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The Impact of Cardiovascular Disease and Intervention through Modifiable Risk Factors in
the Primary Care Behavioral Health (PCBH) Model

by

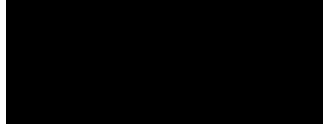
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Dissertation submitted to the Faculty of the
Medical and Clinical Psychology Program
Uniformed Services University of the Health Sciences
In partial fulfillment of the requirements for the degree of
Doctor of Philosophy, 2021

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August 31, 2021

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SUBJECT: Appointment to the Final Doctoral Examination Committee for Kevin Wilfong

In accordance with the School of Medicine Graduate Program Guidelines, you are formally appointed to the Final Examination Committee of Kevin Wilfong, doctoral student in the Department of Medical and Clinical Psychology.

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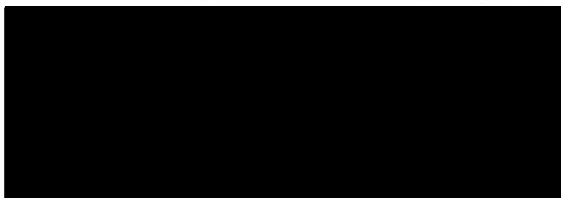
In addition, the Committee will conduct an Oral Examination of the student's scientific knowledge, as well as the knowledge and understanding of the research project and Dissertation. This examination constitutes the Private Defense.

The Private Defense for Kevin Wilfong will take place on Tuesday, August 31, 2021 at 8:00 AM via virtual meeting.

Dr. David Riggs is appointed Chair of the Examination Committee. Dr. Riggs will oversee the examination. Passage of the Private Defense will be by majority vote. If corrections to the Dissertation are needed, the Chair may oversee this process and sign-off after the corrections are made.

The Committee Chair should return the signed and dated Final Examination/Private Defense Form and the Dissertation Approval Form to the Graduate Education Office.

GEO thanks you in advance for your participation in the SOM Graduate Program process.



Saibal Dey, Ph.D.
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ABSTRACT

Title of Dissertation: The Impact of Cardiovascular Disease and Intervention through Modifiable Risk Factors in the Primary Care Behavioral Health (PCBH) Model

Kevin M. Wilfong, Doctoral Candidate, 2021

Dissertation directed by: Jeffrey L. Goodie, Ph.D., ABPP, Professor and Director of Clinical Training, Department of Medical and Clinical Psychology

Cardiovascular disease (CVD) remains the leading cause of mortality in the United States after nearly a century. Although developments in interventions for CVD have improved the survival rate of major adverse cardiac events, the pervasiveness of poor cardiovascular health remains a growing public health burden with deleterious impacts at societal, organizational, and individual levels. Modifiable risk factors for the development of CVD include tobacco use, obesity, and the psychophysiology associated with the onset of depression. The current de facto setting for treating the modifiable risk factors of CVD is primary care; however, the current model is limited by insufficient time and resources, patient motivation or beliefs toward intervention, provider attitudes toward treatment effectiveness, and a reliance on external specialists for evidence-based behavioral interventions.

The Primary Care Behavioral Health (PCBH) model of integrating behavioral health consultants into a primary care setting is one method that may begin to address these limitations through enhancing the assessments and interventions provided for both health behavior and behavioral health problems in primary care. The PCBH model provides

evidence-based brief interventions with enhanced access to care, reduced cost, enriched patient experience, and improved care-team wellbeing. One aspect of the PCBH model that is not well studied, but is identified as a role of behavioral health consultants (BHCs), is chronic disease management through interventions for these modifiable risk factors. Therefore, this study aimed to better understand the effectiveness of the PCBH model by evaluating the penetration within primary care settings of health behavior and depression interventions delivered by BHCs, change in health behaviors and depression across treatment, and the differential impact of CVD and its risk factors on global functioning in the PCBH model.

We anticipated those diagnosed with CVD, obesity, and tobacco use would be referred and targeted for brief intervention by BHCs (i.e., penetration) at a rate of at least 5%, while those diagnosed with depression would be targeted at a higher rate of 25%. It was also hypothesized that the PCBH model would be effective for improving health behaviors as measured by self-reported tobacco use, measured body mass index (BMI), and blood pressure. Finally, it was expected that those diagnosed with CVD would report different levels of global functioning moderated by modifiable risk factors of CVD. The long-term goal of the present study was to examine the effectiveness of the PCBH model to improve interventions targeting modifiable risk factors of CVD so that cardiovascular morbidity and mortality are reduced.

Given the widespread implementation of the PCBH model within the Defense Health Agency and relative impact of the aforementioned conditions on the military population, this study examined the integrated primary care treatment of military health system beneficiaries. To do so, the current study analyzed data previously collected by the

Psychological Health Center of Excellence of the J-9 Directorate at the Defense Health Agency across a 3-year period.

In the monitored three-year period from January 2017 to December 2019, the study revealed penetration rates of 1.47% for CVD, 5.34% for tobacco use, 3.41% for obesity, and 20.00% for depression. This study also revealed significant findings related to the relationship between CVD severity and perceived global functioning. Specifically, this study found that levels of CVD severity did not significantly differ in perceived global functioning; however, interactions with modifiable risk factors suggested that perceived functioning in those with CVD was moderated by tobacco use and depression. Finally, the study compared health outcomes between primary care alone, primary care with PCBH, primary care with specialty mental health, and treatment in all three settings. The results suggested there was a significant difference in treatment group for tobacco use, BMI, and mean arterial blood pressure (MAP). These differences in health outcomes illuminated that primary care (PC) with PCBH demonstrated the best outcomes for tobacco use and MAP, whereas PC with specialty behavioral health demonstrated the best outcomes for BMI.

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CHAPTER 1: Introduction to CVD, Modifiable Risk Factors, and the PCBH Model

Cardiovascular disease (CVD) has been the leading cause of death in the United States for nearly a century, with ischemic heart disease and stroke responsible for 26.7% of all deaths in 2016 (27; 175). Efforts to address the growing public health burden of CVD, such as the American Heart Association's (AHA) 2020 Strategic Impact Goal, expand the focus of cardiovascular health care to include not only the treatment of disease, but also the promotion of cardiovascular health and disease prevention (99). In defining ideal cardiovascular health, the AHA identified critical behavioral markers for promoting health and reducing risk of cardiovascular disease. Ideal health behaviors include nonsmoking and weight management to maintain a body mass index (BMI) less than 25 kg/m² (99). Additionally, strong evidence for the deleterious cardiovascular implications of behavioral health concerns such as depression suggest it, too, is a crucial target for promoting cardiovascular health (122). In total, tobacco use, obesity, and depression make up three important risk factors for CVD with modifiable behaviors that can reduce the risk of adverse outcomes (99; 122). Despite knowledge that addressing health behaviors and behavioral health is vital for prevention of CVD, the current model of treatment includes several limitations to proper implementation (46; 74; 166; 168).

Primary care is one potential setting for providing evidence-based interventions to individuals who may benefit from the protective effects of tobacco cessation, weight management, or brief interventions for depression (52; 93; 167). However, barriers such as insufficient time, limited resources, patient motivation or beliefs toward intervention, and provider attitudes toward treatment effectiveness limit the availability of interventions in traditional primary care (31; 96; 109). The Primary Care Behavioral

Health (PCBH) model of integrating behavioral health consultants into a primary care setting is one method that may begin to address these barriers; the PCBH model is designed to enhance the assessments and interventions provided for both health behavior and behavioral health problems in primary care (132).

This study aims to better understand potential strategies for reducing the morbidity and mortality of CVD - the deadliest disease in the United States (27; 175). The following will review definitions and mechanisms of cardiovascular disease, the associations between cardiovascular disease and major modifiable risk factors (i.e., tobacco use, obesity, and depression) (99; 122), the impact of each on global functioning, the current model for intervention within the primary care setting, and the adaptation of primary care through integration of behavioral health consultants. Given the widespread implementation of the PCBH model within the Department of Defense (DoD), combined with the dearth of available data evaluating the effectiveness of the PCBH model in targeting modifiable risk factors for CVD, the aims of this study have important implications for military health care beneficiaries and other health care systems using the PCBH model (67; 152).

CARDIOVASCULAR DISEASE: DEFINITION AND MECHANISMS

Cardiovascular disease encompasses a wide array of noncommunicable diseases that negatively impact the heart and blood vessels. This section will focus on select types of CVD for their relative burden on the population and how tobacco use, obesity, and depression are related to CVD. Specifically, the following section will define the key features and etiological mechanisms of hypertensive heart disease, atherosclerotic cardiovascular disease, and major adverse cardiac events.

Hypertensive Heart Disease

Hypertensive heart disease is a constellation of cardiovascular symptoms characterized by systolic and diastolic dysfunction and the development of left ventricular hypertrophy as a result of chronic high blood pressure (41). The etiological pathway of primary hypertension is a poorly defined process consisting of genetic, environmental, behavioral, and other medical factors. Medical classification of hypertension stratifies blood pressure into four categories. Normotensive blood pressure is defined by the AHA as a systolic blood pressure (SPB) less than 120 mmHg and diastolic blood pressure (DPB) less than 80 mmHg (156). An SPB of 120 to 129 mmHg is considered elevated, but not hypertensive (156). Stage 1 hypertension includes individuals with an SBP of 130 to 139 mmHg and a DBP of 80 to 89 mmHg; stage 1 hypertension is differentiated from higher stages of hypertension for lower risk of developing more serious cardiovascular disease and a focus on nonpharmacological blood pressure management (73; 156; 170). Stage 2 hypertension is defined by an SBP greater than or equal to 140 mmHg (or 150 mmHg for individuals over 60) and DBP greater than or equal to 90 mmHg (73; 156; 170). The chronicity of elevated blood pressure creates increased stress on the left ventricle — the chamber of the heart responsible for ejecting arterial blood to the body (56). This increased stress can have deleterious effects on the thickness and volume of the left ventricle. The ventricular wall thickens to account for the increase pressure, known as left ventricular hypertrophy; the increased thickness reduces ventricular elasticity, hinders cardiac efficiency, and creates a workload that can lead to congestive heart failure (56). Alternatively, the chronic elevated pressure may dilate the volume of the left ventricular chamber and weaken the cardiac walls, again leading to congestive heart failure (56).

Atherosclerotic Cardiovascular Disease

The development of atherosclerotic cardiovascular disease (ACD) is a multifaceted process involving the buildup of cholesterol, fatty acids, and other substances into intimal lesioning of the arteries (107); this buildup within the inner most lining of the arteries, referred to as atherosclerotic plaque, is thought to be associated with elevated permeability of the arterial walls as a result of abnormally high concentrations of low-density lipoprotein (LDL) cholesterol (107). The resulting atherosclerotic plaque leads to pathological thickening of the arterial wall, which restricts blood flow to major organs including the heart (i.e., coronary artery disease) and brain (i.e., cerebrovascular disease) (59). In addition, the atherosclerotic plaque can rupture into the artery and result in a blockage of the artery, known as arterial thrombosis (14). In more severe manifestations of these conditions, individuals suffer a catastrophic loss of blood flow to the heart (i.e., myocardial infarction) or brain (i.e., stroke) which can prove fatal (2). In total, ACD is estimated to be responsible for 84.3% of CVD-related deaths (2).

Although categorizing types of CVD may be helpful for understanding the differential mechanisms and modifiable risk factors for each constellation of symptoms, the complexity of CVD does not allow for such clean separation of etiological mechanisms. That is, one major risk factor for ACD is the presence of hypertension, especially in conjunction with hypercholesterolemia (71); hypertension increases strain on the arterial wall and promotes the deposit of atherosclerotic plaque into arterial lesions. The blockage created by atherosclerosis contributes to the development of hypertension through increased blood pressure (71). Given the commonalities between hypertensive and atherosclerotic cardiovascular disease, the term CVD will represent all cardiovascular problems related to atherosclerosis and hypertension.

Major Adverse Cardiac Event

Major adverse cardiac events (MACE) refer to a composite of outcomes from CVD that most commonly include stroke, congestive heart failure, myocardial infarction and all-cause mortality in individuals diagnosed with cardiovascular disease (37).

Although individuals with a significant history of MACE are likely captured among those diagnosed with hypertensive heart disease or, more likely, atherosclerotic heart disease, survivors of MACE are important to parse out from these diagnostic groups given the prognostic difference and severity of functional impact (43; 82; 130; 175).

Stroke

Stroke refers to a catastrophic loss of blood supply to portions of the brain that can occur when arteries to the brain are blocked (i.e., ischemic stroke) or burst and begin bleeding into the brain (i.e. hemorrhagic stroke), often leading to death of brain cells (58). Prognostic indicators for stroke recovery in the first 30 days include evident factors such as the severity of the stroke, cardiac history, and age (58). However, indicators in the five years following a stroke focus on modifiable behavioral risk factors for atherosclerotic heart disease — namely tobacco use and obesity (58). In total, only about 40% of individuals survive more than five years after a stroke, with half of those survivors experiencing cognitive decline, disability, or dependence (21; 58).

Congestive Health Failure

Congestive heart failure (CHF) refers to inefficiency of the heart muscles due to chronic hypertension or atherosclerotic buildup in the coronary arteries (97). CHF is most commonly on the left side of the heart and occurs when the heart cannot sufficiently relax to fill properly (i.e., diastolic failure) or when the heart cannot exert the required force to

eject arterial blood (i.e., systolic failure) (97). Diastolic and systolic failure impact the efficiency of the left ventricle in ejecting arterial blood to the body, called the left ventricular ejection fraction (LVEF). The development of CHF is attributed to a complex combination of hypertensive and atherosclerotic heart disease, with the most common pathway through diastolic failure related to left ventricular hypertrophy (97).

CHF is somewhat of a misnomer given it does not exclusively refer to the catastrophic failure of the heart muscles. Rather, there are four classes of CHF that reference the measured LVEF, history of hospitalizations, and the presence and functional impact of cardiac symptoms at states of rest and physical activity (108). Typical symptoms experienced with CHF during ordinary physical activity are breathlessness, fatigue, and chest pain, which act as measures of severity as well as prognostic indicators (43). Ordinary physical activity here refers to actions such as walking at a normal pace, climbing stairs or completing activities of daily living. The New York Heart Association classifications range from no limit to ordinary physical activity (i.e., Class I) to unable to carry out any physical activity without discomfort and symptoms of heart failure (i.e., Class IV) (108).

Myocardial Infarction

Myocardial infarction (MI), or heart attack, refers to the catastrophic loss of blood to the heart muscles due to prolonged ischemia, leading to the death of myocardial tissue (160). MI is classified into five different types based on cause and outcome. Types 1 and 2 refer to an ischemic coronary event, with the former identified as a primary coronary ischemia and the latter considered secondary to insufficient oxygen supply (e.g., coronary artery spasms) (160). Type 3 captures sudden cardiac death, while types 4 and 5 refer to

MI associated with different revascularization procedures. MI can also be classified by location, severity of tissue damage, and stage of recovery (160).

MI is most often the result of coronary heart disease — a term referring to atherosclerotic cardiovascular disease localized to heart tissue (7). Similar to stroke, the process of developing atherosclerotic plaque in myocardial tissue shares many of the same risk factors as arteries in other parts of the body; individuals with poor health behaviors are at particular risk of initial MI and subsequent reinfarction (7). MI also has long-term impacts on the cardiac nervous system, reducing the functional responsiveness of ventricular pacing and hindering healthy modulation of heart functioning (82; 130).

IMPACT OF CARDIOVASCULAR DISEASE

The morbidity and mortality of CVD highlight the importance of examining and implementing strategies for promoting cardiovascular health. The financial and organizational impact illustrate the burden on society through nonclinical outcomes. Finally, the functional impact of CVD provides key insights into the effect on the individual to include quality of life, physical and mental ability, intimate and social engagement, and activity engagement.

Mortality of Cardiovascular Disease

CVD has been the leading cause of death in the United States for nearly a century (175). With ischemic heart disease and stroke responsible for one in four deaths, the global burden of cardiovascular disease is unparalleled (27; 175). Patterns of CVD-related mortality in the United States reveal that although the rates of CVD-related death were falling between 1968 and 2012 with the improvement of assessment and treatment, rates have begun to rise again in the last 7 years (61). The AHA's projected prevalence of

CVD suggests nearly half of Americans will have some form of CVD in the next 15 years (4).

Financial Impact of Cardiovascular Disease

Costs associated with CVD are broken down into direct medical expenses and indirect costs, typically attributed to loss of productivity. Direct medical expenses include treatment of CVD, the sequelae of cardiac events, and related or exacerbated conditions. Lost productivity is calculated into indirect costs through metrics such as days of work missed, days with disability that impact activities of daily living, and lost earnings due to premature death. The combined cost to the US in 2017 is estimated at \$555 billion, with \$318 billion attributed to direct medical costs. This combined cost is expected to double by 2035 to \$1.1 trillion due to the increasing mean age of the population in the United States (4). Even a conservative estimate, isolating personal health care spending (i.e., insurance claims, household spending, and government funding), determined CVD was the most expensive personal health care expense in the United States at \$231.1 billion in 2013 (39).

Functional Impact of Cardiovascular Disease

In 2002, the World Health Organization introduced a common definition for functioning in an effort to emphasize the functional impact of disease beyond presenting symptoms (174). In this context, functioning is defined as all bodily functions, activities, and participation using a biopsychosocial model. This definition extends beyond traditional definitions of health and disability to capture all personal, environmental, contextual, and health factors (174). The inclusion of functioning as a measure of disease impact extends the scope of research to highlight factors such as quality of life, emotional

health, and social engagement. The following section will provide an overview of what is known about the functional impact of hypertensive, atherosclerotic, and cardiovascular disease.

Functional Impact of Hypertensive and Atherosclerotic Cardiovascular Disease

Subjective symptoms experienced with hypertensive and atherosclerotic cardiovascular disease can include fatigue, chest pain, difficulty breathing, headaches, and a host of other symptoms (57; 144). In addition to subjective physiological symptoms, individuals often report a lower overall quality of life including perceptions of lower general health, worse physical functioning, more physical pain, less social engagement, and poorer emotional health than a healthy comparison (12; 65; 163). Objectively, individuals with CVD demonstrated lower work productivity and greater activity impairment as measured by time missed and perceived impairment (33; 65). CVD can also exacerbate the functional impact of comorbid disabilities such as cognitive impairment in older adults (144).

Functional Impact of Major Adverse Cardiac Events

Although MACE is associated with a heightened risk for mortality, the number of individuals surviving long enough to experience the long-term functional impact of such events is increasing with improvements in medicine (35). Survivors of a MACE often experience a reduction in global functioning and quality of life related to their reported symptoms. As mentioned above, individuals with a history of MI or heart failure often report chest pain, difficulty breathing, insomnia, and fatigue, while those with a history of stroke experience an array of other physical disabilities and cognitive decline (7; 43). These symptoms not only impact subjective quality of life, but adversely affect

engagement in physical activity, social and intimate relationships, and physical ability (35; 60). For example, while fatigue and shortness of breath are linked to reductions in physical activity (34), extending the scope of functional impact to include sexual engagement encompasses associated factors such as erectile dysfunction, premature ejaculation, chest pain, fatigue, low libido and patient beliefs regarding the safety of sexual activity with a history of MACE (42; 121; 153; 161).

MODIFIABLE RISK FACTORS OF CARDIOVASCULAR DISEASE

Modifiable risk factors include conditions that contribute to the overall risk of adverse outcomes which can be reduced through preventative or responsive intervention. Although there are several non-modifiable risk factors, such as age, that contribute to CVD, the most significant modifiable risk factors can be targeted through behavioral intervention (177). A global evaluation of modifiable risk factors across 21 countries with variable life expectancies identified tobacco use as the single strongest predictor of cardiovascular death. Physical activity, diet, and obesity were in the top five after alcohol (177); however, alcohol is not a target of the current study given the complicated nature of alcohol use reporting in active duty military populations (81). Finally, behavioral changes associated with depression provides an additional modifiable risk factor for CVD with known deleterious impacts on health behaviors such as tobacco use and obesity (171).

The Influence of Health Behaviors on Cardiovascular Disease

Many health behaviors contribute to the development or prevention of cardiovascular illness. In fact, engaging in recommended health behaviors such as physical activity, healthy diet, abstinence from tobacco use, and healthy weight can triple

the likelihood of preventing premature death associated with diseases such as CVD (100; 172). Unfortunately, only about 2.7% of Americans engage in all four of these recommended health behaviors (101). This section will focus on the impact of tobacco use and obesity for their known contributions to the development of CVD and relevance to the current study.

Tobacco Use and Cardiovascular Disease

Tobacco use includes a range of products categorized into combustible (i.e., smoked) tobacco, e-cigarettes, and smokeless tobacco. The association of tobacco use with all-cause mortality is considered the number one preventable cause of death (165). Although public health practice and research is focused on smoked tobacco, there is a growing emphasis on better understanding the impact of electronic nicotine delivery systems, or e-cigarettes (98); however, this section will focus on the use of combustible tobacco given the lack of available data demonstrating the cardiovascular effect of e-cigarette use (18; 112).

In the United States, 19.3% of the population uses tobacco products, with 16.7% reporting use of combustible tobacco products (169). The pathophysiology of combustible tobacco use in the promotion of CVD development is a complex process involving the prothrombotic and atherogenic effects of nicotine, carbon monoxide, and oxidant gases (25; 75). Nicotine elevates cardiac function (i.e., heart rate, blood pressure, etc.) and increases the arterial concentration of fatty acids and glycerol. Carbon monoxide binds to hemoglobin, creating carboxyhemoglobin and diluting the concentration of oxygenated hemoglobin (25); the diluted arterial blood supply results in compensatory elevation of cardiac function and alters the composition of the blood to include a greater

total concentration of hemoglobin, increasing the blood viscosity and prothrombotic properties (25). Finally, many of the chemicals in combustible tobacco produce oxidant gases, which lead to atherogenesis through oxidation of proteins, lipids and DNA, as well as promotion of oxidative stress by hindering antioxidant defense (75).

In combination, these chemicals rapidly accelerate the processes outlined in both atherosclerotic and hypertensive cardiovascular disease, increasing the risk of sudden cardiac death by 206% compared to those with no history of tobacco use (11). Even individuals who tout the benefits of “light smoking” are at risk of a highly nonlinear dose-response relationship, which suggests even exposure to only 4 to 7 cigarettes per day maintains 70% of the effect of heavy smoking (126; 141). Finally, those with only a history of tobacco use are still at a 38% increased risk of sudden cardiac death compared to complete abstinence (11).

Obesity and Cardiovascular Disease

Overweight and obesity are typically defined using a height-to-weight ratio to estimate body fat called the body mass index (BMI). For adults, obesity is defined as a BMI of 30 or greater, overweight as 25 or higher, and healthy weight between 18.5 and 25. Further, obesity is stratified into Class 1 (BMI 30–35), Class 2 (BMI 35–40), and Class 3 (BMI 40 or higher) obesity (30). In the United States, obesity affects approximately 39.8% of adults and 18.5% of children and adolescents (28-30). Global rates of obesity are estimated closer to 13%, however the global prevalence of obesity has over tripled in the last 40 years (1). In the most basic terms, overweight and obesity are the result of a caloric surplus. However, the factors that impact the basic equation are far more complicated and include cultural, behavioral, social, metabolic, physiologic and

genetic factors that are not completely understood (147). Although the negative health effects of poorly managed weight on health can appear as a bimodal distribution (i.e., overweight and underweight are both related to poorer health), obesity will be the focus of this section for its well-established relation with CVD and relative prevalence in comparison with underweight (1; 28; 29; 47). Additionally, overweight BMI will be reviewed but excluded from the current study given the complicated cardiovascular effects outlined in the obesity paradox.

Obesity has been identified as an independent predictor of hypertensive and atherosclerotic cardiovascular disease by increasing fatty tissue, endocrinological differences, and physical strain on the cardiovascular system (147). The mechanisms of hypertension are not fully understood but are thought to be attributable to endocrinological changes associated with obesity such as hyperinsulinemia or hyperleptinemia. The physical requirements of excess body mass put increased strain on the heart, which can further exacerbate the effects of hypertension and increase risk of congestive heart failure (40). Atherosclerotic cardiovascular disease is explained by the increase in fatty tissue, concentration of LDL cholesterol, and susceptibility to atherosclerotic plaque buildup (40). This increase in atherosclerotic plaque makes obesity a risk factor for stroke and myocardial infarction. For middle-aged adults, the risk of CVD morbidity increases by 21% to 32% for overweight and 153% to 214% for severe obesity compared to healthy weight peers. Overall, overweight and obesity were associated with earlier onset of CVD, greater years lived with disability, and reduced longevity (79).

Obesity Paradox

One anomaly in the relation between weight and CVD has been coined the “obesity paradox.” This consistently replicated phenomenon suggests that individuals with overweight and Class 1 obesity are at reduced risk for cardiovascular mortality compared to their healthy weight peers (64; 146); while Classes 2 and 3 obesity have consistently been associated with significantly higher risk of all-cause mortality (47). The obesity paradox is sustained across chronic and acute heart failure, non-cardiovascular mortality, and across heterogeneous biochemical and clinical profiles (i.e., only differing in BMI) (146).

Skeptics have attempted to explain the paradox using methodological phenomena such as selection, survival, and treatment biases (150). Others have proposed it may be an artifact of BMI as the sole measure of obesity, suggesting individuals with high percentage lean mass may present as overweight on measures of BMI alone (64). Further still, the heterogeneity of the healthy-weight comparison has been cited as a potential explanation, with consideration for complicating factors that increase risk for CVD such as tobacco use, medical conditions that mimic healthy weight and environmental factors (64; 150). Finally, cardiac cachexia (i.e., the loss body fat, muscle and bone following a cardiac event) may artificially classify individuals as a healthy weight with poor cardiovascular outcomes (64; 150). However, no combination of these explanations has fully accounted for the obesity paradox.

Cardiorespiratory Fitness

The current cardiovascular literature suggests cardiorespiratory fitness (CRF), or the combined health of the heart and lungs, is a far greater predictor of cardiovascular outcomes than weight status (44). In cardiovascular mortality, the strength of the

relationship with CRF is nearly double that of BMI (44). Further, healthy CRF can offset and nearly eliminate obesity-associated risk of cardiovascular mortality. Those with combined low CRF and obesity have a cardiovascular mortality risk nearly triple that of fit, healthy-weight individuals (15). The predictive value of CRF suggests that the obesity paradox is likely the result of unmeasured effect of CRF on the impact of overweight and obesity; additionally the predictive value of CRF emphasized the importance of physical activity targeting cardiorespiratory health in tandem with healthy weight management (15; 44; 64).

Cardiovascular Implications of Depression

Despite the well-established cardiovascular benefits of maintaining a healthy weight and abstinence from tobacco, almost 40% of American adults are obese and 20% use tobacco (120; 169). With the inclusion of a healthy diet and physical activity, less than 3% of the population engages in the four most important lifestyle characteristics for prevention of the number one cause of death in the United States (101). One factor in better understanding the low rate of health behavior engagement is the presence of depressive symptoms.

Depressive symptoms are characterized by sad, irritable, or empty mood, accompanied by adverse somatic, physiological, cognitive, and behavioral changes. Symptoms can include changes such as insomnia, fatigue, changes in appetite, social withdrawal, poor concentration, and increased risk of suicide (5). The behavioral changes, particularly changes in engagement of healthy behaviors such as exercise and healthy eating, associated with depressive symptoms have been implicated as a mediating factor in the relationship between depression and CVD (171). Additionally, physiological

changes associated with depression include dysregulation of the sympathetic nervous system and activation of the hypothalamic-pituitary-adrenal (HPA) axis (38).

Physiological Mechanisms of Depression in CVD

The physiological mechanisms in the cardiovascular implications of depression are not well understood but generally include the disturbance of autonomic nervous, neuroendocrine, and autoimmune systems. Disturbance of the autonomic nervous system in depression is characterized by frequent and disproportionate activation of the sympathetic nervous system (38). The cascade initiated through sympathetic hyperactivity elevate heartrate, blood pressure, noradrenaline levels, and manifests in symptoms that mirror hypertensive heart disease. Over time, sympathetic hyperactivity is associated with many of the cardiovascular outcomes outlined above, including left ventricular hypertrophy and congestive heart failure (38).

Alterations in the neuroendocrine system associated with depression are manifested in hyperactivity of the HPA axis. The hyperactive HPA axis leads to glucose intolerance, hyperlipidemia, and elevation of visceral adiposity; combined, the symptoms mirror metabolic syndrome, which accelerates the onset of hypertensive and atherosclerotic heart disease (38; 105). Autoimmune changes displayed in platelet activation and endothelial inflammation, which promote the formation of atherosclerotic plaque (38; 94; 104).

