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DEPRESSIVE SYMPTOMS, BEHAVIORAL HEALTH RISK FACTORS, AND PHYSICAL ILLNESS AMONG OLDER MEXICAN AMERICANS

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DEPRESSIVE SYMPTOMS, BEHAVIORAL HEALTH RISK FACTORS, AND PHYSICAL ILLNESS AMONG OLDER MEXICAN AMERICANS

by

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Dedication

This work is dedicated to the memory of my grandparents, Carlos and Felicitas Talavera, whose unconditional love, support, and encouragement in my formative years is still with me today. It is also dedicated to my children, Gabriela, Carlos, and Marco. You inspire me everyday to be my best.

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Depressive Symptoms, Behavioral Health Risk Factors, and Physical Illness among Older Mexican Americans

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This study utilizes data from Hispanic Established Populations for Epidemiologic Studies of the Elderly (H-EPESE) at two different time points, seven years apart, to examine the relationship between physical illness and depressive symptoms in elderly Mexican Americans. The two physical illnesses studied are coronary artery disease and type II diabetes due to their high prevalence among Mexican Americans. The relationship between physical illness and depressive symptoms is examined longitudinally and prospectively, in both directions. In addition, the relationship between depressive symptoms and three behavioral health risk factors: alcohol use, cigarette smoking, and physical inactivity, at baseline is examined. The roles of gender, acculturation, nativity, and locus of control, are examined as moderators of the key relationships studied in the exploratory analyses. Additionally, self-rated health at baseline is examined as a predictor of physical illness and mortality at follow-up.

Contrary to expectations, baseline physical illness was not significantly related to increased depressive symptomatology at seven-year follow-up. However, consistent with expectations, depressive symptoms at baseline significantly predicted the presence of coronary artery disease seven years later. In addition, baseline depressive symptoms also significantly predicted diabetes and diabetes complications after seven years. Baseline depressive symptoms also significantly predicted mortality. Two mediational models were also examined. The first model explored baseline coronary artery disease as a mediator of the relationship between baseline depressive symptoms and mortality. The results of this model were consistent with the proposed mediation. The results of the second model, which explored baseline alcohol use as a mediator of the relationship between baseline depressive symptoms and mortality, were not consistent with the proposed mediation. In other analyses, nativity was a significant moderator of the relationship between baseline depressive symptoms and diabetes at follow-up and between baseline depressive symptoms and subsequent mortality. In addition, self-rated health was a significant predictor of mortality.

Table of Contents

List of Tables

List of Figures

CHAPTER 1: THE HEALTH OF OLDER MEXICAN AMERICANS

The purpose of the proposed study is to investigate the interrelationship between depressive symptoms and physical illness across a seven-year period in a large nationally representative sample of older Mexican Americans. First, the study investigates chronic physical illness as a predictor of depressive symptomatology. Next, the study investigates depressive symptomatology as a predictor of both chronic physical illness and mortality. Heart disease and diabetes are the chronic physical illnesses proposed for study because Mexican Americans have higher prevalence rates of these diseases compared to the general population. Moreover, the study examines the role of behavioral health risk factors in partially mediating the association between depressive symptomatology and both chronic physical illness and mortality. The behavioral health risk factors to be studied are cigarette smoking, alcohol abuse, and body mass index. The study adds to the literature by elucidating the relationship between depressive symptoms and physical illness in a population at high risk of diabetes and heart disease and the complications associated with these chronic illnesses.

BACKGROUND ON OLDER MEXICAN AMERICANS

Hispanics are the fastest growing minority group in the United States due to a high birth rate and to immigration. Hispanics account for approximately thirteen percent of the total U.S. population. The Hispanic population grew from 22.4 million in 1990 to 35.3 million in 2000. Hispanics are a diverse group and can be divided into five subgroups: Mexican American, Puerto Rican, Cuban, Central or South American, and other Hispanics. Of these subgroups, Mexican Americans made up over fifty-eight percent of all Hispanics in the 2000 census, numbering 20.6 million (Guzman, 2001).

The Census Bureau reported in January 2003 that Hispanics had surpassed African Americans as the largest minority group in the United States (Miller, 2003). Over seventy-five percent of Hispanics live in the southwestern United States. Hispanics have the highest rates of poverty (21.9 percent compared to 12.7 percent for the total population) and the lowest rates of health insurance coverage (67.3 percent compared to 84.1 percent for the total population) (DeNavas-Walt, Proctor, & Lee, 2006). Among Hispanic subgroups in 2003, 24.1 percent of Mexican Americans lived below the poverty level compared to 23.7 percent of Puerto Ricans and 14.4 percent of Cuban Americans. The percent of non-Hispanic Whites that lived below the poverty level in 2003 was 8.4 percent (U.S. Census Bureau, 2004). Poverty and lack of health insurance are two of the greatest barriers faced by Hispanics in terms of access to health care. Two serious consequences of a lack of access to health care are use of emergency departments for primary care and a lack of continuity of care (Council on Scientific Affairs, 1991).

In the U.S., the population aged sixty-five and over is projected to increase substantially by the year 2030. As this population grows, it is expected to reflect the demographic changes in the general population and to become more diverse. In addition, the proportion of elderly from minority groups is growing at a faster rate than that of the majority population (Williams & Wilson, 2001). Of all minority groups, it is estimated that the older Hispanic population will see the biggest change, growing from just over 2 million in 2003 to almost 8 million by the year 2030 (He, Sengupta, Velkoff, & DeBarrios, 2005).

The proposed topic of study is important due to the projected growth of the Mexican American population and the disproportionate incidence and prevalence of heart

disease, diabetes, and depression in this population. The identification and treatment of depressive symptoms in physically ill patients could improve disease management and prevent serious complications from developing. In addition, heart disease or diabetes patients identified and treated for comorbid depression could potentially benefit from an improved quality of life compared to those whose depressive symptoms are never detected.

THE PHYSICAL HEALTH OF MEXICAN AMERICANS

Coronary Artery Disease

Coronary artery disease (CAD), which includes acute myocardial infarction, angina pectoris, atherosclerotic cardiovascular disease and all other forms of chronic ischemic heart disease, was responsible for twenty percent of all deaths in the United States in 2001 and is the leading cause of death for men and women in the U.S. The prevalence of CAD for the total population in 2001 was 6.4 percent. The rate was 6.9 percent for men and 6.0 percent for women. The incidence of coronary artery events was estimated at 700,000 in 2004 with an additional 500,000 Americans estimated to suffer a recurrent event (American Heart Association, 2005).

Over the last thirty years, the prevalence of CAD and stroke has declined by over 50 percent and this decrease is partially responsible for increases observed in the elderly population and for life expectancy (Sundquist, Winkleby, & Pudaric, 2001). Despite these changes, CAD remains a significant public health issue for Americans and for Mexican Americans in particular.

Mexican Americans have a higher prevalence of CAD than non-Hispanic Whites. The prevalence rate for Mexican American men is 7.2 percent while the rate is 6.8

percent for Mexican American women compared to 6.9 percent and 5.4 percent for non-Hispanic White men and women, respectively (American Heart Association, 2005). In addition, an increased risk of mortality due to cardiovascular and coronary artery disease, as well as to all-causes, has been identified in Mexican Americans when compared to non-Hispanic Whites (Hunt et al., 2003).

Researchers studied CAD mortality in Mexican Americans as part of a community-wide regional study, the Corpus Christi Heart Project (Pandey, Labarthe, Goff, Chan, & Nichaman, 2001). The investigators found that for both sexes, mortality rates due to CAD were higher among Mexican Americans than among non-Hispanic Whites. The proportions of deaths due to CAD among Mexican Americans were 36 percent greater among women and 12 percent greater among men compared with non-Hispanic Whites (Pandey et al., 2001).

One complication associated with CAD is a myocardial infarction or MI. Rates of MI have also been found to be elevated in Mexican Americans. Hospitalization rates for MI were studied in Nueces County, Texas as part of the Corpus Christi Heart Project (Goff et al., 1997). These investigators found the highest rates of MI in Mexican American men, followed by non-Hispanic White men, Mexican American women, and finally, by non-Hispanic White women. Mortality due to MI has also been studied in this population. Researchers investigated factors associated with mortality 28 days post-MI in Mexican American and non-Hispanic White men and women (Goff, Ramsey, Labarthe, & Nichaman, 1994). The investigators identified being Mexican American, being female, and to having diabetes as factors associated with increased rates of fatality.

In this study, Mexican American women were found to have the highest 28-day rates of case-fatality of the four groups studied.

Some research has found that Mexican Americans have lower mortality and morbidity rates associated with CAD despite having higher rates of cardiovascular risk factors (Stern et al., 1987; Rewers et al., 1993). For example, one study reviewed the data on five cardiovascular risk behaviors: cigarette smoking, dietary intake, being overweight, physical inactivity, and alcohol consumption among African Americans, Asian/Pacific Islanders, Latinos, and Native Americans (Myers, Kagawa-Singer, Kumanyika, Lex, & Markides, 1995). In this study, Hispanics, and particularly, Mexican Americans were found to have lower mortality due to heart disease. The authors speculate that the population's traditionally low cigarette smoking rates may have contributed to this finding. In another study, data from the 1980s on mortality related to acute myocardial infarction and CAD in Mexican Americans and non-Hispanic Whites in Texas was reviewed (Goff, Ramsey, Labarthe, & Nichaman, 1993). Mortality rates due to both conditions studied were found to be significantly lower among Mexican American men than non-Hispanic White men and were similar for the women studied (Goff et al., 1993).

Currently, however, there is agreement in the literature that the studies that showed lower rates of mortality and morbidity from CAD for Mexican Americans may have been flawed. Many of these early studies relied on vital statistics data that has been found to suffer from such errors as misclassification of ethnicity and incomplete ascertainment of relevant data (Hunt et al., 2003; Goff et al., 1997). For example, in one study reviewed, deaths of older Mexican Americans were under counted when mortality

data collected by the researchers were compared to information from the National Death Index (Patel, Eschbach, Ray, & Markides, 2004). The authors of the review propose that Hispanic mortality rates may be underestimated because that these findings may be generalizable to other data sets that have relied on databases such as the National Death Index.

In addition, some earlier studies also failed to distinguish between Mexico-born and U.S.-born Mexican Americans. Country of birth has been found to be an important predictor of heart disease related mortality. In one study, Mexican Americans born in Mexico had the lowest mortality due to cardiovascular disease while U.S. born Mexican Americans had the highest (Stern & Wei, 1999). In this study, U.S. born Mexican Americans had a mortality ratio of 1.30 for cardiovascular mortality and 1.38 for mortality due to all-causes as compared to non-Hispanic Whites. The lower rate of cardiovascular disease mortality in those born in Mexico is attributed to a healthy migrant effect and to protective factors related to a traditional lifestyle (Stern & Wei, 1999).

