

Psychotherapeutic Interventions for Depression: Reducing Cardiovascular Disease Risk in

Adults

Sadia Ali

University of Missouri, Kansas City

Approved May 2017 by the faculty of UMKC in partial fulfillment of the requirements

for the degree of Doctor of Nursing Practice

©2017

Sadia Ali

All Rights Reserved

Abstract

Depression is often a risk factor for cardiovascular disease, and psychotherapy for depression can result in positive cardiovascular disease outcomes for patients. A correlational study explored the impact of psychotherapy referrals with medication treatment for depression on cardiovascular risk factors of blood pressures, HbA1C, and depression which is measured by the Patient Health Questionnaire (PHQ-9). At three internal medicine clinics in the Midwest, 31 adults with depression and cardiovascular risk factors were included in the psychotherapy intervention. Retrospective data was collected from August 2015-August 2016 prior to the psychotherapy referrals, and prospective data was obtained from September 2016-December 2016 from clients receiving psychotherapy referrals with medication treatment which was initiated for patients with a diagnosis of depression and cardiovascular risk factors. Pre-post risk factors of blood pressure, HbA1C, and depression were measured within the intervention group. The project found a strong positive correlation between HbA1C and PHQ-9 with psychotherapy but no significant correlation between blood pressures and PHQ-9 scores in the intervention group. Regression analysis confirmed that depression treatment positively affects HbA1C and PHQ-9 scores. Treatment of depression can reduce cardiovascular risk factors and favorably impact mortality and morbidity

Keywords: Cardiovascular, depression, disease, intervention, outcomes, relationship

Psychotherapeutic Interventions for Depression Reducing Cardiovascular Disease Risk in Adults

Depression and cardiovascular disease (CVD) associations have been well established and even considered bidirectional in adults (Baune et al., 2012; Nemeroff & Goldschmidt-Clermont, 2012). In a representative U.S. sample, mood disorders were found to have an increased risk of heart disease which is independent of age, geographic location, education, income, race, marital status, employment, smoking, obesity, history of heart disease in family, hypertension, and diabetes (Fiedorowicz, 2014). Furthermore, in developed countries, CVD and depression have recently presented as the two most common causes of disability, and other countries are expected to have the same outcome by 2030 (Hare, Toukhsati, Johansson, & Jaarsma, 2014). Depression alone can increase the risk of CVD up to 1.5 times in adults, and adults with coronary artery disease and depression are two to three times more likely to face risk of non-fatal and fatal cardiac events in the future as compared to adults with coronary artery disease without depression (Baune et al., 2012).

Cardiovascular disease is a significant cause of mortality and reduction in quality of life worldwide (Heijmans, van Lieshout, & Wensing, 2014). According to the U.S. Department of Health and Human Services (DHHS, 2016), the number of adults living with multiple types of CVD is now greater than 1 in 3, or about 81.1 million. In 2010, more than \$500 billion was spent on cardiovascular disease in adults in the United States (U.S. Department of Health and Human Services, 2016). Locally, heart disease mortality rate in Metro Midwest city is significant. As reported by the Kansas Department of Health and Environment (KDHE), for every 100,000 population, approximately 132 adult deaths are related to CVD (KDHE, 2012). The statistics include all ethnicities. Recently, the American Heart Association (AHA, 2013) places Kansas as the 26th highest death rate from cardiovascular disease in the country and also reports heart

disease to be the second highest cause of death in Kansas. The goal of DHHS (2016), *Healthy People 2020*, is to improve health and quality of life for cardiovascular patients using a three-pronged approach: prevent and detect risk factors for heart attack and stroke, identify and treat heart attacks and strokes as early as possible, and prevent repeat cardiovascular events.

Assessment and communication of lifetime risk for cardiovascular disease have become more prominent issues in cardiovascular health (DHHS, 2016). This project site is in Overland Park, Kansas, and the population identifies as Caucasian (78.8%), Asian (6.6%), Hispanic (6.0%), and African American (5.7%). This diverse population belongs to several ethnicities including Indian Asians and non-Hispanic Caucasian. However, patients from surrounding suburban areas also seek care at the internal medicine clinic.

Depression is a common disorder among adults. The National Comorbidity Study, the largest study of psychiatric disorders, reported that major depressive disorder (MDD) has a lifetime prevalence of 16.6% (Nemeroff & Goldschmidt-Clermont, 2012). A recent scientific statement by AHA concludes that acute coronary syndrome followed by depression is a risk factor for all-cause and cardiac mortality in adults, as well as for combined outcomes including mortality or nonfatal cardiac events (Fiedorowicz, 2014). Furthermore, depression also predicts poor outcomes following hospitalization for cardiovascular events (Bradley & Rumsfeld, 2015). In addition to an association with poorer survival rates, depression is also strongly associated with worse patient health status such as symptom affliction, functionality, and quality of life (Bradley & Rumsfeld, 2015). It is imperative to understand the effect of depression on cardiovascular disease in primary care, where the interventions can be provided early and possibly avert the catastrophic effect of depression. Hence, this evidenced based project focuses on reduction of cardiovascular risk factors of blood pressure, HbA1C, and depression as

measured by the Patient Health Questionnaire (PHQ-9) in the adult population with psychotherapeutic intervention for depression in the Midwest Metro City area.

Problem Statement

CVD and depression are the leading cause of health and economic problems across the world (O'Neil, Williams, Stevenson, Oldenburg, & Sanderson, 2012). CVD is a leading cause of death while depression is most commonly associated with disability (O'Neil et al., 2011). Etiological and extrapolative studies denote that depression and CVD are interlinked and mutually influencing; depression can be both a cause or a result of CVD (Möller-Leimkühler, 2010). Therefore, it is vital to learn about the influence of psychological interventions for depression on cardiovascular risk factors to obtain optimum health outcomes for patients.

Furthermore, there is evidence that also suggests that there may be a cardio-protective effect of depression treatment if it is initiated prior to onset of CVD; a study has also suggested that the pathogenesis of CVD can have a harmful effect of depression even in an early phase (Stewart, Perkins, & Callahan, 2014). Providers' reluctance to initiate depression treatment is also a main factor contributing to the problem of increased cardiovascular health risk (Edward H. Wagner, Austin, Davis, Hindmarsh, & al, 2001). Ultimately, a reduction in healthcare cost is an important outcome to consider.

Purpose Statement

This DNP project aimed to explore the impact of psychotherapy referrals with or without medication treatment for depression on cardiovascular risk factors to promote optimum health outcomes for patients.

Facilitators and Barriers

The main facilitators for the project included the Medical Director of Quality Outcomes and the Behavioral Health Lead (psychologist) at the internal medicine clinics. Regular meetings as well as online dialogue supported communication with the project site professionals to discuss implementation issues and computerized information sharing and clinical data. The project site professionals recognized a need for change in primary care for patients with depression and the leadership was strongly engaged in the processes of the project and was a major facilitator for the project. Because the project required minimal cost to the facility and investigators, cost was an important facilitator for the evidence based practice (EBP) initiative.

The internal medicine clinic project sites represented a large organization and achieving support from all providers was challenging in order to change treatment strategies. It was important to consider that provider buy-in can be influenced by the provider understanding of the intervention, especially if there are provider misconceptions about the intervention effectiveness or minimal information to the providers. The leadership role was also central in achieving positive outcomes for the project, limiting lack of support and accountability from senior leadership as a barrier, and sustaining the referral intervention. Timeline constraint was another barrier because it presented an outcome in reduction in sample size and consequently resulted in skewed outcome.

Review of Evidence

PICOT

In adult patients with a diagnosis of depression and CVD, do psychological interventions including psychotherapy with or without psychotherapeutic medications reduce cardiovascular risk factors of blood pressure, HbA1C, and depression as measured by PHQ-9 over three months to six months at four internal medicine clinics?

Search Strategies

The databases searched and search engines used for ascertaining highly relevant current evidence included Cumulative Index to Nursing and Allied Health Literature (CINAHL), Cochrane databases, PubMed, Google Scholar, National Guideline Clearinghouse, and MEDLINE. Combination of search terms included depression, cardiovascular disease, relationship, interventions, and risk factors (see Appendix A for Definition of Terms). Inclusion criteria consisted of peer reviewed scholarly journals, evidence-based or research articles, English language, and a date range of 2007-2016. Exclusion criteria were articles written in a foreign language, research older than 2007, evidence that was not pertinent to the evidence-based project, and sources that represented lower levels of evidence. An initial search using the identified terms yielded more than 10,000 articles. The search was refined with additional terms of randomized control trials, cluster, and retrospective.

Identified studies were 70, and from those, 30 were pertinent to the evidence-based project. Of these research studies, five were applicable to depression and if a relationship with cardiovascular diseases exists. Fifteen studies were applicable to the correlation of depression and cardiovascular disease and the possible outcomes (see Appendix B). From 150 potential guidelines that involved depression and cardiovascular disease, two were identified as most relevant to the EBP project. Prominently, Level I, II, III and IV (Melnyk, 2011) level of evidence studies were selected for synthesis of the evidence to support the project.

Depression and Cardiovascular Disease Risk

Studies have shown that depression can cause an increased risk of incident of coronary artery disease (CAD; Bradley & Rumsfeld, 2015). The relationship between depression and CVD is bidirectional (Baune et al., 2012; Nemeroff & Goldschmidt-Clermont, 2012). Practice

guidelines state that when screening patients for cardiovascular disease risk among special groups such as Asians, Hispanics, and African Americans, attention should be provided to patients with depression (National Guideline Clearinghouse, 2009).

Deschênesa, Burns, and Schmitz (2015) state that diabetes can make patients more susceptible to major depressive disorder (MDD) as well as general anxiety disorder (GAD) which can exacerbate disability in diabetic patients (Deschênes et al., 2015). Results also linked greater disability to MDD when GAD was not present, to GAD when MDD was not present, and to MDD when GAD was present (Deschênes et al., 2015). Chang et al. (2013) conducted a cross-sectional study which reported that the healthy control group in the study had the highest scores for cognitive function, while the group with both late-life depression and cardiovascular disease showed the lowest scores for cognitive function impairment. Furthermore, Chang et al. (2015) reported that late life depression, compared to cardiovascular comorbidities, has a larger impact on worsening cognitive impairment.

Two longitudinal and case controlled meta-analyses identified that major depressive disorder was the most important component that could result in CVD (Van der Kooy et al., 2007; Seldenrijk et al., 2015). Additionally, development of CVD is heavily influenced by presence of MDD (Van der Kooy et al., 2007). Similarly, in a study conducted by Seldenrijk et al. (2015), patients with depressive and anxiety disorders or current depression only were considerably linked with increased CVD incidence. Inflammatory markers and depression has also been linked together and studies have shown a primary relationship between them (Vaccarino et al., 2007). Also, early identification and providing prevention and treatment of cardiovascular disease are important objectives for patients who have depression and a risk of cardiovascular

disease (Nemeroff & Goldschmidt-Clermont, 2012). In contrast, one study reviewed did not find a correlation between depression and cardiovascular disease risk (Wheeler et al., 2012).

Depression increases cardiovascular risk and is also highly prevalent in patients with underlying CVD (Bradley & Rumsfeld, 2015). A meta-analysis conducted by Meijer et al. (2011) concluded that post-MI depression could result in a 1 to 3 fold increased risk of impaired outcomes within 24 months of MI incidence (Meijer et al., 2011). Additionally, over the past 25 years, this observed link has been reasonably constant (Meijer et al., 2011). The effect of depression with cardiovascular in a work place was explored by O'Neal et al. (2012), and the authors concluded that major depressive disorder is linked to a four times increase in decreased performance. Also, individuals with major depressive disorder in presence of another chronic disease, such as arthritis with CVD, reported increased probability of not being able to work (O'Neil et al., 2012).

Similarly, Almas et al. (2015) conducted a longitudinal cohort study which concluded that severity of depression and risk of CVD are directly related, as one increased so did the other (Almas, Forsell, Iqbal, Janszky, & Moller, 2015). A cross-sectional randomized control study showed that there is a higher rate of occurrence of cardiac risk factors in patients with psychological illness (Gardner-Sood et al., 2015). Rude et al. (2014), in a study with hospitalized patients with cardiovascular diseases, concluded that depression is predominant in the Hispanic population compared to non-Hispanic. Also, the study found that for this ethnic population that depression and duration of stay are closely correlated (Rude et al., 2014). The authors state that suitable treatment of depression can reduce duration of stay and hence potentially lower costs which ensures a positive impact in health care settings (Rude et al., 2014).

The American Heart Association/American Council of Capital Foundation (AHA/ACCF) guidelines (2011) recommend routine depression screening and treatment for patients with recent cardiovascular events. The guidelines do not well-define depression treatment in patients in order to reduce cardiovascular risk factors (Smith et al., 2011). The current lack of evidence, as reported by Coventry et al. (2014), has hindered implementation of depression treatment for patients with cardiovascular risk factors.

Psychotherapy Interventions on Cardiovascular Disease Risk Factors

A cluster randomized control trial revealed that psychological brief therapy provided by primary care advanced practice nurses in collaborative care can result in reduced depression in patients with mental and physical comorbidities and in improvement in self-management of chronic disease (Coventry, P. et al., 2015; Coventry, P. A. et al., 2014). The concept of depression treatment prior to CVD onset has also been explored, and a randomized control trial showed that collaborative depression care provided prior to CVD presence reduced the risk of a significant CVD event in older depressed patients by half (Stewart et al., 2014). A study with spiritual intervention for depression concluded that there is a significant modest increase in overall quality of life through a 1-month intervention (Delaney, Barrere, & Helming, 2011). Similarly, among ethnic groups such as African Americans, a pilot study concluded that integrating depression treatment resulted in better outcomes among older African American (Bogner & Vries, 2010).

While there are positive results from psychotherapeutic interventions, other studies have shown contradicting results. A cross-sectional study found that individuals had improved blood pressure control with or without depression treatments (Mejia-Lancheros et al., 2014). Other studies showed that interventions such as psychotherapy referrals and medication treatment

addressing CVD risk may be helpful when they are applied to both depression and cardiovascular disease and HbA1C, and depressive symptoms can be significantly reduced (Echeverry, Duran, Bonds, Lee, & Davidson, 2009; Georgiades et al., 2007). Consequently, studies have not found strong evidence in reduction of mortality with depression intervention (Whalley et al., 2014). A pilot study concluded that improved depression care after an acute coronary event did not improve patients behavior towards health nor their blood pressure (Kronish et al., 2012; see Appendix B for Synthesis of the Evidence Table).

Theory

The theoretical framework that guided this project is the Collaborate Care Model (CCM), a version of the Chronic Care Model (Wagner, 1998). The Chronic Care Model identifies the problems associated with management of chronic disease in a practice; the design of this model was developed to provide a quality improvement framework for chronic diseases (Wagner et al., 2001). The CCM provides a framework for understanding of problems that can hinder quality care for chronic diseases (Johnston, Peppard, & Newton, 2015). The main components of CCM require regular interaction among collaborative team members, combined style of care among teams, a collaborative care plan, and complete patient participation (Johnston et al., 2015). Components of CCM used in this project included support from organization leadership, decision making by providers, evidence based research findings, and electronic databases (Johnston et al., 2015; see Appendix C).

Methods

The primary Institutional Review Board (IRB) for this project was University of Missouri Kansas City (see Appendix D). Site approval was obtained from project site. The EBP project

used electronic health records (EHR) to retrieve data but was designed to improve care instead of generating new knowledge; the project is considered *not human subjects research*.