Disturbance of physiological mechanisms in depression is defined in part by an increase in the allostatic load, or the cost of achieving homeostasis, predominately in the neuroendocrine and autonomic nervous systems (91). An increase in allostatic load can lead to a blunting, exacerbation, or delayed recovery of the physiological stress response.

The greatest evidence for the pathways between increased allostatic load, depression, and CVD suggests that a delayed recovery from the stress response prolongs the aforementioned effects of disturbances to the neuroendocrine and autonomic nervous systems (91). Alternatively, a blunting or delayed recovery of the stress response associated with depression may lead to an increase in poor health behaviors.

Behavioral Mechanisms of Depression

There are several known behavioral and lifestyle changes associated with depression that directly influence the morbidity and mortality of CVD. A synthesis of the known relations between depression and CVD highlights the bidirectional impact of obesity, increased alcohol use, physical inactivity, and tobacco use (151). For example, the onset of depression and CVD are both known to predict a decrease in physical activity; which in turn maintains depressive symptoms, reduces cardiorespiratory fitness, and increases the risk of CVD (15; 142). Some studies suggest these behavioral mechanisms completely mediate the relation between depression and CVD (171). This section will focus on the behavioral mechanisms of tobacco use and obesity as identified health behaviors associated with depression and CVD.

Tobacco Use, Depression, and CVD

The relation between tobacco use, depression, and CVD is so well established that some refer to it as the “tragic triad” (159). Individuals who use tobacco are more likely to endorse depressive symptoms, while those who are depressed are less likely to successfully engage in smoking cessation. Even among individuals with similar cardiovascular-related disability, smokers are more likely to endorse depressive symptoms (149). The correlation between tobacco use and depression is well founded but

the causality is poorly understood. Tobacco use is suggested to increase the likelihood of depression through symptoms of tobacco use such as increased irritability during withdrawals, cardiorespiratory limitations to physically demanding activity, or fatigue (48). Conversely, individuals may pick up smoking to cope with stress or depressive symptoms (48; 95). For example, one study demonstrated poor distress tolerance predicted greater severity and frequency of tobacco cravings and dependence (164). Regardless of the directionality, individuals who successfully quit smoking also report improvement in depressive symptoms (158).

Obesity, Depression, and CVD

The strength of the association between depression and weight is so well founded that the American Psychiatric Association identifies a significant and unintentional change in weight as a criterion of major depressive disorder (5). Individuals with higher weight display a greater incidence of depression from adolescence to adulthood, with the strongest relation in females and in obese BMI ranges (6; 106; 123; 128; 155). The relation between depression and obesity is thought to have bidirectional causality, with greater weight exacerbating depressive symptoms and depressive symptoms hindering healthy weight management (106).

Obesity promotes negative body image, reduced interest in physical activity, and maintenance of a sedentary lifestyle (123; 155). Conversely, the elevated stress response associated with the onset of depression increases the production of biochemical hormones and peptides, driving a heightened appetite (6; 123; 128). Additionally, a depressed mood may lead to altered eating patterns such as binge-eating and seeking calorie-dense foods, further exacerbating weight-gain (123). The effects of treatment targeting depression

provide further insight, as many individuals experience ameliorated weight-related concerns attributed to reductions in the maladaptive patterns (6; 128). Untreated, depression and obesity create a cyclical pattern of self-sustaining symptoms that heighten the risk of CVD-related morbidity and mortality (147).

CURRENT MODEL FOR TREATING MODIFIABLE RISK FACTORS AND LIMITATIONS

Before considering the impact of a model intended to enhance health behavior change in primary care, it is important to first understand the existing model of treatment. In the current model of treatment, primary care functions as the medical home and de facto treatment setting for behavior change intervention to improve health behaviors and mental health conditions. The following will review standard practice addressing health behavior change and limitation to the current model in primary care.

Health Behavior Change in Primary Care

With the onset of hypertensive and atherosclerotic symptoms, the standard practice guideline suggests patients are recommended pharmacologic intervention in addition to nonpharmacologic interventions to reduce symptoms such as high blood pressure, cholesterol, and weight. The standard first line recommendation includes weight loss and smoking cessation with a focus on a heart healthy diet and physical activity (143).

Tobacco Cessation in Primary Care

The American College of Cardiology (ACC) and US Public Health Service (USPHS) decision pathway and treatment guidelines for tobacco cessation recommends the use of a 5-step model based on the 5A's Behavior Change Model (ask, advise, assess,

assist, and arrange). Guidelines recommend all patients presenting to primary care are screened (i.e., *ask*) for tobacco use (16; 46). Providers are then recommended to *assess* tobacco use patterns, quit attempts, relapse risk for former tobacco users, and second-hand exposure for nonsmokers. *Advise* includes providing information on the harmful effect of tobacco use or exposure, benefits of quitting, and potential targets for relapse prevention or reducing second-hand exposure. The ACC and USPHS differ on their stance in care delivery for the last two steps, but both agree these steps involve *assisting* in treatment recommendations for tobacco cessation and *arranging* follow-up (16; 46).

Treatment recommendations for tobacco cessation in primary care most commonly employ pharmacotherapy tools divided into nicotine replacement therapy (e.g., nicotine gums, patches, nasal sprays, inhalers, etc.) and non-nicotine pharmacotherapy (e.g., bupropion, varenicline, etc.), with behavioral strategies often considered supplemental (16; 46; 129). ACC guidelines provide recommend behavioral support resources for tobacco cessation, with limited review of specific behavioral strategies; generally, guidelines leave the implementation of behavioral strategies to behavior specialists, which are differentially available in the current model of treatment (16).

Weight Management in Primary Care

Recommendations and guidelines for weight management are outlined in a combined report by the ACC, AHA, and The Obesity Society (74). Recommended weight loss goals for clinically meaningful change often requires 5% to 10% weight reduction in 6 months; the chief method for weight loss requires an energy deficit achieved with a combination of dietary restriction of calorie intake and physical activity.

Restricted calorie intake is 1200 to 1500 kcal per day for women and 1500 to 1800 kcal per day for men, with special considerations of medical conditions that may interact with weight loss (e.g., hypertension, diabetes, etc.). Guidelines suggest individuals initiating weight management should be referred for high-intensity comprehensive lifestyle intervention, defined as at least 14 individual or group appointments with a behavior change specialist in a six-month period (74). Additionally, individuals with a BMI of 30 or greater, or BMI greater than 27 with comorbid conditions, are also recommended for pharmacotherapy; individuals with a BMI greater than 40 or greater than 35 with comorbid conditions are recommended for bariatric surgical consultation (74).

Limitations in the Current Model

Although the current model of treatment for behavior change outlines empirically founded interventions for effective tobacco cessation and weight management, there are several limitations leaving a gap in the current model. In weight management and tobacco cessation, primary care teams report difficulty discussing required changes, especially if they personally have difficulty with diet, physical activity, or tobacco use (168).

Tobacco Cessation

Some providers also report barriers to addressing tobacco cessation related to beliefs about patient responsibility in initiating quitting, defeated attitudes toward resistance to tobacco cessation, and preference for pharmacotherapy without behavioral intervention (166). Further, light smokers (i.e., four to seven daily cigarettes) are almost half as likely to receive recommendations around tobacco cessation compared to moderate-to-heavy smoking peers, despite sharing nearly 70% of cardiovascular mortality risk (86; 141). When using the recommended 5A's Behavior Change Model,

patients report generally higher satisfaction with the health care services provided; however, broken down by the 5A's, more than half of primary care teams ask, assess, or advise on tobacco cessation, but less than half assist and only 9% arrange for a follow-up. Further, only 18% of individuals are referred to a specialist for behavioral intervention, with most treated using pharmacotherapy and educational materials (129).

Weight Management

Although the equation for weight loss is seemingly a simple energy deficit, the key predictors of successful weight loss and weight maintenance are notably different. During weight loss, success is best achieved with consistent adherence to a sustainable diet in accordance with the recommended calorie deficit and a gradual introduction of physical activity (154); however, this process naturally results in diminishing return as free fat mass is reduced, which precipitates physiological changes that promote craving for calorie dense foods and reduced energy expenditure (154). Therefore, weight loss is best predicted by adherence to a calorie deficit, and weight maintenance is best predicted by behaviors such as regular physical activity and regular self-monitoring of weight. In addition to the inherent challenges associated with effective behavior change in weight management, primary care visits do not adequately address weight. Only 5.8% of primary care visits involve weight management, despite 39.8% of adults in the United States meeting criteria for overweight and obesity (28; 30; 109). Of primary care patients with obesity, nearly 90% acknowledge a need for weight loss, with expressed interest in dietary advice, goal setting, and physical activity recommendations; however, less than half discussed weight with their primary care team (127).

Combined, the current treatment paradigm possesses empirically founded interventions for tobacco cessation and weight management, but each requires a major behavioral component that cannot be adequately addressed by the traditional model. Even without the barriers to implementing effective tobacco cessation or weight management in the current model of primary care intervention, most of the treatment guidelines recommended a referral to behavioral specialists as a part of standard care (16; 74). The recommended referral to behavioral specialists in siloed primary care settings without such an asset, combined with the aforementioned limitations of traditional primary care teams, suggests the integration of a behavioral health professional into primary care teams may enhance treatment over the current model.

PRIMARY CARE BEHAVIORAL HEALTH

A method for addressing the limitations of primary care in promoting positive health behaviors is the integration of behavioral health providers into the primary care setting; integrated primary care is a growing method for delivering behavioral health care in civilian and military facilities (17; 50; 67; 70). The integration of behavioral health providers offers a stepped-care approach that expands the opportunities for patients to receive evidence-based behavioral health care and fills a gap in care where patients are currently undertreated. One commonly used treatment model for delivering care in this setting is the Primary Care Behavioral Health (PCBH) model (138).

In the PCBH model, a behavioral health consultant (BHC) works directly with a primary care provider (PCP) and the primary care team to enhance the holistic approach to health care in a primary care clinic (132). A BHC operates differently than a typical behavioral health care provider in that the BHC serves as a consultant to the PCP but

does not take primary responsibility for the care of the patient. The medical records, treatment plan, and standards of care are shared between the PCP and BHC (69). Given signs of clinically significant distress or a clinically implicated need to improve health behaviors, the PCP will request the consultation of a BHC to assist with the assessment and development of an intervention plan to improve functional outcomes for the patient. The standard model of 45- to 90-minute therapy sessions is substituted with 15- to 30-minute assessment and intervention appointments. During these appointments, the BHC addresses the specific mental health concerns or other health behaviors, such as smoking cessation or weight management (70).

Tobacco Cessation in PCBH

Tobacco cessation with the integration of behavioral health consultants provides specialized skills to the primary care clinic that can improve quit rates through enhanced assessment of behavior patterns related to tobacco use, motivational interviewing, and counseling on effective cessation strategies (68; 88; 131; 152). In one guide for assessment and intervention of tobacco use in the PCBH model, BHCs are recommended to assess previous quit attempts, current patterns of use, and provide strategies to assist the patient in moving toward an effective quit that are consistent with their indicated stage of change; that is, even if patients do not indicate a current desire to quit, BHCs can still promote quitting through motivational enhancement techniques (e.g., eliciting change talk toward quitting). BHCs can also continue to follow patient progress with touchpoints around vital points in the quitting process (68)(p.88). Although evidence from specialty care suggests the use of counseling, in conjunction with pharmacotherapy, yields optimal results, the existing data on effectiveness in the PCBH model is limited

(67; 152). One study, using a university-based sample of integrated primary care patients ($N = 15$), found outcomes more consistent with harm reduction than cessation, with patients cutting tobacco use down by an average of 64% after a mean of 3 appointments ($SD = 1.77$); however, only one participant effectively stopped tobacco use in the monitored period (137).

Weight Management in PCBH

The integration of behavioral health consultants also provides specialized skills to the primary care clinic that can enhance strategies for healthy weight loss by targeting manageable lifestyle changes focused on alteration to patterns of eating and physical activity. In one guide for assessment and intervention of obesity in the PCBH model, BHCs are recommended to assess BMI, eating behaviors, physical activity, and previous efforts targeting weight. Interventions are oriented around goal setting, calorie education, and a sustainable behavior change plan aiming to lose weight at a rate of 1 to 2 pounds per week (68) (p.93). Similar to tobacco cessation, the existing data on effectiveness in the PCBH model in addressing health behavior change around weight management is limited (67). The same study mentioned above using a university-based sample of integrated primary care patients also examined weight management in PCBH. In a subsample of patients seeking intervention for weight management ($N = 9$), Sadock and colleagues (137) found an average weight loss of 7.7 pounds after a mean of 3 appointments ($SD = 1.17$) across 52 days ($SD = 44.76$).

Benefits of the PCBH Model

To understand how the PCBH model addresses the aforementioned limitations in the current model of treatment, the following will outline benefits and shortcomings of

the model within the conceptual framework that guides efforts to reduce the existing treatment gap. The Quadruple Aim is a patient-centered healthcare approach which serves to optimize health system performance through emphasis on improving population health by targeting enhanced access to care, enriching the patient experience, reducing cost of health care, and augmenting the care team to improve well-being (19; 20). An emphasis on these domains also provides a framework for understanding the existing literature on the benefits of the PCBH model.

Enhanced Access to Care

The PCBH model has demonstrated improvements in access to behavioral health care, with some systems increasing penetration of behavioral health services by as much as 391% within one year of implementation (23). The penetration of behavioral health services, or extent to which the health care service encounters the identified patient population, is partly improved by reducing the temporal cost of treatment in the PCBH model. The brevity of interventions and collocation with primary care alone are shown to reduce the time between referral and first appointment from an average of 33 days in specialty behavioral health to an average of 19 minutes in integrated primary care (125). Some clinics, particularly within the military, implement a gateway model in which appointments for consultation within the PCBH model act as a required medical home for referral to specialty behavioral health. This approach to the PCBH model can further improve the penetration of treatment by reducing no-shows and enhancing access to higher levels of care by reducing the burden on specialty behavioral health (89). Still, little is known about the penetration of behavioral health services for improving health behaviors related to CVD.

Reduced Cost

Although the PCBH model is thought to reduce overall health care costs, the benefits of the model are complicated by the scarcity of literature on implementation cost (67). One estimate used patient claims data and suggested that, compared to those who did not have contact with integrated primary care, patients saved an average of 10.8% on annual healthcare costs (134). Part of this cost reduction is attributed to the pattern that those who had seen a BHC had fewer preventable hospitalizations, reducing the overall cost of healthcare (90).

Improved Patient Experience

The patient experience of care is a component of patient-centered care that, here, includes metrics such as satisfaction with services provided and perceived stigma related to seeking or engaging in care.

Satisfaction

Patient satisfaction is at the core of improving the patient experience and quality of care (67). High satisfaction with the PCBH model is the most consistently endorsed outcome across a variety of presenting problems (e.g., insomnia, parenting, depression, etc.) and populations (e.g., general population, students, and active duty military) (10; 49; 53; 54; 76; 135). High satisfaction was frequently attributed to perceived effectiveness and value of the consultation (10; 49; 53). These studies provide consistent evidence that the patient experience of care is not hampered by the brevity or pace in a primary care setting.

Stigma

Although the high patient satisfaction with the PCBH model is associated with a high likelihood of returning for follow-up appointments (49), stigma is consistently cited as the greatest barrier for seeking behavioral health care or presenting to a first appointment (9; 77; 83; 115; 140). The integration of care into a primary care setting and team-based approach attempts to destigmatize the patient experience (63; 132; 178). Additionally, the consultative nature of the PCBH model limits the impact of stigma as a barrier to treatment; the model aims to identify and provide behavioral health consultation with relative immediacy through same day appointments and reduces the need for help seeking behavior. Recent data suggests attitudes toward seeking care in a primary care or medical setting is preferable to specialty behavioral health, especially in those from ethnic minority populations (119). Still, this data examines individuals who completed an initial appointment, limiting generalizability to those with untested attitudes toward seeking behavioral health care.

Care-Team Wellbeing

In addition to improving the patient experience, the PCBH model offers to improve care-team wellbeing through reducing the burden on maintaining the specialized knowledge and skills required for health behavior change and behavioral health interventions. The consultation model allows the specialized behavioral health consultants to both provide and advise relevant clinical work for the primary care team (132). Outcomes of care-team wellbeing are typically evaluated using the primary care team's reported satisfaction with the PCBH model. Primary care teams consistently report high satisfaction with the model. Teams also report perceived value from improved

accessibility, efficiency, accurate assessment, and quality of behavioral health care provided (49; 135; 145; 162).

IMPORTANCE OF MILITARY HEALTH CARE BENEFICIARIES

Military health care beneficiaries encompass a variety of individuals who receive health care services from installations across the Army, Navy, Air Force, and National Capital Region. These individuals provide an opportune population for better understanding the effectiveness of the PCBH model given a high prevalence, deleterious effects, and well-documented impact of tobacco use, obesity, and depression within the military health care system (113; 118; 136).

Tobacco Use and Military Health Care Beneficiaries

Tobacco use in military health care beneficiaries differs largely by beneficiary category and branch of service (13). Prior to 2011, active duty used smoked tobacco at higher rates than the civilian population (13); however more recent surveys suggest rates of smoked tobacco is comparable to the civilian population (i.e., around 16%), with e-cigarette use increasing more than seven-fold in the three-year period from 2011 to 2014 (113). Active duty service members reported nearly four times higher prevalence of smokeless tobacco use than the general civilian population (113). The active duty population is also more likely to use more than one type of tobacco, with approximately 7% using two or more types of tobacco products (118). Although there is a growing perception that that tobacco use is discouraged in the active duty population (113), service members are at an increased risk of starting or expanding tobacco use during and prior to deployments (157). Across branches of service, the Marine Corps has the highest rate of all tobacco use and reports using smoked and smokeless tobacco at nearly twice

the rate of the branch with the lowest prevalence – the Air Force (13); the Army reports tobacco use at rates similar to the Marine Corps, followed by the Navy and Coast Guard (13). Military veterans have not shown the decline in smoked tobacco use noted in the active duty population; most recent surveys show approximately 29.2% of military veterans use tobacco products regularly (118). The global military health system costs associated with medical treatment for tobacco use were estimated at \$1.6 billion per year as recently as 2017 (176). Enhanced strategies to target tobacco use are a valuable pursuit among military health care beneficiaries.

Obesity and Military Health Care Beneficiaries

Despite weight and body composition standards in the active duty military population, only 27.4% of an active duty sample of 11,243 individuals from the Millennium Cohort Study were a healthy weight; 49.1% were overweight and 23.6% were obese, with trends demonstrating a steady increase in the average weight of the population (136). Military dependent demonstrated rates of overweight and obesity at nearly 60%, suggesting higher rates than even the general population in the United States (87). In the decade from 1995 to 2005, rates of obesity in active duty service members increased by 161.8%, negatively impacting the operational readiness as well as overall health and fitness of the United States military (133). Additionally, individuals are at elevated risk of weight gain after discharge from the military related to changes in multiple health behaviors such as diet, sleep, and physical activity (22). Consistent with the literature review above, individuals in the Millennium Cohort Study with BMI classified as obese displayed greater incidence of CVD and depression than their healthy weight peers (136). In the military health system, costs associated with medical treatment

for excess weight and obesity were estimated at \$1.1 billion per year as early as 2007 (36). The high prevalence of obesity in a population when physical readiness is critical for organizational health highlights the value of understanding the PCBH model in military health care beneficiaries.

Depression and Military Health Care Beneficiaries

The rates of depression in the United States military population mirror the United States general population, with 12-month prevalence rates estimated at 9.4% (78; 113). Individuals who receive a mental health related diagnosis are also 80% more likely to attrit from the military than their healthy peers (173). Organizationally, service members with depression are more vulnerable to impaired work performance, more days of missed work, and lower physical health (113; 124). As the United States military continues to expand resources for mental health, understanding the impact of the PCBH model on depression remains a valuable pursuit.

THE PRESENT STUDY

In summary, the current literature supports the continued deleterious impact of CVD on a global, population, and individual level. One potential avenue through which to target CVD is the well-established association between CVD, tobacco use, obesity, and depression. Specifically, the enhancement of available interventions for tobacco use, obesity, and depression may improve CVD-related health outcomes. Integrating behavioral health consultants into primary care using the PCBH model is one alternative that enhances the behavioral health care provided in primary care settings through delivery of evidence-based care by specialized providers. This model serves to improve population health, reduce cost, improve patient experience, and maintain the care team

well-being with an emphasis on addressing global functioning through brief, targeted interventions. Despite the known benefits, growing implementation, and promising health outcomes associated with the PCBH model, the existing literature on health behavior outcomes and chronic disease management is limited (67). The high prevalence, deleterious effects, and well-documented impact of tobacco use, obesity, and depression within the military population, in combination with the large-scale implementation of the PCBH model in the military healthcare system, make the military an ideal setting for examining the efficaciousness of a growing health care model.

Efforts to outline methods for improving the effectiveness and implementation of the PCBH model suggest a need for studies that mirror the brief, time-limited, and infrequent characteristics of appointments in primary care (17). The heterogeneity of the population and pragmatic emphasis on effectiveness over efficacy call for rigorous quasi-experimental design from a data source, such as electronic medical records, that captures the real-world effectiveness of the PCBH model. There is also a need for continued growth in validating the effectiveness of the model using standardized measures of global functioning and behavior change (17; 67). These recommendations suggest a study evaluating a large sample in a quasi-experimental design using a standardized measure of global functioning may contribute to better understanding the effectiveness of the PCBH model.

The overall objective of this study was to evaluate the effectiveness of BHCs to improve modifiable risk factors (i.e., tobacco use, obesity, and depression) among those diagnosed with cardiovascular disease. To do so, this study analyzed data previously collected by the Psychological Health Center of Excellence of the J-9 Directorate at the

Defense Health Agency regarding integrated primary care treatment of military health system beneficiaries. The present study evaluated penetration of the PCBH model; change in health behaviors across treatment groups; and the differential impact of tobacco use, obesity, depression and cardiovascular disease on global functioning in the PCBH model. The goal of the present research was to examine the effectiveness of the PCBH model to improve interventions targeting health behaviors and depression so that CVD-related morbidity and mortality are reduced.

AIMS AND HYPOTHESES

Specific Aim 1

To assess the penetration of behavioral health consultants in addressing health behavior change, mental health intervention, and chronic disease management.

Hypothesis 1a

Among tobacco users seen in a primary care clinic that has a behavioral health consultant, more than 5% have been seen by a BHC for tobacco cessation (129).

Hypothesis 1b

Among those with obesity seen in a primary care clinic that has a behavioral health consultant, more than 5% have been seen by a BHC for weight management (109).

Hypothesis 1c

Among those with depression seen in a primary care clinic that has a behavioral health consultant, 25% have been seen by a BHC for mental health intervention.

Hypothesis 1d

Among those with cardiovascular disease seen in a primary care clinic that has a behavioral health consultant, more than 5% have been seen by a BHC for chronic disease management.

Specific Aim 2

To examine the difference in global functioning, as measured by the BHM-20 GMH scale, between individuals with different severities of cardiovascular disease and the interaction effect of tobacco use, obesity, and depression.

Hypothesis 2a

The main effect of cardiovascular disease on global functioning will demonstrate a significant difference between cardiac groups, with major adverse cardiac events adversely impacting global functioning the greatest.

Hypothesis 2b

Among those with varying severities of cardiovascular disease, the relationship between CVD and global functioning will be different for smokers and nonsmokers as demonstrated by the interaction effect between smoking status and cardiovascular disease severity.

Hypothesis 2c

Among those with varying severities of cardiovascular disease, the relationship between CVD and global functioning will be different for obese and non-obese individuals as demonstrated by the interaction effect between obesity and cardiovascular disease.

Hypothesis 2d

Among those with varying severities of cardiovascular disease, the relationship between CVD and global functioning will be different for depressed and nondepressed individuals as demonstrated by the interaction effect between depression and cardiovascular disease.

Specific Aim 3

To examine the impact of behavioral health consultants on health behavior engagement, as measured by BMI and reported tobacco use, and cardiovascular health, as measured by mean arterial blood pressure, among those with cardiovascular disease, depression, obesity, and tobacco use disorder.

Hypothesis 3a

Among those diagnosed with depression, obesity, tobacco use disorder, and cardiovascular disease, tobacco use will improve more for those seen by all treatment settings followed by primary care with a behavioral health consultant, primary care with specialty behavioral health, and primary care alone.

Hypothesis 3b

Among those diagnosed with depression, obesity, tobacco use disorder, and cardiovascular disease, BMI will improve more for those seen by all treatment settings followed by primary care with a behavioral health consultant, primary care with specialty behavioral health, and primary care alone.

Hypothesis 3c

Among those diagnosed with depression, obesity, tobacco use disorder, and cardiovascular disease, mean arterial blood pressure will improve more for those seen by

all treatment settings followed by primary care with a behavioral health consultant,
primary care with specialty behavioral health, and primary care alone.

CHAPTER 2: Method

RESEARCH DESIGN

The overall objective of this study was to evaluate the effectiveness of BHCs to improve health behaviors in those diagnosed with cardiovascular disease and depression. To do so, this study analyzed data previously collected by Psychological Health Center of Excellence (PHCoE) of the J-9 Directorate at the Defense Health Agency regarding integrated primary care treatment of military health system beneficiaries. The current study used a pretest-posttest quasi-experimental design to examine the change in health outcomes across treatment in a traditional treatment setting (i.e., primary care or specialty behavioral health) or the PCBH model among individuals diagnosed with tobacco use disorder, obesity, depression, or cardiovascular disease. This study also used a cross sectional design to examine the penetration of the PCBH model in providing intervention for health behaviors and the differential impact of tobacco use, obesity, depression and cardiovascular disease on global functioning in the monitored period.

PARTICIPANTS AND RECRUITMENT

The study used existing data provided by the PHCoE of the J-9 Directorate, Research and Development at the Defense Health Agency. These data included beneficiaries of the military health system diagnosed with cardiovascular disease, depression, obesity, or tobacco use disorder who received care from primary care clinics containing behavioral health consultants (BHC) within the Army, Navy, Air Force, and National Capital Region Medical Directorate (NCR-MD). The diagnoses were identified through medical encounter records, which capture recorded diagnoses in accordance with the diagnostic criteria provided in the International Classification of Diseases – 10th

revision (ICD-10) for patients seen from January 2017, through December 2019. The procedures for this study were approved by the USUHS Institutional Review Board.

The inclusion criteria for each aim differed to include the subsample of the population with the appropriated measures to conduct the target analyses. For all aims, individuals were required to be seen in the aforementioned period in the military healthcare system. All individuals were also required to be at least 18 years of age. For the first aim, inclusion criteria for the current sample required at least one primary care visit and access to a BHC at the time of the primary care encounter. The total sample for Aim 1 was 1,321,072 unique individuals; however, on individuals with the target diagnosis were included in calculating the penetration rate described below. For Aim 2, individuals for the current study only met inclusion criteria with at least one BHC visit in the 3-year monitored period and no missing BHM-20 GMH score data. A healthy control (i.e., no CVD or modifiable risk factors) was included to improve the interpretability and comparability of each effect. The total sample for Aim 2 was 112,137 unique individuals. Given the inclusion of those without any of the target diagnoses, the total sample of individuals seen by a BHC was greater for Aim 2 than those capture in the penetration rates of Aim 1. For Aim 3, individuals were required to have at least two primary care appointments for a pre- and post-test measure as well as the presence of vitals data outlined in more detail below. The total sample for Aim 3a was 298,394 unique individuals and for Aim 3b and 3c was 461,909 unique individuals. The difference in these sample sizes was attributed to missing data for the tobacco use vitals data. All sample characteristics are described in more detail below.

PROCEDURES

The two processes to be defined for this study include expected practice of BHCs and data extraction. The practice of the BHCs was inferred from the identified presenting problem in the medical record. Data extraction involved procedures with the Military Health System Management and Reporting Tool (M2) and Air Force Corporate Health Information Processing Service (AFCHIPS) databases.

Behavioral Health Consultant Procedures

Behavioral health consultants (BHC) in the military medical system most commonly include licensed clinical social workers and doctorate level clinical psychologists. Based on BHC competencies (110; 132) and standards published by the services, we expect the following were the common procedures for most BHC encounters. The primary care team diagnosed the patients with tobacco use disorder, obesity, depression, or cardiovascular disease. Patients were then referred to the BHC for behavioral health consultation by their PCP or another medical provider in the clinic. Diagnosing practices were guided by the International Classification of Diseases and Related Health Problems, 10th revision (ICD-10). As a component of standard practice, vitals are recorded including measures such as body mass index (BMI), tobacco use, and blood pressure. Additionally, patients referred to the BHC are required to complete a Behavioral Health Measure - 20 (BHM-20) (85) prior to the behavioral health consultation – a measure that takes less than 2 minutes to complete.