Risk Factors for Coronary Artery Disease

Some of the risk factors associated with CAD are elevated blood pressure, cigarette smoking, hypercholesterolemia, excess body weight, sedentary lifestyle, and diabetes (American Heart Association, 2005). Rates of CAD are believed to be elevated in Mexican Americans compared to the general population because they have more risk factors. Mexican Americans have been found to have higher rates of elevated blood pressure, be substantially more overweight, and to have a higher incidence of diabetes as compared to the general U.S. population (Stern et al., 1981). In addition to these adverse cardiovascular risk factors, higher serum triglyceride levels, higher cholesterol levels,

(including lower HDL and smaller LDL particles) have been identified in Mexican Americans (Stern et al., 1981; Goff et al., 1997). Mexican Americans also have the highest age-adjusted prevalence of metabolic syndrome. People with metabolic syndrome are at increased risk for developing cardiovascular disease and diabetes as well as increased mortality from cardiovascular disease and all-causes (American Heart Association, 2005).

Sundquist et al. (2001) studied the association between ethnicity and cardiovascular disease risk factors among older Black, Mexican-American, and White women and men. These researchers examined the National Health and Nutrition Examination Survey (NHANES III) data and found that those that had the highest prevalence of cardiovascular disease risk factors were Black and Mexican-American women and Black men. The authors concluded that Black and Mexican American women had the highest prevalence of risk factors due to the high prevalence of type II diabetes in these two groups (Sundquist et al., 2001).

Hypertension

Uncontrolled hypertension has been identified as a serious risk factor for cardiovascular disease and has been found to be elevated in Mexican Americans. Researchers studied hypertension in 3050 Mexican Americans aged 65 and older in five Southwestern states (Satish, Stroup-Benham, Espino, Markides, & Goodwin, 1998). The majority of Mexican Americans with hypertension in this study had blood pressure that was inadequately controlled or untreated. Variables associated with under-treatment and poor control of hypertension were underutilization of health care services due to the high cost of medical care and medications and the lack of adequate insurance coverage (Satish

et al., 1998). In a related study, investigators found that being male was related to the unawareness of hypertension in the same sample of Mexican Americans (Satish, Markides, Zhang, & Goodwin, 1997). This finding was attributed to an attitude among males that disease is a normal part of aging with little action to be taken (Satish et al., 1997).

The community of Starr County Texas, a county in south Texas whose population is predominantly Mexican American, has been of interest to researchers because of the elevated incidence of several chronic health conditions, including obesity, diabetes, and gallbladder disease. In one study that examined hypertension in this community, a significant elevation in blood pressure was identified for subjects in Starr County when compared to Mexican American subjects taking part in the San Antonio Heart Study (Hanis, 1996). These findings are believed to be due in part to sociodemographic differences that exist for residents of the two communities including employment, socioeconomic status, and educational attainment. Therefore, it is important to remember that cardiovascular risk factors may vary based on the location of the Mexican American population under study (Hanis, 1996).

Type II Diabetes

The number of people affected with diabetes increased by 3 million to 24 million in only two years (CDC, 2008). Scientists believe that type II diabetes will become one of the main public health problems in the U.S. based on the considerable increases that have been observed in recent years (Burke et al., 1999). The problem of type II diabetes has increased considerably for the Mexican American population. In fact, the incidence of diabetes in Mexican Americans who were evaluated as part of the longitudinal

population-based San Antonio Heart Study tripled from 1979 to 1988, jumping from 5.7 percent to 15.7 percent (Burke et al., 1999). These researchers found that the rate for non-Hispanic Whites increased from 2.8 percent in 1980 to 9.4 percent in 1988 (Burke et al., 1999).

One reason for the increased incidence of type II diabetes in Mexican Americans is believed to be genetic. Mexican Americans, most of whom have Native American ancestry, are thought to be genetically predisposed to developing "New World Syndrome." "New World Syndrome" is a grouping of diseases that includes diabetes, central obesity, gallstones, and gallbladder cancer**.** This genetic predisposition has been theorized to be due to subtle metabolic differences in the original group of Native Americans that survived the harsh migration to the New World. Those who survived the migration are believed to have had an advantage in utilizing central fat storage. Native Americans for many centuries had to adapt to scarce food resources which may have lead to the accumulation of these diabetes susceptibility genes. "New World Syndrome" is now expressed due to a maladaptive interaction between genetics and an environment where food is readily available (Weiss, Ferrell, & Hanis, 1984). This theory is supported by the fact that the risk of developing diabetes has a direct relationship to percentage Amerindian genes. Mexican Americans in Texas have been found to have 30-35 percent of their genes derived from Native Americans. Some groups of Native Americans, such as the Pima Indians, who have almost 100 percent Native American genes, have extremely high rates of diabetes (Weiss et al., 1984). In addition to genetic factors that contribute to the development of diabetes, other risk factors for diabetes are a sedentary lifestyle, low SES, barriers to health care, and a poor diet (Brown et al., 2000). Risk

factors associated with diabetes have been found to vary by sex. For example, in one study, alcohol consumption was established as the most significant risk factor for the development of diabetes in men, while obesity was found to be the most significant factor for women (Monterrosa, Haffner, Stern, & Hazuda, 1995).

One of the complications associated with diabetes is an MI. In a study of the relationship between diabetes and the severity of myocardial infarction conducted in Corpus Christi, Texas, researchers found that individuals who had diabetes endured a more complicated hospital course after suffering a heart attack (Orlander et al., 1994). In addition, those with diabetes had a higher rate of mortality post-MI and Mexican Americans hospitalized for MI were younger when compared to non-Hispanic White subjects (Orlander et al., 1994).

In addition to having an increased incidence of diabetes, the course of diabetes in Mexican Americans is believed to be more severe than in non-Hispanic Whites. For example, a study conducted in San Antonio, Texas found significantly higher glucose values for Mexican Americans with diabetes than for their non-Hispanic White counterparts (Hunt et al., 2002). Mexican Americans also develop diabetes at an earlier age and have more disease complications than the general population (Espino, Parra, & Kriehbiel, 1994; Otiniano, Du, Ottenbacher, Black, & Markides, 2003).

Although diabetes that requires treatment by insulin is believed to be more severe than cases not requiring insulin, Mexican American patients on oral therapy may still be at risk for complications. Researchers who studied diabetes status and insulin use in Mexican Americans with diabetes not treated by insulin were found to have a 2-3 times increased risk of all-cause, cardiovascular, and coronary heart disease mortality than that

of non-Hispanic Whites (Hunt et al., 2003). Therefore, early diabetes for these subjects controlled by diet or oral hypoglycemic therapy may be more severe and more deadly than in the general population (Hunt et al., 2003).

Diabetes is one of the risk factors for CAD that can be controlled or modified by making significant lifestyle changes. The potential for public health intervention programs is especially important with respect to this disease.

Other Cardiovascular Risk Factors

Some researchers have found evidence that socioeconomic disparity accounts for the higher prevalence rate of CAD in Mexican Americans (Johnson et al., 1995). In research on Black, Mexican American and White women, researchers found a relationship between lower SES and a higher prevalence of type II diabetes, physical inactivity, and abdominal obesity in women. In men, lower SES was associated with the risk factors of physical inactivity and cigarette smoking (Sundquist et al., 2001).

However, in other research, ethnicity, not socioeconomic status, was found to play a more significant role in the development of cardiovascular disease. For example, differences with respect to ethnicity persisted even when socioeconomic status was controlled for in one study (Winkleby, Kraemer, Ahn, & Varady, 1998). In this study, ethnic and socioeconomic factors related to cardiovascular disease risk in African American, Mexican American and White women using NHANES III data were examined. After controlling for socioeconomic status, significant differences in BMI, blood pressure, diabetes, and physical inactivity persisted for both Black and Mexican American women as compared to White women (Winkleby et al., 1998). This finding underscores the high-risk of cardiovascular disease that exists for ethnic minority women.

The frequency of diabetic complications and severity of disease has been shown to vary by nativity. U.S.-born Mexican Americans with diabetes had a 66 percent greater risk of cardiovascular mortality than non-Hispanic Whites with diabetes. Mexico-born Mexican Americans with diabetes had similar rates of all-cause and cardiovascular mortality when compared to non-Hispanic Whites with diabetes (Hunt et al., 2002). In another study, those born in Mexico were found to have healthier cardiovascular profiles than both English and Spanish speaking U.S.-born Mexican Americans despite having lower SES and educational attainment (Sundquist & Winkleby, 1999). This disparity is believed to be due to positive social and cultural influences from their country of origin that later disappear as a result of acculturation (Sundquist & Winkleby, 1999).

A disparity between the physical health of Mexico-born versus U.S. born Mexican Americans has also been established in elderly samples. Patel, Peek, Wong, and Markides (2006) found the prevalence of diabetes, cancer, heart attack, stroke, and arthritis was substantially lower in an elderly population in Mexico than in older Mexican Americans from the southwestern United States.

Physical inactivity is among other CAD risk factors that has been found to vary by ethnicity. Researchers using NHANES III data found that Mexican American women had a higher prevalence of physical inactivity than the general population even after controlling for age, education, and income. Inactivity was lower among those who spoke mostly English than among non-English speakers for both men and women. Results for nativity were similar to those for choice of language spoken. Men and women born in Mexico had a higher prevalence of physical inactivity than US-born Mexican American men and women (Crespo, Smit, Carter-Pokras, & Andersen, 2001).

In addition to elevated risk factors, lack of health knowledge may put Mexican Americans at an even greater risk of developing heart disease. In one study, Mexican Americans had less information regarding heart attack prevention and heart attack symptoms as compared to Anglos (Hazuda, Stern, Gaskill, Haffner, & Gardner, 1983). Mexican Americans also engaged in significantly fewer health habits aimed at heart attack prevention than the comparison group. Socioeconomic status was not found to be a significant variable related to heart disease prevention as a large disparity existed between Anglos and Mexican Americans at all socioeconomic levels (Hazuda et al., 1983).