Various ethnicities were included in this study including Asians, Caucasians, African American, and Hispanics without discrimination. The study was based in the Midwest region that is considered a central part of the United States. There was no exclusion criterion for specific cultural background; therefore, the study included all patients from different cultures. HIPAA regulations were followed during the collection and analysis of the data for the study (OCR, 2009). To ensure privacy and confidentiality, data excluded patient identification information, and the data was on a secured electronic drive. No student investigator research conflicts were present that hindered conduction of project. The only major ethical concern for the project included possible student investigator bias with sample selection which was avoided by employing random sampling.

Funding

The cost for the project was expected to be minimal (see Appendix E). For implementation of the DNP project, meetings were held with the project directors of Quality Outcomes at the project sites throughout the project from March 2016-May 2017. The student investigator collected existing patient data from the electronic health record (EHR). The laboratory data and PHQ-9 screening was previously completed; therefore, the project entailed no extra cost to the clinic. Cost also included local travel expense for the student investigator for the retrieval of data from the EHR accessed at the clinic. The budget for this EBP was \$280.00, and no cost was included for the time provided by the student investigator and the hours provided by team members at the project sites. Time provided by the project team was also free of charge as they are working to improve patient outcomes. The student investigator was

awarded the UMKC Women's Council Graduate Assistance Fund and UMKC Travel Award Fund for funding and dissemination for this project, and the student investigator periodically reviewed additional costs such as printing of posters and travel expenses for dissemination which were covered by the funding

Setting and Participants

The sample for this project was patients from internal medicine clinics within the same system. Random sampling, computer generated, was conducted at each site. Three clinics were all located in the greater Midwest city area. Inclusion criteria consisted of the following: (a) adult patients with a diagnosis of depression as identified by the International Classification of Diseases (ICD), 10th revision (WHO, 2016) and ICD 9 and a PHQ-9 screening score >5 ; (b) problem list including diagnosis of diabetes with HbA1c >5 , diagnosis of hypertension, and PHQ-9 score > 5 ; (c) 18 years old or older; and (d) no major psychiatric illness. The PHQ-9 was already a component of the electronic medical record. Exclusion criteria included patients under 18 years of age due to clinic providing care primarily to the adult population, and patients on medications known to exacerbate glycemic control or blood pressure such as atypical antipsychotic agents. Due to anticipated participant size of greater than 30, a priori power calculation with GPower using *t-test*, 0.8 power, alpha 0.05, and medium effect of .5 revealed a total sample size of 34 in the project (see Appendix F for Priori Analysis).

EBP Intervention

The student investigator collected data during the last week of August 2016 from the EHR period of August 1st, 2015 to August 1st, 2016 which represented a cohort prior to psychotherapeutic treatment on patients who meet the inclusion criteria. This retrospective data and a summary of the evidence on depression and CVD was then included in a 30-minute

presentation at the clinics to educate providers on possible improvement in CVD risk factors with psychotherapeutic treatments and referrals. After the first of September 2016, psychotherapeutic treatments and referral was incorporated into patient care. During the first two weeks of January 2017, the student investigator collected existing data from September 1, 2016 – December 31, 2017. The data collected was identical to the baseline inclusion and exclusion criteria with the addition of received psychotherapeutic therapy with treatment medications for depression. The student investigator collected the data and placed into REDCAP, a secure database, and analyzed the data using SPSS (see Appendix G for timeline; Appendix H for Intervention).

The analysis of data was performed during February 15, 2017-March 15, 2017, and several meetings (online) were held at the project sites to discuss results of the depression intervention. The data was presented to the providers in a 30-minute presentation in April 2017 (see Appendix I). The content included data analysis for the depression intervention for the patients with cardiovascular disease risk and outcomes.

Change Process and Evidence Based Practice (EBP) Model

Kotter and Cohen's model of change was utilized in this project to support the change process. This model proposes that the key to organizational change is helping people feel differently (Melnyk, 2011, p. 320). The 8-step model includes need, team selection, vision and strategy, communicating the vision, empowerment, provisional successes, consistent diligence, and nourishment (Melnyk, 2011). Kotter and Cohen's model of change provides a framework to improve communication that can allow individuals to not only identify a problem but also to change their behavior to solve the problems ("The 8-Step Process for Leading Change," 1996; see Appendix J; Appendix K for Logic Model).

The seven-step Iowa model was used as the model for implementing evidence based practice. The Iowa model focuses on organizing, collaborating, and implementing evidence (Doody & Doody, 2011). The steps include selecting an issue, creating a team, supporting evidence retrieval, rating the available evidence, developing an EBP standard, implementing EBP, and evaluating the outcomes (Doody & Doody, 2011). The framework functions in a cyclic fashion and each step presents opportunities to improve the EBP intervention.

Sustainability

The project had major factors promoting sustainability including the possibility of improved patient outcomes and minimal cost associated with the project. There was also no cost to the clinic staff or patients' visit time. After completion of the project, aspects of the project that may result in hindrance are the time commitment for data analysis in the future from a large amount of data from the multiple clinics in order to verify continued benefit of the depression intervention.

Study design

This was a correlational study and provides a framework in which value of a variable is predicted on the data obtained from another variable or variables (Dearholt & Dang, 2012). In the project, the psychotherapy interventions for depression impact on cardiovascular disease risk indicators of blood pressures, HbA1C, depression were explored for associations. The independent variable depression psychotherapy is used to predict the dependent variables of blood pressures, HbA1C, and depression scores.

Validity

The retrospective and prospective design of this evidence-based project provided a time efficient and inexpensive method to collect clinical data which delivered viability problems and

enhanced plans for future studies (Tofthagen, 2012). The EHR and databases foster internal validity because existing data was retrieved from information resources currently used by the project sites and the outcome measurement instruments were standardized across the project clinic and incorporated into the computerized records system. Only the student investigator collected the objective data which excludes patient identification information. For external validity, random selection of samples from the database decreased student investigator selection bias and allowed for the inclusion of all ethnicities who meet inclusion and exclusion criteria. Confounding variables, which may affect the internal validity of the project, were patients with existing depression treatment with medications, poorly controlled hypertension or diabetes, and recent healthy lifestyle interventions of weight control including dieting and exercising. The project can be generalized to other populations of adults with depression at primary care clinics.

Outcomes to be Measured

The primary outcome to be measured was the correlation among depression, blood pressures and HbA1C. Additionally, measured degree of improvement of cardiovascular diseases risk factors of blood pressure, HbA1C, and depression PHQ-9 score after initiation of psychological treatment of referrals and medications for three months and the association of the risk factors related to depression treatment (see Appendix L; M and N).

Measurement Instrument

There were three main instruments for this project. For blood pressures measurement, the AHA guidelines and Eight Joint National Committee 8 (JNC; James et al., 2014) provided the essential procedure to obtain accurate and valid measurements of blood pressure (Pérgola et al., 2007). The project sites followed AHA guidelines and JNC 8 (James et al., 2014) to gather data using manual sphygmomanometer. For HbA1C measurement, the American Diabetic

Association (ADA) guidelines provided the validity to obtain HbA1C measurements through laboratory testing (ADA, 2016). The PHQ-9, a United States Preventative Staff Task Force established tool, was used to screen for depression in adults (USPSTF, 2008). PHQ-9 scores > 10 are reported to have a sensitivity of 88% and a specificity of 88% for Major Depressive Disorder (APA, 2016). The psychometric properties of the tool have been established by reliability and validity with internal consistency of 0.85 (Zhang et al., 2013; APA, 2016). All measurement instruments were in the public domain. The measurements were taken with standardized procedures and the project utilized the existing data.

Quality of Data

Conditional regression analysis was used to determine odds ratio (OR) and a 95% confidence interval (CI) to evaluate depression with cardiovascular risk factors of blood pressures, HbA1C, and depression PHQ-9 score. A *t-test* was used to compare retrospective data collected between August 2015- August 2016 that is prior to the depression intervention and prospective data collected after the intervention. A paired *t-test* analysis was completed for data collected using the intervention during September 2016-December 2016. Analyses included collection and comparisons of blood pressures, HbA1C, and PHQ-9 scores. The study identified for comparison of project results to published studies was a study conducted by Lustman (1998) which reports effective outcomes of cardiovascular disease by improving depression with stepwise behavioral therapy. The study reported cognitive therapy improved glycosylated hemoglobin and depression which is the major risk factors for cardiovascular diseases (Lustman, 1998).

Analysis Plan

Spearman's rank-order correlation was used to explain the correlation in outcomes within the intervention group. The student investigator conducted a primary analysis while using a scaled marginal model that tested for a total effect of the intervention over 3 month outcomes of blood pressure, HbA1C, depression PHQ-9 scores. A *t-test* was used to assess the comparison of outcomes within the intervention group. A *paired t-test* was used to compare data collected before and after the depression intervention. A regression analysis was also conducted to determine how effective independent variables are in predicting the depending variable of blood pressures, HbA1C, and PHQ-9 scores (see Appendix O).

Results

Setting & Participants

Approval for this EBP project was obtained from University of Missouri, Kansas City Institutional Review Board (IRB). A retrospective data set was collected from August 2015-August 2016, which provided the framework of selected patients with required criteria. Providers from three internal medicine clinics were informed of an education intervention before the initiation of prospective data which was collected from September 2016 to January 2017. Randomized patients were selected according to the criterion. Initially, thirty-one patients were randomly selected and data was collected for pre- and post intervention (see Appendix L). The average age of participants was 66 years (standard deviation [SD] = 11.46). There were 17 females and 14 males in the sample.

Intervention Course, Actual

In phase one and two, the student investigator provided a 20-minute educational presentation to internal medicine clinic providers in September 2016 at the monthly provider meeting. The presentation included CVD and depression background, as well as significance of

evidence-based research on reduction of cardiovascular risk by psychotherapeutic intervention with pharmacotherapy. EBP objectives and implementation were explained to providers, and the providers were encouraged to obtain PHQ-9 scores post intervention. Furthermore, initial data including blood pressures, HbA1C, and PHQ-9 scores from a retrospective patient group of thirty-one patients was shared with the providers.

During phase three, pre-intervention data of blood pressures, HbA1C, and PhQ-9 scores was obtained, and psychotherapy was initiated for patients who met criteria during September 2016 and maintained therapy until January 2017. In phase three, total of 31 patients were seen by a behavioral therapist for multiple appointments as determined by the therapist. At the end of the four month intervention, the post intervention blood pressures, HbA1C, and PHQ-9 scores were obtained from the patients (see Appendix L) although post PHQ-9 scores were available for only four of the participants. Phase four included analysis of the data, and in phase five, results were presented to providers at the internal medicine clinics at a monthly meeting in May 2017.

Outcome Data by Sub-Topic

For analysis of the outcome, a correlation between the dependent variable PHQ-9 and independent variables HbA1C and blood pressures was conducted using Spearman's rank-order correlation. Spearman's rank-order correlation coefficient is a nonparametric measure of the strength and direction of associations (Runyon, 2000). Data limitations resulted in correlation with post PHQ-9 scores among only four patients. However, there was a strong, positive correlation between post PHQ-9 and Post A1C scores which approached statistical significance ($r_s = 0.949$, $p = .051$). To predict which variables had greater impact, a regression analysis (See Appendix O) revealed the unstandardized coefficient B for Post A1C, Post Systolic, and Post Diastolic compared to post PHQ-9 were 5.493, 0.322, and -0.938 respectively.

The correlation between post PHQ-9 scores and blood pressures was not statistically significant (Systolic, $r_s = 0.061$, $p = 0.896$; Diastolic, $r_s = -0.09$, $p = 0.847$).

Also, the outcome measured was the degree of improvement of cardiovascular diseases risk factors of blood pressure, HbA1C, and depression PHQ-9 score after initiation of psychological treatment of referrals and medications for three months.

Data for blood pressures and HbA1C was readily available, but PHQ-9 scores were not available for most patients post intervention. The mean values for pre- post systolic blood pressure were 126 (SD = 15.98) and 128 (SD = 11.95), respectively. The mean values for pre- post diastolic blood pressure was 74 (SD = 9.10) and 76 (SD = 8.18), respectively. The pre HbA1C mean was 6.7 (SD = 1.28), and the post-HbA1C was 6.74 (SD = 1.49). The pre-PHQ-9 mean score was 8.2 (SD = 7.31), and the post-PHQ-9 score was 3.5 (SD = 5.83; see Appendix M).

Discussion

Successes, Most Important

The study question for this project posed an investigation of the correlation between depression and cardiovascular risk. This was explored by analyzing correlations among PHQ-9 scores, blood pressures, and HbA1C. Although the sample was small, the project showed that post PHQ-9 scores are directly correlated with post A1C scores. As depression scores decreased A1C scores decreased as well. This finding indicates that individuals with higher depression scores are more likely to have diabetes with higher HbA1Cs. This association increases cardiovascular disease risk for individuals with depression. The results revealed an increase in systolic and diastolic blood pressures, 1.27% and 2.86% respectively. There was a

0.09% reduction in HbA1C scores, and a major change was observed in PHQ-9 scores which was a 60.56% reduction using mean values of the variables.

Regression analysis predicts that for a 5.493 score increase in PHQ-9 score there is a one unit increase in HbA1C scores in post score. While explicating the association between depression and control of cardiovascular risk factors, it is important to understand that among people with diabetes who also have depression that non-compliance and lack of healthy life style may be a common occurrence (Kimbrow, Steers, Mangione, Duru, & Ettner, 2012). There is a cyclical pattern present between diabetes and depression (Kimbrow et al., 2012). Several studies have shown that major depression and diabetes are independently associated with increased coagulation and aggregation of platelets as well as increased response of C-reactive protein (Katon et al., 2004). Presence of depression with diabetes results in additional risk by behavioral mediators such as obesity, sedentary life style, and poor glycemic control, as well as biological mediators (Katon et al., 2004). The project results indicate that psychotherapy can improve cardiovascular risk including HbA1C.

Although there is lack of statistical significance in this project, findings are clinically significant. The reduction in depression scores supports psychotherapeutic referrals. The project shows a correlation between post HbA1C and post PHQ- 9 scores which affirm the reduction in cardiovascular disease risk by psychotherapy. Studies have indicated that simultaneous treatment of depression in patients with diabetes in primary care setting can improve both depression and diabetes (Bogner & Vries, 2010).

Study Strengths

Results of four internal medicine clinics were obtained at one of the three clinic sites by using the EHR. The clinics were based in urban Kansas City. The clinics were fully resourced

with staff to aid in successful completion of the project. The student investigator's site mentor, as well as the administrative staff, were exceptional in moving the project forward. Also, this project provided quality improvement information at this clinic, therefore, the success of the project was important to the internal medicine clinics, providers, and staff.

The project was expected to obtain a sample of at least thirty-four patients, but due to certain barriers including patient drop out and non-compliance, the project was determined to be a pilot study. Data was readily available for the student investigator through secure access. Implementation was successful initially but the post intervention data, especially post PHQ-9 scores, was not obtained at the clinics. Psychotherapy was also an available resource for patients, and there was minimal wait time for patients before they visited the psychotherapist for behavioral intervention.