Although the design of the current study does not make it feasible to know exactly what interventions were implemented, BHCs within the military healthcare system are expected to use brief, targeted, evidence-based or evidence-informed interventions to improve patient functioning. Such interventions are summarized by Hunter and

colleagues (68). Standard transdiagnostic interventions for improving functioning and symptomatology included interventions such as “relaxation, mindfulness exercises, goal setting, cognitive disputation, motivational interviewing strategies, problem solving, self-monitoring, behavioral self-analysis, stimulus control, and assertive communication” (68)(p.27). Interventions specific to tobacco use include preparation for tobacco cessation (i.e., picking a date and preparing to enact their plan), the four A’s of quitting (i.e., avoid, alter, use alternatives, and stay active to reduce cues and urges for smoking), and maintaining cessation. For obesity, BHCs are likely to recommend sustainable lifestyle changes that increase physical activity, improve diet, and provide education on behavior change skills (i.e., goal setting, enacting behavior change, diet planning, and tracking with food/activity logs). For depression, interventions likely included psychoeducation on the interaction of behavioral, cognitive, and physiological changes that maintain depression, behavioral activation, cognitive restructuring, problem solving, and discussing antidepressants with their PCP. For cardiovascular disease, behavioral health consultants are recommended to target the aforementioned modifiable risk factors – tobacco use, obesity, and depression (68).

Data Extraction Procedures

The data extraction procedures for this study included aggregation of data from two independent sources collected as part of standard health care practices over a three-year period. The first included aggregate data from the Military Health System Management and Reporting Tool (M2) database. The latter included more granular data from the Air Force Corporate Health Information Processing Service (AFCHIPS).

M2 Database

For Aims 1 and 3, the PHCoE J-9 Directorate provided data from the M2 database, a data management and reporting tool used for regular analysis of military health systems. The data was comprised of administrative health information and procedures collected as part of regular military health care practice. The M2 included data from standard military electronic recording systems such as the Defense Enrollment Eligibility Reporting System (DEERS) and the Military Health System Data Repository (MDR), which provide data for routine performance and program monitoring. These data were extracted using M2 with no patient identifiers to ensure security and privacy of military health system beneficiaries. M2 data extracts included administrative data (patient demographics, diagnostic and procedure codes, treatment location, etc.) from BHC encounters occurring in the direct-care outpatient setting (Comprehensive Ambulatory Patient Encounter Record [CAPER] Detail Table) as well as measurements of vital signs (e.g., blood pressure, weight, BMI, etc.; Vitals Table). Data were aggregated into a final database with a multifaceted filtration system, simultaneously identifying flags from the full data set for all inclusion criteria. For Aim 1, individuals for the current study met inclusion criteria with at least one primary care visit and access to a BHC. For Aim 3, individuals were required to have at least two primary care appointments for a pre- and post-test measure as well as the presence of vitals data outlined in more detail below. All individuals were required to be at least 18 years old.

AFCHIPS Database

For Aim 2, the PHCoE also provided data from extractions that the AFCHIPS already routinely obtained from the Defense Health Agency, J-9 Directorate under the terms of DSA 16-1617. These data included both administrative and clinical health

information collected as part of regular BHC practice; that is, these data were collected from notes recorded by BHC's following encounters in the PCBH model. The data is typically utilized for routine performance and program monitoring; therefore, the supplied extracts contained no patient identifiers. PHCoE transferred the data for analysis via encrypted electronic mail. To ensure security and privacy of the data during transfer and while it is being analyzed, all data were stored in a password-protected file on a password-protected external hard drive. Again, data were aggregated into a final database with a multifaceted filtration system, simultaneously identifying flags from the full data set for all inclusion criteria. Individuals for the current study only met inclusion criteria with at least one BHC visit in the 3-year monitored period and no missing BHM-20 GMH score data. A healthy control (i.e., no CVD or modifiable risk factors) was included to improve the interpretability and comparability of each effect. All individuals were required to be at least 18 years old.

Variable Coding and Cardiac Grouping

Data provided by the PHCoE included raw ICD-10 diagnostic codes recorded in medical record vitals as part of routine medical care. These data required grouping into cardiac groups by diagnostic codes prior to analysis. These cardiac groupings provided an opportunity to observe any differential impact of the PCBH model on CVD through modifiable risk factors at different severities of disease. Data were sorted in Microsoft Excel and categorized into three cardiac groupings: hypertensive heart disease, atherosclerotic and other cardiovascular disease, and major adverse cardiac events. Specific ICD-10 codes and diagnoses for each cardiac grouping are outlined in Table 1.

MEASURES

Medical Record Vitals

Medical record vitals include a standard set of metrics collected at each primary care appointment and recorded by a trained part of the primary care team. This study drew on select measures from medical record vitals including body mass index (BMI), tobacco use, blood pressure, and ICD-10 diagnostic codes.

Body Mass Index

BMI was obtained from a calculated height-to-weight ratio recorded as a part of medical record vitals. Specifically, BMI is calculated as kilograms of body weight per meters squared of height. Height and weight were measured at each primary care appointment using a stadiometer and calibrated scale.

Tobacco Use

Tobacco use was measured as a binary variable (use/non-use) recorded as a part of medical record vitals given the nonlinear dose-response relationship between tobacco use and CVD (126). The presence of reported tobacco use is acquired as part of standard medical screening at each primary care appointment.

Blood Pressure

Blood pressure consists of a systolic and diastolic reading typically captured in primary care using a calibrated automated sphygmomanometer. Blood pressure was measured using mean arterial pressure (MAP), which provided an average pressure on the arteries and is considered the best predictor of hypertensive precipitants to adverse cardiovascular outcomes (170). MAP was calculated as double the diastolic blood pressure, added with the systolic blood pressure, and divided by 3 (i.e., $MAP =$

$\frac{SBP+2DBP}{3}$). This provided a single continuous variable for the average arterial blood pressure.

ICD-10 Diagnostic Codes

Each encounter included the ICD-10 diagnostic codes associated with the individual's presenting problem and treatment plan. Diseases of the circulatory system will be classified into cardiac groups outlined in Table 1. All modifiable risk factors were also confirmed as relevant medical conditions using ICD-10 codes. Depressive disorders (i.e., F32.0-F34.1) were categorized as the presence of depressive symptoms. Although BMI was calculated for each individual, the presence of ICD-10 codes for obesity (i.e., E66.01-E66.9) were used to confirm individual encounters or consultations were related to obesity. Similarly, although vitals collected current reported tobacco use, the presence of ICD-10 codes for tobacco use (i.e., F172.00-F172.99; O993.30-O993.35; T652.11-T652.94; Z71.6; Z72.0) were used to confirm the individual encounters were related to tobacco use. ICD-10 diagnostic codes tagged for each diagnostic subgroup were extracted from encounters with primary care provider; however, the origins of the diagnoses (i.e., primary versus specialty care medical provider) was not traced. Additionally, ICD-10 diagnostic codes were extracted from BHC encounters for Aim 1 to determine the specific penetration rate (i.e., the rate individuals were seen by the BHC for the same target diagnosis identified by the primary care provider).

Medications

Participant medications were also included as two binary covariates for cardiovascular medications and antidepressant medications for their direct

pharmacological impact on CVD and depression. Participant active medications were extracted as part of standard vitals aggregated as American Hospital Formulary Service (AHFS) codes. Cardiovascular medications included AHFS codes 12:00.00-24:32.40; 28:12.04; 28:12.08; 40:18.17-40:28.92; 52:32.00-52:40.08. Although 24:00.00 (i.e., “Cardiovascular Drugs”) is the generic coding for formulary pharmaceuticals considered first line for cardiovascular medications, the selected range was expanded to include any medication typically prescribed for primary or secondary cardiovascular effects. Antidepressant medications included AHFS codes 28:16.00-28:20.00; 28:24.04-28:36.20. Similarly, although 28:16.04 is the generic coding for antidepressants, the full range of flagged medications was selected to include a full range of medications typically prescribed for antidepressant effects.

Behavior Health Measure – 20 (BHM-20)

Global functioning was measured using the Global Mental Health scale of the BHM-20, a 20-item self-report measure of general psychological distress and functioning intended for repeated measure to track outpatient mental health outcomes (85). This measure is broken down into three domains that are consistent with the phase model of psychotherapy outcomes: Well-Being (3 items), Psychological Symptoms (13 items), and Life Functioning (4 items) (84; 85). The items are scored on a 5-point (0 – 4) Likert Scale, where higher scores indicate higher levels of functioning; the BHM-20 GMH score is an average of all 20 items ranging from 0 to 4. Items targeting distress include questions such as “In the past two weeks, how distressed have you been with your life?” rated from Extremely Distressed to Not at all distressed. Other items targeting functioning ask, “How have you been getting along in the following areas of your life

over the past two weeks?” with social, recreational, work, and intimate relationships rated from terrible to very well. Recommended clinical cutoff scores include *Severe* (0 to 2.08), *Moderate* (2.08 to 2.37), *Mild* (2.38 to 2.93), and *Normal* (2.94 to 4.00). This measure is administered electronically and takes approximately 90 seconds to complete (84). BMH-20 shows good levels of internal consistency (0.89 to 0.90), sensitivity to pathology and change, construct validity, and concurrent validity when measuring global functioning (85). Additionally, the measure has been shown to maintain validity with diverse samples in the primary care setting (24).

CHAPTER 3: Data Analyses

DATA ANALYSES

Data analyses were completed using SAS Version 9.4. All data were examined using descriptive statistics to identify outliers, adherence to assumptions associated with each planned data analysis, and fidelity to expected appointment characteristics in the PCBH model. Extreme outliers to the expected appointment number in the PCBH model (i.e., greater than 9 appointments) were removed, as these cases limited generalizability to the model. Additionally, individuals with only one appointment or missing data were excluded from inferential statistical analyses in aims 2 and 3. Covariates in all inferential analyses included cardiovascular medication, antidepressant medication, age, and sex. Cardiovascular conditions were categorized as hypertensive heart disease, atherosclerotic or other cardiovascular disease, and major adverse cardiac events as outlined in the cardiac groupings below (Table 1).

Aim 1

Specific Aim 1: To assess the penetration of behavioral health consultants in addressing health behavior change, mental health intervention, and chronic disease management.

The first aim was analyzed by computing the rate of individuals seen in primary care who are targeted for a relevant health behavior, mental health intervention, or chronic disease management. Specifically, the analysis identified the number of unique individuals seen by a behavioral health consultant of those seen in a primary care clinic with a chief complaint of tobacco use disorder, obesity, or depression (Figure 1).

Additionally, an exploratory analysis described the consultation questions for those seen by BHCs with a CVD diagnosis. This aim used the M2 database extraction for analysis.

Aim 2

Specific Aim 2: To explore the difference in global functioning, as measured by the BHM-20 GMH scale, between individuals with differences of reported engagement in health behaviors, as measured by BMI and tobacco use, and among those with cardiovascular disease, depression, or comorbid conditions.

Hypothesis 2: A 4x2x2x2 Analysis of Covariance (ANCOVA) was conducted to assess differences between global functioning after controlling for the effects of age, sex, and medication. The covariates for all analyses of age, sex, and medication are chosen for their established moderating effect on CVD and depression (102; 111). The higher order interactions (i.e., 3-way, 4-way interactions) were not the focus on the present study, nor were they supported by the conceptual model; however, the potential for shared variance between two-way interactions suggested the use of a single model would better account for covariance between interaction terms.

In this analysis, differences in global functioning were compared by the main and interaction effects of different severities of cardiovascular disease with the presence of tobacco use, obesity, or depression. First, a main effect for each variable on global functioning was analyzed, with particular emphasis on the main effect of cardiovascular disease, a four-level variable (control, hypertensive heart disease, atherosclerotic and other cardiovascular disease, and major adverse cardiac event; *hypothesis 2a*). Then, the interaction effects of cardiovascular disease and tobacco use (control versus tobacco use; *hypothesis 2b*), cardiovascular disease and obesity, (healthy weight versus obesity;

hypothesis 2c), and cardiovascular disease and depression (control versus depression; *hypothesis 2d*) were analyzed for the hypothesized moderation effects of each modifiable risk factor. Age was included as an ordinal variable, aggregated into age groups (i.e., 18-24, 25-34, 35-44, 45-64, 65+) consistent with standard data extraction from the M2 database to reduce identifiable information. Sex (male versus female), cardiovascular medication (none versus active), and antidepressant medication (none versus active) were each included as binary covariates. This aim used the AFCHIPS database extraction for analysis. The purpose was to parse-out the effects of CVD and modifiable risk factors on global functioning to determine if a main effect of CVD is moderated by the presence of poor health behaviors and depression. Figure 2 provides a conceptual model of this analysis.

Aim 3

Specific Aim 3: To examine the impact of behavioral health consultants on health behavior engagement, as measured by BMI and reported tobacco use, and cardiovascular health, as measured by blood pressure, among those with cardiovascular disease, depression, or comorbid conditions.

For Aim 3, a series of three one-way Analyses of Covariance (ANCOVA) were conducted to assess the difference between treatment groups in health outcomes after controlling for time, age, sex, and medication. Each ANCOVA utilized the same 4-level independent variable of treatment group (primary care [PC] only, PC and PCBH, PC and specialty behavioral health, and a combination of all three treatment types) with different outcome variables including tobacco use (*hypothesis 3a*), BMI (*hypothesis 3b*), and MAP (*hypothesis 3c*). Each outcome variable was calculated as a delta between the first and

last appointment such that negative deltas would demonstrate a reduction in reported tobacco use, BMI, or MAP. Time was included as a continuous variable, measuring total number of days between the first and last appointment. Age was included as an ordinal variable, aggregated into age groups (i.e., 18-24, 25-34, 35-44, 45-64, 65+) consistent with standard data extraction from the M2 database to reduce identifiable information. Sex (male versus female), cardiovascular medication (none versus active), and antidepressant medication (none versus active) were each included as binary covariates. Age and sex were controlled for their established moderating effect on CVD and depression (102; 111). This aim used the M2 database extraction for analysis. Figure 3 provides a conceptual model for this analysis.

POWER

The data set extracted for this study contained greater than 112,000 unique military health care beneficiaries for any given analysis after removal for all exclusion criteria. The analysis to be conducted for Aim 1 is a descriptive rate of penetration, rather than inferential statistical analysis; therefore, the sample size is considered adequately powered for this aim. A priori power analysis for Aims 2 and 3 were conducted using G*Power 3.1 to determine the sample size required to adequately power the study (45). A power analysis for a 4x2x2x2 ANCOVA was conducted, as the most complicated model to be analyzed, using a small effect size (Cohen's $f = 0.1$), power of 80%, and alpha level of 0.05. For the interaction model, there were 8 unique groups (numerator degrees of freedom = 3) and 4 covariates. Based on the power analysis, this study only required 1,396 unique individuals and was overpowered.

CHAPTER 4: Results

AIM 1: PENETRATION OF BEHAVIORAL HEALTH CONSULTANTS

An exploratory analysis was conducted to identify the penetration of BHCs in addressing health behavior change, mental health intervention, and chronic disease management. Specifically, the exploratory analysis identified the number of unique individuals seen in primary care for tobacco use, obesity, major depressive disorder, and CVD; from those seen in primary care for the target diagnoses, BHC penetration was characterized as the percentage of individuals treated under the target diagnosis. All exploratory analyses for this aim are represented in Table 3.

Aim 1 Sample Characteristics

For this aim, a total of 1,321,072 unique individuals were analyzed as part of a routine data extraction from the M2 database for patients seen by primary care from January 2017 to December 2019. This sample included individuals that were predominately married (40.62%), male (56.22%), military dependents (40.91%), between 45 and 64 years old (30.93%). A substantial portion of the analyzed sample did include active duty or activated reservists (37.06%). Participants were seen in Army (42.93%), Navy (21.85%), and Air Force (30.34%) operated clinics. Additional demographic data for the sample is provided in Table 2.

The rates of diagnoses in the analyzed sample were largely consistent with reported rates in the United States general and military population. Tobacco use in the current population was diagnosed at 14.58%, which is consistent with the downward trend projected from the most recent rate of tobacco use in the military estimated around 16% in 2014 (113). The rate of diagnosed obesity in the current sample (19.44%) was

more similar to the military population (23.6%; 119) than the national reported rate of obesity (39.8%; 25; 26). The rate of depression in the current sample (11.78%) was also similar to that in the general and military populations (9.4%) (78; 113). Finally, rates of diagnosed CVD were similar to those observed in the population with hypertensive heart disease [35.29% observed; 33% base rate (156)] and atherosclerotic cardiovascular disease [13.76% observed; 15% base rate (55)] each mirroring the general population. Although there are not many good estimates for the lifetime prevalence for a living population with any history of MACE, the observed rate of 13.19% is similar to the 4-year incidence of MACE estimated around 11.5% (114).

Aim 1a: Tobacco Use Penetration in PCBH

For tobacco use, 192,566 unique individuals were seen in a primary care setting with a diagnosis of tobacco use in the analyzed three-year period. Of those seen in primary care for tobacco use, 8,810 (4.58%) were treated by a BHC related to their tobacco use diagnosis. An additional 1,476 individuals with tobacco use were treated by BHCs for diagnoses unrelated to their tobacco use. Of the 10,286 individuals with a tobacco use diagnosis seen by a BHC, 85.65% were treated for their tobacco use. Therefore, the total penetration of BHCs for individuals in primary care with tobacco use is 5.34%, with a specific penetration rate of 4.58% addressing health behavior change related to tobacco use.

Aim 1b: Obesity Penetration in PCBH

For obesity, 256,852 unique individuals were seen in a primary care setting with a diagnosis of obesity in the analyzed three-year period. Of those seen in primary care for obesity, 5,717 (2.23%) were treated by a BHC related to their obesity diagnosis. An

additional 3,048 individuals with obesity were seen by BHCs for diagnoses unrelated to their weight. Of the 8,765 individuals with an obesity diagnosis seen by a BHC, 65.23% were treated to address weight-related concerns. Therefore, the total penetration of BHCs for individuals in primary care with obesity is 3.41%, with a specific penetration rate of 2.23% addressing health behavior change related to obesity.

Aim 1c: Depression Penetration in PCBH

For major depressive disorder, 155,656 unique individuals were seen in a primary care setting with a diagnosis of depression in the analyzed three-year period. Of those seen in primary care for depression, 30,363 (19.51%) were treated by a BHC related to their major depressive disorder diagnosis. An additional 762 individuals with depression were treated by BHCs for diagnoses unrelated to depression. Of the 31,125 individuals with a major depressive disorder diagnosis seen by a BHC, 97.55% were seen to address their depressive disorder. Therefore, the total penetration of BHCs for individuals in primary care with depression is 20.00%, with a specific penetration rate of 19.51% addressing mental health intervention related to major depressive disorder.

Aim 1d: CVD Penetration in PCBH

For CVD, 583,659 unique individuals were seen in a primary care setting with a CVD diagnosis in the analyzed three-year period. Of those seen in primary care for CVD, 1,090 (0.19%) were treated by a BHC related to their CVD. An additional 7,487 individuals with CVD were treated by BHCs for diagnoses unrelated to their cardiovascular health. Of the 8,577 individuals with a CVD diagnosis seen by a BHC, 12.71% were seen to directly address cardiovascular health concerns. Therefore, the total

penetration of BHCs for individuals in primary care with CVD is 1.47%, with a specific penetration rate of 0.19% addressing chronic disease management related to CVD.

The three subgroups of CVD severity demonstrated similar rates of penetration. Of the 466,167 individuals seen in primary care for hypertensive heart disease, 963 (0.21%) were seen by a BHC related to their CVD; 6,234 individuals with hypertensive heart disease were seen by BHCs for diagnoses unrelated to CVD. The total penetration of BHCs for individuals in primary care with hypertensive heart disease is 1.54%, with a specific penetration rate of 0.21% addressing chronic disease management related to hypertensive heart disease. Of the 181,795 individuals seen in primary care for atherosclerotic and other cardiovascular disease, 107 (0.06%) were seen by a BHC related to their CVD; 2,125 individuals with atherosclerotic and other cardiovascular disease were seen by BHCs for diagnoses unrelated to CVD. The total penetration of BHCs for individuals in primary care with atherosclerotic and other cardiovascular disease is 1.23%, with a specific penetration rate of 0.06% addressing chronic disease management related to atherosclerotic and other cardiovascular disease. Of the 17,426 individuals seen in primary care for major adverse cardiac events, 28 (0.16%) were seen by a BHC related to their CVD; 227 individuals with major adverse cardiac events were seen by BHCs for diagnoses unrelated to CVD. The total penetration of BHCs for individuals in primary care with major adverse cardiac events is 1.46%, with a specific penetration rate of 0.16% addressing chronic disease management related to major adverse cardiac event.

AIM 2: RELATIONSHIP BETWEEN CVD SEVERITY AND GLOBAL FUNCTIONING

A 4x2x2x2 factorial ANCOVA was calculated between 4 levels of CVD severity (control, hypertensive heart disease, atherosclerotic and other cardiovascular disease, and major adverse cardiac event), 2 levels of tobacco use (denied tobacco use versus endorsed tobacco use), 2 levels of obesity (healthy weight versus obesity), and 2 levels of depression (control versus depression diagnosis) to determine if there was a difference in global functioning measured by the BHM-20 GMH score. For simplicity, please note that all following analyses controlled for age, sex, and medication. Additionally, the data was tested for assumptions including normality of distribution, homogeneity of variance, and independence of covariates; all assumptions were met by the data for this aim. The highest-level effects of interest for this analysis were two-way interactions, evaluating the hypothesized moderation of the relationship between CVD severity and global functioning by tobacco use, obesity, and depression. However, all variables were analyzed in a single model to account for any shared variance in these moderating effects; that is, the presence of tobacco use, obesity, and depression were each hypothesized to demonstrate moderating effects on the relationship between CVD and global functioning and therefore required analysis in a single model.

Aim 2 Sample Characteristics

For Aim 2, a total of 112,137 unique individuals were analyzed as part of a routine data extraction from the AFCHIPS database for patients seen by primary care from January 2017 to December 2019. This sample included individuals that were predominately married (40.62%), female (55.90%), military dependents (44.26%), between 25 and 34 years old (26.66%). A substantial portion of the analyzed sample again included active duty or activated reservists (43.36%). Participants were seen in

Army (38.73%), Navy (21.25%), and Air Force (36.28%) operated clinics. Additional demographic data for the sample is provided in Table 4. Diagnostically, the sample maintained expected rates of tobacco use [19.11% observed; 16% base rate (113)] obesity [27.97% observed; 23.6% base rate; (119)], hypertensive heart disease [22.62% observed; 33% base rate (156)] and atherosclerotic cardiovascular disease [14.37% observed; 15% base rate (55)]. However, there was an overrepresentation of depression in the current sample (40.40%) when compared with the expected base rate in the military population (9.4%) (78; 113). This is likely because part of the inclusion criteria for this aim was the presence of a BHM-20 Global Mental Health score, which is typically only administered in the context of treatment with a BHC. Conversely, the rates of lifetime diagnosis of MACE in the observed population (1.78%) did not meet the expected 4-year incidence (11.5%) (114). The underrepresentation of MACE in the observed population is likely due to the low penetration rate in BHC's addressing CVD and health behaviors identified in Aim 1; that is, individuals included in this aim were required to be seen by a BHC, which demonstrated more robust implementation for behavioral health concerns than health behaviors.

Also of note, most individuals were actively prescribed antidepressant (76.79%) or cardiovascular medication (84.95%). The rate of antidepressant medication prescription is likely inflated from the rates in the general population given the inclusion criteria requires an appointment with a BHC, isolating a clinical population with predominately behavioral health concerns. Additionally, the therapeutic classification list allots for the inclusion of medications with antidepressant effects (i.e., bupropion, doxepin, etc.) that are also used treating other common health concerns such as chronic

pain and smoking cessation (66; 139). The rate of CVD medications is less inflated, with typical rates for hypertensive medication reaching 68-70% in a primary care sample (179). Finally, the rates of prescription in the current sample also capture over-the-counter medications with anti-depressant or cardiovascular effects that are not typically recorded in civilian outpatient medical care.

Aim 2a: The effect of CVD on Global Functioning

The first hypothesis examined the main effect of cardiovascular disease on global functioning, hypothesizing that CVD severity would demonstrate a significant difference between cardiac groups, with major adverse cardiac events adversely impacting global functioning the greatest. However, there was no significant main effect of CVD severity ($F(3,112111) = 1.50, p = 0.212, \eta_p^2 < 0.001$) when controlling for all other variables.

Aim 2b: The effect of CVD and Tobacco Use on Global Functioning

The second hypothesis under the present aim posited that among those with varying severities of cardiovascular disease, smokers will experience lower global functioning than nonsmokers as demonstrated by the interaction effect between smoking status and cardiovascular disease severity. There was a significant two-way, cross-over interaction between CVD severity and tobacco use ($F(3,112111) = 10.62, p < 0.001, \eta_p^2 < 0.001$). Although there was no main effect of CVD, there was a very small significant main effect of tobacco use ($F(1,112111) = 10.55, p = 0.001, \eta_p^2 < 0.001$). For tobacco use, a post hoc Tukey test revealed that those who endorsed using tobacco ($M = 2.74$) reported statistically higher global functioning than those who reported no tobacco use ($M = 2.70, p = 0.001$). In this interaction, the main effect of tobacco use on global functioning is reversed at most severe level of CVD. Specifically, individuals with a

history of major adverse cardiac events reported worse functioning in smokers than non-smokers; this differs from all other levels of CVD. Further analysis of twenty-eight multiple pairwise comparisons revealed eight significantly different means in the interaction between CVD severity and tobacco use. The Tukey post hoc test revealed that nonsmokers without CVD (i.e., the healthy population; $M = 2.69$) reported significantly lower perceived global functioning than smokers without CVD ($M = 2.79$; $p < 0.001$), smokers with hypertensive heart disease ($M = 2.75$; $p < 0.001$), and smokers with atherosclerotic and other CVD ($M = 2.75$; $p = 0.001$). Conversely, smokers without CVD ($M = 2.79$) reported significantly higher perceived global functioning than nonsmokers with hypertensive heart disease ($M = 2.70$; $p < 0.001$), nonsmokers with atherosclerotic and other CVD ($M = 2.72$; $p < 0.001$), and nonsmokers with a history of MACE ($M = 2.70$; $p < 0.001$). Even among those with hypertensive heart disease, smoker ($M = 2.70$) reported significantly higher functioning than nonsmokers ($M = 2.75$; $p < 0.001$). Graphical representation of this aim can be found in Figure 4.

Aim 2c: The effect of CVD and Obesity on Global Functioning

The third hypothesis under the present aim posited that among those with varying severities of cardiovascular disease, obese individuals will experience lower global functioning than healthy weight individuals as demonstrated by the interaction effect between obesity and cardiovascular disease severity. However, there was no significant interaction between CVD severity and obesity ($F(3,112111) = 1.37$, $p = 0.250$, $\eta_p^2 < 0.001$). There was a very small significant main effect of obesity ($F(1,112111) = 18.42$, $p < 0.001$, $\eta_p^2 < 0.001$). A post hoc comparison of means revealed that those who met criteria for obesity ($M = 2.74$) reported statistically higher functioning than those who did

not met the criteria for obesity ($M = 2.70, p < 0.001$). Graphical representation of this aim can be found in Figure 5.

Aim 2d: The effect of CVD and Depression on Global Functioning

The final hypothesis under the second aim stated that among those with varying severities of cardiovascular disease, individuals with depression will experience lower global functioning than individuals without depression as demonstrated by the interaction effect between depression and cardiovascular disease severity. There was a significant interaction effect between CVD and depression ($F(3,112111) = 1.37, p = 0.027, \eta_p^2 < 0.001$). As mentioned above, the main effect of CVD severity was insignificant; however, there was a small significant main effect of depression ($F(1,112111) = 1903.71, p < 0.001, \eta_p^2 = 0.021$), with individuals without depression reporting higher global functioning ($M = 2.91$) than those with depression ($M = 2.53, p < 0.001$). A multiple pairwise comparisons revealed sixteen significantly different means in the interaction between CVD severity and depression. The Tukey post hoc test revealed that individuals without depression and CVD (i.e., the healthy population; $M = 2.93$) reported significantly higher perceived global functioning than those with depression without CVD ($M = 2.55; p < 0.001$), with depression and hypertensive heart disease ($M = 2.53; p < 0.001$), with depression and atherosclerotic and other CVD ($M = 2.53; p = 0.001$) and with depression and a history of MACE ($M = 2.51; p = 0.001$). In fact, individuals without depression reported significantly higher functioning than those with depression regardless of CVD condition ($p = 0.001$), accounting for all sixteen significantly different means. Graphical representation of this aim can be found in Figure 6.

Regarding covariates, there was a large significant main effect for depression medication ($F(1,112111) = 1757.79, p < 0.001, \eta_p^2 = 0.015$), very small effect for cardiovascular medication ($F(1,112111) = 4.39, p = 0.036, \eta_p^2 < 0.001$), small effect for age group ($F(7,112111) = 155.95, p < 0.001, \eta_p^2 = 0.010$), and very small effect for sex ($F(1,112111) = 434.29, p < 0.001, \eta_p^2 = 0.004$). A summary of the factorial ANCOVA can be found in Table 5.