In research examining the relationship between health behaviors and ethnicity, Mexican American women with diabetes had the least number of health behaviors (Sundquist et al., 2001). In this study, they were significantly less likely than Black or White women to monitor their diabetes or make efforts to lose weight, in order to control their blood sugar. In addition, Mexican American women with hypertension or high blood cholesterol were much less likely than other women to comply with recommendations by their physicians, such as taking prescribed medicine to lower their blood pressure or cholesterol in this study. Findings from this study also showed that Mexican American men with high cholesterol were less likely to take their prescribed cholesterol lowering medications than were Black or White men. This study also found that Spanish speaking Mexican American women, in particular, were at a significant disadvantage in terms of practicing health behaviors that to control their medical conditions. Language barriers that restricted a patient's ability to be proactive in caring for their own health were proposed as one potential explanation for these findings

(Sundquist et al., 2001). Therefore, in addition to being at an elevated risk for cardiovascular disease, Mexican Americans also practiced many behaviors that made their risk of cardiovascular disease even higher.

Not all ethnic differences regarding CAD are believed to be due to health behaviors. In one study, increased mortality following an MI in Mexican American men and women was established to be due to differences in treatments received by the patients (Goff et al., 1994). Mexican American men were less likely than non-Hispanic White men to receive thrombolytic therapy, angioplasty, and anticoagulants following an MI, even when researchers controlled for heart disease severity (Goff et al., 1994).

In a related study, Herholz et al. (1996) found Mexican Americans were not treated as aggressively as non-Hispanic Whites following an MI. In this study, women received fewer cardiovascular drugs than men and Mexican Americans received all types of medications except for ACE inhibitors and hypoglycemic therapy, less often than non-Hispanic Whites. In another example, Ramsey, Goff, Wear, Labarthe and Nichaman (1997) found that Mexican Americans hospitalized for MI received fewer intervention procedures in addition to receiving less pharmacologic intervention. In this sample, rates of cardiac catheterization and percutaneous transluminal coronary angioplasty were lower for Mexican Americans than non-Hispanic Whites. These findings may reflect ethnic differences in socioeconomic status as well as physician perceptions that Mexican Americans may be less able to afford medications or less compliant with medical recommendations (Herholz et al., 1996; Ramsey et al., 1997).

THE MENTAL HEALTH OF MEXICAN AMERICANS

The mental health of Mexican Americans has been the subject of various empirical studies. The lifetime prevalence rates of 12 DSM-III-R psychiatric disorders in a sample of 3012 adults were studied as part of the Mexican American Prevalence and Services Survey (Vega et al., 1998). In this study, nativity was assessed and results were compared to those of population surveys conducted in the United States and Mexico. Psychiatric disorders were assessed using a modified version of the World Health Organization's Composite International Diagnostic Interview in face-to-face interviews Rates for U.S. born Mexican Americans were similar to those of the general population of the United States while those for Mexican born Mexican Americans were lower and similar to those of Mexican citizens (Vega et al., 1998).

In another study, the prevalence of DSM-III disorders among Mexican Americans and non-Hispanic Whites was assessed using the Diagnostic Interview Schedule as part of the Los Angeles Epidemiologic Catchment Area study (Burnam et al., 1987). These investigators believed Hispanics might be at a higher risk of mental disorders than the general population due to lower educational and income levels. They examined major depression, manic episodes, substance abuse/dependence (alcohol and other drugs), obsessive-compulsive disorder, phobias, panic disorder, schizophrenia and schizophreniform disorder, and antisocial personality. Disorders were considered present if they occurred anytime within six months prior to the study. Very few differences were in fact found between non-Hispanic Whites and Mexican Americans (Burnam et al., 1987). Therefore, country of birth may be a more important variable than ethnicity when studying Mexican American mental health, as established by both of these studies.

Depression

The prevalence of depression in Mexican Americans as compared to other groups was examined utilizing NHANES III data (Riolo, Nguyen, Greden & King, 2005). In this study, White individuals had the highest prevalence of major depressive disorder, while the prevalence of dysthymic disorder was significantly greater among African Americans and Mexican Americans. Although lack of education was a significant predictor of dysthymic disorder in all groups examined, a lack of education was significantly associated with major depressive disorder only for Mexican Americans (Riolo et al., 2005).

Another study examined the prevalence of various depressive disorders across ethnic groups in a population of insured subjects from general medical practices (Jackson-Triche et al., 2000). Physician specialties included in the study were family practice, internal medicine, cardiology, and endocrinology. The variables studied were severity of depressive disorder and their effect on health-related quality of life. Study subjects were screened for depression, coronary heart disease, hypertension, and diabetes. In this study, African Americans and Hispanics were more likely to have depressive symptoms than White subjects or Asian Americans. Hispanics in this study had a higher prevalence of dysthymic disorder compared to Whites (6.8 percent and 3.1 percent, respectively). However, these ethnic group differences disappeared after adjustment for demographic characteristics and gender and socioeconomic status were found to be more significant factors than ethnicity in determining risk for depressive disorder (Jackson-Triche et al., 2000).

Depression in Younger Mexican Americans

Depression in adolescents has been identified as being most prevalent among Hispanics. Hovey and King (1996) found a 23 percent prevalence rate of depressive symptoms and a 25 percent increase in the risk of suicide for Hispanic adolescents. Similar to research on adult depression, some studies have found that differences in depression prevalence are due to ethnicity while others have shown differences to be due to social factors.

A study on ethnically diverse students in middle school examined the prevalence of DSM-IV Major Depression. The prevalence rate of major depression was 8.4 percent without impairment and 4.3 percent with impairment. Depression was higher for females and for those of lower economic status. Only Mexican American adolescents reported significantly higher rates of major depression with impairment, independent of the effects of age, gender, and SES. This study did not find lower status minority youth to be at disproportionate risk of depression compared to lower status majority youth. The authors propose that the elevated risk of major depressive episodes in Mexican American youths could be due to fatalism. Fatalism, or a belief in external control, has been found to be more common in the Mexican culture (Roberts, Roberts, & Chen, 1997).

Depression in Older Mexican Americans

Studies on older Mexican Americans have consistently found higher prevalence rates of depression. Depressive symptoms in older Mexican Americans participating in the Hispanic EPESE study were found to be 25.7 percent bereavement (Black, Markides, & Miller, 1998). In this study, this rate was compared to rates of 9 percent, 16.4 percent, and 13.2 percent found for non-Hispanic White samples in similar (EPESE) studies on

the elderly. Risk factors related to depressive symptoms in this sample of elderly Mexican Americans were lack of insurance coverage, financial difficulties, living arrangements, not having a confidant and recent bereavement (Black, Markides, & Miller, 1998). In another study, Mexican American women were substantially more likely than men to report depressive symptoms. Stressors associated with higher depressive symptomatology in this population were similar to those factors associated with symptoms in Anglo American, African Americans and other groups and nationalities. These stressors included: income, economic events and chronic strains and were found to exert an independent and additive influence on symptoms of depression (Chiriboga, Black, Aranda, & Markides, 2002).

The prevalence of depression in older Mexican Americans was also studied in the Sacramento Area Latino Study on Aging (SALSA) (González, Haan, & Hinton, 2001). Their results were very similar to that reported for subjects in the Hispanic EPESE. Depressive symptoms in this study were also assessed with the CES-D. The prevalence of depression was found to be 25.4 percent in this study. As in the Hispanic EPESE, women were found to be at greater risk of depressive symptoms than men (González, Haan, & Hinton, 2001).

Depression and Acculturation/Nativity

A belief that the socioeconomic disadvantages evident in immigrant populations contributed to poorer mental health than in the general population helped to shape early theories on immigration and mental health (Escobar, Nervi, & Gara, 2000). Recent research on Mexican Americans, however, challenges this idea. A review of recent studies shows that Mexico-born immigrants have better mental health profiles than U.S.-

born Mexican Americans (Escobar et al., 2000). These reviewers state that significant evidence to support of the idea of the negative effect of "acculturation" on the mental health of persons of Mexican descent in the United States continues to grow. In general, a lower level of acculturation and a shorter time since immigration to the United States are believed to be linked to a lower prevalence of psychiatric disorders (Escobar et al., 2000).

In a study that examined mental health in Puerto Ricans, immigrant Mexican Americans, U.S. born Mexican Americans and non-Hispanic Whites, immigrant Mexican Americans had the fewest mental health problems of all the groups (Shrout et al., 1992). These researchers examined the profiles of subjects in the Los Angeles Epidemiologic Catchment Area Study, using the Diagnostic Interview Schedule to assess affective disorders, psychotic disorders, alcoholism, phobia, and somatization. They found that U.S. born Mexican Americans were at highest risk of affective disorder and of alcohol abuse/dependence and had higher rates of all disorders compared to Mexican immigrants. The increased prevalence of these disorders in U.S. born Mexican Americans is attributed to discrimination and relative socioeconomic deprivation faced by this group (Shrout et al., 1992).

Another study differentiated between the effects of acculturation and those of immigration on psychotic disorders (Burnam, Hough, Karno, Escobar, & Telles, 1987). Lifetime prevalence rates of eight major DSM-III psychiatric disorders in Mexican Americans in Los Angeles were examined as part of the same Epidemiologic Catchment Area (ECA) study cited above. In this study, acculturation and country of birth were examined separately. These investigators found that higher acculturation scores were

related to higher rates of phobia, alcohol abuse/dependence, and drug abuse/dependence. They also found with respect to nativity, that U.S. born Mexican Americans, who were likely to have high levels of acculturation, had higher lifetime prevalence of these same disorders than immigrant Mexican Americans and also higher rates of major depression and dysthymia. The lower lifetime prevalence rates of these disorders in immigrant Mexican Americans were unexpected by these researchers due to increased stressors such as lower income and education in this population. The researchers proposed that immigrants may feel less distress due to their low socioeconomic status than those born in the U.S., because although it is lower than the general population's, it still surpasses the standard of living in Mexico. They similarly posit that U.S. born Mexican Americans may experience a greater sense of deprivation because they have higher expectations in terms of their standard of living. Both a theory of selective migration, which accounts for lower prevalence rates of disorders among Mexican immigrants and a social stress theory, which explains a higher prevalence rate of disorders among U.S. born Mexican Americans, support these findings (Burnam, Hough, Karno, Escobar, & Telles, 1987).

Although researchers believe that being born in Mexico and the preservation of traditional culture may have a positive impact on the health and mental health of Mexican Americans, some studies on depression in elderly Mexican Americans show opposite relationships. Low levels of acculturation in elderly Mexican Americans have been found to be related to a higher risk of depressive symptoms (Black et al., 1998). With respect to nativity, Gonzalez et al. (2001) found depressive symptomatology to be higher in older immigrants than in Mexican Americans born in the U.S.