Results Compared to Evidence in the Literature

Research is limited with the studies that focus on depression treatment, either psychotherapy or pharmaceutical, with cardiovascular disease risk reduction. The results of this project are comparable to other studies published on depression intervention for cardiovascular risk reduction. A study by Lustmen (1998) concluded that although there was no immediate changes in post intervention measures, there was a significant improvement in follow-up mean glycosylated hemoglobin. The result of this current project reports similar findings about depression and A1C correlation.

Another study concluded that psychotherapy reduces depression and results in improved blood pressures for patients, and there was significant improvement observed after 8-weeks of psychotherapy (Ahmadpanah et al., 2016). This evidenced-based project did not show significant improvement in blood pressures for patients. However, results are mixed as some patients

showed improvement with several sessions and some did not show improvement in systolic or diastolic blood pressures. Additionally, some patients had improvement in blood pressures with only a few sessions. These findings suggest that there is a need for further research on the impact of psychotherapeutic and pharmacotherapeutic treatments of depression among patients at risk for cardiovascular disease. This is especially important to achieve the goal of decreasing depression among vulnerable population with multiple chronic co-morbidities.

Limitations

Internal Validity Effects

Utilization of the EHR to retrieve existing data and use of standardized instruments at the clinic promoted internal validity of the project. Confounding variables, which could have affected the internal validity of the project, were patients with existing depression treatment with medications, poorly controlled hypertension or diabetes, recent healthy lifestyle interventions of weight control including diet and exercise, non-compliant patients, changes in primary care practices and collection of data in winter months (Rohan et al., 2013). These variables could have exacerbated depression results (Rohan et al., 2013).

External validity effects

For external validity, random selection of samples from the database decreased student investigator selection bias and allowed for the inclusion of all ethnicities that met inclusion and exclusion criteria. The project was expected to be generalized to other populations of adults with depression at primary care clinics but small sample size affected the strength of the project. The project was conducted over four months which is a relatively short time frame for an intervention to make a positive effect. Although this project focused on a population with existing chronic conditions and multiple co-morbidities, the depression intervention can be applied to other

healthier populations for the prevention of cardiovascular diseases such as hypertension and diabetes.

Sustainability of effects and plans to maintain effects

Internal medicine clinics were involved in this project and were working towards a policy change to include psychotherapeutic intervention in collaboration with pharmacotherapy. This project will provide strength for the depression intervention in the future as the findings indicated improved patient outcome. Aspects of the project that may result in hindrance of sustainability are the time commitment for data analysis in the future from a large amount of data from the multiple clinics in order to verify continued benefit of the depression intervention. There is an ongoing effort at the clinics to maintain the improvement in patient outcome and remind providers to refer patients, who have depression and at risk for cardiovascular disease, immediately for psychotherapy.

Efforts to minimize the study limitations

There were several improvements that were made to minimize project limitations including a presentation to providers to explain the importance of the project and improved outcomes for patients. During the presentation to clinic staff, the importance of proper technique to measure blood pressure was also stressed. Biases were minimized by randomized selection. The effect of limitations resulted in reduced sample size which in turn provided skewed data. This affected the comparison of blood pressures with PHQ-9 as well as with A1C and PHQ-9.

Interpretation

Expected & Actual outcomes

Expected outcomes included improved blood pressures, HbA1C, and PHQ-9 scores with psychotherapeutic and pharmacotherapeutic intervention and correlation among depression,

hypertension, and diabetes. The project showed that there is a strong correlation between PHQ-9 scores and HbA1C values in post interventional results. Unexpected results included no correlation between post PHQ-9 scores and post blood pressures. Blood pressures are taken manually at the clinic; hence, human error is one possible reasoning for finding no correlation between PHQ-9 scores and blood pressures. Also, baseline blood pressures at pre intervention were close to normal values, therefore, there was reduced chance of improvement with intervention. Furthermore, PHQ-9 scores were not available for the post intervention. This is possibly due to educational deficit about the importance of the intervention and project. Although, there was a presentation prior to initiation of project to explain the importance and relevance of the project and possible outcomes for patients, data was not collected by the clinics.

Intervention's Effectiveness (Inferences)

Due to lack of post intervention PHQ-9 data, the project is now presented as a pilot study. However, the strong correlation between PHQ-9 scores and HbA1C scores infer that depression treatment greatly influences improvement in HbA1C values. Consequently, psychotherapeutic and pharmacotherapeutic interventions can reduce cardiovascular disease risk factors in primary care clinics. Causality has met all three conditions in this EBP project but cannot be implied due to small sample size; as the time-order is met, there is a correlation seen between PHQ-9 scores and HbA1C, and no other variable in the project intervention can explain this correlation. This project did not show any correlation between blood pressures and PHQ-9 score.

The project intervention can be effective in any internal medicine clinic as well as family medicine clinic. The psychotherapy resource is the major barrier for implementation of the intervention as it initially increases cost to the practice. Furthermore, psychotherapist availability is limited for patients and sometimes require long wait before the care is available for them. As

long as referrals are made and patients are seen in a timely fashion, the depression intervention is a viable adjunct treatment option for reducing cardiovascular disease risk.

Intervention revision

There are several changes possible for improved positive outcomes. First, the gathering of data prior to the intervention can be improved by obtaining blood pressures, HbA1C, and PHQ-9 scores at the same time to reduce possibility of skewed data. Second, intervention time extension can be highly valuable in explaining if more psychotherapy appointments make a larger impact on cardiovascular disease risk factors. Third, the project was limited to a population older than 50-years; it would be noteworthy to study the impact of this intervention on a younger population. Finally, improvement can be measured by inclusion of cholesterol levels because of the link with cardiovascular disease risk (Katon et al., 2004).

Expected and actual impact to health system, costs, and policy

This evidence based intervention was shown, at least partially, to be successful in supporting prior evidence indicating correlation between depression and cardiovascular risks. It is important to keep in mind that these patients were simultaneously being treated with pharmacotherapy. The project provides a strong framework for change in policy and procedures at the primary care clinics to improve patient outcomes. The project was conducted with minimal cost, and the cost remained the same as predicted prior to initiation of project. Due to minimal cost of project, sustainability of the project is high. The primary site where the project was conducted already has psychotherapists employed as a team. Quality improvement teams can reinforce education to primary care providers about persistent referrals to a psychotherapist to improve cardiovascular risk factors which can easily become part of their monthly meetings, as

this student investigator has done. Current funding by Women's Graduate Assistance Fund (GAF) provided the cost of conducting the project as well as dissemination at conferences.

Conclusion

This project explored the effect of psychological treatment and medications for depression in adult patients on cardiovascular disease risk factors measuring blood pressure, HbA1C, and depression PHQ-9 scores. This EBP initiative showed that effective treatment for depression can decrease the risk of cardiovascular disease in the short term. This project strengthens the findings of other studies and reports results that support use of both medication and behavioral therapy to improve outcomes for patients. However, it does not distinguish which therapy is superior to the other.

The depression screening can easily be implemented with patients although referral services may need to be addressed by the site if services that are convenient to the patient are absent. Further studies should be performed to explore long-term effects of depression treatment in cardiovascular risk factors.

The project results were presented at Advanced Practice Nurses of Ozarks (APNO) annual conference in November 2016 and Midwest Nurses Research Society's (MNRS) annual conference in April 2017 as a poster presentation to educate providers and nurse researchers about importance of depression treatment which can ultimately foster reduction in cardiovascular risk factors. The providers at the project sites have data information available so that further evaluation or studies may be conducted. The results from this project can provide a framework for other studies that might have specific ethnicities or populations. Also, depression intervention can be separated into either psychotherapy or medications, and impacts on cardiovascular disease risk factors can be studied instead of the combined intervention within this project. Proper and

timely treatment of depression may result in decreased cardiovascular risk factors for patients as well as reduction in overall healthcare cost and quality of life.

References

- Ahmadpanah, M., Paghale, S. J., Bakhtyari, A., Kaikhavani, S., Aghaei, E., Nazaribadie, M., ... Brand, S. (2016). Effects of psychotherapy in combination with pharmacotherapy, when compared to pharmacotherapy only on blood pressure, depression, and anxiety in female patients with hypertension. *Journal of Health Psychology, 21*(7), 1216–1227.
<https://doi.org/10.1177/1359105314550350>
- Almas, A., Forsell, Y., Iqbal, R., Janszky, I., & Moller, J. (2015). Severity of Depression, Anxious Distress and the Risk of Cardiovascular Disease in a Swedish Population-Based Cohort. *PLOS ONE, 10*(10), e0140742. <https://doi.org/10.1371/journal.pone.0140742>
- APA. (2016). Patient Health Questionnaire (PHQ-9 & PHQ-2). Retrieved July 10, 2016, from <http://www.apa.org/pi/about/publications/caregivers/practice-settings/assessment/tools/patient-health.aspx>
- Baune, B. T., Stuart, M., Gilmour, A., Wersching, H., Heindel, W., Arolt, V., & Berger, K. (2012). The relationship between subtypes of depression and cardiovascular disease: a systematic review of biological models. *Translational Psychiatry, 2*(3), e92.
<https://doi.org/10.1038/tp.2012.18>
- Bogner, H. R., & Vries, H. F. de. (2010). Integrating Type 2 Diabetes Mellitus and Depression Treatment Among African Americans A Randomized Controlled Pilot Trial. *The Diabetes Educator, 36*(2), 284–292. <https://doi.org/10.1177/0145721709356115>
- Bradley, S. M., & Rumsfeld, J. S. (2015). Depression and cardiovascular disease. *Trends in Cardiovascular Medicine, 25*(7), 614–622.
<https://doi.org/http://dx.doi.org.proxy.library.umkc.edu/10.1016/j.tcm.2015.02.002>

Coventry, P. A., Hudson, J. L., Kontopantelis, E., Archer, J., Richards, D. A., Gilbody, S., ...

Bower, P. (2014). Characteristics of Effective Collaborative Care for Treatment of Depression: A Systematic Review and Meta-Regression of 74 Randomised Controlled Trials: e108114. *PLoS One*, 9(9), e108114.

<https://doi.org/http://dx.doi.org.proxy.library.umkc.edu/10.1371/journal.pone.0108114>

Coventry, P., Lovell, K., Dickens, C., Bower, P., Chew-Graham, C., McElvenny, D., ... Gask, L.

(2015). Integrated primary care for patients with mental and physical multimorbidity: cluster randomised controlled trial of collaborative care for patients with depression comorbid with diabetes or cardiovascular disease. *BMJ*, 350, h638.

<https://doi.org/10.1136/bmj.h638>

Dearholt, S., & Dang, D. (2012). *Johns Hopkins Nursing Evidence-based Practice: Models and*

Guidelines. Sigma Theta Tau.

Delaney, C., Barrere, C., & Helming, M. (2011). The Influence of a Spirituality-Based

Intervention on Quality of Life, Depression, and Anxiety in Community-Dwelling Adults With Cardiovascular Disease A Pilot Study. *Journal of Holistic Nursing*, 29(1), 21–32.

<https://doi.org/10.1177/0898010110378356>

Deschênes, S. S., Burns, R. J., & Schmitz, N. (2015). Associations between diabetes, major

depressive disorder and generalized anxiety disorder comorbidity, and disability:

Findings from the 2012 Canadian Community Health Survey — Mental Health (CCHS-MH). *Journal of Psychosomatic Research*, 78(2), 137–142.

<https://doi.org/10.1016/j.jpsychores.2014.11.023>

Doody, C., & Doody, O. (2011). Introducing evidence into nursing practice: using the IOWA model. Retrieved from

<https://ulir.ul.ie/bitstream/handle/10344/1801/Doody.pdf?sequence=2>

Echeverry, D., Duran, P., Bonds, C., Lee, M., & Davidson, M. B. (2009). Effect of Pharmacological Treatment of Depression on A1C and Quality of Life in Low-Income Hispanics and African Americans With Diabetes A randomized, double-blind, placebo-controlled trial. *Diabetes Care*, *32*(12), 2156–2160. <https://doi.org/10.2337/dc09-0785>

Fiedorowicz, J. G. (2014). Depression and Cardiovascular Disease: An Update on How Course of Illness May Influence Risk. *Current Psychiatry Reports*, *16*(10), 1–12.

<https://doi.org/10.1007/s11920-014-0492-6>

Gardner-Sood, P., Lally, J., Smith, S., Atakan, Z., Ismail, K., Greenwood, K. E., ... Gaughran, F. (2015). Cardiovascular risk factors and metabolic syndrome in people with established psychotic illnesses: baseline data from the IMPaCT randomized controlled trial.

Psychological Medicine, *45*(12), 2619–2629.

<https://doi.org/http://dx.doi.org.proxy.library.umkc.edu/10.1017/S0033291715000562>

Georgiades, A., Zucker, N., Friedman, K. E., Mosunic, C. J., Applegate, K., Lane, J. D., ...

Surwit, R. S. (2007). Changes in Depressive Symptoms and Glycemic Control in

Diabetes Mellitus: *Psychosomatic Medicine*, *69*(3), 235–241.

<https://doi.org/10.1097/PSY.0b013e318042588d>

Hare, D. L., Toukhsati, S. R., Johansson, P., & Jaarsma, T. (2014). Depression and cardiovascular disease: a clinical review. *European Heart Journal*, *35*(21), 1365–1372.

<https://doi.org/10.1093/eurheartj/eh462>

- James, P. A., Oparil, S., Carter, B. L., Cushman, W. C., Dennison-Himmelfarb, C., Handler, J., ... Ortiz, E. (2014). 2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report From the Panel Members Appointed to the Eighth Joint National Committee (JNC 8). *JAMA*, *311*(5), 507.
<https://doi.org/10.1001/jama.2013.284427>
- Katon, W. J., Lin, E. H. B., Russo, J., Von Korff, M., Ciechanowski, P., Simon, G., ... Young, B. (2004). Cardiac Risk Factors in Patients with Diabetes Mellitus and Major Depression. *Journal of General Internal Medicine*, *19*(12), 1192–1199.
<https://doi.org/10.1111/j.1525-1497.2004.30405.x>
- Kimbro, L. B., Steers, W. N., Mangione, C. M., Duru, O. K., & Ettner, S. L. (2012). The Association of Depression and the Cardiovascular Risk Factors of Blood Pressure, HbA1c, and Body Mass Index among Patients with Diabetes: Results from the Translating Research into Action for Diabetes Study. *International Journal of Endocrinology*, *2012*, e747460. <https://doi.org/10.1155/2012/747460>
- Kronish, I. M., Rieckmann, N., Burg, M. M., Edmondson, D., Schwartz, J. E., & Davidson, K. W. (2012). The effect of enhanced depression care on adherence to risk-reducing behaviors after acute coronary syndromes: Findings from the COPES trial. *The American Heart Journal*, *164*(4), 524–9.
<https://doi.org/http://dx.doi.org.proxy.library.umkc.edu/10.1016/j.ahj.2012.07.024>
- Lustman, P. J. (1998). Cognitive Behavior Therapy for Depression in Type 2 Diabetes Mellitus: A Randomized, Controlled Trial. *Annals of Internal Medicine*, *129*(8), 613.
<https://doi.org/10.7326/0003-4819-129-8-199810150-00005>

Meijer, A., Conradi, H. J., Bos, E. H., Thombs, B. D., van Melle, J. P., & de Jonge, P. (2011).

Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta-analysis of 25 years of research. *General Hospital Psychiatry*, *33*(3), 203–216. <https://doi.org/10.1016/j.genhosppsych.2011.02.007>

Mejia-Lancheros, C., Estruch, R., Martínez-González, M., Salas-Salvadó, J., Corella, D.,

Gómez-Gracia, E., ... the PREDIMED Study Investigators. (2014). Blood pressure values and depression in hypertensive individuals at high cardiovascular risk. *BMC Cardiovascular Disorders*, *14*(1), 109. <https://doi.org/10.1186/1471-2261-14-109>

Melnik, B. M. (2011). *Evidence-based practice in nursing & healthcare: a guide to best practice* (2nd ed). Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.

Möller-Leimkühler, A. M. (2010). Higher comorbidity of depression and cardiovascular disease

in women: A biopsychosocial perspective. *The World Journal of Biological Psychiatry*, *11*(8), 922–933. <https://doi.org/10.3109/15622975.2010.523481>

Nemeroff, C. B., & Goldschmidt-Clermont, P. J. (2012). Heartache and heartbreak—the link

between depression and cardiovascular disease. *Nature Reviews Cardiology*, *9*(9), 526–539. <https://doi.org/10.1038/nrcardio.2012.91>

O’Neil, A., Williams, E. D., Stevenson, C. E., Oldenburg, B., Berk, M., & Sanderson, K. (2011).

Co-morbid cardiovascular disease and depression: sequence of disease onset is linked to mental but not physical self-rated health. Results from a cross-sectional, population-based study. *Social Psychiatry and Psychiatric Epidemiology*, *47*(7), 1145–1151.

<https://doi.org/10.1007/s00127-011-0421-5>

O’Neil, A., Williams, E. D., Stevenson, C. E., Oldenburg, B., & Sanderson, K. (2012). Co-

morbid depression is associated with poor work outcomes in persons with cardiovascular

- disease (CVD): A large, nationally representative survey in the Australian population. *BMC Public Health*, 12, 47. <https://doi.org/10.1186/1471-2458-12-47>
- Pérgola, P. E., White, C. L., Graves, J. W., Coffey, C. S., Tonarelli, S. B., Hart, R. G., & Benavente, O. R. (2007). Reliability and validity of blood pressure measurement in the Secondary Prevention of Small Subcortical Strokes study. *Blood Pressure Monitoring*, 12(1), 1–8. <https://doi.org/10.1097/MBP.0b013e3280858d5b>
- Rohan, K. J., Evans, M., Mahon, J. N., Sitnikov, L., Ho, S.-Y., Nillni, Y. I., ... Vacek, P. M. (2013). Cognitive-behavioral therapy vs. light therapy for preventing winter depression recurrence: study protocol for a randomized controlled trial. *Trials*, 14, 82. <https://doi.org/10.1186/1745-6215-14-82>
- Rude, J., Azimova, K., Dominguez, C., Sarosiek, J., Edlavitch, S., & Mukherjee, D. (2014). Depression as a Predictor of Length of Stay in Patients Admitted to the Cardiovascular Intensive Care Unit at a University Medical Center. *Angiology*, 65(7), 580–584. <https://doi.org/10.1177/0003319713496868>
- Runyon, R. P. (2000). *Fundamentals of behavioral statistics* (9th ed). Boston, Mass: McGraw-Hill.
- Seldenrijk, A., Vogelzangs, N., Batelaan, N. M., Wieman, I., van Schaik, D. J. F., & Penninx, B. J. W. H. (2015). Depression, anxiety and 6-year risk of cardiovascular disease. *Journal of Psychosomatic Research*, 78(2), 123–129. <https://doi.org/10.1016/j.jpsychores.2014.10.007>
- Smith, S. C., Benjamin, E. J., Bonow, R. O., Braun, L. T., Creager, M. A., Franklin, B. A., ... Taubert, K. A. (2011). AHA/ACCF Secondary Prevention and Risk Reduction Therapy

for Patients With Coronary and Other Atherosclerotic Vascular Disease: 2011 Update.

Retrieved from <http://circ.ahajournals.org>

Stewart, J. C., Perkins, A. J., & Callahan, C. M. (2014). Effect of Collaborative Care for Depression on Risk of Cardiovascular Events: Data From the IMPACT Randomized Controlled Trial. *Psychosomatic Medicine*, 76(1), 29–37.

<https://doi.org/10.1097/PSY.0000000000000022>

The 8-Step Process for Leading Change. (1996). Retrieved November 26, 2014, from

<http://www.kotterinternational.com/the-8-step-process-for-leading-change/>

Tofthagen, C. (2012). Threats to Validity in Retrospective Studies. *Journal of the Advanced Practitioner in Oncology*, 3(3), 181–183.

U.S. Department of Health and Human Services. (2016, February 12). Healthy People 2020:

Topics. Retrieved from <http://www.healthypeople.gov/2020/topics-objectives/topic/heart-disease-and-stroke>

USPSTF. (2008). Final Update Summary: Lipid Disorders in Adults (Cholesterol,

Dyslipidemia): Screening - US Preventive Services Task Force. Retrieved April 15, 2016, from

<http://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/lipid-disorders-in-adults-cholesterol-dyslipidemia-screening>

Vaccarino, V., Johnson, B. D., Sheps, D. S., Reis, S. E., Kelsey, S. F., Bittner, V., ... Bairey

Merz, C. N. (2007). Depression, Inflammation, and Incident Cardiovascular Disease in Women With Suspected Coronary Ischemia: The National Heart, Lung, and Blood

Institute-Sponsored WISE Study. *Journal of the American College of Cardiology*, 50(21),

2044–50.

<https://doi.org/http://dx.doi.org.proxy.library.umkc.edu/10.1016/j.jacc.2007.07.069>

Van der Kooy, K., van Hout, H., Marwijk, H., Marten, H., Stehouwer, C., & Beekman, A. (2007). Depression and the risk for cardiovascular diseases: systematic review and meta analysis. *International Journal of Geriatric Psychiatry*, *22*(7), 613–626.

<https://doi.org/10.1002/gps.1723>

Wagner, E H. (1998). Chronic Disease Management: what will it take to improve care for chronic illness. *Effective Clinical Practice*, *1*(1), 2.

Wagner, Edward H., Austin, B. T., Davis, C., Hindmarsh, M., & al, et. (2001). Improving chronic illness care: Translating evidence into action. *Health Affairs*, *20*(6), 64–78.

Whalley, B., Thompson, D. R., & Taylor, R. S. (2014). Psychological Interventions for Coronary Heart Disease: Cochrane Systematic Review and Meta-analysis. *International Journal of Behavioral Medicine*, *21*(1), 109–21.

<https://doi.org/http://dx.doi.org.proxy.library.umkc.edu/10.1007/s12529-012-9282-x>

Wheeler, A., Beltrame, J., Tucker, G., Air, T., Ling, L.-H., & Schrader, G. (2012). Depression and 5-year mortality in patients with acute myocardial infarction: Analysis of the IDACC database. *Australian and New Zealand Journal of Psychiatry*, *46*(7), 669–675.

<https://doi.org/10.1177/0004867412449875>

Zhang, Y.-L., Liang, W., Chen, Z.-M., Zhang, H.-M., Zhang, J.-H., Weng, X.-Q., ... Zhang, Y.-L. (2013). Validity and reliability of Patient Health Questionnaire-9 and Patient Health Questionnaire-2 to screen for depression among college students in China. *Asia-Pacific Psychiatry: Official Journal of the Pacific Rim College of Psychiatrists*, *5*(4), 268–275.

<https://doi.org/10.1111/appy.12103>

Appendix A: Definition of Terms

Prevalence - The percentage of a population that is affected with a particular disease at a given time.

Psychotherapy – In respect to this project this includes pharmaceutical and behavioral psychological therapy for patients with depression.

Cardiovascular disease – This term includes diseases related to cardiac function including hypertension, diabetes, chronic kidney disease, and cardiac anatomy change due to blood flow, atherosclerosis as well as other cardiac related diseases.

Appendix B: Synthesis of Evidence Table

First author, Year, Title, Journal	Purpose	Research Design ¹ , Evidence Level ² & Variables	Sample & Sampling, Setting	Measures & Reliability (if reported)	Results & Analysis Used	Limitations & Usefulness
1) Correlation between depression and cardiovascular diseases risk						
Seldenrijk (2015). Depression, anxiety and 6-year risk of cardiovascular disease. Journal of Psychosomatic Research	To examine 6-year associations between depressive and anxiety disorders, clinical characteristics and newly-developed CVD	Quantitative, Level IV	6 year incidence assessment	CI 95%	Cox regression analyses. Current depressive (but not anxiety) disorder independently contributed to CVD in our sample of initially CVD-free participants. CVD incidence over 6 years of follow-up was particularly increased in subjects with more symptoms, and in those using benzodiazepines.	
Deschênesa (2015). Associations between diabetes, major depressive disorder and generalized anxiety	To examine the associations between diabetes, disability, and the likelihood of comorbid major	Level IV	17,623 surveys	CI 95%	Individuals with diabetes may be particularly vulnerable to comorbid MDD and GAD, and MDD-	-Cross-sectional and thus temporal relationships between MDD, GAD, and diabetes cannot

<p>disorder comorbidity, and disability: Findings from the 2012 Canadian Community Health Survey — Mental Health (CCHS-MH). Journal of Psychosomatic Research</p>	<p>depressive disorder (MDD) and generalized anxiety disorder (GAD).</p>				<p>GAD comorbidity may exacerbate disability in persons with diabetes. - Composite International Diagnostic Interview 3.0.</p>	<p>be identified. - Diabetes status was assessed via self-reported medical diagnoses</p>
<p>Fiedorowicz (2014). Depression and Cardiovascular Disease: An Update on How Course of Illness May Influence Risk. Curr Psychiatry Rep.</p>	<p>Literature review linking depressive disorders to cardiovascular mortality with a focus on how the course of illness of mood disorders may influence this risk</p>	<p>Level VII</p>	<p>N/A</p>	<p>N/A</p>	<p>Persons with depressive disorders and other risk factors for vascular disease represent a neglected, high-risk group for cardiovascular events.</p>	
<p>Hare (2014). Depression and cardiovascular disease: a clinical Review. European Heart Journal</p>	<p>To explore relationship between depression and cardiovascular diseases.</p>	<p>Level IV</p>	<p>N/A</p>	<p>N/A</p>	<p>Depression is probably the most important driver of overall quality of life.</p>	
<p>Chang (2013). Cognitive performance in older elderly men with late-life</p>	<p>Whether depression or cardiovascular disease would have a greater</p>	<p>Cross sectional, Level IV</p>	<p>207 cognitively impaired older elderly (≥ 75 years old) men</p>		<p>Late life depression worsens neuropsychological function more than cardiovascular</p>	

<p>depression and cardiovascular comorbidities: symptomatological correlation. Annals of General Psychiatry</p>	<p>effect on worsening cognitive impairment in the burgeoning older elderly population</p>				<p>comorbidities do.</p>	
<p>Wheeler (2012). Depression and 5-year mortality in patients with acute myocardial infarction: Analysis of the IDACC database. Australian and New Zealand Journal of Psychiatry</p>	<p>Study aimed to determine whether depression during hospitalization for acute MI (AMI) predicted 5-year all-cause or cardiac mortality.</p>	<p>Quantitative, RCT, Level IV</p>	<p>Database of 337 hospitalized patients with AMI.</p>	<p>CI 95%</p>	<p>Relationship between mortality and depression severity is not linear and that the association only becomes evident when the severity reaches a threshold level of CES-D \geq 27, consistent with major depression. - SPSS statistical software package version 11.0.4</p>	
<p>Baune (2012). The relationship between subtypes of depression and cardiovascular disease: a systematic review of biological models. Translational Psychiatry</p>	<p>Indications of depression subtype specific biological mechanisms.</p>	<p>Systemic Literature review. Level I</p>	<p>147 articles</p>	<p>CI 95%</p>	<p>Diagnostic subtypes rather than a unifying model of depression should be considered when investigating the bidirectional biological relationship between CVD and depression.</p>	<p>- Studies with small number of subjects - Variation in used instruments and criteria to classify depression</p>

<p>Meijer (2011). Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta-analysis of 25 years of research. Science direct</p>	<p>To investigate changes in this association over time and to investigate subgroup effects between MI and depression.</p>	<p>Meta analysis RCT, quantitative, Level I</p>	<p>29 studies</p>	<p>CI 95%</p>	<p>Post-MI depression is associated with a 1.6- to 2.7-fold increased risk of impaired outcomes within 24 months. This association has been relatively stable over the past 25 years</p>	<p>-Could not provide a pooled association that is consistently adjusted for the same variables across studies.</p>
<p>(2011). AHA/ACCF Secondary Prevention and Risk Reduction Therapy for Patients With Coronary and Other Atherosclerotic Vascular Disease: 2011 Update. AHA</p>	<p>Evidence based practice guideline.</p>	<p>Guidelines, Level I,</p>				
<p>American Heart Association - Professional Association, Effectiveness-based guidelines for the prevention of</p>		<p>Guidelines, level 1</p>	<p>Studies from 2006-2010, including randomized control trials</p>			

<p>cardiovascular disease in women—2011 update: a guideline from the American Heart Association.</p>						
<p>Vaccarino (2007). Depression, Inflammation, and Incident Cardiovascular Disease in Women With Suspected Coronary Ischemia: The National Heart, Lung, and Blood Institute-Sponsored WISE Study. Journal of the American College of Cardiology</p>	<p>Whether the level of 2 inflammatory biomarkers, interleukin-6 and C-reactive protein, explained the relationship between depression and CVD in 559 women referred for coronary angiography for suspected coronary ischemia</p>	<p>Quantitative, Randomized control Trials, Level 1</p>	<p>935 participants from which 674 were used who filled out depression scale</p>	<p>CI 95%</p>	<p>In women with suspected coronary ischemia, inflammation is independently correlated with depression</p>	<ul style="list-style-type: none"> - Sample might not be generalized to all women. - Sample was heterogeneous - No info on MDD through interview - Delayed implementation of the psychosocial questionnaire reducing the sample size
<p>Van der Kooy (2007). Depression and the risk for the cardiovascular diseases: systematic review and meta analysis. International</p>	<p>To estimate the risk of depression as an independent risk factor for various cardiovascular diseases (CVD) and explore the</p>	<p>Quantitative, longitudinal and case controlled, Level 1</p>	<p>11 studies were selected from a pool of 28 studies</p>	<p>Confidence interval 95%.</p>	<p>Overall combined risk of depression for the onset of myocardial infarction was homogenous and major depressive disorder was the</p>	<p>Possibly missed some studies from non-journal publication. Presence of substantial heterogeneity could produce</p>

Journal of geriatric psychiatry	effects of the				most important risk factor for developing CVD.	spurious results. Pooling of studies can also be a limitation.
2) Relationship between depression and cardiovascular outcomes						
Almas (2015). Severity of Depression, Anxious Distress and the Risk of Cardiovascular Disease in a Swedish Population-Based Cohort. PloS one.	To determine the association between depression of varying severity and risk for CVD and to study the effect of concomitant anxious distress on this association.	Quantitative, Cohort longitudinal study, Level I	Mental health, work and relations among adults (20–64 years), with a total of 10,443 individuals. Depression and anxious distress were assessed using psychiatric rating scales and defined according to DSM-5	CI 95%	Severity level of depression seems to be of significance for increased risk of CVD among depressed persons. Higher risk of CVD among depressed individuals with symptoms of anxious distress	- Low response rate - Low follow up rate could have caused potential underestimation of CVD outcomes. - No access to the migration or death register
Gardner-Sood (2015). Cardiovascular risk factors and metabolic syndrome in people with established psychotic illnesses: baseline data from the IMPaCT randomized	To determine the prevalence of cardiometabolic risk factors and establish the proportion of people with psychosis meeting criteria for the metabolic syndrome (MetS)	RCT, Cross-sectional, Level I	450 randomly selected out-patients, aged 18-65 years with established psychotic illness	P<0.001	The prevalence of cardiometabolic risk factors in individuals with psychotic illnesses is much higher than that observed in national general population studies	Cross-sectional design, and the lack of a control group