AIM 3: IMPACT OF TREATMENT GROUP ON HEALTH OUTCOMES

A series of analyses of covariance were conducted to determine the extent to which treatment group predicted health behavior change including reported tobacco use (*hypothesis 3a*), BMI (*hypothesis 3b*), and mean arterial blood pressure (*hypothesis 3c*) when controlling for age, gender, and time. The three one-way ANCOVAs were calculated between 4 levels of treatment group (primary care [PC] only, PC and PCBH, PC and specialty behavioral health, and a combination of all three treatment types). All analyses controlled for age, sex, baseline, and time. Additionally, the data was tested for assumptions including normality of distribution, homogeneity of variance, and independence of covariates; all assumptions were met by the data for this aim. These analyses are summarized in Table 8.

Aim 3 Sample Characteristics

Although Aim 3 analyzed the same M2 database and time period (January 2017 to December 2019) as Aim 1, the required vital data reduced the analyzed sample to 461,909 unique individuals. The sample was further reduced for Aim 3a, which required reported tobacco use and was only available in 298,394 unique individuals. Both samples

included individuals that were predominately married (Aim 3a: 51.67%; Aim 3b,c: 48.09%), male (Aim 3a: 54.51%; Aim 3b,c: 54.04%), military dependents (Aim 3a: 39.51%; Aim 3b,c: 41.20%), between 45 and 64 years old (Aim 3a: 36.90%; Aim 3b,c: 37.04%). Again, a substantial portion of the analyzed sample included active duty or activated reservists (Aim 3a: 32.61%; Aim 3b,c: 31.34%). For Aim 3a, participants were seen with expected distribution in Army (46.90%), Navy (22.39%), and Air Force (24.73%) operated clinics. However, for Aims 3b and 3c, the Navy (16.93%) accounted for a disproportionately smaller number of the encounters than the Army (43.36%) or Air Force (35.22%). Finally, individuals predominately presented to primary care only (Aim 3a: 65.18%; Aim 3b,c: 65.07%) followed by PC and Specialty BH (Aim 3a: 18.39%; Aim 3b,c: 17.60%), PC and PCBH (Aim 3a: 8.77%; Aim 3b,c: 9.33%), or all three treatment groups (Aim 3a: 7.65%; Aim 3b,c: 8.00%). Additional demographic data for the sample is provided in Table 6 and Table 7 for Aims 3a and 3b,c, respectively.

Aim 3a: Impact of Treatment Group on Tobacco Use

There was a significant main effect of treatment group on tobacco use ($F(3,298380) = 32.62, p < 0.001, \eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline tobacco use. In a post hoc Tukey test of estimated marginal means, individuals seen by PC and PCBH reported the significantly greater reduction in reported tobacco use at the final reported primary care appointment ($M = -0.017$) than those seen by PC alone ($M = -0.012, p = 0.050$), PC and specialty mental health ($M = 0.001, p < 0.001$), or all three treatment settings ($M = -0.009, p = 0.017$). Conversely, those seen by PC and specialty behavioral health reported significantly less change in reported tobacco use at the final recorded primary care appointment ($M = 0.001$) than those seen by PC alone (M

= -0.012, $p < 0.001$) or all three treatment settings ($M = -0.009$, $p < 0.001$). Put differently, treatment in PC and PCBH demonstrated a significantly greater reduction in reported tobacco use than any other treatment setting, with PC and specialty mental health demonstrating a mean increase in reported tobacco use. Regarding covariates, there was a significant main effect for baseline tobacco use ($F(1,298380) = 88539.4$, $p < 0.001$, $\eta_p^2 = 0.229$), age group ($F(7,298380) = 926.81$, $p < 0.001$, $\eta_p^2 = 0.021$), sex ($F(1,298380) = 1902.53$, $p < 0.001$, $\eta_p^2 = 0.006$), and time ($F(1,298380) = 434.29$, $p < 0.001$, $\eta_p^2 < 0.001$). A graphic representation of this aim is available in Figure 7.

In a post hoc analysis of treatment group on tobacco use, additional ANCOVAs were computed using an identical model but with populations isolated from the original sample ($N = 298,394$) for identified presence of CVD ($N = 186,371$; 62.46% of Aim 3a sample), tobacco use ($N = 41,753$; 13.99% of Aim 3a sample), obesity ($N = 47,949$; 16.07% of Aim 3a sample), and depression ($N = 39,841$; 13.35% of Aim 3a sample). For additional information on these post hoc analyses, see Table 9.

For the subgroup with identified CVD, there was a significant main effect of treatment group on tobacco use ($F(3,186357) = 5.76$, $p = 0.001$, $\eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline tobacco use. PC alone ($M = -0.011$) demonstrated a significantly greater reduction in tobacco use than all three treatment settings ($M = 0.000$; $p = 0.001$). All other comparisons were insignificant.

For the subgroup with identified tobacco use, there was a significant main effect of treatment group on tobacco use ($F(3,41739) = 8.46$, $p < 0.001$, $\eta_p^2 = 0.001$) when controlling for age, sex, time, and baseline tobacco use. PC and PCBH ($M = -0.147$) demonstrated significantly greater reduction in tobacco use than PC alone ($M = -0.117$, p

= 0.001). Additionally, the condition with all three treatment settings ($M = -0.146$) demonstrated significantly greater reduction in tobacco use than PC alone ($M = -0.117, p < 0.001$) or PC and specialty behavioral health ($M = -0.126, p = 0.033$).

For the subgroup with identified obesity, there was a significant main effect of treatment group on tobacco use ($F(3,47935) = 8.93, p < 0.001, \eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline tobacco use. PC alone ($M = -0.022$) demonstrated a significantly greater reduction in tobacco use than PC and specialty mental health ($M = -0.007, p < 0.001$). All other comparisons were insignificant.

For the subgroup with identified depression, there was a significant main effect of treatment group on tobacco use ($F(3,39828) = 16.00, p < 0.001, \eta_p^2 = 0.001$) when controlling for age, sex, time, and baseline tobacco use. PC alone ($M = -0.011$) and PC and PCBH ($M = -0.017$) demonstrated significantly greater reductions in tobacco use than PC and specialty mental health ($M = 0.011; p < 0.001$ for each) or the condition with all three treatment settings ($M = 0.005; p < 0.001$ for each).

Aim 3b: Impact of Treatment Group on BMI

There was a significant main effect of treatment group on BMI ($F(3,461894) = 23.00, p < 0.001, \eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline BMI. The post hoc comparison of estimated marginal means revealed individuals seen by PC and specialty behavioral health reported the significantly greater reduction in BMI at the final reported primary care appointment ($M = -3.008$) than those seen by PC alone ($M = -2.388, p < 0.001$) or PC and PCBH ($M = -2.397, p < 0.001$). Similarly, those seen by all three treatment settings reported greater reduction in BMI at the final reported primary care appointment ($M = -2.740$) than those seen by PC alone ($M = -2.388, p = 0.005$) or

PC and PCBH ($M = -2.397, p = 0.043$). In other words, treatment in PC and specialty behavioral health or all three treatment settings demonstrated a significantly greater reduction in BMI than PC alone or PC and PCBH. Regarding covariates, there was a significant main effect for baseline BMI ($F(1,461894) = 299800000, p < 0.001, \eta_p^2 = 0.998$), age group ($F(7, 461894) = 461.15, p < 0.001, \eta_p^2 = 0.007$), and time ($F(1,461894) = 39.82, p < 0.001, \eta_p^2 < 0.001$). A graphic representation of this aim is available in Figure 8.

In a post hoc analysis of treatment group on BMI, additional ANCOVAs were computed using an identical model but with populations isolated from the original sample ($N = 461,909$) for identified presence of CVD ($N = 284,078$; 61.50% of Aim 3b sample), tobacco use ($N = 58,452$; 12.65% of Aim 3b sample), obesity ($N = 76,884$; 16.64% of Aim 3b sample), and depression ($N = 65,825$; 14.25% of Aim 3b sample). For additional information on these post hoc analyses, see Table 10.

For the subgroup with identified CVD, there was a significant main effect of treatment group on BMI ($F(3,284064) = 12.17, p < 0.001, \eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline BMI. PC and specialty mental health ($M = -5.122$) demonstrated a significantly greater reduction in BMI than PC alone ($M = -4.784; p = 0.003$), PC and PCBH ($M = -4.325; p < 0.001$), or all three treatment settings ($M = -4.503; p = 0.001$). PC alone also demonstrated significantly greater reduction in BMI than PC and PCBH ($M = -4.325; p < 0.001$).

For the subgroup with identified tobacco use, there was a significant main effect of treatment group on BMI ($F(3,58438) = 4.75, p = 0.003, \eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline BMI. PC alone ($M = -0.170$) demonstrated a significantly

greater reduction in BMI than the condition with all three treatment settings ($M = 0.076$; $p = 0.001$). All other comparisons were insignificant.

For the subgroup with identified obesity, there was a significant main effect of treatment group on BMI ($F(3,76870) = 5.10$, $p = 0.002$, $\eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline BMI. PC and specialty mental health ($M = -1.79$) demonstrated a significantly greater reduction in BMI than PC alone ($M = -0.872$; $p = 0.003$) or PC and PCBH ($M = -0.615$; $p = 0.010$).

For the subgroup with identified depression, there was no significant main effect of treatment group on BMI ($F(3,65811) = 0.92$, $p = 0.430$, $\eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline BMI. Additionally, there were no significant comparisons.

Aim 3c: Impact of Treatment Group on Mean Arterial Blood Pressure

There was a significant main effect of treatment group on MAP ($F(3,461895) = 92.91$, $p < 0.001$, $\eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline MAP. A post hoc analysis of estimated marginal means highlighted those individuals seen by PC and PCBH demonstrated a significantly greater reduction in MAP at the final reported primary care appointment ($M = -3.981$) than those seen by PC alone ($M = -3.332$, $p < 0.001$) or PC and specialty mental health ($M = -3.635$, $p < 0.001$). Conversely, those seen by PC alone demonstrated significantly less change in MAP at the final recorded primary care appointment ($M = -3.332$) than those seen by PC with specialty mental health ($M = -3.635$, $p < 0.001$) or all three treatment settings ($M = -4.001$, $p < 0.001$). Regarding covariates, there was a significant main effect for baseline MAP ($F(1,461895) = 233744$, $p < 0.001$, $\eta_p^2 = 0.336$), age group ($F(7,461895) = 1962.34$, $p < 0.001$, $\eta_p^2 = 0.029$), sex

($F(1,461895) = 1580.18, p < 0.001, \eta_p^2 = 0.003$), and time ($F(1,461895) = 90.61, p < 0.001, \eta_p^2 < 0.001$). A graphic representation of this aim is available in Figure 9.

In a post hoc analysis of treatment group on MAP, additional ANCOVAs were computed using an identical model but with populations isolated from the original sample ($N = 461,909$) for identified presence of CVD ($N = 284,079$; 61.50% of Aim 3c sample), tobacco use ($N = 58,452$; 12.65% of Aim 3c sample), obesity ($N = 76,885$; 16.64% of Aim 3b sample), and depression ($N = 65,825$; 14.25% of Aim 3c sample). For additional information on these post hoc analyses, see Table 11.

For the subgroup with identified CVD, there was a significant main effect of treatment group on MAP ($F(3,284065) = 15.71, p < 0.001, \eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline MAP. PC alone ($M = -5.007$) demonstrated significantly less reduction in MAP than PC and PCBH ($M = -5.399; p < 0.001$), PC and specialty mental health ($M = -5.195; p = 0.014$), and all three treatment settings ($M = -5.456; p < 0.001$).

For the subgroup with identified tobacco use, there was a significant main effect of treatment group on MAP ($F(3,58438) = 3.18, p = 0.023, \eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline MAP. PC and PCBH ($M = -2.136$) demonstrated significantly greater reduction in MAP than PC and specialty behavioral health ($M = -1.778; p = 0.042$) or all three treatment settings ($M = -1.681; p = 0.016$).

For the subgroup with identified obesity, there was a significant main effect of treatment group on MAP ($F(3,76871) = 4.30, p = 0.005, \eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline MAP. The condition with all three treatment settings ($M = -$

2.221) demonstrated significantly greater reduction in MAP than PC alone ($M = -1.884$; $p = 0.005$). All other comparisons were insignificant.

For the subgroup with identified depression, there was a significant main effect of treatment group on MAP ($F(3,65811) = 12.77$, $p < 0.001$, $\eta_p^2 < 0.001$) when controlling for age, sex, time, and baseline MAP. PC and specialty mental health ($M = -2.563$) demonstrated significantly less reduction in MAP than PC alone ($M = -3.052$; $p < 0.001$), PC and PCBH ($M = -2.961$; $p < 0.001$), or all three treatment settings ($M = -3.038$; $p < 0.001$).

CHAPTER 5: Discussion

The PCBH model is designed to provide brief, behavioral interventions addressing population health for a myriad of presenting problems including CVD and related modifiable risk factors (70). Although the existing literature provides a solid foundation for the concept of integrated behavioral health, there are substantial gaps related to the penetration and effect of the PCBH model in addressing behaviors germane to health conditions (67). The current study provides exploratory analyses of the penetration of BHCs across the Defense Health Agency in addressing concerns related to CVD and its modifiable risk factors (i.e., tobacco use, obesity, and depression). This study also revealed significant findings related to the relationship between CVD severity and perceived global functioning. Specifically, this study found that levels of CVD severity did not significantly differ in perceived global functioning; however, interactions with modifiable risk factors suggested that perceived functioning in those with CVD was moderated by tobacco use and depression. Finally, the study compared health outcomes between primary care alone, primary care with PCBH, primary care with specialty mental health, and treatment in all three settings. The results suggested there were significant differences in treatment group for tobacco use, BMI, and MAP. These differences in health outcomes illuminated that PC with PCBH demonstrated the best outcomes for tobacco use and MAP, whereas PC with specialty behavioral health demonstrated the best outcomes for BMI.

PENETRATION RATES OF BEHAVIORAL HEALTH CONSULTANTS

The analysis of penetration rates was parsed out into diagnostic categories to illustrate the frequency at which BHCs treated individuals with CVD, tobacco use,

obesity, and depression. Given the dearth of relevant data, each health behavior was hypothesized to demonstrate a conservative penetration rate of more than 5% (67; 109; 129). Similarly, CVD was expected to demonstrate a penetration rate of more than 5% as an extension of treatment for the related health behaviors. However, depression was hypothesized to demonstrate a much higher rate of penetration at greater than 25% given the more salient role of the BHCs as a stepped care model for specialty behavioral health; that is, it was anticipated that PC would more readily adopt the PCBH model for increasing penetration of mental health diagnoses (67).

In the monitored three-year period from January 2017 to December 2019, only 1.47% of individuals diagnosed with any severity of CVD were seen by a BHC for any reason. Further, only 0.19% of those with CVD were seen by a BHC related to their CVD diagnosis. The rate of penetration was similar at for each level of CVD severity including hypertensive heart disease (1.54%), atherosclerotic and other CVD (1.23%), and major adverse cardiac events (1.46%). Regardless of the severity, over 98% of individuals with any form of CVD and access to a BHC are not receiving care the from PCBH model.

The single greatest modifiable risk factor of CVD (i.e., tobacco use) demonstrated penetration rates consistent with the hypothesis of greater than 5%. Of all individuals diagnosed with tobacco use in the three-year period, 5.34% were subsequently seen by BHCs. A more conservative representation of the penetration rate looking at BHC encounters using tobacco use as the presenting problem suggests the rate falls slightly to 4.58%. There is no definitive rationale for the increased penetration rate for those with tobacco use; however, it seems feasible that defeated attitudes and beliefs that patients are responsible for initiating smoking cessation may encourage PC provider to engage in

higher adoption of the PCBH model to address tobacco use than for other health behaviors examined in this study (166). Additionally, as many as 18% of individuals are referred to specialists for behavioral intervention, suggesting utilization of behavioral consultation has an existing precedent (129). Finally, recommended practice for BHCs within the military healthcare system includes managing promotional material such as handouts for tobacco cessation; making pamphlet prominently available may prompt conversations about intervention for tobacco cessation (68). Still, nearly 95% of individuals with recorded tobacco use and access to a BHC are not receiving care from the PCBH model.

In the United States, only 5.8% of PC visits involve weight management, despite nearly 2 in 5 adults meeting criteria for obesity, which is partly attributed to factors such as physician discomfort in addressing weight and low perceived efficacy in weight loss interventions without specialty care intervention (28; 30; 109). The penetration of BHCs in treating individuals with obesity reflected similar rates to those addressing weight management in traditional primary care, capturing 3.41% of individuals with an obesity diagnosis. Again, a more conservative representation looking at BHC encounters using obesity as a presenting problem shows a decline to 2.23% penetration of the total population with an obesity diagnosis. Although the existing literature on weight management in the PCBH model is limited, existing studies suggest brief intervention with a BHC can provide efficacious weight loss intervention (137). Additionally, high patient satisfaction with the PCBH model, combined with high patient interest in addressing weight loss at PC appointments, suggests the low penetration rate is not likely related to patient disinterest (10; 49; 53). Rather, it seems to illustrate a perpetuation of

the aforementioned provider obstacles (i.e., low perceived efficacy and physician discomfort) as well as continued opportunity for adoption of the PCBH model in addressing obesity (28; 30; 109).

Finally, BHCs were hypothesized to demonstrate penetration rates of greater than 25% for individuals diagnosed with depression; the PCBH model was anticipated to reveal a higher rate for depression because it was developed first and foremost as an augmentation to PC intended to provide a stepped care model for mental health treatment (46). The current rate of penetration for individuals with a depression diagnosis was 20%, with a specific penetration for encounters using depression as a presenting problem maintaining at 19.51%. This rate of penetration was lower than expected, especially given some clinics, particularly within the military, implement a gateway model in which appointments for consultation within the PCBH model act as a required medical home for referral to specialty behavioral health (89).

In the context of providing intervention for CVDs modifiable risk factors, the adoption of behavioral health specialists in a primary care setting provides an opportunity to augment care as usual to address challenging behavior modifications. Mental health concerns such as depression have presented a salient target for BHC intervention, yielding exponential increases in penetration of behavioral health services upon implementation (23). Similarly, intervention for health behaviors such as tobacco use and obesity include referral to specialized behavioral intervention as first-line standard of care (16; 74). Yet, the current penetration rates show that only 1 in 5 individuals with a depression diagnosis will receive care from a BHC and only 1 in 20 with a modifiable health behavior will receive the same treatment.

CVD SEVERITY, MODIFIABLE RISK FACTORS, AND GLOBAL FUNCTIONING

The second aim evaluated the impact of CVD and its modifiable risk factors on perceived global functioning through a self-report measure captured at all encounters with BHCs. It was hypothesized that individuals with increasing severity of CVD would report increasingly lower global functioning, where global functioning is defined as a combination of perceived well-being, presence of psychological symptoms, and functional difficulties across common domains of daily life (84). This aim further hypothesized that the relationship between CVD and global functioning would be moderated by modifiable risk factors. The main effect of CVD severity suggested there was no significant difference in perceived global functioning. However, the relationship was significantly moderated by tobacco use and depressive symptoms.

CVD and Tobacco Use on Global Functioning

The significant interaction between CVD severity and tobacco use demonstrates the relationship between CVD severity and global functioning was different for smokers than nonsmokers. Specifically, those who endorsed tobacco use perceived progressively lower functioning with more severe CVD, while nonsmokers demonstrated no statistical difference in perceived functioning across CVD severity. On average, smokers reported higher perceived functioning than nonsmokers until the major adverse cardiac events (i.e., the highest level of severity). It was anticipated that smokers would perceive lower global functioning given the subjective health complaints typically associated with tobacco use independent of CVD (103). Perhaps smokers reported higher perceived functioning due to the positive affective experience associated with active sedative and stimulant effects of nicotine (3). That is, smokers may have perceived higher global

functioning due to perceived benefits of tobacco use (i.e., weight loss, satiated craving, etc.) without perceived costs to their functioning (i.e., shortness of breath, fatigue, etc.). Therefore, smokers with lower severity of CVD may be less likely to endorse readiness to change with generally higher perceived functioning (3). Additionally, it may be that the functional impact of CVD becomes acutely salient after a cardiac event, making the physical disabilities and cognitive decline associated with MACE more adversely affect functioning that was less notable in earlier stages of CVD (35; 60). In total, it is likely that those with tobacco use reported higher global functioning on average until the major adverse impact of a cardiac event made functional decline irrefutable.

It is worth noting that the statistically observed difference may not be clinically meaningful. Although the statistically analyses identified differences global functioning across groups, those differences were marginal in the context of determining clinically meaningful improvement of deterioration for an individual patient. First, the means across all conditions range from 2.68 to 2.79, which maintains averages in the *mild* range of perceived global functioning. Further, the effect size of the interaction effect is very small ($\eta_p^2 < 0.001$), suggesting the variance explained by this interaction is negligible. Finally, clinically meaningful difference characterized by gross change in accordance with a reliable change index ($RCI = \frac{posttest - pretest}{S_{diff}}$) would require a mean difference between groups of greater than 0.76 in BHM-20 GMH score (72). Reliable change index is a conservative statistical method for characterizing clinically meaningful difference between groups or change across time (72). Given the greatest difference between groups was only 0.11, there was no indication of clinically meaningful differences in groups.

CVD and Depression on Global Functioning

The significant interaction between CVD severity and depression suggests the relationship between CVD severity and global functioning was different for those with depression than those without depression. Although the interaction was significant, there was no apparent cross-over or statistically significant difference within the two levels of the depression variable. Additionally, the effect size was very small ($\eta_p^2 < 0.001$) and the p-value for the interaction effect ($p = 0.027$) suggests a probability that statistical significance may have derived from an overpowered analysis. In combination, this interaction was statistically significant for an alpha level of 0.05, but the statistical generalizability is highly questionable.

IMPACT OF TREATMENT GROUP ON HEALTH OUTCOMES

The third aim was to examine the impact of BHCs on health behavior outcome through comparison of treatment outcomes in primary care alone, primary care with BHC encounter, primary care with specialty care, and treatment in all three settings. To simplify discussion, these groups will be referred to as primary care, PCBH, specialty mental health, and all treatment settings. Overall, treatment groups demonstrated significantly different health outcomes for each targeted health behavior outcome (i.e., tobacco use, BMI, and MAP). The hypothesis for this aim stated that those seen in all treatment settings would demonstrate the greatest improvement for each health behavior outcome, followed by PCBH, specialty mental health, and finally primary care. Although the models each yielded significant results for treatment group, the treatment group with the greatest improvement was not always consistent with the hypothesized order. The effect size of treatment group in each model ($\eta_p^2 < 0.001$) also suggests the significant differences were only detectable given the substantial size of the data sample; that is,

statistically significant differences typically did not constitute clinically meaningful change at the individual level. Even from a population health perspective, the literature uses similar thresholds for identifying clinically meaningful trends in healthcare outcomes for tobacco cessation, weight loss, and blood pressure change (51; 117; 148). Therefore, trends in the mean change outline below will be defined below in the context of typical treatment guidelines for clinically meaningful change. In analysis by diagnostic subgroup for CVD, tobacco use disorder, obesity, and depression the analyses revealed similar result, with most models reporting statistically significant difference in treatment group but clinically insignificant effect sizes.

Treatment Group and Tobacco Use

Reported tobacco use was hypothesized to demonstrate different outcomes for each treatment group such that individuals seen in all treatment settings would yield the greatest reduction in tobacco use, followed by PCBH. Although the model was statistically significant for difference in treatment group, the hypothesis was refuted as individuals seen by PCBH revealed the greatest reduction in in reported tobacco use; additionally, PCBH was not statistically different from primary care.

The interpretation of these results is complicated by the meaning of the dependent variable (i.e., change in reported tobacco use); this value represents the average number of individuals that started or stopped smoking while controlling for their baseline reported tobacco use. This means the average change in reported tobacco use for PCBH ($M = -0.017$) does not represent an odds ratio, but rather the average between all individuals that started (i.e., $\Delta\text{Tobacco Use} = 1$), stopped (i.e., $\Delta\text{Tobacco Use} = -1$) or had no change in tobacco use. Therefore, the best interpretation is the statistically significant

difference in average tobacco use between groups. The interpretation of these results is further complicated by the very small effect size ($\eta_p^2 < 0.001$), which combined with the categorical nature of reported tobacco use, limits the interpretability for clinically meaningful interpretation of outcomes.

Treatment by PCBH yielded the greatest reduction in reported tobacco use, with significantly greater reduction than specialty mental health ($p < 0.001$) and all treatment settings ($p = 0.017$). However, one perspective in interpreting the results of health outcomes to determine if the addition of a BHC improved the health care outcome was that between primary care and PCBH; for reported tobacco cessation in the total population, PCBH was not significantly different from primary care. This is likely due to the precedent that primary care is the identified home for tobacco cessation (166). Therefore, it is reasonable to expect primary care providers are at least familiar with clinical practice guidelines for tobacco cessation. Still, those who are treated in PCBH are would theoretically be more likely to receive specialized behavioral intervention for tobacco use at a higher rate than primary care alone (129), even with the low penetration rate for BHCs noted in Aim 1 (i.e., 4.58%).

Tobacco Use Outcomes in those with Tobacco Use Disorder

For individuals with identified tobacco use, the model was significant for difference in health outcomes between the treatment groups. Like the larger model, PCBH yielded the greatest reduction in reported tobacco use. However, primary care was statistically significant as the lowest reduction in reported tobacco use. This diagnostic subgroup also produced the greatest magnitude in change values, with estimate marginal means ranging from -0.117 to -0.147. This is partly attributable to the diagnostic

subgroup isolating identified smokers; therefore, the complication of the change variable is limited to those who stopped (i.e., $\Delta\text{Tobacco Use} = -1$) or maintained (i.e., $\Delta\text{Tobacco Use} = 0$) their smoking status. One potential interpretation of these means would be that smokers who were seen in PCBH ($M = -0.147$) reported tobacco cessation at a rate of 14.7% when controlling for age, sex, time, and baseline tobacco use. However, this would likely be an overinterpretation of the data given the relative effect size of treatment group ($\eta_p^2 < 0.001$) and confounding aspects of data collection that are not controlled without using relative comparison to similar groups.

For example, an individual may have been identified as quitting tobacco use at their final monitored primary care encounter without ever being asked. Given the data for all four treatment group conditions required data collection in the primary care setting, the incidents of false reporting would ostensibly be distributed between treatment conditions. Therefore, comparing trends between treatment settings would reduce the confounding nature of data collection, but to deduce clinically meaningful rates of tobacco cessation would be overstating the data. To suggest the current data demonstrate a tobacco cessation rate of 14.7% is largely inconsistent with the literature (8; 92). Still, studies evaluating the impact of electronic health records similar to the one used in this study suggest substantial improvements in screening rates, accurate reporting of tobacco use, and sustained tobacco cessation at follow up (116).

It is worth highlighting that this model isolating those with identified tobacco use disorder is the most appropriate for interpreting generalizable results. In the comparison between the primary care and PCBH, the insignificant difference in the larger sample became statistically significant in this diagnostic subgroup ($p = 0.001$). This difference

suggests the addition of a BHC in the primary care setting demonstrates a trend of improved health outcomes. More rigorous examination of BHC practices, fidelity to the PCBH model, and tobacco cessation outcomes would be required for definitive clinically meaningful difference.

Tobacco Use Outcomes in those with CVD, Obesity, or Depression

For individuals diagnosed with CVD, the difference in treatment group on tobacco use outcomes was statistically significant. This diagnostic subgroup differed from the larger sample, with primary care demonstrating significantly greater reduction in reported tobacco use than treatment in all settings. However, there were no other statistically significant differences. Similarly, diagnostic subgroups for obesity and depressive disorders each demonstrated statistically significant difference in treatment groups with primary care demonstrating the greatest reduction in tobacco use. The trend of primary care yielding among the greatest reduction in reported tobacco use persisted across the entire sample and each diagnostic subgroup except for those with identified tobacco use disorder.

One possibility for this trend is that those diagnosed with a tobacco use disorder received more specialized behavioral intervention for tobacco use as the target for intervention. However, it is more likely that this trend is an artifact of the data given the diagnostic subgroup with tobacco use disorder was the only subset of the data with consistent baseline tobacco use. That is, all models other than the diagnostic subgroup for tobacco use disorder included individuals with a reported increase in smoking at the final monitored appointments or maintained abstinence with an identical change score (i.e., $\Delta\text{Tobacco Use} = 0$) to someone who maintained their status as a smoker. In sum, the

consistency of the trend across the entire sample and each diagnostic subsample is more likely attributable to confounding factors than something clinically meaningful; the deviation from this trend in the tobacco use disorder diagnostic subgroup highlights that it is the most methodologically reliable model.