Smoking and Alcohol Abuse

Although smoking rates among Mexican Americans have been historically low, a 2004 report by the CDC found Mexican American men to have slightly higher smoking rates than non-Hispanic White men (29.8 percent compared to 29.2 percent). Rates for Mexican American women were significantly lower than non-Hispanic White women, 15.6 percent compared to 25.9 (CDC, 2004).

In another study, rates of cigarette smoking were lower in Latinos compared to non-Hispanic Whites and African Americans (Perez-Stable et al., 2001). In this study, among Hispanics, Mexican Americans had lower smoking rates than Puerto Ricans and Cubans, however, the lower rate was due to the lower rate of smoking among Mexican American women. In this investigation, the relationship between acculturation and smoking varied according to gender. Men with higher acculturation scores were less likely to smoke cigarettes, while women with higher acculturation scores were more likely to smoke (Perez-Stable et al., 2001). Wilkinson et al. (2005) found that higher levels of acculturation were related to ever having smoked in women. However, they found no relationship between smoking status and acculturation in men.

The effect of nativity on smoking behavior has also been explored. Differences in smoking behavior between Mexican Americans born in the United States and those born in Mexico were examined as part of a study by Wilkinson et al. (2005). These researchers found that there were more current smokers born in the U.S. than in Mexico (31.3 percent compared to 27.2 percent).

In a study that examined experimental smoking and intention to smoke in a Chinese/Chinese American, Latino, Persian/Iranian, and White sample of adolescents,

researchers found that Latinos were most likely to have started experimenting with cigarette smoking and were also the most likely to have the intention to smoke in the next year (Nezami et al., 2005).

With respect to alcohol consumption, trends from 1984 to 1995 revealed that there was a drop in the proportion of drinkers who drank five or more drinks per occasion for Whites but not for Blacks and Hispanics (Caetano, 2003). In addition, the proportion of Hispanic men reporting three or more alcohol related problems almost doubled between 1984 and 1995 (Caetano, 2003). In other research, Hispanic men had the highest rates of ever having been arrested for DUI (19 percent) compared to White (13 percent) and Black (11 percent) men and they also had the highest cirrhosis death rates (20.1 per 100,000) compared to 12.4 per 100,000 for Black men and 10.2 per 100,000 for White men (Stinson, Grant, & Dufour, 2001).

Mexican Americans have been found to have more alcohol-related problems among Hispanics. In a study that examined drinking patterns in Cubans, Mexican Americans, Puerto Ricans and other Hispanics, significant differences were found among the groups (Nielsen, 2000). In this study, Mexican American men reported the highest frequency of drinking alcohol and also reported drinking the heaviest as compared to other groups. With respect to gender, Mexican American women were most likely to abstain from heavy drinking than the other groups (Nielsen, 2000).

In a study that examined alcohol consumption in three generations of Mexican Americans, younger and middle-aged men were found to drink most frequently compared to the oldest generation (Markides, Krause, & Mendes de Leon, 1988). However, alcohol consumption problems have been identified as persisting among Mexican American men

until well into late middle age when it is hypothesized that they are forced to abstain or reduce their consumption levels due to health problems (Markides & Black, 1996).

Two different models have been proposed regarding the relationship between drinking behavior and acculturation. According to the "acculturative stress" model, increases in drinking frequency and amount are due to stresses from acculturation into the larger society (Markides et al., 1988). In the "acculturation" model, patterns of alcohol consumption reflect the degree to which Mexican Americans have adopted the attitudes and behaviors of mainstream society (Markides et al., 1988). Findings from a study on drinking behaviors among Puerto Rican, Cuban-American, and Mexican American women supported the acculturation model (Black & Markides, 1993). In this study, higher levels of acculturation were related to the probability of being a drinker and to more frequent consumption in all three groups of women. Hispanic women adopt the less stringent drinking norms of larger society as they become more acculturated (Black & Markides, 1993; Markides et al., 1988). In the study that examined alcohol consumption and acculturation in three generations of Mexican Americans, there was a significant finding only for men in the middle-aged generation, whose higher acculturation scores were related to lower levels of alcohol consumption (Markides et al., 1988). These researchers propose that this finding supports the "acculturative stress" model because those who are least acculturated may turn to alcohol to cope with the stresses of acculturation.

CHAPTER 2:

DEPRESSION, PHYSICAL ILLNESS, AND HEALTH RISK BEHAVIORS

Rates of psychiatric disorders have been found to be significantly elevated in those with comorbid medical problems (Kessler, Ormel, Demler, & Stang, 2003). Patients with arthritis, heart disease, high blood pressure, and chronic lung disease have been found to have higher prevalence rates of affective, substance use, and anxiety disorders in their lifetime compared to persons with none of these chronic medical conditions (Wells, Golding, & Burnam, 1989).

The association between mental health and physical health is important because poor mental health has been found to affect the outcome and course of chronic illness by contributing to illness complications, increased health care costs, and increased mortality when assessed in those with a comorbid medical condition. In addition, there is increasing evidence that the relationship between mental health and physical health is bidirectional. Prospective studies have identified higher incidences of conditions including pulmonary disease, cancer, heart disease, and diabetes in subjects with pre-existing depression (Pembroke, Rasul, Hary, Smith, & Stansfield, 2006; Friedman, 1994; Hippisley-Cox, Fielding, & Pringle, 1998; van den Akker, Schuurman, Metsemakers, & Buntinx, 2004).

The presence of increased health risk behaviors, such as cigarette smoking, alcohol abuse, physical inactivity, and poor adherence to medical regimens have been implicated as possible causes of poor outcomes in those with comorbid mental and physical health disorders.

DEPRESSION AND PHYSICAL ILLNESS

Depression is the most common disorder associated with chronic medical illness. In a longitudinal study that evaluated the risk of depression after onset of a range of chronic medical illnesses, the risk of depression was found to be greatest in participants following the onset of cancer, arthritis, chronic lung disease, and heart disease (Polsky et al., 2005). Similarly, Patten (2001) demonstrated an increased risk of developing major depression with virtually any chronic medical illness.

The presence of depression also has important implications for physical health outcomes in the chronically ill. Blazer, Hybels, and Pieper (2001) found that significant depressive symptomatology in older adults was associated with a two-fold increase in mortality assessed after three years. Depression in those aged 85 and over was found to contribute to an increase of both cardiovascular and non-cardiovascular mortality (Vinkers Stek, Gussekloo, van der Mast, & Westendorp, 2004). In this study, depression was still associated with a two-fold increase in all-cause mortality after controlling for comorbid medical conditions.

The relationship between depression and mortality has also been identified in studies on Mexican American elders. Black and Markides (1999) examined depressive symptoms in elderly Hispanics with diabetes, cardiovascular disease, hypertension, stroke, and cancer. They found that mortality rates were two to four times greater in those study participants that reported the comorbidity of depressive symptomatology with a chronic physical illness.

In addition to affecting the course of an illness, depression has been associated with higher medical costs in the chronically ill. Unutzer et al. (1997) in a 4-year

prospective study examined whether depressive symptoms in older adults (assessed with the CES-D) increased the cost of general medical services. In this investigation, health care costs were about 50% higher in participants with significant depressive symptoms than in those without.

Researchers have also examined the impact of varying levels of depressive symptomatology. The baseline data from an intervention study of 1801 depressed people aged 60 and over revealed that severity of depression was significantly associated with four indicators of general health status (Noel et al., 2004). In this study, increased severity of depression was associated with poorer physical and mental functioning, lower quality of life, and increased disability. The relationships between increased depression and poorer general health in this study remained even after controlling for sociodemographic variables, other psychological conditions, and eleven comorbid medical conditions. Therefore, the severity of depressive symptoms was more strongly associated with general health indicators in depressed elders than most chronic physical illnesses (Noel et al., 2004). The effects of both minor and major depression on mortality among older persons aged 55-85 was evaluated utilizing the CES-D to measure depressive symptoms and the Diagnostic Interview Schedule (DIS) to assess the presence of major depression (Penninx et al., 1999). In this study, both minor depression and major depression increased the risk of dying. Minor depression was found to increase the risk of dying for older men and major depression for both older men and women even after adjustment for socidemographics variables, health status, and health behaviors (Penninx et al., 1999).

Depression and Coronary Artery Disease

The presence of depressive symptoms is common in patients with CAD and has been studied extensively. Hance, Carney, Freedland, and Skala (1996) administered the DIS to 200 patients under the age of 75 who were being evaluated for the presence of CAD. These researchers found that 17 percent of these patients met the DSM-IV criteria for major depression and that an additional 17 percent met criteria for minor depression.

In patients with CAD, the presence of depression has often been found to be stable and enduring. Holahan, Moos, Holahan, and Brennan (1995) found that those with acute and chronic cardiac illness reported higher rates of depressive symptoms even 1 year after an initial interview. In the Hance et al. (1996) study, patients who met criteria for either major or minor depression at the baseline evaluation were contacted by telephone and interviewed at 3 weeks, and at 3, 6, 9, and 12 months following the initial interview. The results of the study showed that fifty percent of the patients with major depression at baseline remained depressed or remitted only to relapse at follow-up.

Rates of depression have also been found to be elevated in patients following coronary artery bypass graft (CABG) surgery and following a myocardial infarction (MI). In one study, the CES-D was administered to patients who underwent CABG surgery pre- and post-procedure (Pirraglia, Peterson, Williams-Russo, Gorkin, & Charlson, 1999). The results of the study found that pre-operatively, the rate of significant depressive symptoms was 43.1 percent. Six months following surgery, the rate of depression was still elevated at 23.4 percent (Pirraglia et al., 1999). Schleifer et al. (1989) conducted a prospective study on the prevalence and course of depression in patients who had suffered an MI. Patients in this study were administered the Schedule for Affective

Disorders and Schizophrenia eight to ten days post- MI and three to four months post-MI. Research Diagnostic Criteria was used to identify major and minor depressive disorders by researchers and they found that 27 percent of the sample met criteria for major depression and that 18 percent met criteria for minor depression eight to ten days post-MI. Additionally, 83 percent of those diagnosed with major depressive disorder one week post-MI continued to meet criteria for either major or minor depression three months later (Schleifer et al, 1989).

Depressive symptoms in patients with CAD increase the risk of cardiac morbidity and mortality. Depression has been implicated as a risk factor for poorer health outcomes following a CABG. In a study done prospectively on patients who had undergone a CABG, investigators studied the relationship between depression and coronary outcomes one year later (Connerney, Shapiro, McLaughlin, Bagiella, & Sloan, 2001). As part of this research, patients who had undergone coronary surgery were administered a modified version of the DIS as well as the Beck Depression Inventory. The investigators identified that 20 percent of the these patients met modified diagnostic criteria for major depression, while 28 percent exhibited significant depressive symptoms on the BDI. Those patients who met criteria for major depressive disorder were more than two times as likely to die or to be re-hospitalized in the year following discharge from the hospital than those who were not depressed (Connerney et al., 2001).