<p>controlled trial. Psychological Medicine</p>	<p>and to identify the key lifestyle behaviours associated with increased risk of the MetS and to investigate whether the MetS is associated with illness severity and degree of functional impairment.</p>					
<p>Seldenrijk (2014). Depression, anxiety and 6-year risk of cardiovascular disease. Journal of Psychosomatic Research</p>	<p>Examined 6-year associations between depressive and anxiety disorders, clinical characteristics and newly-developed CVD</p>	<p>Quantitative, Longitudinal study RCT, Level II</p>	<p>106 subjects</p>	<p>CI 95%</p>	<p>Current depressive (but not anxiety) disorder independently contributed to CVD in our sample of initially CVD-free participants. CVD incidence over 6 years of follow-up was particularly increased in subjects with more symptoms, and in those using benzodiazepines.</p>	<p>Limitations: presence of CVD was not verified by general practice or hospitalization records. findings might have been affected by criterion contamination</p>
<p>Mejia-Lancheros (2014). Blood pressure values and depression in</p>	<p>To analyze whether depression may influence the</p>	<p>Quantitative, Cross-sectional Study, Level</p>	<p>5954 hypertensive patients with high</p>	<p>CI 95%</p>	<p>Depressive patients, with and without antidepressant treatment, had better</p>	<p>- Does not allow causal inferences to be drawn - Smaller sample</p>

<p>hypertensive individuals at high cardiovascular risk. BMC cardiovascular disorders.</p>	<p>control of blood pressure in hypertensive individuals at high cardiovascular risk.</p>	<p>IV</p>	<p>cardiovascular risk factor from 7447 PREDIMED study participants</p>		<p>blood pressure control. Multivariate analysis (logistic and log-linear regression)</p>	<p>size in relation with depression - Lack of family history of depression and hypertension.</p>
<p>Rude (2013). Depression as a Predictor of Length of Stay in Patients Admitted to the Cardiovascular Intensive Care Unit at a University Medical Center. Angiology</p>	<p>To assess the impact of depression on length of stay (LOS) in a Hispanic population admitted to a cardiovascular intensive care unit (CVICU).</p>	<p>Quantitative, Level IV</p>	<p>151 consecutive patients admitted to hospital</p>	<p>P=0.001</p>	<p>Multivariate analysis. - Depression has a significant impact on LOS in a Hispanic population. Appropriate treatment of depression may decrease LOS and has the potential to be cost effective in the current health care environment.</p>	
<p>O'Neal (2012). Co-morbid depression is associated with poor work outcomes in persons with cardiovascular disease (CVD): A large, nationally representative</p>	<p>To examine the association of co-morbid MDD with work outcomes in persons with and without CVD.</p>	<p>Quantitative, Cross-sectional-longitudinal, Level IV</p>	<p>2007 Australian National Survey of Mental Health and Wellbeing n=8841.</p>	<p>CI 95%</p>	<p>MDD was associated with a four-fold increase in impaired functioning. individuals with co-morbid MDD and CVD reported greatest likelihood</p>	<p>- Self report measures used, may have led to recall bias, misclassification or incorrect identification of CVD - Under reporting</p>

survey in the Australian population. BMC Public Health.					of workplace absenteeism.	of MDD
O'Neil (2011) Co-morbid cardiovascular disease and depression: sequence of disease onset is linked to mental but not physical self-rated health. Results from a cross-sectional, population-based study. Social Psychiatry and Psychiatric Epidemiology	To identify the prevalence of major depressive disorder (MDD) preceding CVD and secondly determine whether sequence of disease onset is associated with mental and physical self-rated health	Quantitative, Cross-sectional, Level IV	224 respondents of the 2007 Australian National Survey of Mental Health and Wellbeing (NSMHWB)	CI 95%	The proportion of individuals in whom MDD preceded CVD was 80.36% (CI: 72.57–88.15). One-fifth (19.64%, CI: 11.85–27.42) reported MDD onset at the time of, or following, CVD. MDD is most common prior to the onset of CVD.	- CVD diagnosis self reported. - People with other circulatory conditions could have been included.
Alderson (2011). How patients understand depression associated with chronic physical disease – a systematic review. BMC Family Practice. Biomed Central	To understand people's beliefs about depression, particularly in the presence of chronic physical disease	Mixed method systematic review. Level I.	65 studies	CI 95%	Approaches to detection of depression in physical illness need to be receptive to the range of beliefs held by patients. Patient beliefs have implications for engagement with depression screening.	- Limit scope to primary care

(2011). AHA/ACCF Secondary Prevention and Risk Reduction Therapy for Patients With Coronary and Other Atherosclerotic Vascular Disease: 2011 Update. AHA	Update on evidenced-based practice guideline	Level I				
3) Impact of psychotherapy interventions on reduction of cardiovascular disease risk factors						
Coventry (2015). Integrated primary care for patients with mental and physical multimorbidity: cluster randomised controlled trial of collaborative care for patients with depression comorbid with diabetes or	To test the effectiveness of an integrated collaborative care model for people with depression and long term physical conditions.	Cluster randomized controlled trial. Level II	387 patients with a record of diabetes or heart disease, or both.	CI 95%	Collaborative care that incorporates brief low intensity psychological therapy delivered in partnership with practice nurses in primary care can reduce depression and improve self management of chronic disease in people with mental	- Could not recruit and assess participants before practices were randomized - Were able to assess only the short term effectiveness of collaborative care

cardiovascular disease. BMJ					and physical multimorbidity.	
Whalley (2014). Psychological Interventions for Coronary Heart Disease: Cochrane Systematic Review and Meta-analysis. International Journal of Behavioral Medicine	Aims to estimate effects of psychological interventions on mortality and psychological symptoms in this group, updating an existing Cochrane Review.	Systematic review and meta-regression analyses of randomized trials. Level I.	Sixteen studies (27 publications) from the 2004 Cochrane Review	CI 95%	No strong evidence that psychological intervention reduced total deaths, risk of revascularization, or non-fatal infarction. Psychological intervention did result in small/moderate improvements in depression and anxiety, and there was a small effect for cardiac mortality.	- lack of methodological detail reported by authors - excluding our only Chinese study altered the inference for anxiety outcomes
Coventry (2014). Characteristics of Effective Collaborative Care for Treatment of Depression: A Systematic Review and Meta-Regression of 74 Randomized Controlled Trials: e108114. PLoS one.	A meta-regression to identify factors in collaborative care associated with improvement in patient outcomes and the process of care.	Randomized control Trials, Level I	Seventy four trials were identified (85 comparisons, across 21,345 participants)	CI 95%	Trials of collaborative care that included psychological treatment, with or without anti-depressant medication, appeared to improve depression more than those without psychological treatment.	Observational nature of meta-regression, incomplete data reporting, and the use of study aggregates

					Systematic review with meta-regression	
Stewart (2013). Effect of collaborative care for depression on risk of cardiovascular events: data from the IMPACT randomized controlled trial. Psychosomatic Medicine	To test depression treatment delivered before clinical CVD onset reduces risk of CVD events.	Randomized control trial, Level II	235 primary care patients 60 years or older with major depression or dysthymia in 12-month collaborative care program.	CI 95%	Collaborative depression care delivered before CVD onset halved the excess risk of hard CVD events among older, depressed patients.	- The IMPACT trial was not designed to test hypothesis of this project - Randomization was not stratified by CVD status
Kronish (2012). The effect of enhanced depression care on adherence to risk-reducing behaviors after acute coronary syndromes: Findings from the COPES trial. The American Heart Journal	Evaluates the impact of the depression intervention on health behavior and blood pressure control.	RCT, Level II	80 patients	CI 95%	Enhanced depression care after an ACS did not improve health behavior or blood pressure control compared to usual care.	- Post hoc analyses - adherence behaviors were measured using single-item, dichotomous, self-report questions and thus were imprecise - self report
Delaney (2011). The Influence of a Spirituality-Based Intervention on Quality of Life,	To determine the feasibility and preliminary efficacy of an individualized	Qualitative, pre-experimental pilot study. Level IV	Three community-based organizations, (N = 27)	N/A	Patients who participated in the 1-month intervention demonstrated a significant modest	ANOVA to compare the groups on demographic variables

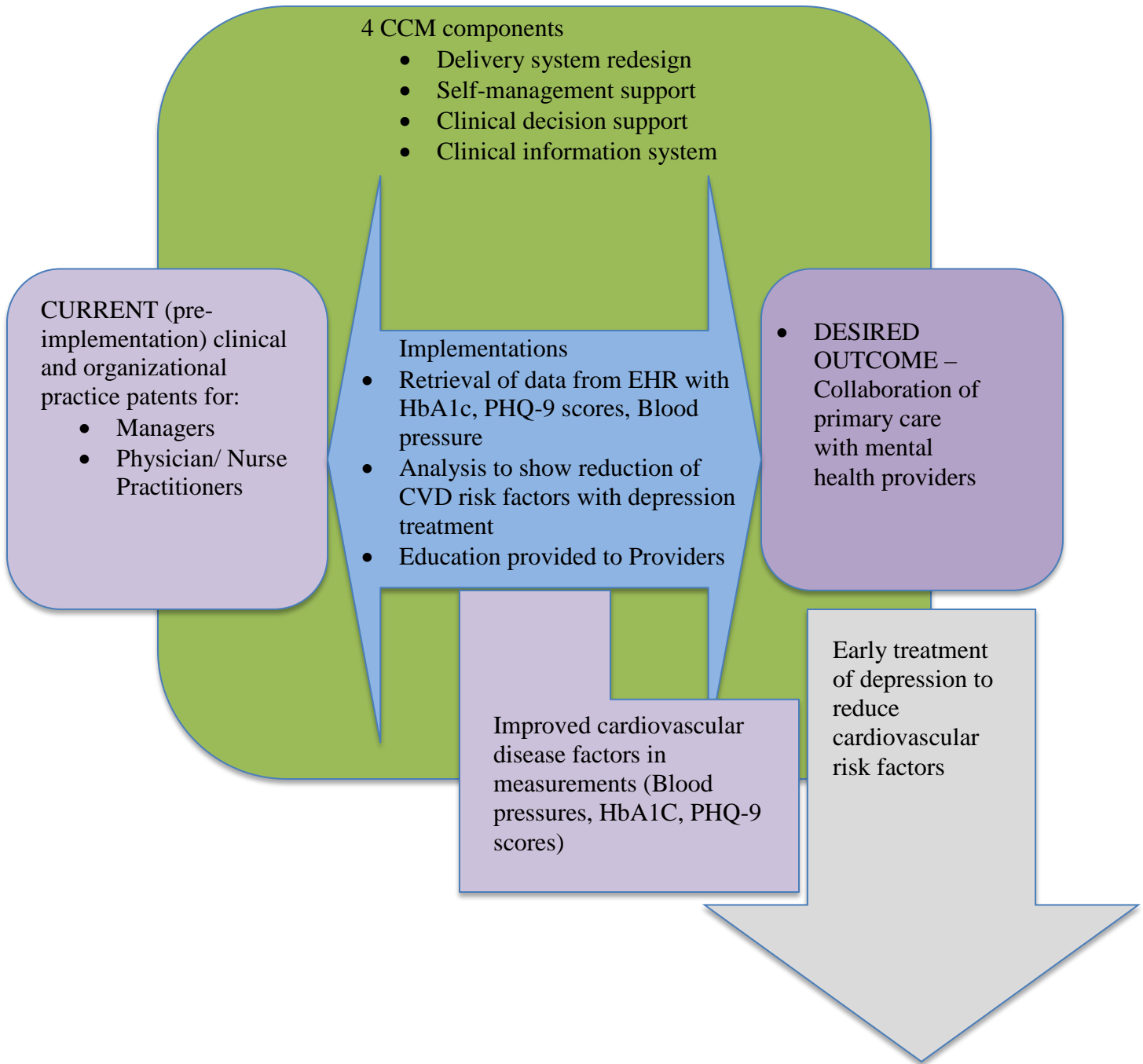
<p>Depression, and Anxiety in Community-Dwelling Adults With Cardiovascular Disease A Pilot Study. Journal of Holistic Nursing</p>	<p>spirituality-based intervention on health-related outcomes, depression, and anxiety) in community-dwelling patients with cardiovascular disease (CVD)</p>				<p>increase in overall quality of life.</p>	
<p>Bogner (2010). Integrating Type 2 Diabetes Mellitus and Depression Treatment Among African Americans A Randomized Controlled Pilot Trial. The diabetic educator</p>	<p>To examine whether integrating depression treatment into care for type 2 diabetes mellitus among older African Americans improved medication adherence, glycemic control, and depression outcomes.</p>	<p>Quantitative, Pilot study RCT, Level II</p>	<p>58 participants aged 50 to 80 years</p>	<p>Not reported</p>	<p>Integrating type 2 diabetes mellitus treatment and depression was successful in improving outcomes among older African Americans</p>	
<p>Echeverry (2009). Effect of Pharmacological Treatment of Depression on A1C and Quality of Life</p>	<p>To determine whether pharmacological treatment of depression in low-income</p>	<p>Quantitative, RCT, level II</p>	<p>150 subjects</p>	<p>Not reported</p>	<p>Pharmacological treatment of depression significantly improved A1C and systolic blood</p>	

<p>in Low-Income Hispanics and African Americans With Diabetes A randomized, double-blind, placebo-controlled trial. Diabetes care</p>	<p>minorities with diabetes improves A1C and quality of life.</p>				<p>pressure levels compared with placebo.</p>	
<p>Georgiades (2007). Changes in Depressive Symptoms and Glycemic Control in Diabetes Mellitus. Psychosomatic Medicine.</p>	<p>To investigate if changes in depressive symptoms would be associated with changes in glycemic control over a 12-month period in patients with Type 1 and Type 2 diabetes</p>	<p>RCT, Level II I don't know how could have a depressive intervention. Level IV?</p>	<p>90 patients</p>	<p>Not reported</p>	<p>Changes in depressive symptoms were not associated with changes in HbA1c or fasting glucose levels over a 1-year period in either patients with Type 1 or Type 2 diabetes.</p>	

¹ Design: Indicate if qualitative with specific design, quantitative with specific design, SR qualitative with meta-synthesis, SR quantitative. ² 1 1. 1 State the specific research design or state EBPG/guideline. 2 State the Hierarchy of Evidence per the Melnyk 7 level.

Appendix C: Theory to Application Diagram

Adapted for DNP Project -The Collaborate Care Model (CCM)



Appendix D: IRB Approval Letter



PROTOCOL
Unified IRB Form
UMKC

Protocol # 16-241
Date Printed: 08/06/2016

Protocol Title: Psychotherapeutic Interventions for Depression: Reducing Cardiovascular Disease Risk in Adults
Protocol Type: Unified IRB Form
Date Submitted: 07/25/2016
Important Note: This Print View may not reflect all comments and contingencies for approval. Please check the comments section of the online protocol. Questions that appear to not have been answered may not have been required for this submission. Please see the system application for more details.