Treatment Group and BMI

The hypothesis regarding treatment group and health behavior outcomes for BMI posited that individuals seen in all treatment settings would yield the greatest reduction in BMI, followed by PCBH. Although the model was statistically significant for difference in treatment group, the hypothesis was refuted as individuals seen by specialty behavioral health revealed the greatest reduction in BMI. Further, the effect size for treatment group was very small ($\eta_p^2 < 0.001$), requiring continued caution in definitive statement about clinically meaningful outcomes.

Clinically significant weight loss is typically considered a reduction of 5% to 10% in body weight over a 6- to 12-month period (154). Given the analyzed sample was mostly males between 18 and 44 years old with a baseline BMI of 29.5 ($M = 29.5$, $SD = 6.14$), the average male in the population with a height of 5 feet 9 inches would weigh approximately 200 pounds; therefore, they would need to lose approximately 10 to 20 pounds (i.e., a BMI change of -0.7 to -1.4) to constitute clinically significant weight loss. When controlling for all other factors, treatment in specialty behavioral health yielded an average reduction in BMI of 3.008, or approximately 21 pounds for an average height male. Controlling for the same factors, treatment with PCBH ($M = -2.397$) or primary care ($M = -2.388$) yielded an average reduction in BMI equal to approximately 16 pounds of weight loss for an average height male. This means the statistically significant

difference between these treatment groups also approaches a clinically significant difference of about 5 pounds difference in weight loss. The other statistically significant difference between all treatment settings ($M = -2.740$) and those seen by primary care ($M = -2.388$) or PCBH ($M = -2.397$) only amounted to about a 2-pound difference in average weight loss; therefore, the statistically significant difference does not translate to be clinically meaningful. In the continued comparison of primary care and PCBH, this model suggests the addition of a BHC did not yield statistically or clinically significant difference in BMI outcomes ($p = 1.000$); this may be due to the low BHC penetration rate for obesity (2.23%) limiting opportunities to impact BMI.

BMI Outcomes in those with Obesity

For individuals diagnosed with obesity, the significant main effect of treatment group on BMI continued the pattern outlined by the complete sample; specialty mental health ($M = -1.790$) averaged the greatest reduction in BMI including statistically better outcomes than primary care ($M = -0.872$) or PCBH ($M = -0.615$). Although the mean reduction in BMI was less for those diagnosed with obesity than it was for the complete sample, the difference between treatment settings was greater than any other diagnostic subgroup. It is unclear why this subgroup demonstrated less reduction in BMI than the general population, especially considering regression to the mean would suggest those with more weight would intrinsically show more weight loss. One consideration may be the typical weight fluctuation seen in military populations in tandem with weight standards; individuals in the full sample may have been less likely to be diagnosed with obesity as an active duty servicemember and subsequently had to lose weight to meet body composition standards (32; 133).

The mean BMI reduction for specialty mental health equates to approximately 12 pounds of weight loss for a male with average height. Conversely, primary care and PCBH equate to approximately 4 to 6 pounds of weight loss for a male with average height. For individuals diagnosed with obesity, treatment in specialty mental health produced 6 to 8 pounds of weight loss greater than primary care or PCBH; meanwhile, the addition of a BHC to the primary care setting (i.e., primary care vs PCBH) did not produce statistically or clinically meaningful difference. Overall, BMI outcomes for those diagnosed with obesity demonstrated clinically meaningful difference, with those treated in specialty mental health yielding the greatest results.

BMI Outcomes in those with CVD, Tobacco Use, or Depression

For individuals diagnosed with CVD, the difference in treatment group on BMI outcomes was statistically significant. This diagnostic subgroup mirrored the larger sample, with specialty mental health yielding the greatest reduction in BMI. Different from the entire sample, PCBH was statistically significant as the lowest reduction in BMI across treatment settings. Clinically, the difference between specialty mental health ($M = -5.122$) and PCBH ($M = -4.325$; difference of 0.797) was approximately 5 pounds of difference in average weight loss. This suggests that, again, treatment in specialty behavioral health approached clinically superior health outcomes to those in PCBH. Although specialty behavioral health showed statistically more reduction in BMI than any other treatment group, this was the only comparison that produced a difference in BMI greater than 0.7 points.

For individuals with identified tobacco use, the model was significant for difference in health outcomes between the treatment groups. However, the estimated

marginal means revealed only the difference between primary care and treatment in all settings was statistically significant, with primary care yielding the greatest reduction in BMI. The difference in BMI change between primary care ($M = -0.170$) and all treatment settings ($M = 0.076$) was clinically marginal. In fact, no treatment setting produced a clinically meaningful change in BMI for individuals with identified tobacco use, and treatment in all settings averaged an increase in weight.

For individuals diagnosed with depression, the difference in treatment group on BMI outcomes failed to refute the null hypothesis, as the mean difference in BMI outcomes had a range of 0.41 across all four levels of treatment group. Put differently, the average weight change across the four levels differed by approximately 3 pounds for an average height male; not only is that a statistically insignificant difference between treatment groups, 3 pounds constitutes normal fluctuation in weight without targeted intervention (154). The insignificant results combined with the low average BMI change in this model was particularly surprising given well founded relationship between weight management and amelioration in depressive symptoms (142). Several common psychotherapeutic treatments for depression include some form of increase in pleasurable or activity (26; 68), with the referenced guide for expected practices by BHCs including common behavioral activation strategies (68) (p.60); although these strategies do not directly target weight loss, the crossover in weight management and behavioral activation provides a multifaceted approach to treating comorbid modifiable risk factors for CVD. This presents an opportunity to improve delivery of care at every treatment level.

Treatment Group and MAP

The hypothesis regarding treatment group and health behavior outcomes for MAP posited that individuals seen in all treatment settings would yield the greatest reduction in MAP, followed by PCBH. The hypothesis was supported in that treatment in all settings and PCBH were not significantly different but greater than all other treatment settings. Again, the effect size for this model was very small ($\eta_p^2 < 0.001$), which will require continued caution in interpreting the clinical meaningfulness of the results. To that end, clinically meaningful change in blood pressure is typically defined as a 5 mmHg reduction in systolic and diastolic blood pressure (57; 62). Given the equation for mean arterial pressure ($MAP = \frac{SBP+2DBP}{3}$) provides a linear calculation for mean pressure change, a reduction of 5mmHg in both systolic and diastolic pressure would be equivalent to a reduction of 5 mmHg in mean arterial pressure.

When controlling for all other factors, treatment in PCBH yielded an average reduction in MAP of 3.981 mmHg. Controlling for the same factors, treatment with specialty mental health ($M = -3.635$) or primary care ($M = -3.332$) yielded a difference of 0.346 and 0.649 mmHg, respectively. Therefore, the statistically significant difference between treatment settings did not equate to a clinically meaningful difference in blood pressure change. Further, the total change in MAP for each treatment setting ranged from 3.332 mmHg to 4.010 mmHg when controlling for all other variables. Therefore, the estimated marginal means suggest that for the entire population, there was not clinically meaningful reduction in blood pressure in any single treatment setting. It is worth noting that the mean MAP at baseline for the entire population was 96.217 mmHg, which is considered within the healthy range (170). Therefore, the mean reduction in blood pressure and relative difference in blood pressure change may have been dampened a

healthy mean, making the diagnostic subgroup for those with CVD (i.e., the next section) particularly important for clinically meaningful change.

MAP Outcomes in those with CVD

For individuals diagnosed with CVD, the difference in treatment group on MAP outcomes was statistically significant. This diagnostic subgroup mirrored the larger sample. Again, PCBH and treatment in all settings yielded the greatest reduction in MAP, with no significant difference between the two conditions. Also like the total sample, the difference between the greatest (i.e., treatment in all settings, $M = -5.399$) and smallest reduction in MAP (i.e., primary care alone, $M = -5.007$) did not yield clinically meaningful difference at 0.392 mmHg. However, the difference between primary care and PCBH was statistically significant ($p < 0.001$), suggesting at least a marginal trend that the addition of a BHC yielded greater improvement in MAP. One notable difference from the total population was the magnitude of average reduction in MAP. On average, individuals diagnosed with some level of CVD demonstrated a reduction in blood pressure of more than 5 mmHg across all treatment settings. As mentioned above, the diagnostic subgroup with a CVD diagnosis provides more opportunity to illustrate clinically meaningful change given blood pressure was directly clinically relevant. Therefore, it stands to reason the those with CVD produced a greater magnitude in MAP reduction than any other diagnostic subgroup or the total population.

MAP Outcomes in those with Tobacco Use, Obesity, or Depression

For individuals with identified tobacco use, the model was significant for difference in health outcomes between the treatment groups. The trend was maintained, with the greatest reduction in MAP demonstrated in PCBH. The magnitude of MAP

reduction was more similar to the total sample with change in MAP ranging from 1.681 mmHg to 2.136 mmHg; although tobacco use has known deleterious effects on hypertension (25; 75), it seems reasonable that the magnitude of change would be more similar to the total population without focus on intervention for cardiovascular symptoms. Overall, no treatment setting produced a clinically meaningful change in MAP for individuals with identified tobacco use. Additionally, the difference in MAP change between PCBH ($M = -2.136$) and all treatment settings ($M = -1.681$) created the greatest difference (i.e., 0.455 mmHg) but was clinically marginal (57; 62).

For individuals diagnosed with obesity, the significant main effect of treatment group on MAP deviated from the trends in the previous populations. For this diagnostic subgroup, treatment in all three settings ($M = -2.221$) demonstrated significantly greater reduction in MAP than primary care ($M = -1.884$) with no other significant comparisons. Again, the statistically significant difference did not equate to clinical significance (i.e., 0.337 mmHg). Overall, the magnitude of change in each treatment group did not suggest clinically significant reduction in MAP, with a range of 1.88 mmHg to 2.221 mmHg. Like the tobacco use disorder group, obesity has known deleterious effect on blood pressure (147). However, without targeted focus on blood pressure, it is understandable that the clinical subgroup for obesity did not demonstrate clinically meaningful change or clinically meaningful difference between groups.

For individuals diagnosed with depression, the significant main effect of treatment group on MAP continued the pattern outlined by the complete sample; PCBH ($M = -2.961$) averaged the greatest reduction in with statistically equivalent outcomes to primary care ($M = -3.052$) and treatment in all settings ($M = -3.038$). Realistically, the

statistically significant difference in treatment groups for this model was largely attributable to the outcomes in specialty mental health ($M = -2.563$), which demonstrated statistically, but not clinically, lower outcomes than all other treatment groups. This difference may be due to the focus on depressive symptoms, rather than cardiovascular symptoms, for individuals treated in specialty mental health. Still, as mentioned above in health outcomes related to BMI, several common treatments for depression include some form of increase in activity (26; 68). With increased physical activity, individuals would experience cardiovascular benefits. Nonetheless, the very small effect size and clinically insignificant difference between treatment groups suggests no treatment group stands out in cardiovascular benefits.

Treatment Group in the context of Penetration Rates

Throughout the third aim examining the impact of BHCs on health outcomes germane to CVD, the trends suggested there was no single treatment setting responsible for the greatest change. It seemed that the diagnostic subgroup most relevant to the analyzed health outcome (i.e., tobacco use disorder for tobacco use, obesity for BMI, and CVD for MAP) demonstrated the most substantial results. Still, the health outcomes with tangibly interpretable results, namely BMI and MAP, demonstrated consistent difficulty yielding clinically meaningful outcomes in a setting with evidence-based and evidence-informed interventions for the targeted modifiable risk factors (68).

It is important to understand the results of the third aim in the context of the first. As was outlined in the first aim, less than 95% of individuals with access to a BHC are seen for behavioral intervention to manage their tobacco use, obesity, or CVD diagnosis. This means that the current data is a snapshot of a healthcare system with ample

opportunity to expand the scope of the PCBH model into addressing critical health behaviors. Previous literature demonstrates interventions in the PCBH model are efficacious (16; 46; 50; 67; 74), even with the current study failing to demonstrate clinical meaningful change an a health system perspective. Perhaps part of the inconsistency in which treatment group yields the greatest health outcome is the low rate of addressing critical modifiable risk factors at any level. It is well established that tobacco cessation and weight management are some of the most difficult behaviors to change (129; 154). However, the integration of BHCs augments usual care with specialized providers for the purpose of improving health outcomes related to these difficult behaviors (132). In the context of penetration rates, the health outcomes illustrate an opportunity for better engagement through the PCBH model in targeting modifiable risk factor of the deadliest disease in the world (27; 175).

STUDY LIMITATIONS AND FUTURE DIRECTIONS

The current study provided several novel opportunities to explore the penetration rate of the PCBH model in combination with the effectiveness in addressing CVD and its modifiable risk factors. However, there were several important limitations that constrained both the interpretability and generalizability of the current data. An important limitation of this study was the quasi-experimental method using retrospectively extracted data. This is particularly important for the third aim evaluating health outcomes for different treatment groups and diagnostic subgroups. Although there were levels for each diagnostic variable that constituted a quasi-experimental control (i.e., no CVD, no depression, no obesity, and no tobacco use), even the group of individuals without any of the target diagnoses ($N = 46,070$) could not be definitively identified as a healthy control.

Without a control group, it is unknown whether the comparison group provides a healthy constant for defining statistically and clinically meaningful change. This would be particularly important for identifying individuals that may have relevant clinical conditions that were not captured in the course of typical care in the military healthcare system. Future studies should look to address this limitation by identifying individuals who constitute a more definitive healthy control, reasonably ruling out any confounding presentations. Additionally, it may benefit future studies to evaluate health outcomes across the course of treatment using a natural experimental design; this could track health outcomes as participants are naturally referred to higher levels of care, improve at their current level of care, or even monitor those who elect to drop out of treatment without showing improvement.

Related, the aggregate of retrospective data collected from the natural course of care provides a strength for this study as a focus on population health outcomes; despite the limited data on employed interventions in any specific treatment setting, the aggregation of data at the universal level provides generalizable information on trends across the healthcare system. However, the nature of the interventions for primary care, BHCs, and specialty behavioral health required speculation based on the identified problem for each encounter. This is a major limitation for this study because it substantially reduces the interpretability in identifying the mechanisms of change for treatment groups. That is, individuals that were seen at any level of treatment under a presenting problem of tobacco use disorder may have used the 5A's Behavior Change Model (16; 46), or the provider may have coded the encounter for tobacco use after discussing something completely unrelated. Future studies looking to address this

limitation will require far more granular data to monitor fidelity to the model of care and qualitatively examine mechanisms of change. One potential method for addressing this limitation while maintaining a population health sample is the categorical recording of the employed intervention at each encounter by the respective provider; this would still limit quantifying fidelity or competency in executing the intervention but could drastically improve several aspects interpretability that are limited in the current model.

Additionally, the three-year period analyzed in the current study provided an ample span of monitored care to capture the population of healthcare beneficiaries. Across this span of time, treatment in any given setting was classified as a binary variable to promote comparison of very different modalities of care. Controlling for a dosage effect may penalize treatment in settings with more frequent appointments like specialty mental health. Conversely, the natural course of primary care often includes infrequent appointments that make episodes of care difficult to characterize. Therefore, evaluating health outcomes at a universal level in a quasi-experimental design for such a long period of time required flexible categorization to examine natural trends. Still, the study design limits the ability to identify episodes of care and longitudinal outcomes after termination from care. An episode of care would capture an entire course of treatment for a presenting problem until the healthcare event is complete. For example, in weight management this might include all appointments from the initial primary care encounter until the individual has either met their goal weight or sufficiently maintained the recommended behavior change to terminate from continued care. In the monitored three-year period, some individuals may have received several episodes of care for the same problem or be partly through an episode of care at either end of the monitored period.

Future studies hoping to address this limitation in the cross comparison of such different modalities should either conduct a natural experiment with defined standards of care in each condition or develop treatment protocols that are consistent with standard practices at each level of care. With either approach, defining a start, end, and follow up evaluation of health outcomes would be recommended. Although each would have unique benefits and limitation, both would address the limitation of the current study.

Finally, these methods do not account for individuals who may have received care from other sources. Although a review of medical records captures any treatment within the DoD, there is potential for individuals to have received care from other sources during, or prior to, the monitored period. This creates a major confounding variable that is unaccounted for in analyzing health outcomes. Future studies should either exclude or control for individuals receiving care outside the examined model of care to better understand the impact of treatment received.

CONCLUSIONS

The current study contributes to the existing literature through population level data demonstrating the penetration rates and relative effectiveness of the PCBH model for the treatment of CVD and related modifiable risk factors. The PCBH model presents a wide-reaching opportunity to provide brief, behavioral interventions for the leading cause of death in the United States and improve the aggregate health of the population at the medical home – primary care (27; 70; 175). Specifically, the study provides exploratory analyses for the penetration of BHCs in addressing CVD, tobacco use, obesity, and depression as well as health outcomes for related vitals (i.e., reported tobacco use, BMI, and MAP). The contributions of the current study are consistent with the outlined gaps in

the call to action suggesting a need for better understanding of comparative data, penetration rates for BHCs, and health behavior impact in real-world practice (67). The study revealed specific penetration rates around 3% for obesity and CVD, 5% for tobacco use, and 20% for depression. Although the PCBH model was predominately developed as a stepped care model for managing mental health conditions as part of the primary care medical home, the results suggest health behaviors that receive first line treatment in primary care are largely untouched by the PCBH model (16; 74; 132). When comparing health outcomes between treatment settings, treatment with the PCBH model yielded statistically better reduction in MAP and tobacco use than primary care alone; this was especially true when isolating populations with diagnosed CVD or tobacco use disorder. Conversely, treatment in primary care alone was not statistically different from the PCBH model for BMI, even for those diagnosed with obesity. With improved penetration rates, perhaps these statistically significant differences could become clinically meaningful opportunities to employ the PCBH model in reducing cardiovascular deaths.

One opportunity for consideration in the application of these findings comes in the context of the Quadruple Aim. As mentioned before, this framework is a patient-centered healthcare approach which serves to optimize health system performance (19; 20). The development of clinical pathways to inform the application of evidence-informed interventions may increase the rate at which military healthcare beneficiaries receive health behavior interventions (80). This would provide a vehicle for guiding clinical practice to address the low penetration rates identified in this study. Clinical pathways would guide providers in a growing interdisciplinary model on methods for optimal application of clinical practice guidelines to address modifiable risk factors of

CVD; pathways would address identifiers for the target population, procedures for increasing the penetration rate of BHCs in those target populations, and methods of monitoring outcomes to maintain adherence to an appropriate level of care (68; 80).

Ultimately, proper implementation would require additional research to better inform each factor of a proposed clinical pathway. Studies should further examine the effectiveness of the PCBH model in addressing health behaviors utilizing more rigorous methodology. This could include monitoring of appointment characteristics, fidelity to the PCBH model, interventions employed by BHCs, or follow-up measures at variable intervals after the final appointment (67). Monitoring the fidelity to the PCBH model and interventions employed by BHCs would provide more standardized information on best practices and mechanisms of change. Studies should also continue to examine the limitations of the PCBH model to identify appropriate criteria for treatment in the PCBH model and referral to higher levels of care. Finally, it will be important to revisit the exploration of trends in health outcomes once penetration rates for modifiable risk behaviors suggest the PCBH model is reaching beyond behavioral health and addressing critical health behaviors.

TABLES

Table 1. Cardiovascular ICD-10 Codes and Cardiac Grouping

ICD-10 Codes	Diagnosis
Hypertensive Heart Disease	
I10	Essential Hypertension
I11	Hypertensive Heart Disease
I12	Hypertensive Chronic Kidney Disease
I13	Hypertensive Heart and Chronic Kidney Disease
I15	Secondary Hypertension
I16	Hypertensive Crisis
Atherosclerotic and Other Cardiovascular Disease	
I20	Angina Pectoris
I25	Chronic Ischemic Heart Disease
I26 - I28	Pulmonary Heart Disease
I42-I43	Cardiomyopathy
I60-I69*	Other Cerebrovascular Disease
I70	Atherosclerosis
I71-I79	Other Diseases of the Arteries, Arterioles, and Capillaries
I30 - I99*	Other Cardiovascular Disease
Major Adverse Cardiac Event	
I21-I24	Myocardial Infarction
I46	Cardiac Arrest
I50	Heart Failure
I63	Stroke (Cerebral Infarction)

Note: *Includes ICD-10 codes in the specified range not elsewhere classified. All specified codes include the subordinate ICD-10 diagnostic codes and specifiers.

Table 2. Sample Characteristics for Aim 1 (N = 1,321,072)

Variable	Category	Total (%)
Gender	Female	578,332 (43.78%)
	Male	742,740 (56.22%)
Age	18-24	315,476 (23.88%)
	25-34	253,799 (19.21%)
	35-44	210,486 (15.93%)
	45-64	408,542 (30.93%)
	65+	127,544 (9.65%)
	Unknown	5,225 (0.40%)
Marital Status	Married	536,598 (40.62%)
	Separated or Divorced	31,739 (2.40%)
	Single	128,064 (9.69%)
	Widowed	14,466 (1.10%)
	Unknown	610,205 (46.19%)
Clinic Branch	Army	567,084 (42.93%)
	Navy	288,689 (21.85%)
	Air Force	400,799 (30.34%)
	Other	64,500 (4.88%)
Beneficiary Category	Active Duty or Active Reserve	489,561 (37.06%)
	Dependent	540,506 (40.91%)
	Retiree	260,787 (19.74%)
	Other	30,218 (2.29%)
Diagnosis	Tobacco Use	192,566 (14.58%)
(Overlapping % of N)	Obesity	256,852 (19.44%)
	Major Depressive Disorder	155,656 (11.78%)
	CVD All	583,659 (44.18%)
	Hypertensive Heart Disease	466,167 (35.29%)
	Atherosclerotic & Other CVD	181,795 (13.76%)
	Major Adverse Cardiac Event	17,426 (3.19%)

Note: Demographic information is determined by existing data in the Defense Enrollment Eligibility Reporting System (DEERS) included as part of the routine data extraction by the SDD. Reported sample characteristics are dependent on accurate reporting and maintenance of DEERS.

Table 3. Penetration of Behavioral Health Consultants

Diagnosis	Sample Size	Total Penetration	Specific Penetration
Tobacco Use			
	192,566	10,286 (5.34%)	8,810 (4.58%)
Obesity			
	256,852	8,765 (3.41%)	5,717 (2.23%)
Major Depressive Disorder			
	155,656	31,125 (20.00%)	30,363 (19.51%)
All CVD			
	583,659	8,577 (1.47%)	1,090 (0.19%)
Hypertensive Heart Disease			
	466,167	7,197 (1.54%)	963 (0.21%)
Atherosclerotic and Other CVD			
	181,795	2,232 (1.23%)	107 (0.06%)
Major Adverse Cardiac Event			
	17,426	255 (1.46%)	28 (0.16%)

Note: Total penetration is characterized as the number of individuals with the target diagnosis who were then treated by a BHC for any reason, whereas specific penetration is the number of individuals with the target diagnosis who were treated by a BHC for that same diagnosis.

Table 4. Sample Characteristics for Aim 2 (N = 112,137)

Variable	Category	Total (%)
Gender	Female	62,685 (55.90%)
	Male	49,452 (44.10%)
Age	18-24	25,575 (22.81%)
	25-34	29,894 (26.66%)
	35-44	24,231 (21.61%)
	45-64	27,303 (24.35%)
	65+	5,134 (4.58%)
Marital Status	Married	46,434 (41.41%)
	Separated or Divorced	3,024 (2.70%)
	Single	9,611 (8.57%)
	Widowed	906 (0.81%)
	Unknown	52,162 (46.52%)
Clinic Branch	Army	43,430 (38.73%)
	Navy	23,833 (21.25%)
	Air Force	40,686 (36.28%)
	Other	4,188 (3.73%)
Beneficiary Category	Active Duty or Active Reserve	48,620 (43.36%)
	Dependent	49,628 (44.26%)
	Retiree	12,780 (11.40%)
	Other	1,109 (0.99%)
Tobacco Use	Denied Tobacco Use	90,712 (80.89%)
	Endorsed Tobacco Use	21,425 (19.11%)
Obesity	Not Obese	80,769 (72.03%)
	Obesity	31,368 (27.97%)
Depression	Depression Diagnosis Absent	66,830 (59.60%)
	Depression Diagnosis Present	45,307 (40.40%)
CVD Severity	No CVD	68,658 (61.23%)
	Hypertension	25,367 (22.62%)
	Atherosclerotic & Other CVD	16,118 (14.37%)
	Major Adverse Cardiac Event	1,994 (1.78%)

Table 4 (continued). Sample Characteristics for Aim 2 (N = 112,137)

Variable	Category	Total (%) or Mean $\pm$ SD
CVD Medication	No CVD Medication	16,877 (15.05%)
	Active CVD Medication	95,260 (84.95%)
Depression Medication	No Antidepressant	26,026 (23.21%)
	Active Antidepressant	86,111 (76.79%)
Mean BHM-20 Score	Total Population	2.67 $\pm$ 0.72
	Tobacco Use	2.79 $\pm$ 0.75
	Obesity	2.73 $\pm$ 0.69
	Depression	2.38 $\pm$ 0.69
	Any CVD	2.76 $\pm$ 0.69
	Hypertension	2.76 $\pm$ 0.70
	Atherosclerotic	2.76 $\pm$ 0.68
	MACE	2.79 $\pm$ 0.66

Note: Demographic information is determined by existing data in the Defense Enrollment Eligibility Reporting System (DEERS) included as part of the routine data extraction by the SDD. Reported sample characteristics are dependent on accurate reporting and maintenance of DEERS.

Table 5. Summary of ANCOVA for CVD Severity and BHM-20 GMH Score (N = 112,137)

Effects	Type III SS	<i>df</i>	<i>F</i>	<i>p</i>	Partial Eta ²
Interaction Terms					
CVD Severity * Tobacco Use	14.158	3	10.62	< 0.001	< 0.001
CVD Severity * Obesity	1.824	3	1.37	0.250	< 0.001
CVD Severity * Depression	4.098	3	3.07	0.027	< 0.001
Main Effects					
CVD Severity	2.003	3	1.50	0.212	< 0.001
Tobacco Use	4.686	1	10.55	0.001	< 0.001
Obesity	8.185	1	18.42	< 0.001	< 0.001
Depression	845.960	1	1903.71	< 0.001	0.021
Covariates					
CVD Medication	1.951	1	4.39	0.036	< 0.001
Depression Medication	781.117	1	1757.79	< 0.001	0.015
Age Group	485.092	7	155.95	< 0.001	0.010
Patient Sex	192.990	1	434.29	< 0.001	0.004
Error	49819.374	112,111			
Total	57963.232	112,136			

Note: Partial Eta² is a measure of effect size for each variable (Cohen et al., 2001). Effect sizes are classified as small (≥ 0.01), medium (≥ 0.06), and large (≥ 0.14).

Table 6. Sample Characteristics for Aim 3a [Tobacco Use] (N = 298,394)

Variable	Category	Total (%) or Mean $\pm$ SD
Gender	Female	135,730 (45.48%)
	Male	162,664 (54.51%)
Age	18-24	42,806 (14.34%)
	25-34	47,610 (15.95%)
	35-44	46,264 (15.50%)
	45-64	110,112 (36.90%)
	65+	51,602 (17.29%)
Marital Status	Married	154,202 (51.67%)
	Separated or Divorced	9,035 (3.03%)
	Single	22,986 (7.70%)
	Widowed	6,348 (2.13%)
	Unknown	105,823 (35.47%)
Clinic Branch	Army	139,976 (46.90%)
	Navy	66,810 (22.39%)
	Air Force	73,802 (24.73%)
	Other	178,806 (5.97%)
Beneficiary Category	Active Duty or Active Reserve	97,330 (32.61%)
	Dependent	117,906 (39.51%)
	Retiree	78,339 (26.25%)
	Other	4,819 (1.61%)
Treatment Group	Primary Care Only	194,502 (65.18%)
	PC and PCBH	26,158 (8.77%)
	PC and Specialty BH	54,893 (18.39%)
	PC, PCBH, and Specialty BH	22,841 (7.65%)
Baseline Tobacco	Primary Care Only	0.14 $\pm$ 0.35
	PC and PCBH	0.17 $\pm$ 0.38
	PC and Specialty BH	0.24 $\pm$ 0.43
	PC, PCBH, and Specialty BH	0.23 $\pm$ 0.42

Note: Demographic information is determined by existing data in the Defense Enrollment Eligibility

Reporting System (DEERS) included as part of the routine data extraction by the SDD. Reported sample characteristics are dependent on accurate reporting and maintenance of DEERS.