Depression following an MI has also been linked to increased cardiac mortality. Frasure-Smith, Lesperance, & Talajic (1993) examined the relationship between major depression and cardiac mortality in patients hospitalized following an MI. These researchers administered a modified version of the DIS and the Beck Depression

Inventory to subjects between 5 and 15 days after an MI to evaluate the presence of major depressive episode and the presence of depressive symptoms. Patients in this study were followed over the course of 6 months in order to assess survival status. Major depression was a significant predictor of mortality 6 months following a myocardial infarction and as important a risk factor for mortality as other traditional clinical risk factors, such as left ventricular dysfunction and a history of previous MI (Frasure-Smith, et al, 1993).

These same patients were followed for an additional twelve months as part of another investigation (Frasure-Smith et al, 1995). Researchers found that by 18 months post-MI, both baseline major depression and depressive symptomatology continued to be a predictor of mortality (Frasure-Smith et al., 1995).

In a study with similar results, the BDI was administered to patients aged 65 and older who had suffered an acute myocardial infarction (Bush et al., 2001). In this study, severe depressive symptoms were associated with the highest mortality rates. However, it was also revealed in this study that patients who exhibited very low levels of depressive symptoms, as indicated by scores ranging from 4 to 9 on the BDI, also had elevated rates of mortality (Bush et al., 2001). Similarly, an increased risk of cardiac mortality in those with BDI scores lower than those customarily used to diagnose mild depression was observed for patients who had suffered an MI during initial hospitalization and one year later (Lesperance, Frasure-Smith, Talajic, & Bourassa, 2002).

In addition to being a risk factor for poor outcomes in patients with CAD, depression has also been identified as a risk factor for the development of CAD in healthy individuals. Ford et al. (1998) found that clinically depressed men free of cardiac illness at baseline were at a moderately increased risk of developing CAD when studied

over a period of 37 years. This risk was found to remain by the researchers, even after controlling for multiple cardiovascular risk factors.

Depression was also found to be an independent risk factor for the development of coronary heart disease and mortality after six years in a prospective study conducted with elderly people free of cardiovascular disease at baseline (Ariyo, 2000). Twenty percent of the subjects in this study met criteria for major depressive disorder at baseline. The risks of developing coronary heart disease and of mortality increased as the level depressive symptomatology increased (Ariyo et al., 2000).

In another study, recurrent major depression was found to be associated with a two- to three-fold increased risk of coronary and aortic calcification in a group of depressed women (Agatisa et al., 2005). Middle-aged healthy women with a history of recurrent major depression were studied as part of this research and were found to be at elevated risk of atherosclerosis. Women in this study with a history of recurrent major depression were more likely to be classified in the groups at the highest level of calcification of the coronaries and aorta compared with women with a single episode of depression or with no history of major depression. These results suggest one potential pathway of clinical depression in the early development of sub-clinical heart disease (Agatisa et al., 2005).

In addition to clinical depression, the presence of sub-clinical depressive symptoms has also been shown to increase the risk of developing heart disease. In one study, depressive symptoms, as measured by the CES-D, were related to the incidence of coronary heart disease in men and women (Ferketich, Schwartzbaum, Frid, & Moeschberger, 2000). In another study that examined depressive symptoms utilizing questions from the General Well-Being Schedule, an increased risk of both fatal and non-

fatal heart disease events was observed with increased depressive symptomatology (Anda et al., 1993). The increased risk of fatal and non-fatal events was evident for those with both moderate and severe levels of depressive symptoms, delineating the importance of even sub-threshold levels of depression (Anda et al., 1993).

Depression and Diabetes

Higher rates of depressive symptomatology have been found in those with diabetes when compared to the general population. Peyrot and Rubin (1997) found that more than forty percent of patients with diabetes had levels of depressive symptoms consistent with a clinical diagnosis of depression. Lustman, Griffith, and Clouse (1988) studied depression in patients with diabetes and identified the course of depression in these subjects as more severe and persistent than in those without diabetes.

The increased incidence of depressive symptoms in those with diabetes may be due to in part to poor glycemic control in patients with diabetes. Gross et al. (2005) found that poor glycemic control was three times as likely among diabetes patients with major depression compared to those without depression. The authors propose several behavioral and biological mechanisms that might be responsible for the association between depression and poor glycemic control. One reason for poor glycemic control in those with depression may be a result of noncompliance with medical treatment recommendations (Gross et al., 2005).

The impact of depression on patients with diabetes may be especially important because of the psychological and behavioral self-care demands associated with this illness (Ciechanowski, Katon, & Russo, 2000). The potential impact of self-care motivation was examined in a study on the effects of depression and diabetes on the

incidence of diabetes related complications, functional disability, and mortality among older Mexican Americans (Black, Markides, & Ray, 2003). In this investigation, the interaction between diabetes and depressive symptoms predicted increased mortality, complications, and disability, as well as earlier occurrence of these outcomes. The risks of these adverse outcomes in this study increased with the increasing severity of depression. One manner in which depression is believed to influence the course of diabetes is through decreased motivation to maintain proper self-care behaviors in order to protect against the development of diabetes or progression of the disease (Black, Markides, & Ray, 2003).

Although the increased severity of depressive symptoms has been linked to worsening outcomes, the importance of both severe and moderate levels of depressive symptoms in those with diabetes has also been established. For example, a high depressive symptoms score, as assessed by the CES-D, was more strongly related to diabetes specific distress and diabetes management than a diagnosis of major depressive disorder (Fisher et al., 2007). And, although researchers found that the odds of having died among diabetics with high levels of depressive symptoms was three times that of diabetics without high levels of depressive symptoms, an increased risk of mortality was also evident for those with moderate levels of depressive symptoms (Black & Markides, 1999). In addition, Katon et al. (2005) found that both major and minor depression were related to significantly higher mortality in patients with type 2 diabetes compared to non-depressed type 2 patients.

Epidemiological research indicates not only that depression is common in patients with diabetes and affects its course, it also implicates depression as an independent risk

factor for the development of type II diabetes. Kawakami, Takatsuka, Shimizu, & Ishibashi (1999) studied the relationship between depressive symptoms and the incidence of type II diabetes and found that subjects with either moderate or severe levels of depressive symptoms at baseline had an 2.3 times increased risk of having type II diabetes at 8 year follow-up. These researchers suggest that physicians should closely monitor metabolic changes in depressed patients for the prevention and early detection of type II diabetes.

DEPRESSION AND HEALTH RISK BEHAVIORS

Preventable modifiable behavioral health risk factors are the leading causes of death in the United States and smoking persists as the leading modifiable cause of death (Mokdad, Marks, Stroup, & Gerberding, 2004). There is substantial evidence that personal health behaviors are associated with depressive symptoms and psychological well-being. Depressed mood has been linked to cigarette smoking and alcohol consumption. Depressive symptoms in young men and women have been associated with several unhealthy personal behaviors including smoking, lack of physical exercise, poor sleeping habits, not eating breakfast, and not using a seat belt (Allgöwer, Wardle, & Steptoe, 2001).

Personal health behaviors take on a new significance in those with chronic medical illnesses. Adherence to medical regimens and recommendations could potentially make the difference between positive and negative medical outcomes. Researchers have shown that depression is associated with poor adherence to medical treatment regimens in patients with diabetes and heart disease (Carney, Freedland, Eisen, Rich, & Jaffe, 1995; Gonzalez et al., 2007). In addition, a higher proportion of depressed

patients with diabetes report health risk behaviors including infrequent exercise, an unhealthful diet, and increased rates of cigarette smoking (Lin et al., 2004).

Depression and Cigarette Smoking

The relationship between depression and the health risk behavior cigarette smoking has been well established. Glassman et al. (1990) studied major depressive disorder (MDD) in smokers and never smokers. They found the lifetime prevalence of MDD for those who had ever smoked for a least one month to be 6.6 percent compared to 2.9 percent for never smokers.

A relationship between depression and smoking has been found across different age groups. In a study that assessed smoking and depressive symptoms in adults aged 65 and older, Salive and Blazer (1993) found that the percentage of those with clinically significant CES-D scores was highest among current smokers as compared to never smokers and former smokers.

Additionally, the relationship between depression and cigarette smoking has also been observed in Latino adults and adolescents. Depression and smoking was evaluated in 2090 adults of Mexican, Puerto Rican, and Cuban descent using the Hispanic Health and Nutrition Examination Survey Data (Escobedo, Kirch, & Anda, 1996). This national survey used the CES-D to assess depressive symptomatology and the DIS to assess major depression. Both depressive symptomatology and major depression were found to be associated with the likelihood of smoking across all Latino sub-groups examined as part of this investigation. The associations between depressive states and smoking were found to be most consistent among Mexican Americans, women, and those with less than a high school education (Escobedo, Kirch, & Anda, 1996). In a study on smoking and

depressive symptomatology in adolescents of Mexican American descent, investigators found that both male and female smokers reported feelings of depression more frequently than their non-smoking counterparts (Pesa, Cowdery, Wang, & Fu, 2001).

Increased depressive symptomatology has also been implicated as a precursor to initiating smoking behavior. Nezami et al. (2005) studied the relationship between depressive symptoms and intention to smoke or experiment with smoking in a sample of Chinese/Chinese American, Latino, Persian/Iranian, and non-Hispanic White adolescents. Depressive symptoms in this study were associated with an increased probability of experimentation with smoking and with intention to smoke across all ethnic groups.

Depression and Alcohol Abuse

The association between major depression and alcohol use disorders has been examined in several studies. One study involved a large national sample and utilized the Alcohol Use Disorder and Associated Disabilities Interview Schedule (Grant & Harford, 1995). In this study, the increased risk of having an alcohol use disorder was four times greater among those with major depression than in those without major depression. Alcohol dependence in this study was more strongly related to major depression than alcohol abuse. Additionally, over 21 percent of subjects with major depression reported either alcohol use or dependence in the year preceding the interview (Grant & Harford, 1995).

The relationship between depression and alcohol abuse has also been established in women. Using longitudinal data from the Canadian National Health Survey, investigators in one study assessed the role that alcohol consumption plays in major and minor depression (Wang and Patten, 2002). Subjects without major depression at

baseline were selected for this study and were interviewed regarding their alcohol use in the previous year. The prevalence of major depression was assessed in these subjects two years later. Researchers found that the incidence of major depression was more likely for women who drank heavily at least once a month compared to those who did not (Wang and Patten, 2002). Another study found the risk of major depression to be three times as high in women with alcohol use disorders compared to those without (Golding, Burnam, Benjamin, & Wells, 1992).

