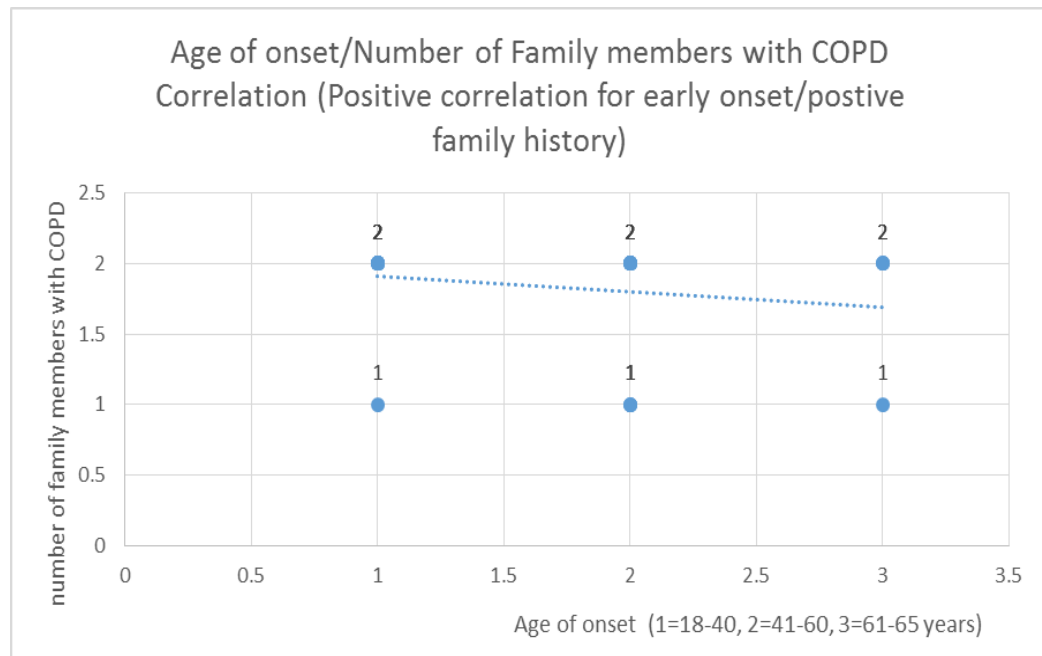


Figure 7. Positive correlation for early onset symptoms with increased number of family with symptoms ($n = 31$, $r = 0.5272$, $p = 0.0023$).