Table 7. Sample Characteristics for Aim 3b [BMI] and Aim 3c [MAP] (N = 461,909)

Variable	Category	Total (%) or Mean $\pm$ SD
Gender	Female	212,304 (45.96%)
	Male	249,605 (54.04%)
Age	18-24	73,420 (15.89%)
	25-34	72,771 (15.75%)
	35-44	71,649 (15.51%)
	45-64	171,109 (37.04%)
	65+	72,960 (15.80%)
Marital Status	Married	222,113 (48.09%)
	Separated or Divorced	13,057 (2.83%)
	Single	35,346 (7.65%)
	Widowed	8,686 (1.88%)
	Unknown	182,707 (39.55%)
Clinic Branch	Army	200,295 (43.36%)
	Navy	78,187 (16.93%)
	Air Force	162,703 (35.22%)
	Other	20,724 (4.49%)
Beneficiary Category	Active Duty or Active Reserve	144,769 (31.34%)
	Dependent	190,318 (41.20%)
	Retiree	119,671 (25.91%)
	Other	7,151 (1.55%)
Treatment Group	Primary Care Only	300,575 (65.07%)
	PC and PCBH	43,095 (9.33%)
	PC and Specialty BH	81,273 (17.60%)
	PC, PCBH, and Specialty BH	36,966 (8.00%)
Baseline BMI	Primary Care Only	30.97 $\pm$ 6.22
	PC and PCBH	29.75 $\pm$ 6.53
	PC and Specialty BH	28.79 $\pm$ 5.71
	PC, PCBH, and Specialty BH	28.69 $\pm$ 5.67
Baseline MAP	Primary Care Only	97.10 $\pm$ 11.96
	PC and PCBH	95.20 $\pm$ 11.30
	PC and Specialty BH	94.68 $\pm$ 11.01
	PC, PCBH, and Specialty BH	93.59 $\pm$ 10.43

Note: Demographic information is determined by existing data in the Defense Enrollment Eligibility

Reporting System (DEERS) included as part of the routine data extraction by the SDD. Reported sample characteristics are dependent on accurate reporting and maintenance of DEERS.

Table 8. Summary of ANCOVAs for Treatment Group and Health Outcomes

Effects	Type III SS	<i>df</i>	<i>F</i>	<i>p</i>	Partial Eta ²
Treatment Group on Tobacco (<i>N</i> = 298,394)					
Treatment Group	8.355	3	36.62	< 0.001	< 0.001
Tobacco Baseline	7560.291	1	88539.4	< 0.001	0.229
Time	2.859	1	33.48	< 0.001	< 0.001
Age Group	553.978	7	926.81	< 0.001	0.021
Patient Sex	162.455	1	1902.53	< 0.001	0.006
Error	25478.378	298380			
Total	33166.661	298393			
Treatment Group on BMI (<i>N</i> = 461,909)					
Treatment Group	22786	3	23.00	< 0.001	< 0.001
BMI Baseline	9.90x10 ¹⁰	1	2.998x10 ⁸	< 0.001	0.998
Time	13150	1	39.82	< 0.001	< 0.001
Age Group	1066000	7	461.15	< 0.001	0.007
Patient Sex	1150	1	3.48	0.062	< 0.001
Error	1.53x10 ⁸	461894			
Total	9.92x10 ¹⁰	461907			
Treatment Group on MAP (<i>N</i> = 461,909)					
Treatment Group	25611.64	3	92.91	< 0.001	< 0.001
MAP Baseline	21477144.01	1	233744	< 0.001	0.336
Time	8325.35	1	90.61	< 0.001	< 0.001
Age Group	1262141.02	7	1962.34	< 0.001	0.029
Patient Sex	145192.09	1	1580.18	< 0.001	0.003
Error	42440347.13	461895			
Total	64444329.47	461908			

Note: Partial Eta² is a measure of effect size for each variable (Cohen et al., 2001). Effect sizes are classified as small (≥ 0.01), medium (≥ 0.06), and large (≥ 0.14).

Table 9. Summary of Post Hoc ANCOVAs for Treatment Group and Tobacco Use

Effects	Type III SS	<i>df</i>	<i>F</i>	<i>p</i>	Partial Eta ²
Treatment Group on Tobacco with CVD (<i>N</i> = 186,371)					
Treatment Group	0.909	3	5.76	0.001	< 0.001
Tobacco Baseline	3045.189	1	3045.189	< 0.001	0.237
Time	0.527	1	10.03	0.001	< 0.001
Age Group	25.356	7	68.95	< 0.001	0.003
Patient Sex	18.242	1	68.95	< 0.001	0.002
Error	9790.246	186357			
Total	12839.863	186370			
Treatment Group on Tobacco with Tobacco Use (<i>N</i> = 41,753)					
Treatment Group	5.418	3	8.46	< 0.001	0.001
Tobacco Baseline	6322.421	1	29628.3	< 0.001	0.415
Time	18.475	1	86.58	< 0.001	0.002
Age Group	8.817	7	5.90	< 0.001	0.001
Patient Sex	11.365	1	53.26	< 0.001	0.001
Error	8906.741	41739			
Total	15455.026	41752			
Treatment Group on Tobacco with Obesity (<i>N</i> = 47,949)					
Treatment Group	1.813	3	8.93	< 0.001	< 0.001
Tobacco Baseline	1149.539	1	16979.8	< 0.001	0.262
Time	0.001	1	0.01	0.924	< 0.001
Age Group	41.715	7	88.03	< 0.001	0.013
Patient Sex	27.855	1	411.45	< 0.001	0.009
Error	3245.213	47935			
Total	4407.986	47948			
Treatment Group on Tobacco with Depression (<i>N</i> = 39,841)					
Treatment Group	4.296	3	16.00	< 0.001	0.001
Tobacco Baseline	1046.966	1	11700.2	< 0.001	0.227
Time	0.005	1	0.06	0.805	< 0.001
Age Group	29.445	7	54.84	< 0.001	0.008
Patient Sex	23.387	1	261.36	< 0.001	0.007
Error	3563.916	39828			
Total	4628.672	39840			

Note: Partial Eta² is a measure of effect size for each variable (Cohen et al., 2001). Effect sizes are classified as small (≥ 0.01), medium (≥ 0.06), and large (≥ 0.14).

Table 10. Summary of Post Hoc ANCOVAs for Treatment Group and BMI

Effects	Type III SS	<i>df</i>	<i>F</i>	<i>p</i>	Partial Eta ²
Treatment Group on BMI with CVD (<i>N</i> = 284,078)					
Treatment Group	9323	3	12.17	< 0.001	< 0.001
BMI Baseline	9.90x10 ¹⁰	1	3.88x10 ⁸	< 0.001	0.993
Time	11703	1	45.84	< 0.001	< 0.001
Age Group	701902	7	392.72	< 0.001	0.010
Patient Sex	19136	1	74.95	< 0.001	< 0.001
Error	7.25x10 ⁷	284064			
Total	9.91x10 ¹⁰	284077			
Treatment Group on BMI with Tobacco Use (<i>N</i> = 58,452)					
Treatment Group	366.778	3	4.75	0.003	< 0.001
BMI Baseline	184855.111	1	7185.30	< 0.001	0.109
Time	2540.110	1	98.73	< 0.001	0.002
Age Group	3631.480	7	20.17	< 0.001	0.002
Patient Sex	44.879	1	44.88	0.187	< 0.001
Error	1.50x10 ⁶	58438			
Total	1.70x10 ⁶	58451			
Treatment Group on BMI with Obesity (<i>N</i> = 76,884)					
Treatment Group	12381.82	3	5.10	0.002	< 0.001
BMI Baseline	7.36x10 ⁷	1	90949.3	< 0.001	0.542
Time	12822.71	1	15.85	< 0.001	< 0.001
Age Group	381598.37	7	67.40	< 0.001	0.006
Patient Sex	47548.38	1	58.79	< 0.001	0.001
Error	6.21x10 ⁷	76870			
Total	1.36x10 ⁸	76883			
Treatment Group on BMI with Depression (<i>N</i> = 65,825)					
Treatment Group	1378.36	3	0.92	0.430	< 0.001
BMI Baseline	556633.85	1	1113.96	< 0.001	0.017
Time	13347.21	1	26.71	< 0.001	< 0.001
Age Group	20591.94	7	5.89	< 0.001	0.001
Patient Sex	375.67	1	0.75	0.386	< 0.001
Error	3.29x10 ⁷	65811			
Total	3.35x10 ⁷	65824			

Note: Partial Eta² is a measure of effect size for each variable (Cohen et al., 2001). Effect sizes are classified as small (≥ 0.01), medium (≥ 0.06), and large (≥ 0.14).

Table 11. Summary of Post Hoc ANCOVAs for Treatment Group and MAP

Effects	Type III SS	<i>df</i>	<i>F</i>	<i>p</i>	Partial Eta ²
Treatment Group on MAP with CVD (<i>N</i> = 284,079)					
Treatment Group	3965.33	3	15.71	< 0.001	< 0.001
MAP Baseline	1.68x10 ⁸	1	155984	< 0.001	0.354
Time	1867.88	1	17.39	< 0.001	< 0.001
Age Group	609273.53	7	810.20	< 0.001	0.020
Patient Sex	8995.89	1	83.74	< 0.001	< 0.001
Error	3.05x10 ⁷	284065			
Total	4.74x10 ⁷	284078			
Treatment Group on MAP with Tobacco Use (<i>N</i> = 58,452)					
Treatment Group	762.05	3	3.18	0.023	< 0.001
MAP Baseline	2266731.01	1	28343.3	< 0.001	0.327
Time	8836.90	1	110.50	< 0.001	0.002
Age Group	56211.68	7	100.41	< 0.001	0.012
Patient Sex	44900.84	1	561.44	< 0.001	0.010
Error	4.67x10 ⁶	58438			
Total	7.02x10 ⁶	58451			
Treatment Group on MAP with Obesity (<i>N</i> = 76,885)					
Treatment Group	944.05	3	4.30	0.005	< 0.001
MAP Baseline	2.94x10 ⁶	1	40205.7	< 0.001	0.343
Time	3526.91	1	48.20	< 0.001	0.001
Age Group	279416.93	7	545.49	< 0.001	0.047
Patient Sex	50636.81	1	691.99	< 0.001	0.009
Error	5.63x10 ⁶	76871			
Total	8.66x10 ⁶	76884			
Treatment Group on MAP with Depression (<i>N</i> = 65,825)					
Treatment Group	2838.08	3	12.77	< 0.001	0.001
MAP Baseline	2.28x10 ⁶	1	30761.2	< 0.001	0.319
Time	8574.07	1	115.78	< 0.001	0.002
Age Group	129321.93	7	249.47	< 0.001	0.026
Patient Sex	74610.82	1	1007.49	< 0.001	0.015
Error	4.87x10 ⁶	65811			
Total	7.19x10 ⁶	65824			

Note: Partial Eta² is a measure of effect size for each variable (Cohen et al., 2001). Effect sizes are classified as small (≥ 0.01), medium (≥ 0.06), and large (≥ 0.14).

FIGURES

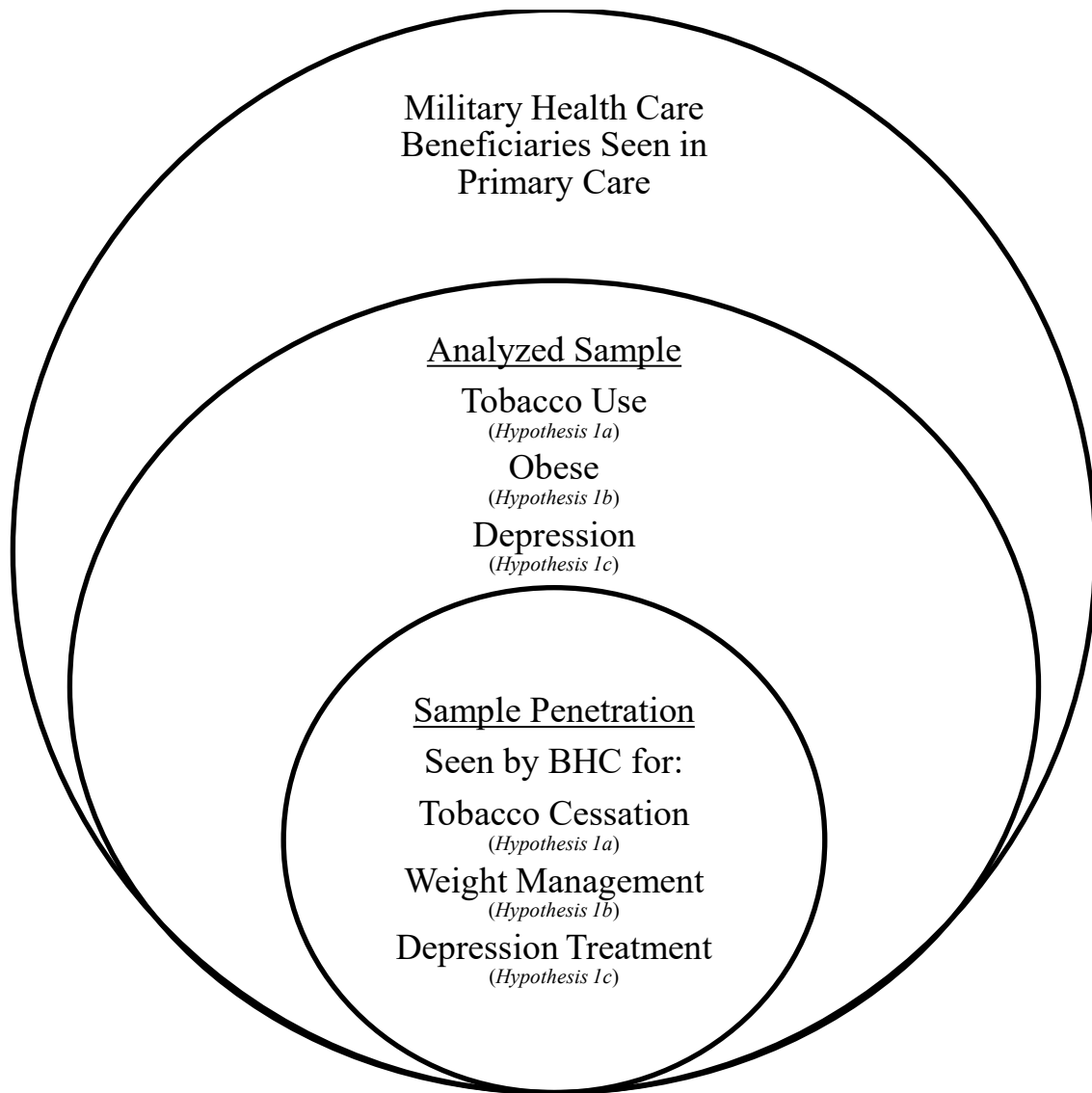


Figure 1. BHC Penetration in Tobacco Use, Obesity, and Depression. For Specific Aim 1, the total analyzed sample for each hypothesis included military health care beneficiaries seen in primary care with tobacco use, obesity, or depression. From that analyzed sample, penetration rates included all individuals seen by a BHC for tobacco cessation, weight management, or depression treatment.

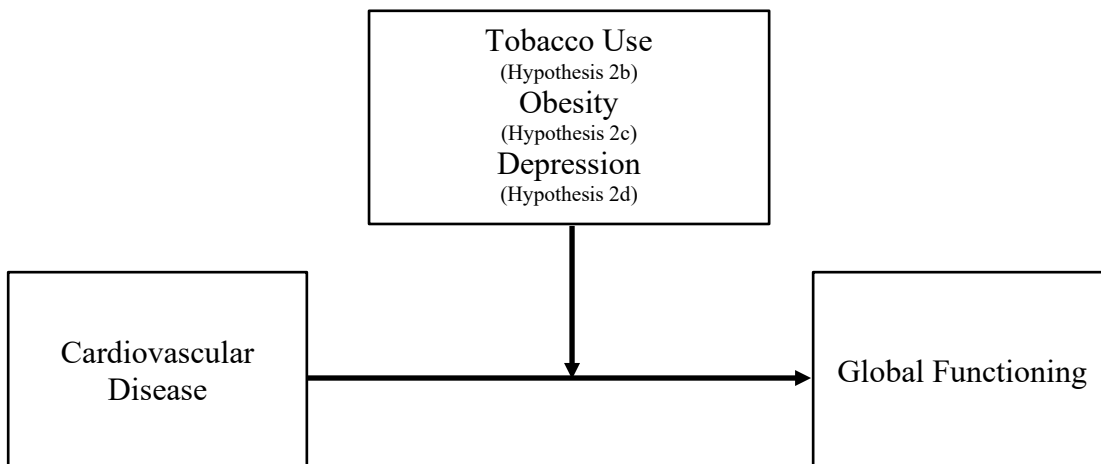
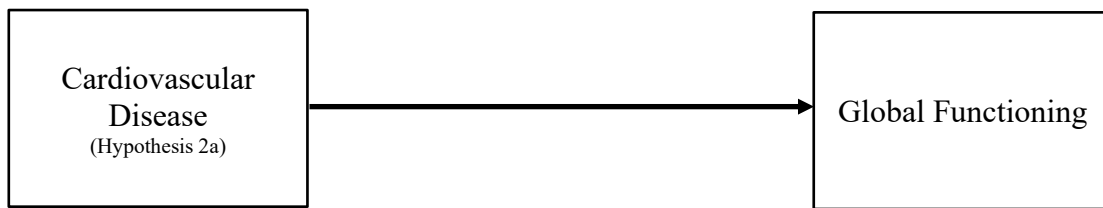


Figure 2. Conceptual Models for Specific Aim 2. The first hypothesis analyzed the main effect of CVD on global functioning. The remaining hypotheses focused on the interaction effect between CVD and tobacco use, obesity, or depression; conceptually, this moderation effect suggests the impact of CVD on global functioning is different in the presence of tobacco use, obesity, or depression.

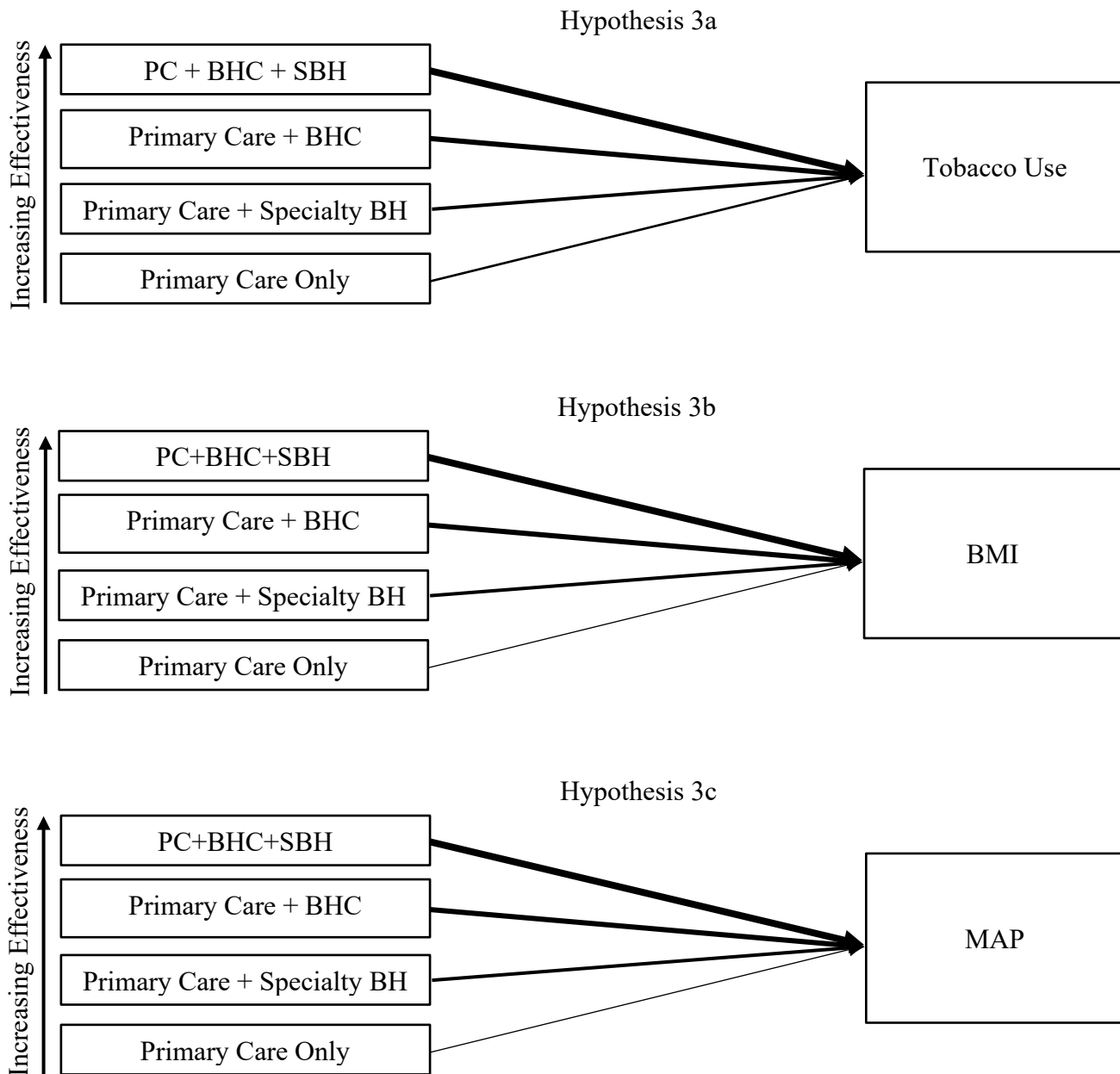


Figure 3. Conceptual Models for Specific Aim 3. Each hypothesis evaluated the predictive value of an episode of care with primary care (PC), behavioral health consultation (BHC), or specialty behavioral health (SBH) in improving tobacco use, BMI, and MAP. For each, an episode of care with all treatment types was hypothesized to show the greatest predictive value, followed by PC with BHC, PC with SBH, and PC only.

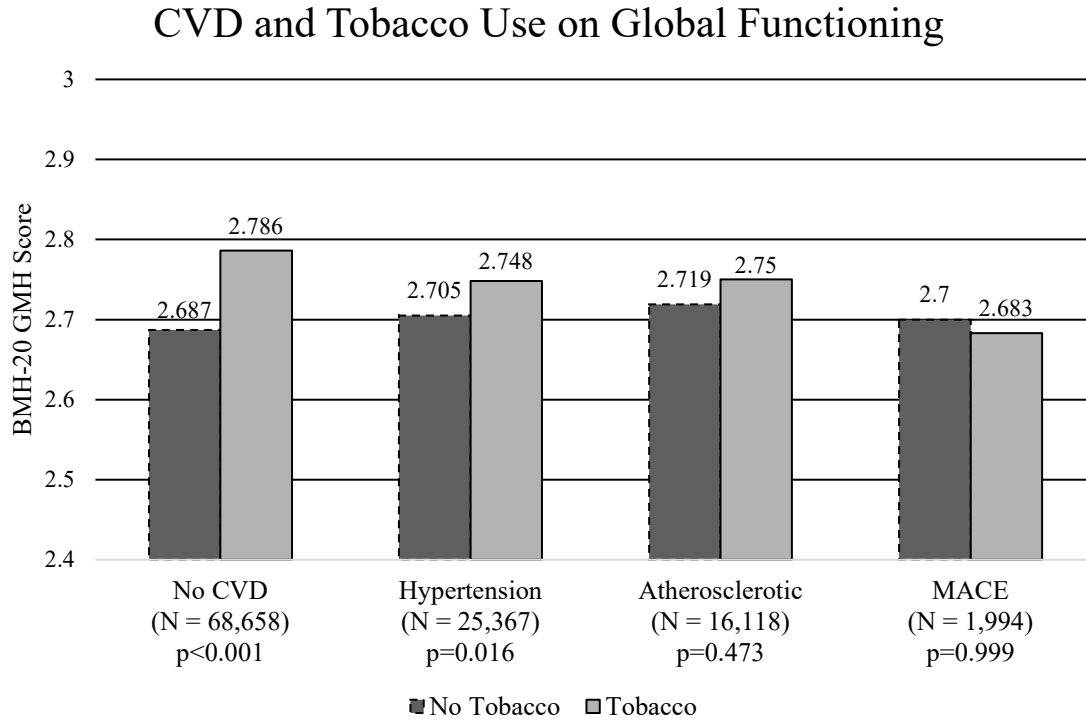


Figure 4. Mean BHM-20 GMH Scores for CVD and Tobacco Use, Aim 2b.

The interaction between CVD severity and tobacco use is significant ($p < 0.001$, $\eta_p^2 < 0.001$). The difference between smokers and no smokers was significant for No CVD ($p < 0.001$) and Hypertension ($p = 0.016$) but not Atherosclerotic CVD ($p = 0.473$) and MACE ($p = 0.999$). Note, the independent variable is BMH-20 GMH scores, which are categorized as *Normal* (2.94 to 4.00), *Mild* (2.38 to 2.93), *Moderate* (2.08 to 2.37), *Severe* (0 to 2.08). The above means are in the *Mild* range.

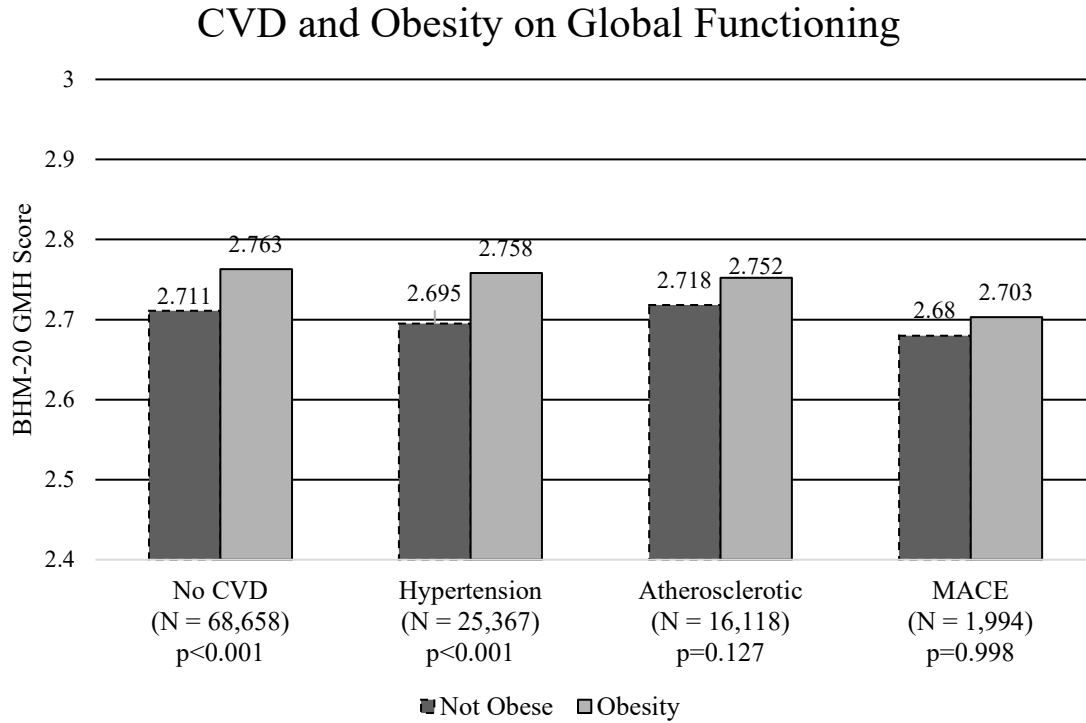


Figure 5. Mean BHM-20 GMH Scores for CVD and Obesity, Aim 2c.

The interaction between CVD severity and obesity is not significant ($p = 0.250$, $\eta_p^2 < 0.001$). The difference between individuals with and without obesity was significant for No CVD ($p < 0.001$) and Hypertension ($p < 0.001$) but not Atherosclerotic CVD ($p = 0.127$) and MACE ($p = 0.998$). Note, the independent variable is BMH-20 GMH scores, which are categorized as *Normal* (2.94 to 4.00), *Mild* (2.38 to 2.93), *Moderate* (2.08 to 2.37), *Severe* (0 to 2.08). The above means are in the *Mild* range.