In a study that examined the prevalence of depression in community residents with alcohol use disorders, the highest rate of absolute risk of major depression was highest for Mexican American women, followed by non-Hispanic White women, non-Hispanic White men, and finally, by Mexican American men (Golding, Burnam, Benjamin, & Wells, 1992). Low acculturation was also observed in this study to be related to developing secondary depression in those with alcohol use disorders.

Depression and Physical Inactivity

Physical inactivity has been identified as an important risk factor for morbidity and mortality associated with CAD. Physical inactivity is inversely associated with mental health, including depression. The relationship between physical activity and depressive symptomatology in men and women was examined as part of a longitudinal study (Galper, Trivedi, Barlow, Dunn, & Kampert, 2006). In this study, which utilized the CES-D to assess depressive symptoms, investigators observed a dose-response relationship between depressive symptoms and physical inactivity. In another study, Wassertheill-Smoller et al. (2004), studied correlates of cardiovascular disease in women

and also found that lower rates of depressive symptomatology were associated with lower rates of physical activity, and therefore, lower rates of cardiovascular disease.

A relationship between depression and obesity has also been established in adolescents. Researchers studying obesity used data from a nationally representative cohort of more than 9000 adolescents (Goodman and Whitaker, 2002). They found that depressed mood at baseline was associated with the development of obesity in those not yet obese at baseline. Depressed mood was also associated with an increase in BMI in those already obese at baseline (Goodman and Whitaker, 2002).

HYPOTHESES

All hypotheses will be tested both longitudinally and prospectively (i.e., controlling for outcome variables at baseline) across a seven-year period.

Physical Illness as a Predictor of Depressive Symptomatology

Hypothesis 1: Extending previous research on the relationship between cardiovascular disease and depressive symptoms (Hance et al., 1996; Schleifer et al., 1989), it is hypothesized that cardiovascular illness at Time 1 will predict depressive symptomatology at Time 2.

Hypothesis 2: It is hypothesized that more cardiovascular illness complications at Time 1 will add unique variance, beyond that predicted by cardiovascular illness diagnosis alone, in predicting depressive symptomatology at Time 2.

Hypothesis 3: Extending previous research on the relationship between diabetes and depression (Peyrot & Rubin, 1997), it is hypothesized that diabetic illness at Time 1 will predict depressive symptomatology at Time 2.

Hypothesis 4: (Katon et al., 2005), it is hypothesized that more diabetic illness complications at Time 1 will add unique variance, beyond that predicted by diabetic illness diagnosis alone, in predicting depressive symptomatology at Time 2.

Hypothesis 5: Extending and integrating previous research on cardiovascular illness, diabetic illness, and depressive symptoms (Hance et al., 1996; Peyrot & Rubin, 1997), it is hypothesized that the comorbidity of cardiovascular illness with diabetic illness at Time 1 will add unique variance, beyond that predicted by either disease alone, in predicting depressive symptomatology at Time 2.

Figure 1 presents a graphic overview of Hypotheses 1-5 in an integrative conceptual model.

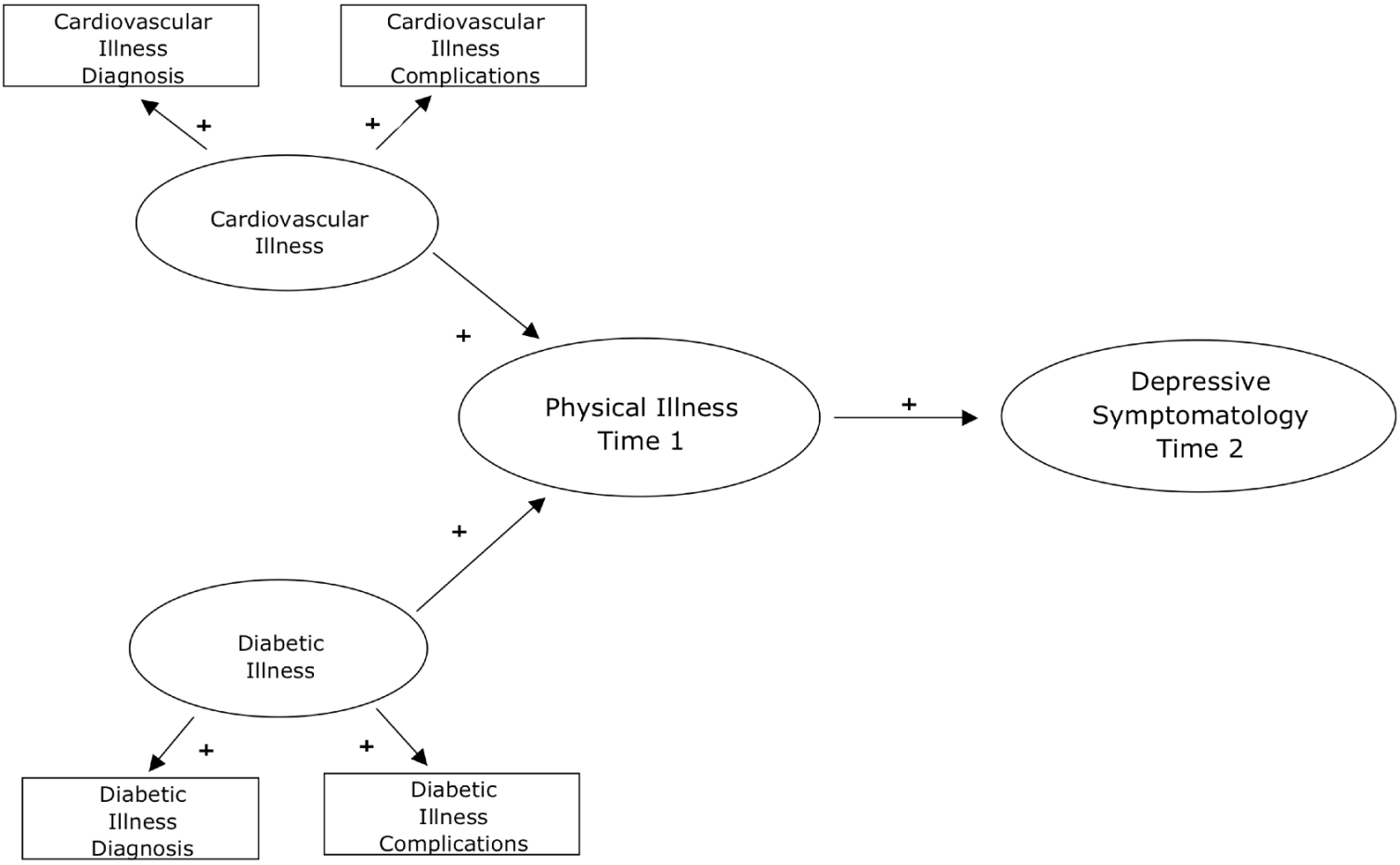


Figure 1: Physical Illness as a Predictor of Depressive Symptomatology

Depressive Symptomatology as a Predictor of Physical Illness and Mortality

Hypothesis 6: Extending previous research on depressive symptoms and cardiovascular illness (Ariyo et al., 2000), it is hypothesized that depressive symptomatology at Time 1 will be positively related to both cardiovascular illness diagnosis and cardiovascular disease complications at Time 2.

Hypothesis 7: Extending previous research on depressive symptoms and diabetes (Kawakami et al., 1999), it is hypothesized that depressive symptomatology at Time 1 will be positively related to both diabetic illness diagnosis and diabetic illness complications at Time 2.

Hypothesis 8: Extending previous research on depression and mortality (Schulz et al., 2000), it is hypothesized that depressive symptomatology at Time 1 will be positively related to mortality at Time 2.

Hypothesis 9: Extending previous research on depressive symptomatology and cigarette smoking in non-Hispanic samples (Glassman et al., 1990; Salive & Blazer, 1993), it is hypothesized that depressive symptomatology at Time 1 will be positively associated with cigarette smoking at Time 1.

Hypothesis 10: Extending previous research on depressive symptomatology and alcohol abuse in non-Hispanic samples (Grant & Harford, 1995; Golding et al., 1995), it is hypothesized that depressive symptomatology at Time 1 will be positively associated with alcohol abuse at Time 1.

Hypothesis 11: Extending previous research on depressive symptomatology and physical activity in non-Hispanic samples (Galper et al., 2006), it is hypothesized that

depressive symptomatology at Time 1 will be positively associated with body mass index at Time 1.

Hypothesis 12: Integrating hypothesis 6 with hypotheses 9 through 11, it is hypothesized that the relationship between depressive symptomatology at Time 1 and cardiovascular illness at Time 2 will be partially mediated by behavioral health risk factors at Time 1.

Hypothesis 13: Integrating hypothesis 7 with hypotheses 9 through 11, it is hypothesized that the relationship between depressive symptomatology at Time 1 and diabetic illness at Time 2 will be partially mediated by behavioral health risk factors at Time 1.

Hypothesis 14: Integrating hypothesis 8 with hypotheses 9 through 11, it is hypothesized that the relationship between depressive symptomatology at Time 1 and mortality at Time 2 will be partially mediated by behavioral health risk factors at Time 1.

Figures 2 and 3 present graphic overviews of Hypotheses 6-14 in integrative conceptual models.