*** Personnel Information ***

Starred items indicate required fields whenever that section is completed.

Principal Investigator

UMKC defines "Investigator" as an individual who conducts a research study. If the study is conducted by a team of individuals, the Investigator is the responsible leader of the team. Students, fellows and residents may not act as a Principal Investigator.

Name of Principal Investigator	Degree (MD/PhD/BSN/etc.)	Title
Lyla Lindholm	DNP, R	Clinical Assistant Professor
Email	Phone	Fax
lindholm1@umkc.edu		
Research Department	UMKC Status Check ALL that apply	Mailing Address
School of Nursing	<input checked="" type="checkbox"/> Faculty <input type="checkbox"/> Staff <input type="checkbox"/> Other	UMKC Health Sciences Building

ALL research personnel are required to complete Human Subject Research training from CITI within the last 2 years prior to engaging in any research-related activities. Go to CITI Program to complete.

The Research Compliance Office will verify the last date of completion below.

CITI Training Date	Type of CITI training completed.
01/13/2016	Group 1 Biomedical

Other Investigator(s)

Name of Other Investigator	Degree (MD/PhD/BSN/etc.)	Title	Research Department	Type of Investigator
Sadia Ali	BSN-DNP	Graduate Student	School of Nursing	Student Investigator

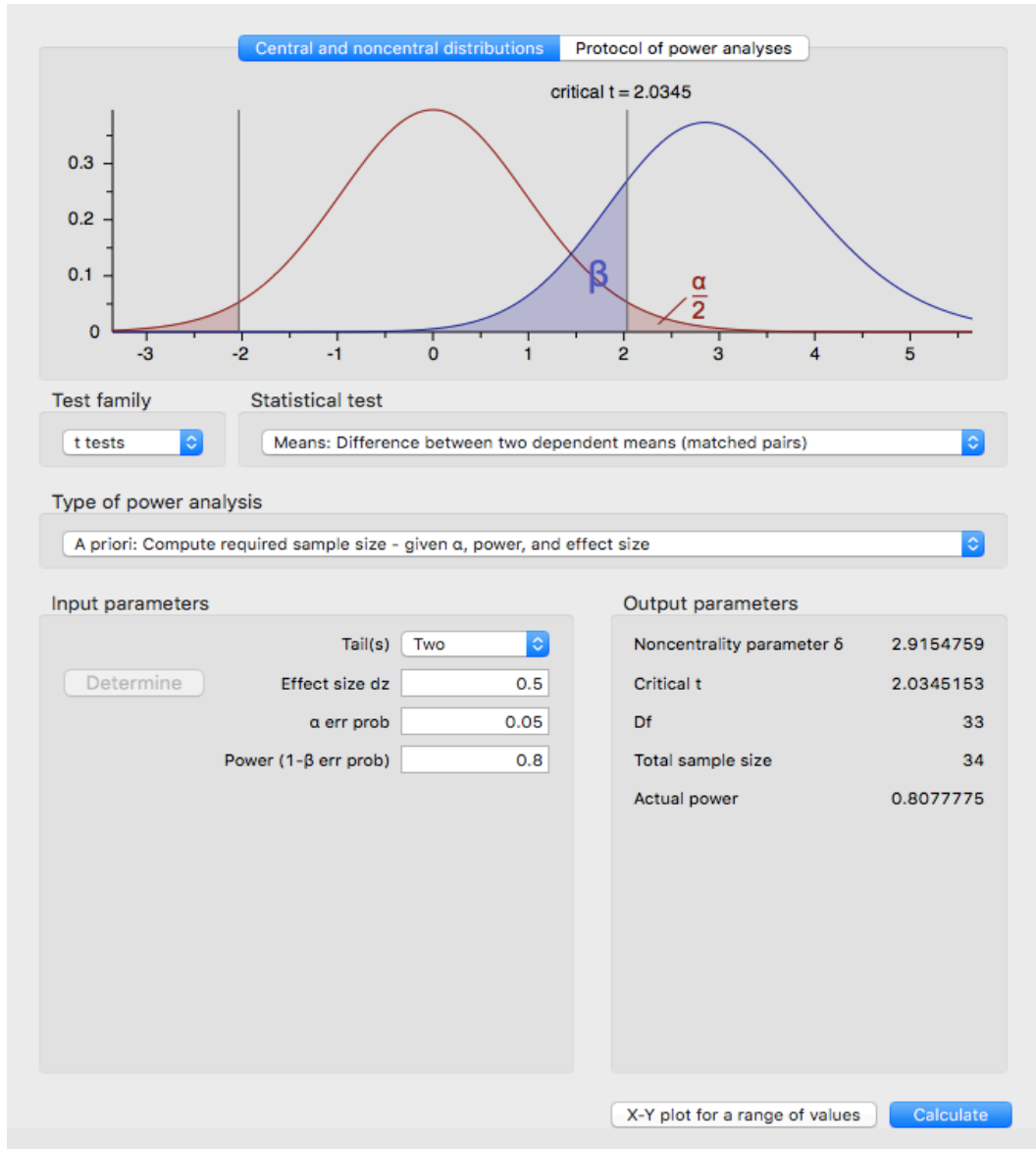
Other Personnel

Appendix E: Cost Table for project

Direct Cost s	Amount
Gasoline cost to and from to Internal	\$200.00
Medicine Clinic	
\$2.00/gallon for 35 miles each way/trip	
10 trips	
Material for presentation to Providers	\$55.00
Total Direct Costs	\$280.00

Indirect Costs	Amount
APNO and MNRS Presentation cost	\$575.00
Poster cost (Office Max)	\$25.00
Total Indirect costs	\$600.00

Appendix F: Priori Power Analysis for Sample Size

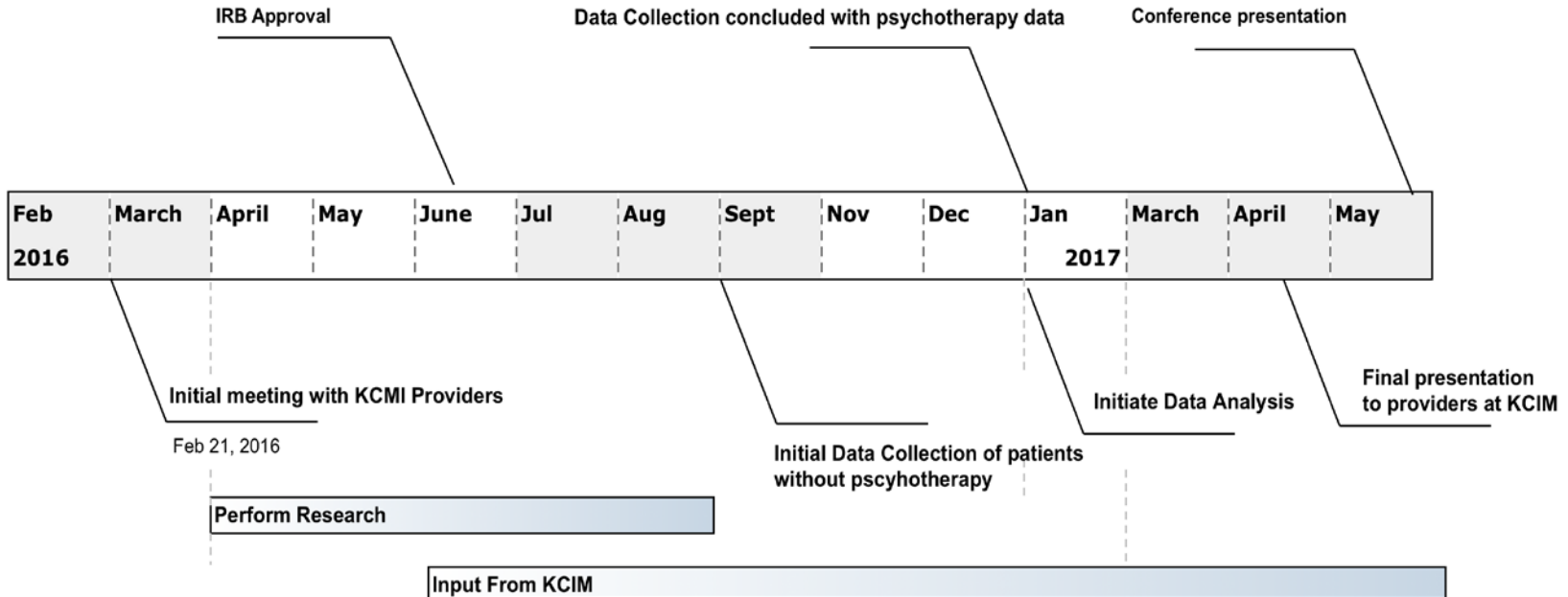


Appendix G: Project Timeline

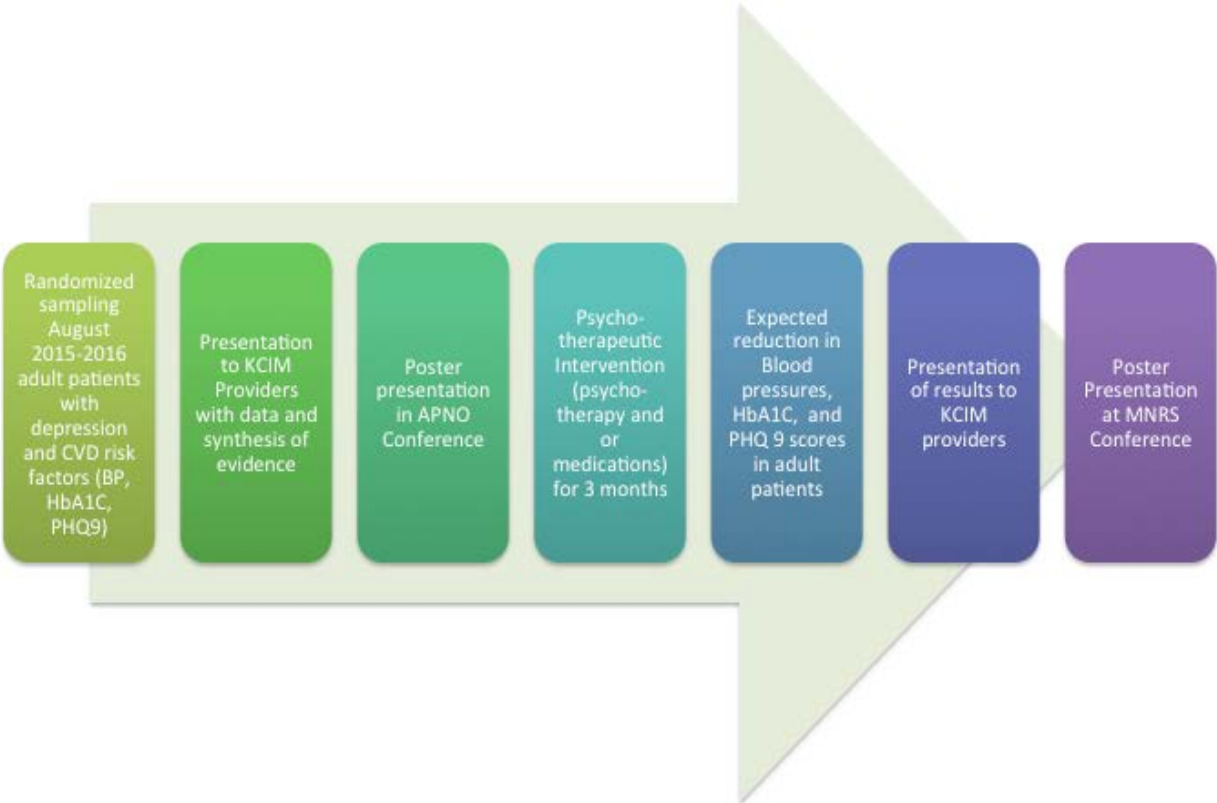
Sadia Ali

Depression Intervention and Reduction in Cardiovascular Risks

EBP Project Schedule



Appendix H: Intervention Flow Diagram



Appendix I: Intervention and Educational Materials

The intervention plan for the program: Early treatment of depression to reduce cardiovascular risk factors (blood pressures, PHQ-9 scores, HbA1C)

Phase One: August 2016

Presentation to providers with evidence to treat depression early resulting in reduced cardiovascular risk factors.

Phase Two: September 2016

Initiation of intervention with patients treated with psychotherapy or pharmaceutical therapy or both.

1. Patients will be identified by their cardiovascular risk factors with diagnosis of hypertension and diabetes:
 - a. HbA1C > 5.5
 - b. Blood Pressures (Systolic 120-130 mmHg, Diastolic 80-90 mmHg)
 - c. PHQ-9 Depression severity >5

Phase Three: December 2016 – January 2017

Completion of intervention and collection of data

Phase four: January 2017- March 2017

Analysis of Data

Phase five: May 2017

Data analysis presented to providers at Internal Medicine Clinic to show results of treatment of depression on cardiovascular risk factors.

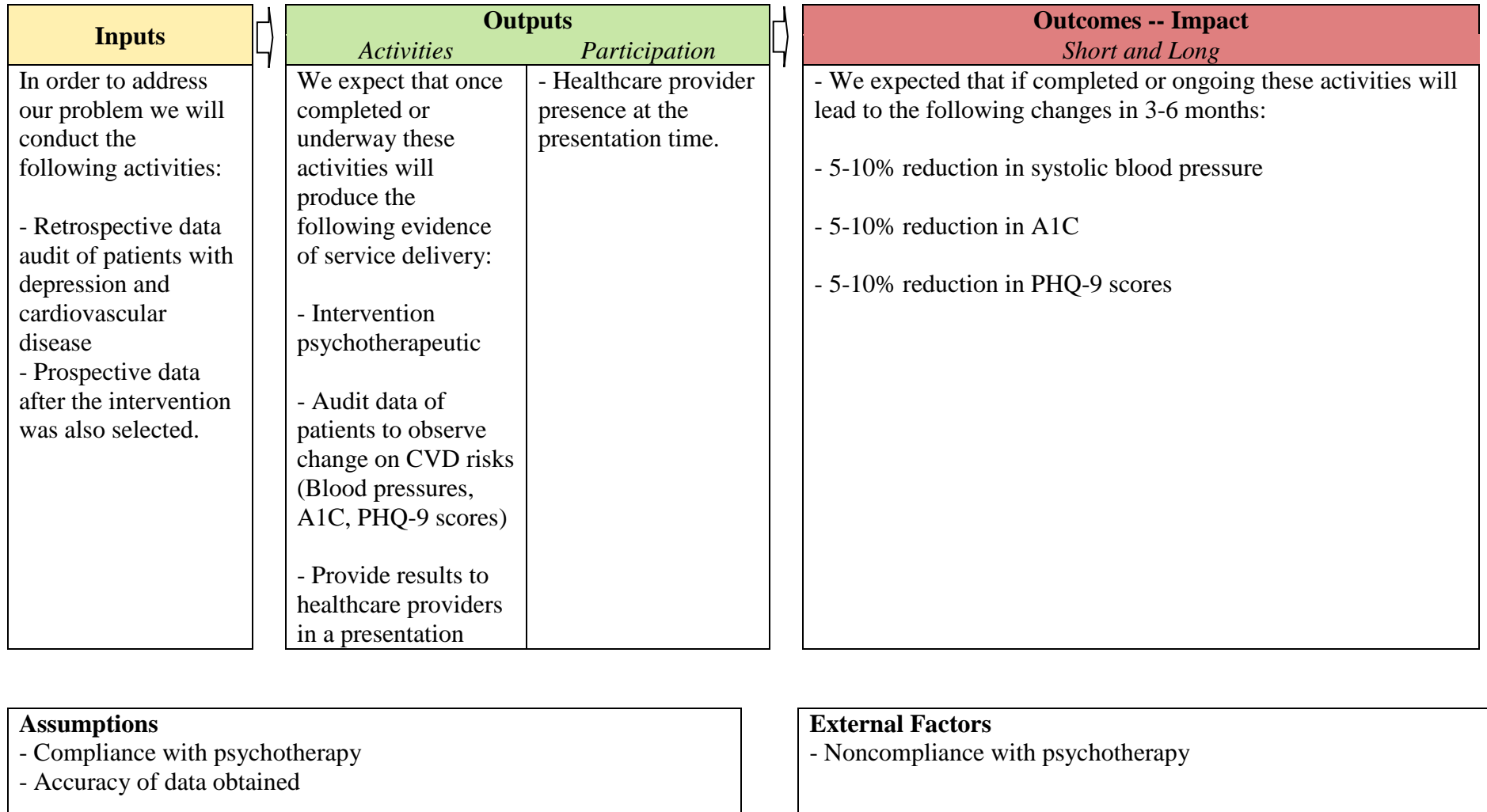
Educational Materials

I used power point presentation as well as poster presentation to provide education to providers with importance of early treatment of depression prior to intervention. Providers are the main audience for the presentations. The verbal and visual power point presentation did not exceed 30 minutes including question/answer session. The presentation was developed in accordance to providers' education level with serif font (Calibri) to be easily read as well as prior approval from quality management team. Total of 7 providers attended the presentation session in September 2016.