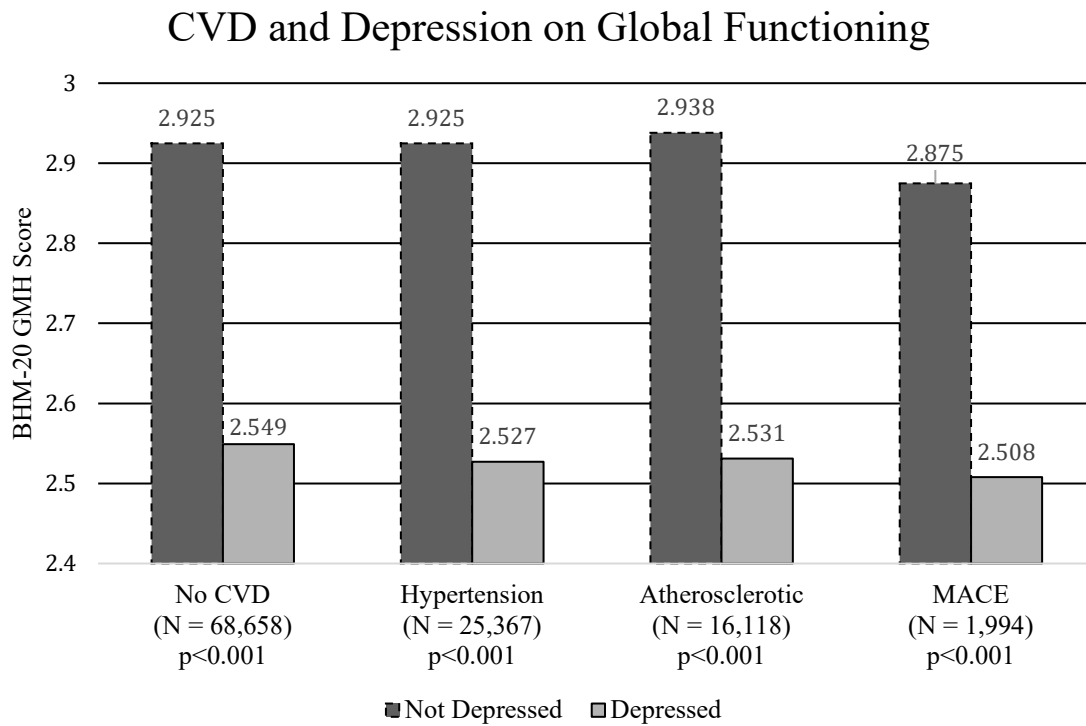


Figure 6. Mean BHM-20 GMH Scores for CVD and Global Functioning, Aim 2d. The interaction between CVD severity and depression is significant ($p = 0.027$, $\eta_p^2 < 0.001$). The difference between individuals with and without depression was significant at all levels of CVD severity ($p < 0.001$). Note, the independent variable is BMH-20 GMH scores, which are categorized as *Normal* (2.94 to 4.00), *Mild* (2.38 to 2.93), *Moderate* (2.08 to 2.37), *Severe* (0 to 2.08). The above means are in is the *Normal* to *Mild* range.

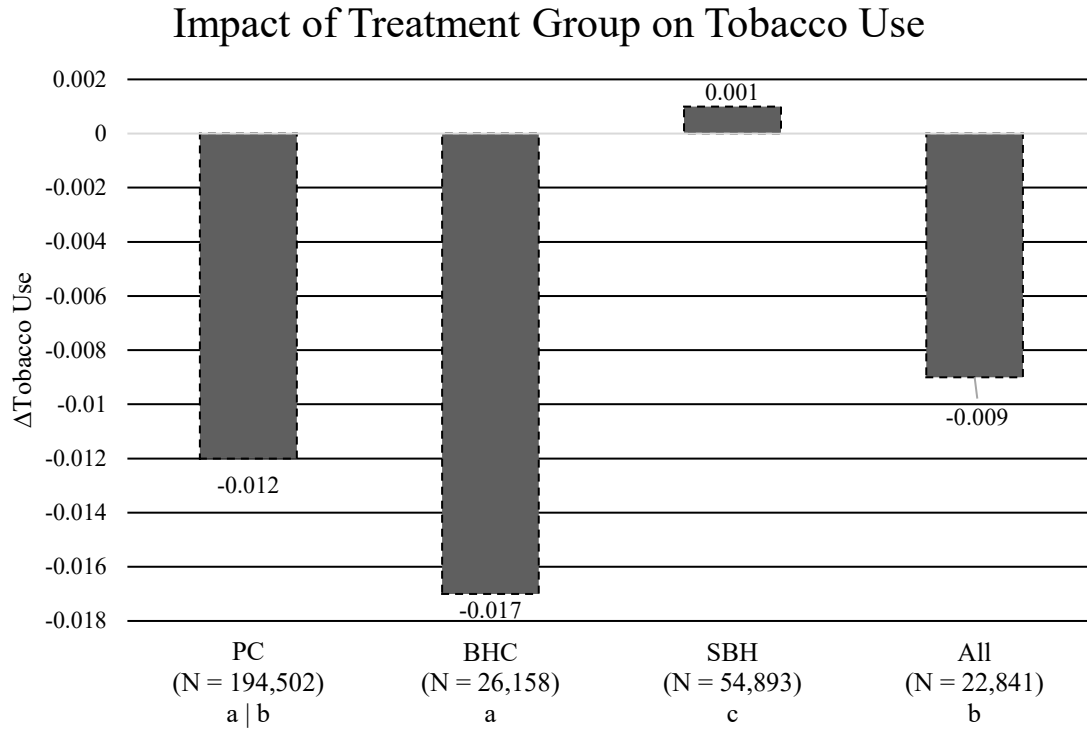


Figure 7. Impact of Treatment Group on Tobacco Use, Aim 3a.

The main effect of treatment group on tobacco use was significant ($p < 0.001$, $\eta_p^2 < 0.001$). The difference between treatment groups was significant for PC vs SBH ($p < 0.001$), BHC vs SBH ($p < 0.001$), BHC vs All ($p = 0.017$), and SBH vs All ($p < 0.001$). Bars that share a letter we not significantly different. Note, the independent variable is change in reported tobacco use, so more negative scores resemble better health outcomes.

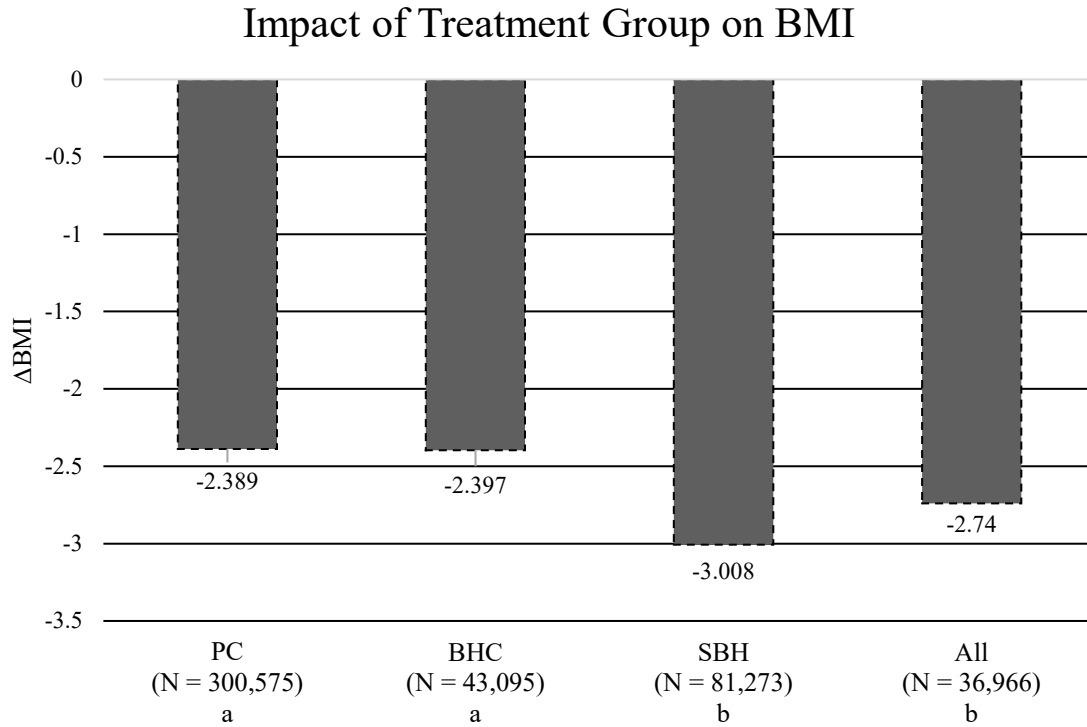


Figure 8. Impact of Treatment Group on BMI, Aim 3b.

The main effect of treatment group on BMI was significant ($p < 0.001$, $\eta_p^2 < 0.001$). The difference between treatment groups was significant for PC vs SBH ($p < 0.001$), PC vs All ($p = 0.005$), BHC vs SBH ($p < 0.001$), and BHC vs All ($p = 0.043$). Bars that share a letter are not significantly different. Note, the independent variable is change in body mass index, so more negative scores resemble better health outcomes.

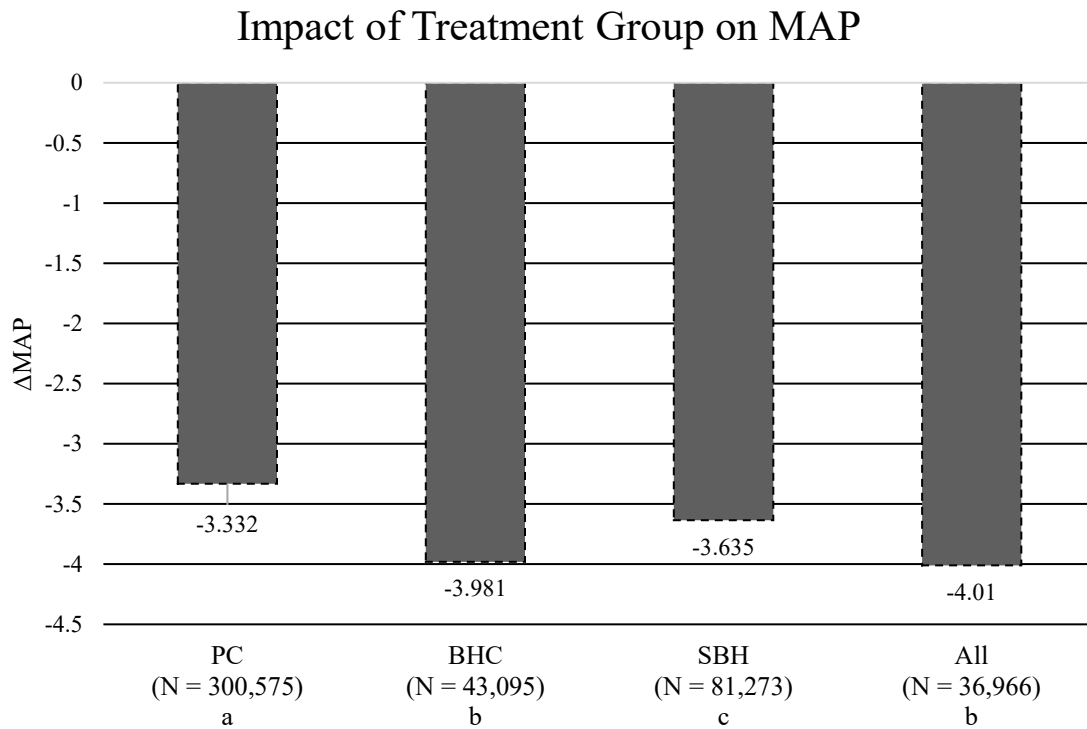


Figure 9. Impact of Treatment Group one MAP, Aim 3c.

The main effect of treatment group on MAP was significant ($p < 0.001$, $\eta_p^2 < 0.001$). The difference between treatment groups was significant for all comparisons ($p < 0.001$) except BHC vs All ($p = 0.977$). Bars that share a letter we not significantly different. Note, the independent variable is change in mean arterial pressure, so more negative scores resemble better health outcomes.

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APPENDIX

SAS Syntax Aim 2 ANCOVA Analysis

```
347 libname ds
"C:\Users\dzhaosi\projects\Wilfong code
review\Aim2retry";
348
349 *proc contents data=ds.aim2;
350 run;
351 /*
352 Alphabetic List of Variables and
Attributes
353 # Variable Type Len
354 5 CVDflag Num 8
355 2 VALUE Char 16
356 4 c_med Char 1
357 3 d_med Char 1
358 8 depressionflag Num 8
359 1 newid Char 16
360 7 obesityflag Num 8
361 6 tobaccoflag Num 8
362 */
363
364 data aim2;
365 set ds.aim2;
366 *value is DV, delete rows without
VALUES*/
367 if newid = '1PPNALQ3QP' then
value = 1.33;
368 if newid = '9PZU0LZJ9Z' then
value = 3;
369 if newid = 'EDZ46F6OT5' then
value = 2.7;
370 if newid = 'EVJ0A4D18E' then
value = 1.67;
371 if newid = 'KHFQ5CIJEI' then
value = 3.7;
372 if newid = 'MU8HQBYYWMK' then
value = 2.33;
373 if newid = 'Q6XSF3QPW3' then
value = 2.40;
374 if newid = 'SRQTLL50MD' then
value = 1.70;
375 if newid = 'W9N9F6DU2O' then
value = 4;
376 if newid = 'WPW37TG891' then
value = 1.33;
377 if newid = 'YVZ7LXGOI6' then
value = 1.33;
378 if newid = 'ZVXOOATXFP' then
value = 0.33;
379 if newid = 'PO5OW1XIQ2' then
value = 1.33;
380 BHM20 = value * 1;
381 if BHM20 ne .;
382
383 *setting 1 or 0 dummay values;
384 c_med2=0;
385 if c_med = 'Y' then c_med2=1;
386
387 d_med2=0;
388 if d_med = 'Y' then d_med2=1;
389
390 depressionflag2 = 0;
391 if depressionflag = 1 then
depressionflag2 = 1;
392 obesityflag2 = 0;
393 if obesityflag = 1 then obesityflag2
= 1;
394 tobaccoflag2 = 0;
395 if tobaccoflag = 1 then tobaccoflag2
= 1;
396 cvdflag2 = 0;
397 if cvdflag = 1 then cvdflag2 = 1;
398
399 run;
400 /*
401 NOTE: There were 146786
observations read from the data set
WORK.AIM2.
402 NOTE: The data set WORK.AIM2
has 146786 observations and 19
variables.
403 */
404
405 *getting bhc from AIM3;
406 proc sort data=aim2; by newid; run;
```

```

407 proc sort data=ds.aim3_merge
out=BHC nodupkey; where BHC=1; by
newid; run;
408 /*
409 NOTE: There were 1561685
observations read from the data set
DS.AIM3_MERGE.
410 WHERE BHC=1;
411 NOTE: 1371188 observations with
duplicate key values were deleted.
412 (!!!ds note: may need to look into
what records being deleted here)
413 NOTE: The data set WORK.BHC
has 190497 observations and 28
variables.
414 */
415
416
417 data aim2_BHC;
418 merge aim2 (in=a) bhc (in=b);
419 by newid;
420 if a=1 and b=1;
421
422 agegrp2=.;
423 if AGEGRP = "A" then
AGEGRP2 = 1;
424 if AGEGRP = "B" then
AGEGRP2 = 2;
425 if AGEGRP = "C" then
AGEGRP2 = 3;
426 if AGEGRP = "D" then
AGEGRP2 = 4;
427 if AGEGRP = "E" then
AGEGRP2 = 5;
428 if AGEGRP = "F" then
AGEGRP2 = 6;
429 if AGEGRP = "G" then
AGEGRP2 = 7;
430 if AGEGRP = "H" then
AGEGRP2 = 8;
431
432 if AGEGRP = "X" then delete;
433
434 if PATSEX = "F" then patsex2 =
1;

```

```

435 else if PATSEX = "M" then
patsex2 = 0;
436 else delete;
437
438 run;
439 /*
440 NOTE: There were 146786
observations read from the data set
WORK.AIM2.
441 NOTE: There were 190497
observations read from the data set
WORK.BHC.
442 NOTE: The data set
WORK.AIM2_BHC has 112200
observations and 40 variables.
443 */
444
445 data aim2_bhc;*aim2_bhc2;
446 set aim2_bhc;
447 if 0 < bhm20 < 2038; /*remove
extrem BHM20 values*/
448 * if 0 < bhm20 < 40; /*remove
extrem BHM20 values*/
449
450 /*
451 assign people with cardiovascular
disease to the category reflecting
452 their MOST severe CV issue
(in descending order):
453 Level 4 - Major Adverse Cardiac
Events
454 Level 3 - Atherosclerotic and Other
CVD
455 Level 2 - Hypertensive Heart
Disease
456 Level 1 ? Control
457 */
458
459 if majorcardiacflag = 1 then
CVD_level = 4;
460 else if atherosclflag = 1 then
CVD_level = 3;
461 else if hypertensionflag = 1 then
CVD_level = 2;
462 else CVD_level = 1;
463

```

```

464 keep newid bhm20 cvd_level
cvdflag2 tobaccoflag2 obesityflag2
465 depressionflag2 c_med2 d_med2
agegrp2 patsex2;
466
467 run;
468 /*
469 NOTE: There were 112200
observations read from the data set
WORK.AIM2_BHC.
470 NOTE: The data set
WORK.AIM2_BHC has 112137
observations and 40 variables.
471 NOTE: The data set
WORK.AIM2_BHC2 has 112136
observations and 40 variables.
472 */
473
474 data ds.aim2_model_data;
475 set aim2_bhc;
476 run;
477
478 proc export data =
ds.aim2_model_data
479 outfile =
"C:\Users\dzhaosi\projects\Wilfong code
review\aim2retry\aim2modeldata.csv"
480 dbms=csv
481 replace;
482 run;
483
484 * PRODUCT: SAS
485 * VERSION: 9.4
486 * CREATOR: External File
Interface
487 * DATE: 08JUN21
488 * DESC: Generated SAS
Datastep Code
489 * TEMPLATE SOURCE: (None
Specified.)
490
491 data _null_;
492 %let _EFIERR_ = 0; /* set the
ERROR detection macro variable */
493 %let _EFIREC_ = 0; /* clear
export record count macro variable */

```

```

494 file
'C:\Users\dzhaosi\projects\Wilfong code
review\aim2retry\aim2modeldata.csv'
494! delimiter=',' DSD DROPOVER
lrec1=32767;
495 if _n_ = 1 then /* write
column names or labels */
496 do;
497 put
498 "newid"
499 ','
500 "BHM20"
501 ','
502 "c_med2"
503 ','
504 "d_med2"
505 ','
506 "depressionflag2"
507 ','
508 "obesityflag2"
509 ','
510 "tobaccoflag2"
511 ','
512 "cvdflag2"
513 ','
514 "agegrp2"
515 ','
516 "patsex2"
517 ','
518 "CVD_level"
519 ;
520 end;
521 set DS.AIM2_MODEL_DATA
end=EFIEOD;
522 format newid $16. ;
523 format BHM20 best12. ;
524 format c_med2 best12. ;
525 format d_med2 best12. ;
526 format depressionflag2 best12.
;
527 format obesityflag2 best12. ;
528 format tobaccoflag2 best12. ;
529 format cvdflag2 best12. ;
530 format agegrp2 best12. ;
531 format patsex2 best12. ;
532 format CVD_level best12. ;

```

```

533 do;
534     EFIOUT + 1;
535     put newid $ @;
536     put BHM20 @;
537     put c_med2 @;
538     put d_med2 @;
539     put depressionflag2 @;
540     put obesityflag2 @;
541     put tobaccoflag2 @;
542     put cvdflag2 @;
543     put agegrp2 @;
544     put patsex2 @;
545     put CVD_level ;
546     ;
547 end;
548 if _ERROR_ then call
symputx('_EFIERR_',1); /* set ERROR
detection macro variable */
549 if EFIEOD then call
symputx('_EFIREC_',EFIOUT);
550 run;
551
552 %macro sampleout;
553 /*random sample 1k*/
554 proc sql OUTOBS=1000;
555 create table aim2sample1k as
556 select *
557 from aim2_bhc
558 order by RANUNI(4321);
559 quit;
560
561
562 data ds.aim2sample1k;
563 set aim2sample1k;
564 run;
565
566 proc export data = aim2sample1k
567     outfile =
"C:\Users\dzhaosi\projects\Wilfong code
review\aim2retry\aim2datasample1k.csv
"
568     dbms=csv
569     replace;
570 run;
571 %mend sampleout;

2051 libname ds
"C:\Users\dzhaosi\projects\Wilfong code
review\Aim2retry";
2052
2053 *proc contents
data=ds.aim2_model_data;
2054 run;
2055 /*
2056 Data Set Name
DS.AIM2_MODEL_DATA
Observations 112137
2057 # Variable Type Len
2058 2 BHM20 Num 8
2059 14 CVD_level Num 8
2060 12 agegrp2 Num 8
2061 3 c_med2 Num 8
2062 8 cvdflag2 Num 8
2063 4 d_med2 Num 8
2064 5 depressionflag2 Num 8
2065 1 newid Char 16
2066 6 obesityflag2 Num 8
2067 13 patsex2 Num 8
2068 7 tobaccoflag2 Num 8
2069 */
2070
2071 /*-----Multicollinearity-----
-----*/
2072
2073 *%macro corROUT;
2074 proc CORR
data=ds.aim2_model_data;
2075 var bhm20 cvd_level tobaccoflag2
obesityflag2 depressionflag2
2076     d_med2 c_med2 agegrp2
patsex2;
2077 title "Correlation Coefficients";
2078 run;
2079 *%mend corROUT;
2080 /*
2081 Pearson Correlation Coefficients,
N = 112137
2082 BHM20 CVD_level tobaccoflag2
obesityflag2 depressionflag2
2083     d_med2 c_med2 agegrp2
patsex2

```

2084 BHM20 VS CVD_LEVEL
 0.08877 <.0001
 2085 BHM20 VS TOBACCOFLAG2
 0.07580 <.0001
 2086 BHM20 VS OBESITYFLAG2
 0.04992 <.0001
 2087 BHM20 VS
 DEPRESSIONFLAG2 -0.33133 <.0001
 2088 BHM20 VS D_MED2 -0.21537
 <.0001
 2089 BHM20 VS C_MED2 0.01640
 <.0001
 2090 BHM20 VS AGEGRP2 0.10948
 <.0001
 2091 BHM20 VS PATSEX2 -0.13079
 <.0001 (patsex2 F/1, M/2)
 2092
 2093 CVD_LEVEL VS
 TOBACCOFLAG2 -0.11012 <.0001
 2094 CVD_LEVEL VS
 OBESITYFLAG2 -0.05263 <.0001
 2095 CVD_LEVEL VS
 DEPRESSIONFLAG -0.15161 <.0001
 2096 CVD_LEVEL VS D_MED2 -
 0.00968 0.0012
 2097 CVD_LEVEL VS C_MED2
 0.20770 <.0001
 2098 CVD_LEVEL VS AGEGRP2
 0.42522 <.0001
 2099 CVD_LEVEL VS PATSEX2 -
 0.03453 <.0001
 2100
 2101 TOBACCOFLAG2 VS
 OBESITYFLAG2 -0.10164 <.0001
 2102 TOBACCOFLAG2 VS
 DEPRESSIONFLAG2 -0.12880 <.0001
 2103 TOBACCOFLAG2 VS D_MED2
 -0.02581 <.0001
 2104 TOBACCOFLAG2 VS C_MEDS
 -0.01792 <.0001
 2105 TOBACCOFLAG2 VS
 AGEGRP2 -0.10651 <.0001
 2106 TOBACCOFLAG2 VS PATSEX2
 -0.17797 <.0001
 2107

2108 OBESITYFLAG2 VS
 DEPRESSIONFLAG2 -0.10934 <.0001
 2109 OBESITYFLAG2 VS D_MED2 -
 0.03590 <.0001
 2110 OBESITYFLAG2 VS C_MED2
 0.03662 <.0001
 2111 OBESITYFLAG2 VS AGEGRP2
 -0.03451 <.0001
 2112 OBESITYFLAG2 VS PATSEX2
 0.05428 <.0001
 2113
 2114 DEPRESSIONFLAG2 VS
 D_MED2 0.29971 <.0001
 2115 DEPRESSIONFLAG2 VS
 C_MED2 -0.05043 <.0001
 2116 DEPRESSIONFLAG2 VS
 AGEGRP2 -0.11121 <.0001
 2117 DEPRESSIONFLAG2 VS
 PATSEX2 0.17992 <.0001
 2118
 2119 D_MED2 VS C_MED2 0.11272
 <.0001
 2120 D_MED2 VS AGEGRP2 0.02579
 <.0001
 2121 D_MED2 VS PATSEX2 0.15815
 <.0001
 2122
 2123 C_MED2 VS AGEGRP2 0.24603
 <.0001
 2124 C_MED2 VS PATSEX2 -0.00125
 0.6760
 2125
 2126 AGEGRP2 VS PATSEX2
 0.01292 <.0001
 2127 */
 2128
 2129
 2130 /*-----GLM ANCOVA with
 Post Hoc(Tukey Kramer)-----*/
 2131
 2132 proc glm
 data=ds.aim2_model_data;
 2133
 2134 class cvd_level tobaccoflag2
 obesityflag2 depressionflag2

```

2135          c_med2 d_med2 agegrp2
patsex2;
2136
2137 model bhm20 = cvd_level
tobaccoflag2 obesityflag2
depressionflag2
2138          cvd_level*tobaccoflag2
cvd_level*obesityflag2
cvd_level*depressionflag2
2139          c_med2 d_med2 agegrp2
patsex2;
2140
2141 lsmeans cvd_level tobaccoflag2
obesityflag2 depressionflag2
2142          cvd_level*tobaccoflag2
cvd_level*obesityflag2
cvd_level*depressionflag2
2143          c_med2 d_med2 agegrp2
patsex2
2144          /ADJUST=TUKEY
pdiff;* stderr cl cov;
2145          *Post Hoc (Tukey-
Kramer) test;
2146
2147 means cvd_level tobaccoflag2
obesityflag2 depressionflag2
2148          cvd_level*tobaccoflag2
cvd_level*obesityflag2
cvd_level*depressionflag2
2149          c_med2 d_med2 agegrp2
patsex2
2150          /hovtest=levene welch;
2151          /*Homogeneity of
variance: BAR, BARTLETT, BF,
LEVENE, OBRIEN*/
2152          *WARNING:
Homogeneity of variance testing and
Welch's ANOVA
2153          are only available for
unweighted one-way models;
2154
2155 OUTPUT OUT = pred p=ybar
r=resid;
2156
2157 title "aim2 model:
4main+3x+4cv";

```

```

2158
2159 run;
2161 /*
2162 Source DF Sum of Squares Mean
Square F Value Pr > F
2163 Model 25 8143.85804 325.75432
733.06 <.0001
2164
2165 R-Square Coeff Var Root MSE
BHM20 Mean
2166 0.140500 24.93823 0.666615
2.673064
2167 */
2168
2169 /*-----QQplot and Shapiro-
Wilk test for normality-----*/
2170
2171 %macro normout;
2172
2173 proc sql OUTOBS=2000;
2174 create table pred2k as
2175 select *
2176 from pred
2177 order by RANUNI(6789);
2178 quit;
2179
2180 PROC UNIVARIATE
data=pred2k normal;
2181 var resid;
2182 QQPLOT resid / normal(mu=est
sigma=est);
2183 title "aim2mdl glm pred2k:
Normal Q-Q Plot of Residuals and
Shapiro-Wilk test";
2184 run;
2185 /*pred2k -- Tests for Normality
2186 Test Statistic p Value
2187 Shapiro-Wilk W 0.992932 Pr < W
<0.0001
2188
2189 p-values <0.05, reject H0 that
residual is normally distributed.
2190 */
2191
2192 %mend normout;

```

SAS Syntax Aim 3 ANCOVA Analysis

```

1  libname ds
"C:\Users\dzhaosi\projects\Wilfong code
review\Aim3retry";
2
3  *proc contents
data=ds.aim3_model_data;run;
4  /*
5  Alphabetic List of Variables and
Attributes
6  # Variable Type Len Label
7  16 BMI_baseline Num 8
8  13 BMI_days_final Num 8
9  10 BMI_delta Num 8
10  3 CVDflag Num 8
11  12 MAP_days_final Num 8
12  9 MAP_delta Num 8
13  2 PATSEX Char 1 Patient Gender
14  17 TOB_baseline Num 8
15  8 agegrp2 Num 8
16  7 cohort Num 8
17  6 depressionflag Num 8
18  15 map_baseline Num 8
19  1 newid Char 16
20  5 obesityflag Num 8
21  14 tobacco_days_final Num 8
22  11 tobacco_delta Num 8
23  4 tobaccoflag Num 8
24  */
25
26  data aim3mdl;
27  set ds.aim3_model_data;
28  if patsex = "F" then patsex2 = 0;
29  if patsex = "M" then patsex2 = 1;
30  run;
31
32  /*-----Multicollinearity-----
-----*/
33
34  *%macro corrout;
35  proc corr data=aim3mdl;
36
37  var cohort AGEGRP2 PATSEX2
38      map_baseline map_days_final
39      BMI_baseline BMI_days_final
40      tob_baseline tobacco_days_final
41      ;
42
43  title "aim3: Correlation
Coefficients";
44  run;
45  *%mend corrout;
46
47
48  /*-----GLM ANCOVA with
Post Hoc(Tukey Kramer)-----*/
49  *%macro glmout;
50
51  proc glm data=aim3mdl;
52
53  class cohort AGEGRP2 PATSEX2;
54
55  model bmi_delta = bmi_baseline
bmi_days_final cohort AGEGRP2
PATSEX2;
56
57  lsmeans cohort agegrp2 patsex2
58      /ADJUST=TUKEY pdiff;
59      *Post Hoc (Tukey-Kramer)
test;
60
61  means cohort AGEGRP2 PATSEX2
62      /hovtest=levene welch;
63      /*Homogeneity of
variance: BAR, BARTLETT, BF,
LEvene, OBRIEN*/
64      *WARNING:
Homogeneity of variance testing and
Welch's ANOVA
65      are only available for
unweighted one-way models;
66
67  OUTPUT OUT = pred p=ybar
r=resid;
68