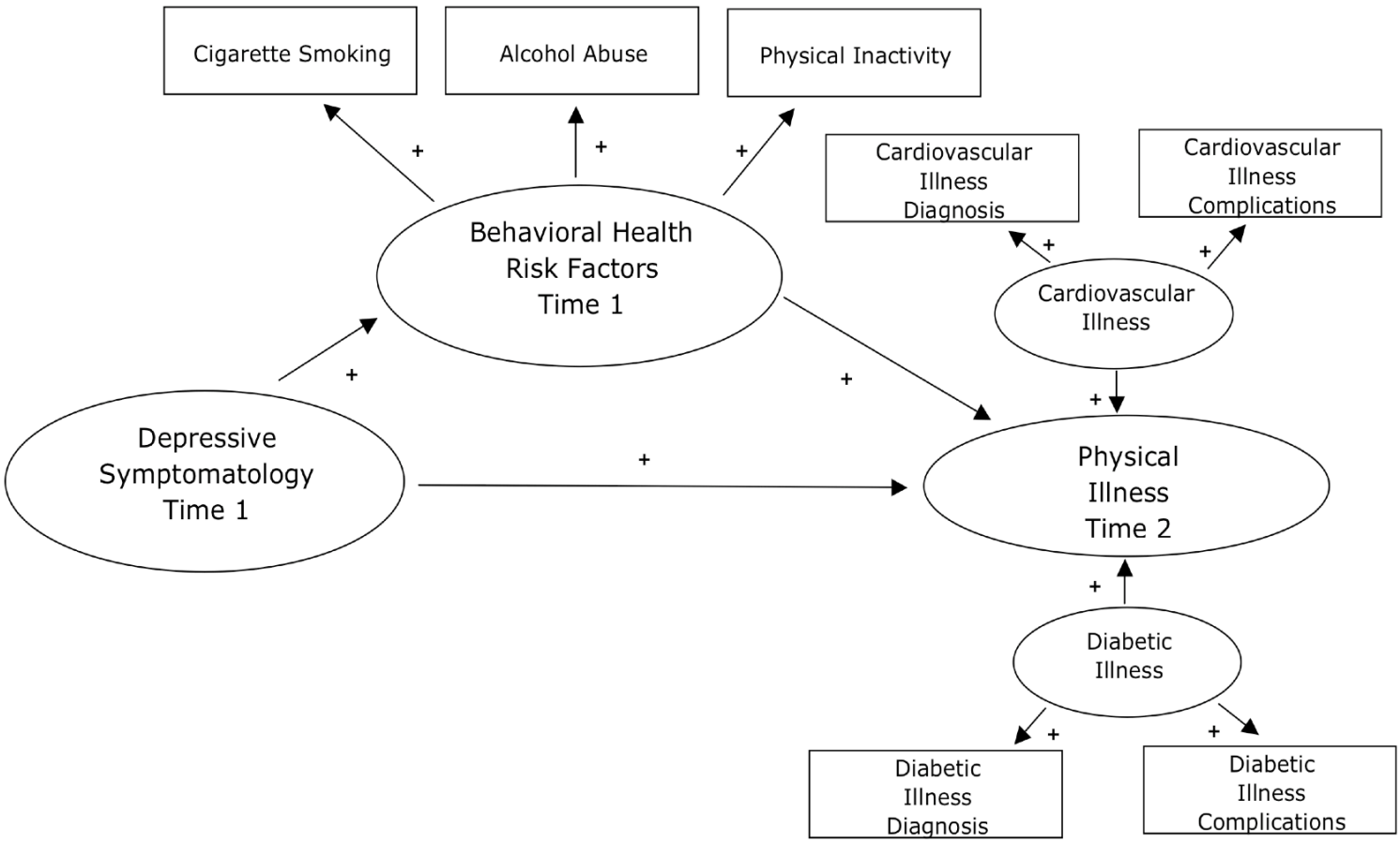


Figure 2: Depressive Symptomatology, Behavioral Risk Factors, and Physical Illness. Body Mass Index was used as a proxy measure for Physical Inactivity.

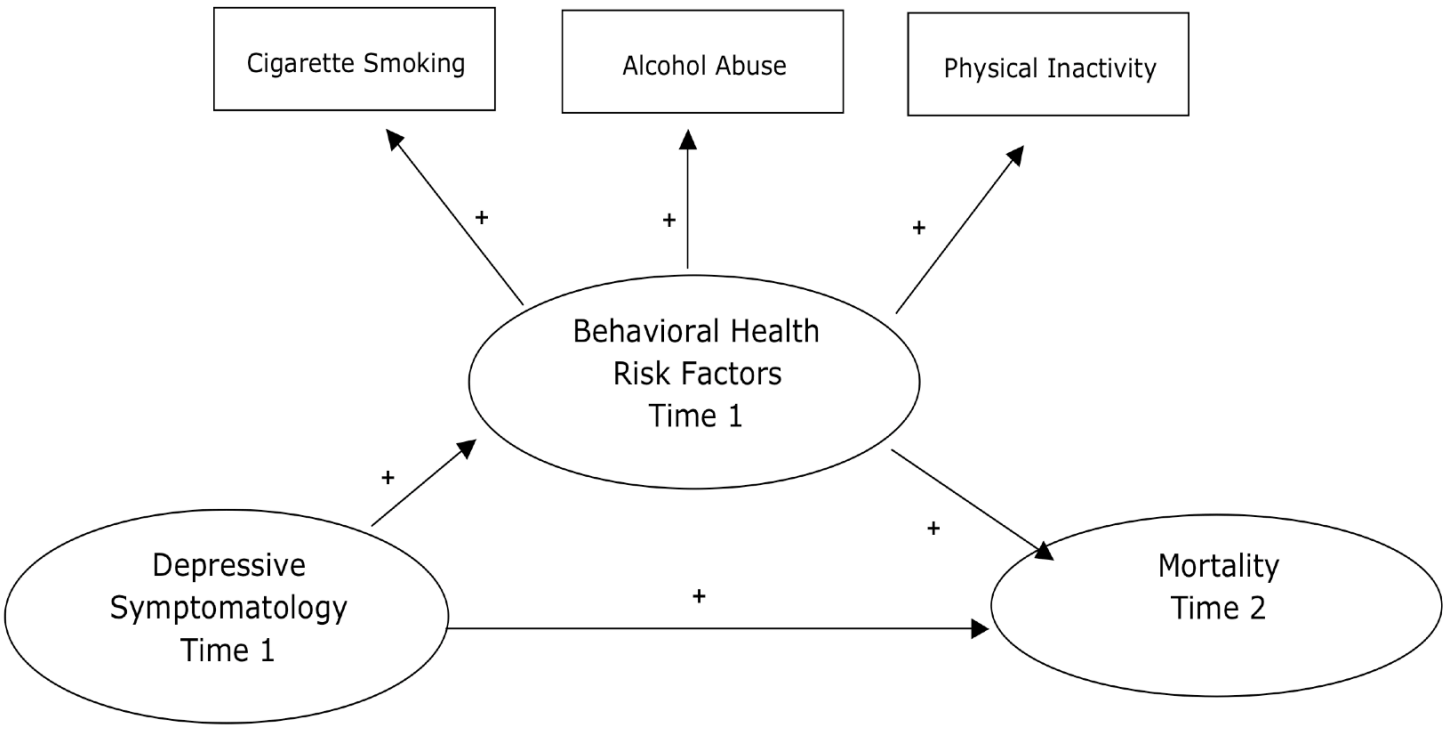


Figure 3: Depressive Symptomatology, Behavioral Health Risk Factors, and Mortality

EXPLORATORY QUESTIONS

Exploratory question 1: The study will explore the role of gender as a moderator of the relationships in Figures 1 through 3.

Exploratory question 2: The study will explore the role of acculturation as a moderator of the relationships in Figures 1 through 3.

Exploratory question 3: The study will explore the role of perceived health locus of control as a moderator of the relationships in Figures 1 through 3.

Exploratory question 4: The study will explore the relative contribution of threshold depression in contrast to sub-threshold depression in predicting the relationships in Figures 1 through 3.

Exploratory question 5: The study will explore the relative contribution of self-rated health at Time 1 in contrast to Physical Illness Indicators (i.e., cardiovascular illness diagnosis, cardiovascular illness complications, diabetic illness diagnosis and diabetic illness complications) at Time 1 in predicting depressive symptomatology at Time 2.

Exploratory question 6: The study will explore the relative contribution of self-rated health at Time 1 in contrast to Physical Illness Indicators (i.e., cardiovascular illness diagnosis, cardiovascular illness complications, diabetic illness diagnosis and diabetic illness complications) at Time 1 in predicting mortality at Time 2.

CHAPTER 3: METHOD

The proposed study will use data from the Hispanic Established Populations for Epidemiologic Studies of the Elderly (H-EPESE) at two different time points, seven years apart. Depressive symptomatology and physical illness will be measured at baseline and at a seven-year follow-up. Behavioral health risk factors will be measured at baseline. Baseline measures of acculturation, health locus of control, and self-rated health will also be included in exploratory analyses. Mortality, cause of death, and survival time since baseline will be assessed using follow-up data collected at the seven-year follow-up.

H-EPESE DATA

The first wave of data for the H-EPESE was collected September 1993 through June 1994 (Markides, 1999). Data were collected on a sample of non-institutionalized Mexican American elderly, aged 65 years and older from the southwestern states of Arizona, California, Colorado, New Mexico and Texas (Markides, 1999). The follow-up data used here were collected in 2000-2001, seven years later. The original full cohort of 3,050 participants, which is used here, is generalizable to over 500,000 Mexican Americans living in the southwestern United States (Markides, 1999).

The design of the H-EPESE was modeled after the Established Populations for Epidemiologic Studies of the Elderly (EPESE) studies that took place between 1981 and 1993 in East Boston, Massachusetts, Iowa and Washington Counties, Iowa, New Haven Connecticut, and North Central North Carolina (Markides, 1999). These studies were

established by the National Institutes of Health to ascertain community-based data on the physical and mental health of older Americans (Markides, 1999). The H-EPESE was initiated in order to gather data on the Hispanic elderly to compare to the epidemiologic data on elderly non-Hispanic Whites and African Americans gathered at the other EPESE sites (Markides, 1999). The goal of the H-EPESE was to provide data on the "prevalence of physical health conditions, mental health conditions, and functional impairments in older Mexican Americans and to compare these estimates with those for other populations" (Markides, 1999). Additionally, the researchers wanted to investigate predictors and correlates of important physical health outcomes (Markides, 1999).

SUBJECTS

Subjects for the study were chosen based on a multi-stage probability sampling procedure (Markides, 1999). Based on Census Bureau data, counties were first ranked by the number of Mexican American elderly. A cutoff was established based on the cumulative total of Mexican American elderly. The universe from which counties were selected was based on this cutoff. Census tracts were then selected from these counties. Households were selected from these census tracts by interviewers who went door-to-door to screen subjects. Eligible subjects were chosen based on age, self-identification as Mexican American, birthplace, and a review of the ethnic background of the person's parents and grandparents. These screenings yielded a total of 3050 subjects. The number of subjects interviewed in person was 2,873 while 177 were interviewed by a proxy who in most cases was a family member close to the subject. The response rate was 86 percent which is similar to rates achieved in the other EPESE studies. Data were

available on 1,685 subjects at seven years. The total number deceased was 940 at seven years (Markides, 1999).

PROCEDURE

Interviews at baseline and at the follow-up interview were conducted in the respondent's home by trained bilingual interviewers. Respondents were able to choose whether to be interviewed in Spanish or in English. The interviews took an average of 1 hour and 45 minutes to complete (Markides, 1999).