Appendix J: Kotter and Cohen's Change Model



Appendix K: Depression Intervention and Reduced Cardiovascular Disease Risks Logic Model

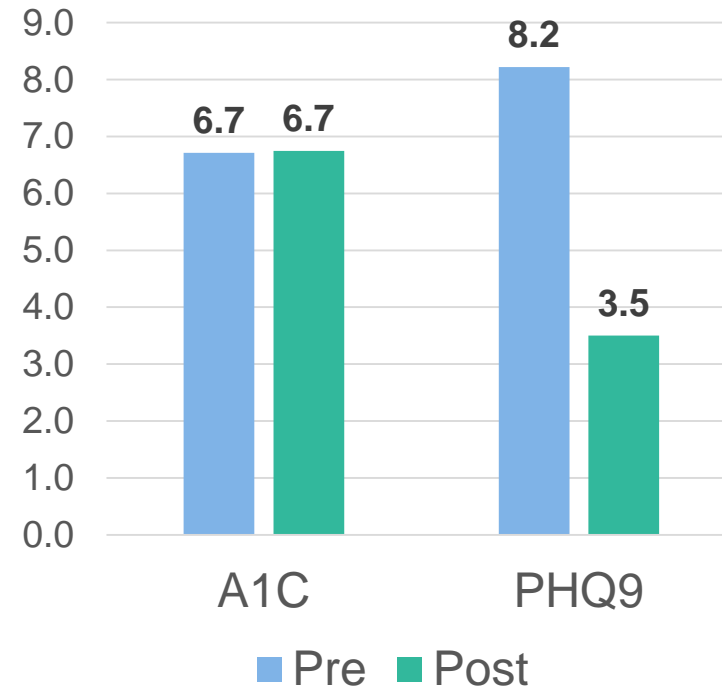
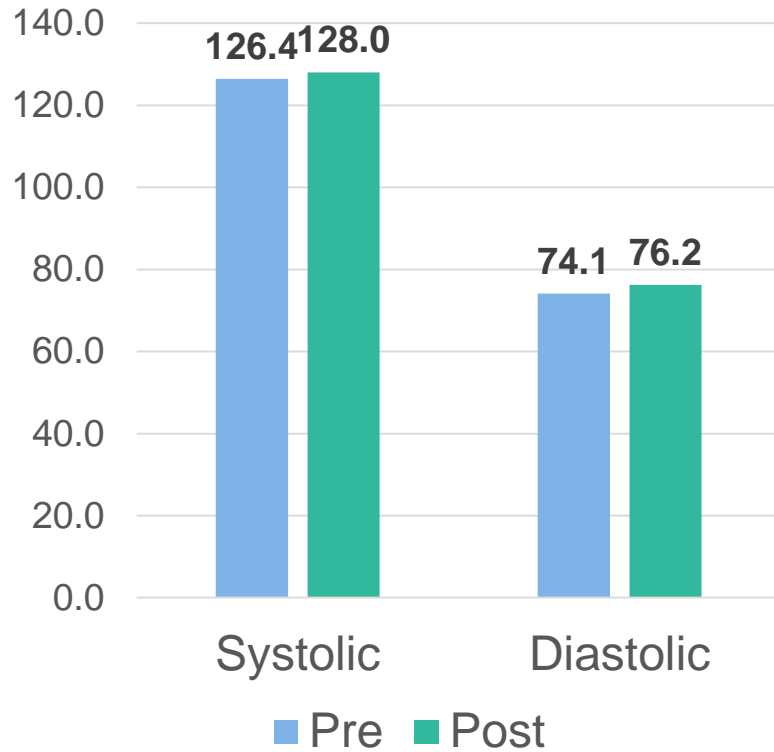


Appendix L: Data Collection Table

Total_Apts	Age	Sex	Qualifies	Dx	Date initial Data collected	Pre Systolic	Post_Systolic	Pre_Diastolic	Post_Diastolic	Pre_A1C	Post_A1C	Pre_PHQ9	Post_PHQ9	Meds
12	51	F	Yes	Dep, HTN	3/2016	138	132	86	82	5.9	5.5	14		Escitalopram 5 mg, Adderal 30 mg, L
8	69	M	Yes	Dep, DMII	3/2016	122	128	70	76	5.6	5.1	12	0	Cimetidine (ADR depression,
6	68	M	Yes	Dep, HTN, DMII	08/17/16	104	132	60	58	6.5	6.3	6		Zolpidem, Lantus, Metformin, Lisinopril
3	76	F	Yes	Dep, DMII, HTN	05/10/16	162	128	72	76	6.9	7.7	8		Glimipride, Lisinopril
7	74	M	Yes	Dep, DMII	07/13/15	138	150	80	80	7.6	7.1	12	12	Glipizide, Metformin, Actos,
5	69	F	Yes	Dep, DMII, HTN	8/2016	132		82		6.3		0		Bupropion, HCTZ, Lisinopril,
7	62	F	Yes	Dep, DMII, HTN	9/2016	132	130	80	80	7	6.8	3		Escitalopram, Bupropion, Trazidone,
0	76	M	Yes	Dep, DMII?, HTN	7/2016	122	126	64	74	4.9	5.3	0	0	Citalopram, Perindopril,
8	67	F	Yes	Dep, HTN	8/2016	118	122	82	68	5.2				Venlafaxine, Losartan, Zolpidem(Inso
2	67	F	Yes	Dep, DMII, HTN	9/2016	108	124	70	70	5.9	5.8	17		Amlodipine, Losartan, Zolpidem, So
0	77	F	Yes	Dep, DMII	8/2016	132	138	84	62	6.3	5.4	0		Mirtazipine, Prednisone
0	58	M	Yes	Dep, DMII, HTN	7/2016	84		62		8.1				Glipizide, Metformin, humalog, meto
8	58	F	Yes	Dep, DMII, HTN	8/2016	140	162	90	84	9.2	11			Novolog, Duloxetine, Levemir, Metfo
0	50	F	Yes	Dep, HTN	3/2016	126	114	80	72	5.7	5.7	0		Spirolactone
0	66	M	Yes	Dep, HTN	8/2016	106		74		6.1		10		Sertraline, Carvedilol, lisinopril, Metf
0	61	M	Yes	Dep, DMII	8/2016	118	124	62	78	6.2	6.1	0	0	Januvia, Lisinopril, Furosemide
0	65	F	Yes	Dep, DMII, HTN	8/2016	130	144	70	78	8.2	8.3	7		Amylodipine, Bystolic, Furoside, L
3	84	M	Yes	Dep, HTN	10/2016	124	124	60	84	N/A		0	0	Benzopril-HCTZ, Doxazosin, Paroxit
5	74	F	Yes	Dep, DMII	10/2016	122	120	54	70	10.1		?		Metformin, Flouxetine, Furoside, h
4	74	F	Yes	Dep, HTN	8/2016	132	144	70	86	N/A		3		Indapamide, Lisinopril,
14	59	M	Yes	Dep, HTN	4/2016	120	124	84	80	5.8		12	1	Bupropion, Trazadone, Vbyrd, Benza
0	79	M	yes	Dep, DMII, HTN	5/2015	104	128	62	70	8.6	8.7			Synthroid, Wellbutrin, humalog, vit D
10	54	F	Yes	Dep, HTN, Pro-Diab	9/2016	132	130	84	88	5.9	6.3	24		Escitalopram, Alprazolam,
7	69	F	Yes	Dep, DMII	7/2016	152	120	76	70	7.3	7.7	24	15	Novolog, Lantus, Amlodipine,
2	37	M	Yes	Dep, HTN	10/2016	110	110	72	74			11		Alprazolam
1	85	F	Yes	Dep, DMII, HTN	11/2016	149	110	73	73	7.2		15		Glyburide-metformin, Trazedone
1	47	F	Yes	Dep, DMII, HTN	05/31/16	132	122	82	90					Amylodipine, Lisinopril, Vit. D, Al
3	87	F	Yes	Dep, DMII, HTN	08/2016	148	123	78	71	5.5	5.9			Bupropion, Metformin, losartan
4	59	M	Yes	Dep, HTN	9/2016	124	110	80	70	5.8		1	0	Bupropion, Trazadone, Vbyrd, Benza
0	64	M	Yes	Dep, HTN	8/2016	132	138	80	94			10		

Appendix M:

Paired Sample Statistics, Intervention Group



Appendix N: Statistical Analysis Variable View

Name	Type	Width	Decimals	Label	Values	Missing	Columns	Align	Measure	Role
Participant_number	Numeric	8	2	Participant number	None	None	8	Right	Nominal	Input
Age	Numeric	8	2	Age of participant	None	None	8	Right	Nominal	Input
Gender	Numeric	8	2	Gender of partic...	{1.00, Fema...	None	8	Right	Nominal	Input
BP_Preintervention	Numeric	8	2	Blood Pressure ...	None	None	8	Right	Scale	Input
BP_Post_intervention...	Numeric	8	2	Blood Pressure ...	None	None	8	Right	Scale	Input
BP_Pre_intervention	Numeric	8	2	Blood Pressure ...	None	None	8	Right	Scale	Input
BP_Post_intervention...	Numeric	8	2	Blood Pressure ...	None	None	8	Right	Scale	Input
HbA1C_Pre_Intervention	Numeric	8	2	Hemoglobin A1C	None	None	8	Right	Scale	Input
HbA1C_Post_interven...	Numeric	8	2	Hemoglobin A1...	None	None	8	Right	Scale	Input
PHQ9_pre_Intervention	Numeric	8	2	PHQ 9 score Pre	None	None	8	Right	Scale	Input
PHQ9_Post_Intervention	Numeric	8	2	PHQ 9 score Post	None	None	8	Right	Scale	Input

Appendix O: Regression Analysis

Case Processing Summary

		Post_A1C	Post_PHQ9
Series or Sequence Length		30	30
Number of Missing Values in the Plot	User-Missing	0	0
	System-Missing	13	23

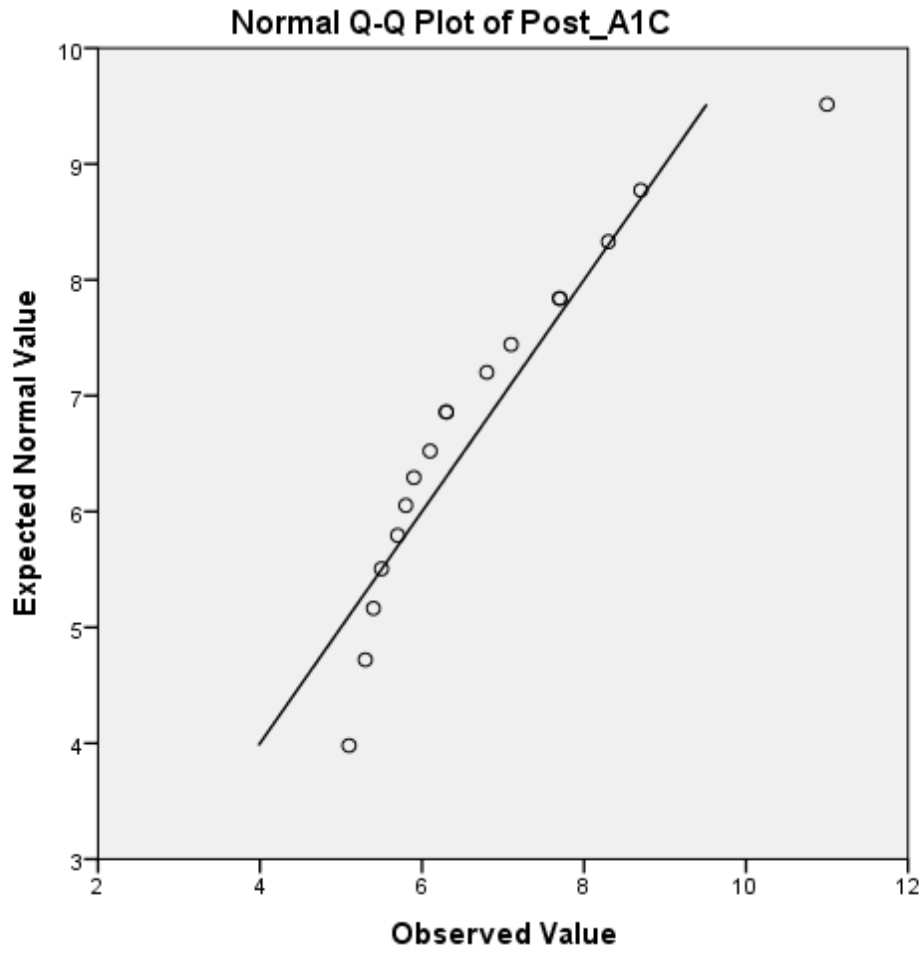
The cases are unweighted.