```

```

69 title "aim3mdl bmi_delta";
70
71 run;
72 quit;
73
74 /*
75 Source DF Sum of Squares Mean
Square F Value Pr > F
76 Model 13 99007470014
7615959232 2.306E7 <.0001
77
78 R-Square Coeff Var Root MSE
BMI_delta Mean
79 0.998462 -4781.423 18.17233 -
0.380061
80 */
81 %mend glmout;
82
83
84 /*-----QQplot and Shapiro-
Wilk test for normality-----*/
85
86 %macro normout;
87
88 proc sql OUTOBS=2000;
89 create table pred2k as
90 select *
91 from pred
92 order by RANUNI(6789);
93 quit;
94
95 PROC UNIVARIATE data=pred2k
normal;
96 var resid;
97 QQPLOT resid / normal(mu=est
sigma=est);
98 title "aim3mdl bmidelta pred2k:
Normal Q-Q Plot of Residuals and
Shapiro-Wilk test";
99 run;
100 /*pred2k -- Tests for Normality
101 Test Statistic p Value
102 Shapiro-Wilk W 0.30581 Pr < W
<0.0001
103
104 p-values <0.05, reject H0 that
residual is normally distributed.
105 */
106
107 %mend normout;
108
109
110 /*-----Additional models
based on type of disease-----*/
111
112 %macro runby(condition);
113
114 proc glm data=aim3mdl;
115
116 class cohort AGEGRP2 PATSEX2;
117
118 model bmi_delta = bmi_baseline
bmi_days_final cohort AGEGRP2
PATSEX2;
119
120 where &condition = 1;
121
122 lsmeans cohort agegrp2 patsex2
123 /ADJUST=TUKEY pdiff;
124 *Post Hoc (Tukey-Kramer)
test;
125
126 OUTPUT OUT = pred p=ybar
r=resid;
127
128 title "aim3 bmi_delta model with
&&condition = ture";
129
130 run;
131 quit;
132 %mend runby;
133 %runby(cvdflag)
134 %runby(tobaccoflag);
135 %runby(obesityflag);
136 %runby(depressionflag);
137 libname ds
"C:\Users\dzhaosi\projects\Wilfong code
review\Aim3retry";
138
139 *proc contents
data=ds.aim3_model_data;run;

```

```

140 /*
141 Alphabetic List of Variables and
Attributes
142 # Variable Type Len Label
143 16 BMI_baseline Num 8
144 13 BMI_days_final Num 8
145 10 BMI_delta Num 8
146 3 CVDflag Num 8
147 12 MAP_days_final Num 8
148 9 MAP_delta Num 8
149 2 PATSEX Char 1 Patient Gender
150 17 TOB_baseline Num 8
151 8 agegrp2 Num 8
152 7 cohort Num 8
153 6 depressionflag Num 8
154 15 map_baseline Num 8
155 1 newid Char 16
156 5 obesityflag Num 8
157 14 tobacco_days_final Num 8
158 11 tobacco_delta Num 8
159 4 tobaccoflag Num 8
160 */
161
162 data aim3mdl;
163 set ds.aim3_model_data;
164 if patsex = "F" then patsex2 = 0;
165 if patsex = "M" then patsex2 = 1;
166 run;
167
168 /*-----Multicollinearity-----
-----*/
169
170 /*%macro corROUT;
171 proc CORR data=aim3mdl;
172
173 var cohort AGEGRP2 PATSEX2
174     map_baseline map_days_final
175     BMI_baseline BMI_days_final
176     tob_baseline tobacco_days_final
177     ;
178
179 title "aim3: Correlation
Coefficients";
180 run;
181 /*%mend corROUT;
182

```

```

183
184 /*-----GLM ANCOVA with
Post Hoc(Tukey Kramer)-----*/
185 /*%macro glmOUT;
186
187 proc GLM data=aim3mdl;
188
189 class cohort AGEGRP2 PATSEX2;
190
191 model map_delta = map_baseline
map_days_final cohort AGEGRP2
PATSEX2;
192
193 lsmeans cohort agegrp2 patsex2
194     /ADJUST=TUKEY pdiff;
195     *Post Hoc (Tukey-Kramer)
test;
196
197 means cohort AGEGRP2
PATSEX2
198     /hovtest=levene welch;
199     /*Homogeneity of
variance: BAR, BARTLETT, BF,
LEVENE, OBRIEN*/
200     *WARNING:
Homogeneity of variance testing and
Welch's ANOVA
201     are only available for
unweighted one-way models;
202
203 OUTPUT OUT = pred p=ybar
r=resid;
204
205 title "aim3 MAP_delta model";
206
207 run;
208 quit;
209
210 /*
211 Source DF Sum of Squares Mean
Square F Value Pr > F
212 Model 13 99007470014
7615959232 2.306E7 <.0001
213
214 R-Square Coeff Var Root MSE
BMI_delta Mean

```

```

215 0.998462 -4781.423 18.17233 -
0.380061
216 */
217 *%mend glmout;
218
219
220 /*-----QQplot and Shapiro-
Wilk test for normality-----*/
221
222 *%macro normout;
223
224 proc sql OUTOBS=2000;
225 create table pred2k as
226 select *
227 from pred
228 order by RANUNI(6789);
229 quit;
230
231 PROC UNIVARIATE data=pred2k
normal;
232 var resid;
233 QQPLOT resid / normal(mu=est
sigma=est);
234 title "aim3mdl MAPdelta pred2k:
Normal Q-Q Plot of Residuals and
Shapiro-Wilk test";
235 run;
236 /*pred2k -- Tests for Normality
237 Test Statistic p Value
238 Shapiro-Wilk W 0.30581 Pr < W
<0.0001
239
240 p-values <0.05, reject H0 that
residual is normally distributed.
241 */
242
243 *%mend normout;
244
245
246 /*-----Additional models
based on type of disease-----*/
247
248 %macro runby(condition);
249
250 proc glm data=aim3mdl;
251

```

```

252 class cohort AGEGRP2 PATSEX2;
253
254 model map_delta = map_baseline
map_days_final cohort AGEGRP2
PATSEX2;
255
256 where &condition = 1;
257
258 lsmeans cohort agegrp2 patsex2
259 /ADJUST=TUKEY pdiff;
260 *Post Hoc (Tukey-Kramer)
test;
261
262 OUTPUT OUT = pred p=ybar
r=resid;
263
264 title "aim3_map_delta model with
&&condition = ture";
265
266 run;
267 quit;
268 %mend runby;
269 %runby(cvdflag)
270 %runby(tobaccoflag);
271 %runby(obesityflag);
272 %runby(depressionflag);
273 libname ds
"C:\Users\dzhaosi\projects\Wilfong code
review\Aim3retry";
274
275 *proc contents
data=ds.aim3_model_data;run;
276 /*
277 Alphabetic List of Variables and
Attributes
278 # Variable Type Len Label
279 16 BMI_baseline Num 8
280 13 BMI_days_final Num 8
281 10 BMI_delta Num 8
282 3 CVDflag Num 8
283 12 MAP_days_final Num 8
284 9 MAP_delta Num 8
285 2 PATSEX Char 1 Patient Gender
286 17 TOB_baseline Num 8
287 8 agegrp2 Num 8
288 7 cohort Num 8

```

```

289 6 depressionflag Num 8
290 15 map_baseline Num 8
291 1 newid Char 16
292 5 obesityflag Num 8
293 14 tobacco_days_final Num 8
294 11 tobacco_delta Num 8
295 4 tobaccoflag Num 8
296 */
297
298 data aim3mdl;
299 set ds.aim3_model_data;
300 if patsex = "F" then patsex2 = 0;
301 if patsex = "M" then patsex2 = 1;
302 run;
303
304 /*-----Multicollinearity-----
-----*/
305
306 %macro corrout;
307 proc corr data=aim3mdl;
308
309 var cohort AGEGRP2 PATSEX2
310     map_baseline map_days_final
311     BMI_baseline BMI_days_final
312     tob_baseline tobacco_days_final
313     ;
314
315 title "aim3: Correlation
Coefficients";
316 run;
317 %mend corrout;
318
319
320 /*-----GLM ANCOVA with
Post Hoc(Tukey Kramer)-----*/
321 %macro glmout;
322
323 proc glm data=aim3mdl;
324
325 class cohort AGEGRP2 PATSEX2;
326
327 model tobacco_delta = tob_baseline
tobacco_days_final cohort AGEGRP2
PATSEX2;
328
329 lsmeans cohort agegrp2 patsex2
330         /ADJUST=TUKEY pdiff;
331         *Post Hoc (Tukey-Kramer)
test;
332
333 means cohort AGEGRP2
PATSEX2
334         /hovtest=levene welch;
335         /*Homogeneity of
variance: BAR, BARTLETT, BF,
LEVENE, OBRIEN*/
336         *WARNING:
Homogeneity of variance testing and
Welch's ANOVA
337         are only available for
unweighted one-way models;
338
339 OUTPUT OUT = pred p=ybar
r=resid;
340
341 title "aim3 tobacco_delta model";
342
343 run;
344 /*
347 Source DF Sum of Squares Mean
Square F Value Pr > F
348 Model 13 7688.28402 591.40646
6926.02 <.0001
349
350 R-Square Coeff Var Root MSE
tobacco_delta Mean
351 0.231808 -13796.66 0.292214 -
0.002118
352 */
353 %mend glmout;
354
355
356 /*-----QQplot and Shapiro-
Wilk test for normality-----*/
357
358 %macro normout;
359
360 proc sql OUTOBS=2000;
361 create table pred2k as
362 select *
363 from pred

```

```

364 order by RANUNI(6789);
366
367 PROC UNIVARIATE data=pred2k
normal;
368 var resid;
369 QQPLOT resid / normal(mu=est
sigma=est);
370 title "aim3mdl tobaccodelta
pred2k: Normal Q-Q Plot of Residuals
and Shapiro-Wilk test";
371 run;
372 /*pred2k -- Tests for Normality
373 Test Statistic p Value
374 Shapiro-Wilk W 0.784289 Pr < W
<0.0001
375
376 p-values <0.05, reject H0 that
residual is normally distributed.
377 */
378
379 *%mend normout;
380
381
382 /*-----Additional models
based on type of disease-----*/
383
384 %macro runby(condition);
385
386 proc glm data=aim3mdl;
387
388 class cohort AGEGRP2 PATSEX2;
389
390 model tobacco_delta = tob_baseline
tobacco_days_final cohort AGEGRP2
PATSEX2;
391
392 where &condition = 1;
393
394 lsmeans cohort agegrp2 patsex2
395 /ADJUST=TUKEY pdiff;
396 *Post Hoc (Tukey-Kramer)
test;
397
398 OUTPUT OUT = pred p=ybar
r=resid;
399

```

```

400 title "aim3 tobacco_delta model
with &&condition = ture";
401
402 run;
403 quit;
404 %mend runby;
405 %runby(cvdflag)
406 %runby(tobaccoflag);
407 %runby(obesityflag);
408 %runby(depressionflag);
725 libname ds
"C:\Users\dzhaosi\projects\Wilfong code
review\Aim3retry";
726
727 *proc contents
data=ds.aim3_model_data;run;
728 /*
729 Alphabetic List of Variables and
Attributes
730 # Variable Type Len Label
731 16 BMI_baseline Num 8
732 13 BMI_days_final Num 8
733 10 BMI_delta Num 8
734 3 CVDflag Num 8
735 12 MAP_days_final Num 8
736 9 MAP_delta Num 8
737 2 PATSEX Char 1 Patient Gender
738 17 TOB_baseline Num 8
739 8 agegrp2 Num 8
740 7 cohort Num 8
741 6 depressionflag Num 8
742 15 map_baseline Num 8
743 1 newid Char 16
744 5 obesityflag Num 8
745 14 tobacco_days_final Num 8
746 11 tobacco_delta Num 8
747 4 tobaccoflag Num 8
748 */
749
750 data aim3mdl;
751 set ds.aim3_model_data;
752 if patsex = "F" then patsex2 = 0;
753 if patsex = "M" then patsex2 = 1;
754 run;
755

```

```

756 /*-----data for Tobacco
model, with type of disease-----*/
757
758 data aim3mdl_tob_data;
759 set aim3mdl;
760
761 *model tobacco_delta =
tob_baseline tobacco_days_final cohort
AGEGRP2 PATSEX2;
762
763 run;
764 /*
765 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL.
766 NOTE: The data set
WORK.AIM3MDL_TOB_DATA has
461942 observations and 18 variables
767 */
768
769 data aim3mdl_tob_cvd;
770 set aim3mdl_tob_data;
771
772 if cvdflag=1;
773
774 run;
775 /*
776 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_TOB_DATA.
777 NOTE: The data set
WORK.AIM3MDL_TOB_CVD has
284093 observations and 18 variables.
778 */
779
780 data aim3mdl_tob_obe;
781 set aim3mdl_tob_data;
782
783 if obesityflag=1;
784
785 run;
786 /*
787 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_TOB_DATA.

```

```

788 NOTE: The data set
WORK.AIM3MDL_TOB_OBE has
76885 observations and 18 variables.
789 */
790
791 data aim3mdl_tob_dep;
792 set aim3mdl_tob_data;
793
794 if depressionflag=1;
795
796 run;
797 /*
798 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_TOB_DATA.
799 NOTE: The data set
WORK.AIM3MDL_TOB_DEP has
65827 observations and 18 variables.
800 */
801
802 data aim3mdl_tob_tob;
803 set aim3mdl_tob_data;
804
805 if tobaccoflag=1;
806
807 run;
808 /*
809 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_TOB_DATA.
810 NOTE: The data set
WORK.AIM3MDL_TOB_TOB has
58453 observations and 18 variables.
811 */
812
813 /*-----data for MAP model,
with type of disease-----*/
814
815 data aim3mdl_map_data;
816 set aim3mdl;
817
818 *model map_delta = map_baseline
map_days_final cohort AGEGRP2
PATSEX2;
819
820 run;

```

```

821 /*
822 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL.
823 NOTE: The data set
WORK.AIM3MDL_MAP_DATA has
461942 observations and 18 variables
824 */
825
826 data aim3mdl_map_cvd;
827 set aim3mdl_map_data;
828
829 if cvdflag=1;
830
831 run;
832 /*
833 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_MAP_DATA.
834 NOTE: The data set
WORK.AIM3MDL_MAP_CVD has
284093 observations and 18 variables
835 */
836
837 data aim3mdl_map_obe;
838 set aim3mdl_map_data;
839
840 if obesityflag=1;
841
842 run;
843 /*
844 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_MAP_DATA.
845 NOTE: The data set
WORK.AIM3MDL_MAP_OBE has
76885 observations and 18 variables
846 */
847
848 data aim3mdl_map_dep;
849 set aim3mdl_map_data;
850
851 if depressionflag=1;
852
853 run;
854 /*

```

```

855 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_MAP_DATA.
856 NOTE: The data set
WORK.AIM3MDL_MAP_DEP has
65827 observations and 18 variables.
857 */
858
859 data aim3mdl_map_tob;
860 set aim3mdl_map_data;
861
862 if tobaccoflag=1;
863
864 run;
865 /*
866 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_MAP_DATA.
867 NOTE: The data set
WORK.AIM3MDL_MAP_TOB has
58453 observations and 18 variables.
868 */
869
870
871 /*-----data for BMI model,
with type of disease-----*/
872
873 data aim3mdl_bmi_data;
874 set aim3mdl;
875
876 *model bmi_delta = mbi_baseline
mbi_days_final cohort AGEGRP2
PATSEX2;
877
878 run;
879 /*
880 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL.
881 NOTE: The data set
WORK.AIM3MDL_BMI_DATA has
461942 observations and 18 variables
882 */
883
884 data aim3mdl_bmi_cvd;
885 set aim3mdl_bmi_data;

```

```

886
887 if cvdflag=1;
888
889 run;
890 /*
891 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_mbi_DATA.
892 NOTE: The data set
WORK.AIM3MDL_mbi_CVD has
284093 observations and 18 variables
893 */
894
895 data aim3mdl_bmi_obe;
896 set aim3mdl_bmi_data;
897
898 if obesityflag=1;
899
900 run;
901 /*
902 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_mbi_DATA.
903 NOTE: The data set
WORK.AIM3MDL_mbi_OBE has
76885 observations and 18 variables
904 */
905
906 data aim3mdl_bmi_dep;
907 set aim3mdl_bmi_data;
908
909 if depressionflag=1;
910
911 run;
912 /*
913 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_mbi_DATA.
914 NOTE: The data set
WORK.AIM3MDL_mbi_DEP has
65827 observations and 18 variables.
915 */
916
917 data aim3mdl_bmi_tob;
918 set aim3mdl_bmi_data;
919

```

```

920 if tobaccoflag=1;
921
922 run;
923 /*
924 NOTE: There were 461942
observations read from the data set
WORK.AIM3MDL_bmi_DATA.
925 NOTE: The data set
WORK.AIM3MDL_bmi_TOB has
58453 observations and 18 variables.
926 */
950 libname save
"C:\Users\dzhaosi\projects\Wilfong code
review\aim3retry";
NOTE: Libref SAVE refers to the same
physical library as DS.
NOTE: Libref SAVE was successfully
assigned as follows:
      Engine:      V9
      Physical Name:
C:\Users\dzhaosi\projects\Wilfong code
review\Aim3retry
951
952 *proc contents
data=save.aim3_merge;run;
953 /*Data Set Name
SAVE.AIM3_MERGE Observations
7501525
954 Alphabetic List of Variables and
Attributes
955 # Variable Type Len Label
956 3 AGEGRP Char 1 Age Group
957 21 APPTDT Num 8
APPOINTMENT DATE
958 4 BHC Num 8
959 8 CVD_days Num 8
960 7 CVDflag Num 8
961 26 DIASTOLIC_SIT Num 8
DIASTOLIC BLOOD PRESSURE
MEASURED WHILE SITTING
962 23 HEIGHT_INCHES Num 8
HEIGHT (IN INCHES)
963 6 MH Num 8
964 2 PATSEX Char 1 Patient Gender
965 5 PC Num 8

```

```

966 27 RATE_SIT Num 8 HEART
RATE MEASURED WHILE SITTING
967 25 SYSTOLIC_SIT Num 8
SYSTOLIC BLOOD PRESSURE
MEASURED WHILE SITTING
968 24 TOBACCO Char 1
CURRENTLY USING A TOBACCO
PRODUCT
969 22 WEIGHT_LBS Num 8
WEIGHT (IN POUNDS)
970 28 all_days Num 8
971 18 atheroscl_days Num 8
972 17 atherosclflag Num 8
973 14 depression_days Num 8
974 13 depressionflag Num 8
975 16 hypertension_days Num 8
976 15 hypertensionflag Num 8
977 20 majorcardiac_days Num 8
978 19 majorcardiacflag Num 8
979 1 newid Char 16
980 12 obesity_days Num 8
981 11 obesityflag Num 8
982 10 tobacco_days Num 8
983 9 tobaccoflag Num 8
984 */
985
986 data aim3_newvar;
987 set save.aim3_merge
(rename=(TOBACCO=TOBACCOchar)
);
989 /*
990 NOTE: There were 7501525
observations read from the data set
SAVE.AIM3_MERGE.
991 NOTE: The data set
WORK.AIM3_NEWVAR has 7501525
observations and 28 variables.
992 */
993
994 data aim3_newvar;
995 set aim3_newvar;
996
997 if all_days GE 2;
998
999 run;
1000 /*

```

```

1001 NOTE: There were 7501525
observations read from the data set
WORK.AIM3_NEWVAR.
1002 NOTE: The data set
WORK.AIM3_NEWVAR has 5467921
observations and 7 variables.
1003 */
1004
1005
1006 Aim 3 (See Figure 3) Requested
Data:
1007 Inclusion Criteria: Seen by
Primary Care.
1008 At least 2 vitals measurements
Categorize by:
1009 Seen by PC only
1010 Seen by PC and Specialty BH
1011 Seen by PC and BHC
1012 Seen by all three
1013 Dependent Variable: Vitals
1014 Control Variables: Age, Sex, Time
between first and last
1015
1016
1017 data aim3_newvar;
1018 set aim3_newvar;
1019
1020 cohort = .;
1021 if PC=1 and BHC = . and MH =
. then cohort = 1;
1022 if PC=1 and BHC = 1 and MH
= . then cohort = 2;
1023 if PC=1 and BHC = . and MH =
1 then cohort = 3;
1024 if PC=1 and BHC = 1 and MH
= 1 then cohort = 4;
1025
1026 agegrp2=.;
1027 if AGEGRP = "A" then
AGEGRP2 = 1;
1028 if AGEGRP = "B" then
AGEGRP2 = 2;
1029 if AGEGRP = "C" then
AGEGRP2 = 3;
1030 if AGEGRP = "D" then
AGEGRP2 = 4;

```

```

1031   if AGEGRP = "E" then
AGEGRP2 = 5;
1032   if AGEGRP = "F" then
AGEGRP2 = 6;
1033   if AGEGRP = "G" then
AGEGRP2 = 7;
1034   if AGEGRP = "H" then
AGEGRP2 = 8;
1035
1036   if AGEGRP = "X" then
AGEGRP2 = 9;
1037
1038   /*3 vitals: tobacco, BMI AND
MAP*/
1039
1040   map = DIASTOLIC_SIT + ((1/3)
* (SYSTOLIC_SIT -
DIASTOLIC_SIT));
1041
1042   BMI = (WEIGHT_LBS *
0.453592) / ((HEIGHT_INCHES *
0.0254)**2);
1043
1044   tobacco = .;
1045   if TOBACCOchar = "Y" then
TOBACCO = 1;
1046   if TOBACCOchar = "N" then
TOBACCO = 0;
1047
1048   run;
1049
1050
1051   data aim3_newvar no3vitals;
1052   set aim3_newvar;
1053   if map = . and BMI = . and
tobacco = . then output no3vitals;
1054   else output aim3_newvar;
1055
1056   /*
1057   assign people with cardiovascular
disease to the category reflecting
1058   their MOST severe CV issue
(in descending order):
1059   Level 4 - Major Adverse Cardiac
Events

```

```

1060   Level 3 - Atherosclerotic and
Other CVD
1061   Level 2 - Hypertensive Heart
Disease
1062   Level 1 ? Control
1063   */
1064
1065   if majorcardiacflag = 1 then
CVD_level = 4;
1066   else if atherosclflag = 1 then
CVD_level = 3;
1067   else if hypertensionflag = 1
then CVD_level = 2;
1068   else CVD_level = 1;
1069
1070   run;
1071
1072   /*
1073   NOTE: There were 5467921
observations read from the data set
WORK.AIM3_NEWVAR.
1074   NOTE: The data set
WORK.AIM3_NEWVAR has 5383191
observations and 33 variables.
1075   NOTE: The data set
WORK.NO3VITALS has 84730
observations and 33 variables.
1076   */
1077
1078   proc sort data=aim3_newvar;
1079   by newid descending apptdt;
1080   run;
1081
1082   data aim3_newvar;
1083   set aim3_newvar;
1084
1085   by newid descending apptdt;
1086
1087   seq=.;
1088   if FIRST.newid then seq=1;
1089   if LAST.newid then seq=2;
1090
1091   run;
1092
1093   data aim3_newvar2;
1094   set aim3_newvar;

```

```

1095
1096 if seq ^= .; /*take first and last
obs per newid*/
1097
1098 run;
1099 /*
1100 NOTE: There were 5383191
observations read from the data set
WORK.AIM3_NEWVAR.
1101 NOTE: The data set
WORK.AIM3_NEWVAR2 has 1402419
observations and 34 variables.
1102 */
1103
1104 data aim3_newvar3;
1105 set aim3_newvar2;
1106
1107 if depression_days eq 1 then
delete;
1108 if obesity_days eq 1 then delete;
1109 if tobacco_days eq 1 then
delete;
1110 if CVD_days eq 1 then delete;
1111
1112 proc format;
1113 value cohort 1 = "PC only"
1114             2 = "PC and BHC"
1115             3 = "PC and Specialty
BHC"
1116             4 = "PC, BHC, and
Specialty BHC";
1117 /*
1118 NOTE: There were 1402419
observations read from the data set
WORK.AIM3_NEWVAR.
1119 NOTE: The data set
WORK.AIM3_NEWVAR3 has 975481
observations and 34 variables.
1120 */
1121
1122 data aim3_newvar4;
1123 set aim3_newvar3;
1124
1125 newid1 = lag(newid);
1126 agegrp21 = lag(agegrp2);
1127 apptdt1=lag(apptdt);

```

```

1130 bmi1 = lag(bmi);
1131 map1 = lag(map);
1132 tob1 = lag(tobacco);
1133 cohort1 = lag(cohort);
1134
1135 if newid1 = newid; *eliminate
mismatched lags;
1136
1137 run;
1138 /*
1139 NOTE: There were 975481
observations read from the data set
WORK.AIM3_NEWVAR.
1140 NOTE: The data set
WORK.AIM3_NEWVAR4 has 461942
observations and 41 variables.
1141 */
1142
1143 data aim3_newvar5;
1144 set aim3_newvar4;
1145
1146 by newid descending apptdt;
1147
1148 if LAST.newid;
1149
1150 run;
1151 /*
1152 NOTE: There were 461942
observations read from the data set
WORK.AIM3_NEWVAR4.
1153 NOTE: The data set
WORK.AIM3_NEWVAR5 has 461942
observations and 41 variables.
1154 */
1155
1156 data aim3_clean; /*final
modeling dataset*/
1157 set aim3_newvar5;
1158
1159 MAP_delta = MAP1-MAP;
1160 BMI_delta = BMI1-BMI;
1161
1162 tobacco_delta = tob1 - tobacco;
1163
1164 MAP_days_final = apptdt1 -
apptdt;

```

```

1165 BMI_days_final = apptdt1 -
apptdt;
1166
1167 if tobacco ^= . and tob1 ^= .
then do;
1168     tobacco_days_final =
apptdt1 - apptdt;
1169 end;
1170
1171 map_baseline = MAP;
1172 BMI_baseline = BMI;
1173 TOB_baseline = tobacco;
1174
1175 run;
1176 /*
1177 NOTE: There were 461942
observations read from the data set
WORK.AIM3_NEWVAR5.
1178 NOTE: The data set
WORK.AIM3_CLEAN has 461942
observations and 47 variables.
1179 */
1180
1181 data save.aim3_model_data;
1182 set aim3_clean;
1183
1184 format apptdt date10. apptdt1
date10.;
1185 /*
1186 if tob_baseline ^= . and tob1 ^= .;
1187
1188 if map_baseline ^= . and map1 ^=
.;
1189
1190 if bmi_baseline ^= . and bmi1 ^=
.;
1191 */
1192
1193 keep newid cohort AGEGRP2
PATSEX
1194 CVDflag obesityflag
tobaccoflag depressionflag
1195 map_delta map_baseline
map_days_final
1196 BMI_delta BMI_baseline
BMI_days_final

```

```

1197 tobacco_delta tob_baseline
tobacco_days_final
1198 ;
1199
1200 run;
1201 /*
1202 NOTE: There were 461942
observations read from the data set
WORK.AIM3_CLEAN.
1203 NOTE: The data set
SAVE.AIM3_MODEL_DATA has
461942 observations and 17 variables.
1204 */
1205
1206 %macro dataout;
1207 proc export data =
save.aim3_model_data
1208     outfile =
"C:\Users\dzhaosi\projects\Wilfong code
1208!
review\aim3retry\aim3modeldata0608.cs
v"
1209     dbms=csv
1210     replace;
1211     run;
1212
1213
1214
1215 /*random sample 1k*/
1216 proc sql OUTOBS=1000;
1217 create table aim3sample1k as
1218 select *
1219 from aim3_model_data
1220 order by RANUNI(4321);
1221 quit;
1222
1223
1224 data save.aim3sample1k;
1225 set aim3sample1k;
1226 run;
1227
1228
1229 proc export data = aim3sample1k
1230     outfile =
"C:\Users\dzhaosi\projects\Wilfong code

```

```
1230!  
review\aim3retry\aim3datasample1k.csv  
"  
1231      dbms=csv  
1232      replace;  
1233      run;  
1234 %mend dataout;
```