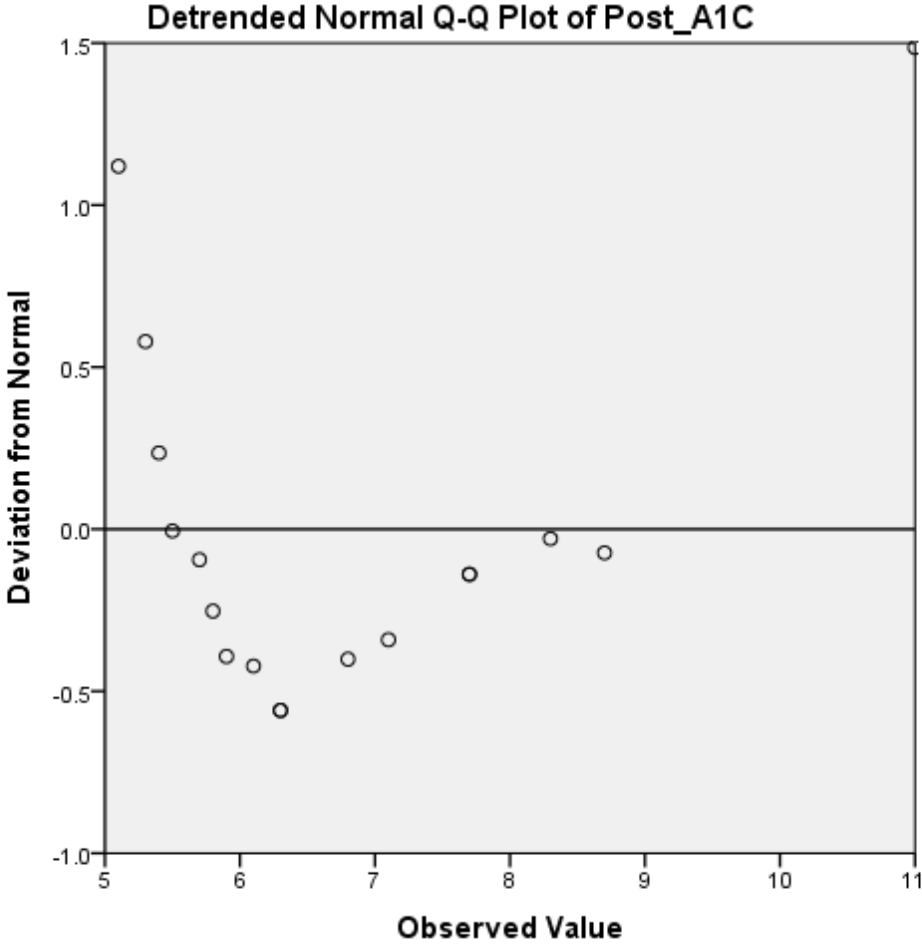
Estimated Distribution Parameters

		Post_A1C	Post_PHQ9
Normal Distribution	Location	6.75	4.00
	Scale	1.540	6.557

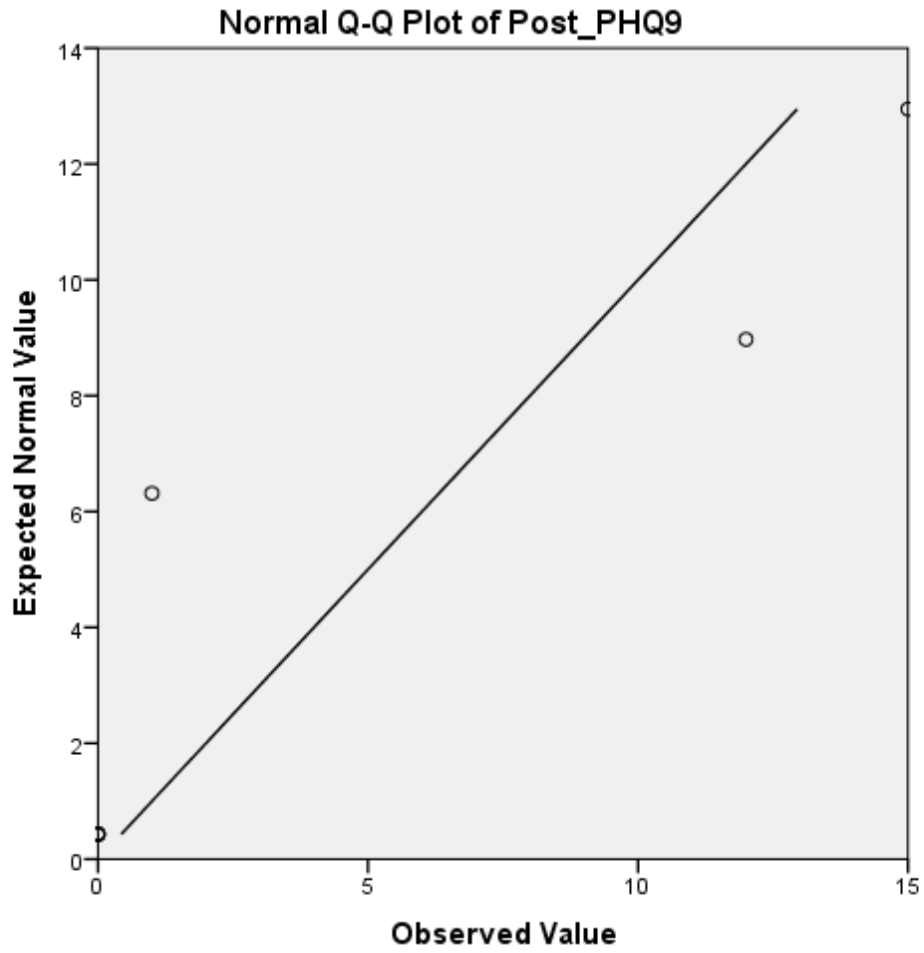
The cases are unweighted.

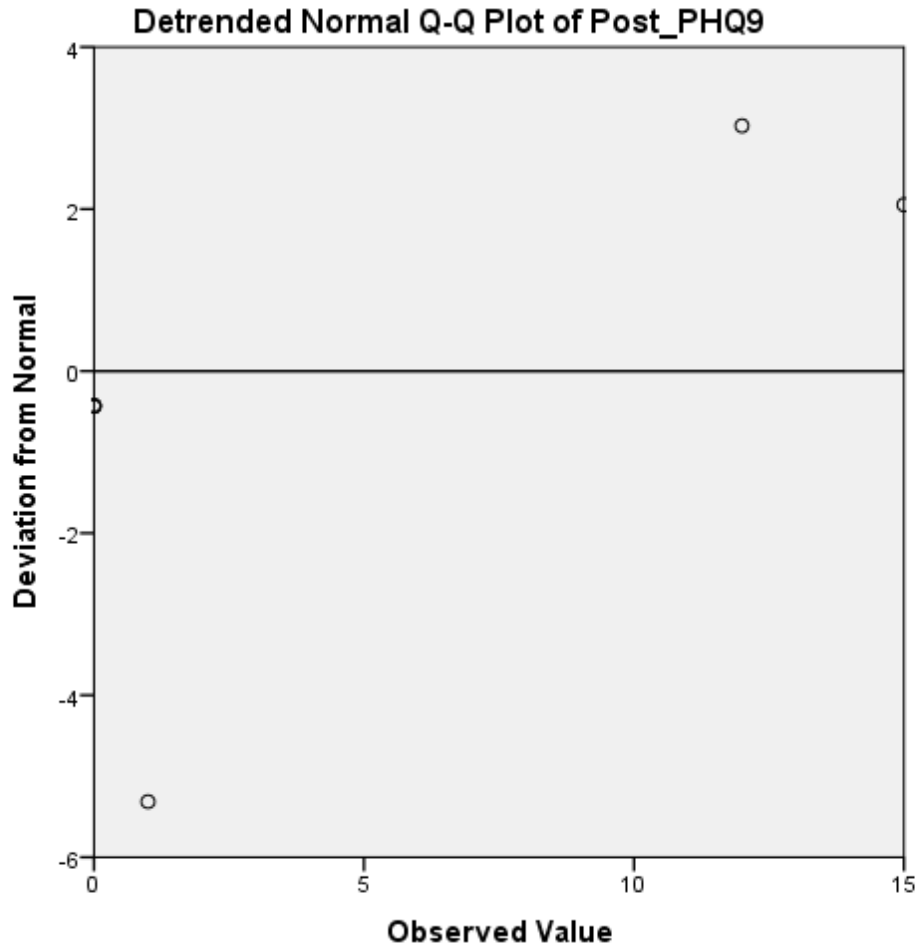
Post_A1C





Post_PHQ9





Regression

Variables Entered/Removed^a

Model	Variables Entered	Variables Removed	Method
1	Post_A1C ^b		. Enter
2	Post_Systolic ^b		. Enter
3	Post_Diastolic ^b		. Enter

a. Dependent Variable: Post_PHQ9

b. All requested variables entered.

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.948 ^a	.898	.848	3.079
2	.956 ^b	.914	.742	4.009
3	1.000 ^c	1.000	.	.

a. Predictors: (Constant), Post_A1C

b. Predictors: (Constant), Post_A1C, Post_Systolic

c. Predictors: (Constant), Post_A1C, Post_Systolic, Post_Diastolic

ANOVA^a

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	167.794	1	167.794	17.704	.052 ^b
	Residual	18.956	2	9.478		
	Total	186.750	3			
2	Regression	170.679	2	85.339	5.310	.293 ^c
	Residual	16.071	1	16.071		
	Total	186.750	3			
3	Regression	186.750	3	62.250	.	. ^d
	Residual	.000	0	.		
	Total	186.750	3			

a. Dependent Variable: Post_PHQ9

b. Predictors: (Constant), Post_A1C

c. Predictors: (Constant), Post_A1C, Post_Systolic

d. Predictors: (Constant), Post_A1C, Post_Systolic, Post_Diastolic

Coefficients^a

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	-39.332	11.060		-3.556	.071
	Post_A1C	7.035	1.672	.948	4.208	.052
2	(Constant)	-47.842	24.718		-1.936	.304
	Post_A1C	6.880	2.208	.927	3.116	.198

	Post_Systolic	.073	.173	.126	.424	.745
3	(Constant)	-.359	.000		.	.
	Post_A1C	5.493	.000	.740	.	.
	Post_Systolic	.322	.000	.554	.	.
	Post_Diastolic	-.938	.000	-.527	.	.

a. Dependent Variable: Post_PHQ9

Excluded Variables^a

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics Tolerance
1	Post_Systolic	.126 ^b	.424	.745	.390	.972
	Post_Diastolic	-.067 ^b	-.209	.869	-.205	.952
2	Post_Diastolic	-.527 ^c	.	.	-1.000	.310

a. Dependent Variable: Post_PHQ9

b. Predictors in the Model: (Constant), Post_A1C

c. Predictors in the Model: (Constant), Post_A1C, Post_Systolic

Regression

Variables Entered/Removed^a

Model	Variables Entered	Variables Removed	Method
1	Post_A1C ^b	.	Enter
2	Post_Systolic ^b	.	Enter
3	Post_Diastolic ^b	.	Enter

a. Dependent Variable: Post_PHQ9

b. All requested variables entered.

Model Summary^d

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Durbin-Watson
1	.948 ^a	.898	.848	3.079	

2	.956 ^b	.914	.742	4.009	
3	1.000 ^c	1.000	.	.	.400

- a. Predictors: (Constant), Post_A1C
- b. Predictors: (Constant), Post_A1C, Post_Systolic
- c. Predictors: (Constant), Post_A1C, Post_Systolic, Post_Diastolic
- d. Dependent Variable: Post_PHQ9

ANOVA^a

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	167.794	1	167.794	17.704	.052 ^b
	Residual	18.956	2	9.478		
	Total	186.750	3			
2	Regression	170.679	2	85.339	5.310	.293 ^c
	Residual	16.071	1	16.071		
	Total	186.750	3			
3	Regression	186.750	3	62.250	.	. ^d
	Residual	.000	0	.		
	Total	186.750	3			

- a. Dependent Variable: Post_PHQ9
- b. Predictors: (Constant), Post_A1C
- c. Predictors: (Constant), Post_A1C, Post_Systolic
- d. Predictors: (Constant), Post_A1C, Post_Systolic, Post_Diastolic

Coefficients^a

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	-39.332	11.060		-3.556	.071
	Post_A1C	7.035	1.672	.948	4.208	.052
2	(Constant)	-47.842	24.718		-1.936	.304
	Post_A1C	6.880	2.208	.927	3.116	.198
	Post_Systolic	.073	.173	.126	.424	.745
3	(Constant)	-.359	.000		.	.
	Post_A1C	5.493	.000	.740	.	.
	Post_Systolic	.322	.000	.554	.	.

Post_Diastolic	-938	.000	-527	.	.
----------------	------	------	------	---	---

Coefficients^a

Model		95.0% Confidence Interval for B		Collinearity Statistics	
		Lower Bound	Upper Bound	Tolerance	VIF
1	(Constant)	-86.918	8.254		
	Post_A1C	-.159	14.230	1.000	1.000
2	(Constant)	-361.909	266.225		
	Post_A1C	-21.177	34.937	.972	1.028
	Post_Systolic	-2.125	2.272	.972	1.028
3	(Constant)	-.359	-.359		
	Post_A1C	5.493	5.493	.697	1.434
	Post_Systolic	.322	.322	.317	3.159
	Post_Diastolic	-.938	-.938	.310	3.227

a. Dependent Variable: Post_PHQ9

Excluded Variables^a

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics	
						Tolerance	VIF
1	Post_Systolic	.126 ^b	.424	.745	.390	.972	1.028
	Post_Diastolic	-.067 ^b	-.209	.869	-.205	.952	1.050
2	Post_Diastolic	-.527 ^c	.	.	-1.000	.310	3.227

Excluded Variables^a

Model		Collinearity Statistics	
		Minimum Tolerance	
1	Post_Systolic	.972	
	Post_Diastolic	.952	
2	Post_Diastolic	.310	

a. Dependent Variable: Post_PHQ9

b. Predictors in the Model: (Constant), Post_A1C

c. Predictors in the Model: (Constant), Post_A1C, Post_Systolic

Collinearity Diagnostics^a

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions		
				(Constant)	Post_A1C	Post_Systolic
1	1	1.990	1.000	.00	.00	
	2	.010	14.300	1.00	1.00	
2	1	2.983	1.000	.00	.00	.00
	2	.013	15.119	.05	.95	.15
	3	.004	27.589	.95	.05	.85
3	1	3.979	1.000	.00	.00	.00
	2	.016	15.732	.00	.62	.01
	3	.004	31.393	.15	.01	.35
	4	.000	91.792	.85	.37	.64

Collinearity Diagnostics^a

Model	Dimension	Variance Proportions	
			Post_Diastolic
1	1		
	2		
2	1		
	2		
	3		
3	1		.00
	2		.01
	3		.00
	4		.99

a. Dependent Variable: Post_PHQ9

Residuals Statistics^a

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	.00	15.00	6.75	7.890	4
Residual	.000	.000	.000	.000	4
Std. Predicted Value	-.856	1.046	.000	1.000	4
Std. Residual	0

a. Dependent Variable: Post_PHQ9



July 20, 2016

UMKC Institutional Review Board
University of Missouri-Kansas City
Kansas City, MO 64108

UMKC IRB,

This letter serves to provide documentation regarding Sadia Ali's Doctor of Nursing Practice (DNP) Project proposal. Ms. Ali obtained approval for her project proposal, *Psychotherapeutic Interventions for Depression: Reducing Cardiovascular Disease Risk in Adults*, from the School of Nursing DNP faculty committee on July 20, 2016.

If I can provide any further information, please feel free to contact me.

Sincerely,

A handwritten signature in cursive script that reads "Susan J. Kimble".

Susan J. Kimble, DNP, RN, ANP-BC, FAANP
Clinical Associate Professor
DNP Programs Director
UMKC School of Nursing and Health Studies
816-235-5962

kimbles@umkc.edu