MEASURES

Items on the baseline interview included self-reports of sociodemographic, cultural, and health-related measures as well as cognitive and physical assessments (Markides, 1999). In addition to the items on the original interview, vital status was established during the follow-up interviews (Markides & Ray, 2009).

Sociodemographic Characteristics

Questions on age, gender, years of education, employment status, marital status, bereavement status, living arrangements, personal and household income and type of health insurance were included in the interview.

Physical Illness

The presence of self-reported chronic medical conditions was assessed during the interviews.

Coronary Artery Disease Diagnosis. There was one question that established the presence of CAD. It was: "Has a doctor ever told you that you had a heart attack, or coronary, or myocardial infarction, or coronary thrombosis?"

Coronary Artery Disease Complications. Subjects answered eight questions on chest pain and shortness of breath including: "Have you ever had pain or discomfort in your chest?" and "Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?"

Diabetic Illness Diagnosis. The following question was asked to determine if the subject had diabetes: "Have you ever been told by a doctor that you have diabetes, sugar in your urine, or high blood sugar?"

Diabetic Illness Complications. Subjects answered eight questions regarding diabetes related complications including: "As a result of your diabetes, have you ever had any problems with your kidneys?" and "As a result of your diabetes, have you ever had any problems with your eyes?"

Subjects were also asked if they had ever had the following chronic illnesses: hypertension, stroke, cancer, hip and other bone fractures, arthritis, and gallbladder disease.

Depressive Symptomatology

Depressive symptoms were measured with the Center for Epidemiologic Studies Depression Scale (CES-D). This scale was designed to assist in the identification of depressive symptoms in community surveys. The CES-D has been widely used as a survey measure of psychological distress in studies involving older adults. It has been found to have high reliability and validity in the older adult population (Radloff & Teri, 1986).

The scale is made up of 20 items that ask questions about how frequently specific symptoms were present during the past week. Sample questions include, "I was bothered by things that usually don't bother me," "I felt sad," "People were unfriendly," and "I enjoyed life." Responses on this measure are scored using a 4-point scale, with total scores ranging from 0-60. A score of 16 or greater the CES-D is generally used to indicate depressive symptomatology in the clinical range (Boyd, Weissman, Thompson, & Myers, 1982). This scale has been shown to predict both current depression as well as future clinical depression, but does not provide a diagnosis of depression (Roberts & Vernon, 1983).

Behavioral Health Risk Factors

The health behaviors of smoking, alcohol use, and obesity were assessed as part of the H-EPESE.

Smoking. The smoking measure consisted of 5 questions and included items on current smoking status, age at onset of smoking current smoking, and number of cigarettes currently or previously smoked on a regular basis. Ever-smoking was assessed based on the subject's response to the question: "Have you ever smoked at least 100 cigarettes in your entire life?"

Alcohol Consumption. Nine items made up the alcohol consumption measure. These items assessed ever- drinking, current drinking, amount of alcohol in the last month, and problem drinking. Questions on problem drinking included "Have you ever felt you should cut down on your drinking?" and "Have you ever felt you should cut down on your drinking?"

Body Mass Index. Obesity was defined as a body mass index (BMI) greater than 30. BMI was assessed using the subject's height and weight which were obtained at the time of the interview.

Mortality, Cause of Death, and Survival Time

Mortality, cause of death, and survival time were ascertained from vital status data gathered at the each of the three follow-up waves. Proxy respondents were interviewed if a subject was missing at follow-up. They were asked whether the subject had died, had moved, or had been admitted to a hospital, nursing home or hospice facility. If the subject had died, proxies were asked questions from a death questionnaire that asked the date, location, and cause of death. This questionnaire also included questions about admissions to the hospital or nursing home that had occurred prior to the subject's death. The overwhelming majority (86 percent) of proxies were family members of the missing subject (Markides & Ray, 2009).

Vital status information obtained from interviews with proxy respondents was compared with information from death records in the National Death Index database (Patel et al, 2004).

Potential Moderators

Acculturation. Levels of acculturation and assimilation were measured using a modified version of the Acculturation Scale (Hazuda, Stern, & Haffner, 1988). The acculturation measure was made up of 10 items that assessed English and Spanish language use, including questions on language spoken with spouse, language spoken with children, language spoken with friends, and language of television programs watched.

Possible scores on this measure ranged from 3 to 50, with lower scores indicating lower acculturation.

The assimilation measure consisted of 3 items that assessed integration into mainstream society. Questions regarding ethnicity of neighbors, coworkers, and close personal friends make up this measure. Possible scores ranged from 3 to 9 and lower scores were indicative of lower assimilation (Black et al., 1998).

Self-rated health. An assessment of the subject's self-rated health was assessed in response to the question: Overall, how would you rate your health? excellent, good, fair or poor?

Health locus of control. Health locus of control was ascertained based on the subject's response to the following questions:

1) How concerned are you about being unable to be independent and take care of yourself and your affairs in the future? Would you say you are very concerned, somewhat concerned, or not concerned at all?
2) To what extent do you feel you can control the general state of your health through your own actions—a great deal, somewhat or not at all? How is this scaled?

H-EPESE SAMPLE CHARACTERISTICS

The average age of participants in the H-EPESE at baseline was 72.6 years, for both genders. Women made up 56.8 percent of the total sample and men accounted for 43.2 percent. The respondents in the sample had a low level of education with 51.6 percent reporting less than five years of education. More than one-third of the

participants (34.3%) had an income of less than $5000 and 9 percent had no type of insurance coverage (Rudkin, McColgin, & Peek, 2003).

Depressive Symptoms

High levels of depressive symptoms on the CES-D were reported at baseline by over one fourth of the respondents (25.6 percent). Women reported a rate of depressive symptoms much higher than men (28.6 percent compared to 17.4 percent) (Black & Ray, 2003).

Health Risk Behaviors

Cigarette Smoking. While 12 percent of the respondents reported being current smokers 43.1 percent reported smoking at any time during their life. Rate of current smoking was 17.3 percent for men and 8.2 percent for women. Males were also more likely to have ever smoked as compared to females (61.4 percent compared to 29.3 percent (DiNuzzo & Randolph, 2003).

Alcohol Consumption. Over one-half of the respondents reported never consuming alcohol in their lifetime, with 70 percent of women and 24.2 percent of men being lifetime abstainers. Only 16.4 percent reported drinking within the past month and only 10 percent consumed 2 or more ounces per day. Males reported drinking 2 or more ounces of alcohol at a rate of 13.1 percent and women at 1.7 percent (DiNuzzo & Randolph, 2003).

Physical Health Outcomes at Baseline

Diabetes. The prevalence of self-reported diabetes was 27 percent. The prevalence for males was 28.2 percent compared to 26.1 percent for women. Only 67.7

percent of males and 73 percent of females who reported having diabetes were currently taking diabetes medication (Miles et al., 2003).

Coronary artery disease. A history of heart attack was reported by 11 percent of the sample. Males (12.0 percent) had a slightly higher prevalence of history of heart attack compared to females (10.3 percent) (Miles et al, 2003).

Hypertension. A diagnosis of hypertension was reported by 45.8 percent of the respondents, 52.7 percent of women and 36.6 percent of men. Medication for the treatment of hypertension was reported by 74 percent of those who had been told they had hypertension (Miles et al., 2003).

Stroke. Stroke was reported by 7.8 percent of the total sample, with an 8.4 percent prevalence in males and 7.3 percent in females (Miles et al., 2003).

Cancer. A self-reported history of cancer was made by 6.9 percent of the sample. Females had a slightly higher rate with 7.3 percent while men had a rate of 6.3 percent (Miles et al., 2003).

Hip and Other Bone Fractures. Hip fractures were reported by 4.3 percent of the participants and 14 percent reported other types of fractures. Women reported higher rates of hip fractures than men (5.4 percent versus 3.0 percent). They also had higher rates of fractures other than hip with a 16.5 percent rate compared to 11.4 percent of men (Miles et al., 2003).

Arthritis. Overall, 40.6 percent of the respondents reported having arthritis. Females had a significantly higher prevalence than males (49.6 percent compared to 28.7

percent). Of those who reported having arthritis, 76.8 percent had been prescribed medication for this condition (Miles et al, 2003).

Gallbladder disease. Gallbladder disease was found to be more prevalent in this sample as compared to national samples with 19 percent of participants reporting a history of gallbladder surgery. Women reported a rate twice that of men (24.8 percent compared to 11.4 percent) (Miles et al., 2003).

PLANNED STATISTICAL ANALYSES

In addition to descriptive and correlational analyses, hypotheses will be tested using multiple regression analyses and, where appropriate, structural equation modeling. Direct effects of predictive factors will be tested using multiple linear regression and (for binary outcomes) multiple logistic regression. Mediation effects will be tested using structural equation modeling. Moderating effects will be tested using product terms in the multiple regression analyses and multi-sample models in the structural equation analyses.

CHAPTER 4: RESULTS

This chapter is divided into eight main sections. The first section summarizes the demographic characteristics of subjects at both time points utilized for the study. The second section presents a descriptive summary of the primary study variables. The third section gives an overview of the relationships among the primary study variables by examining them cross-sectionally and longitudinally at the two time points studied. The fourth section addresses the first five hypotheses proposed in Chapter 2. These hypotheses relate to physical illness as a predictor of depressive symptoms. The fifth section covers hypotheses six through eight. These hypotheses address depressive symptomatology as a predictor of physical illness and mortality. The sixth section is on hypotheses nine through eleven which examine the relationship between depressive symptoms and behavioral risk factors at Time 1. Section seven presents the results of the integrative models proposed by hypotheses twelve through fourteen. These hypotheses examine the role of behavioral risk factors at Time 1 as mediators between Time 1 depressive symptoms and Time 2 outcomes. Section eight examines the exploratory analyses proposed at the end of Chapter 2.

SUBJECT CHARACTERISTICS

Sociodemographic Characteristics

There were 3050 subjects in the total sample at baseline. The Time 1 characteristics are summarized in Table 1. At Time 1, the total sample was made up of more women than men (57.6% vs. 42.4%). The average age of participants at baseline

was 73 years of age. Age results were almost identical when analyzed separately for men and women. There were no gender differences in age when a one-way ANOVA was calculated ($F(1,3048) = .001, p > .05$). Fifty-six percent of the sample was born in the U.S., while the rest was born in Mexico. Results for nativity were similar for both men and women (55.3% compared to 56.4%). A chi-square was calculated for nativity by gender and was not found to be significant ($x^2(1) = .46, p > .05$).

Table 1
Sample characteristics at baseline for full sample and men and women

Characteristic	Total Sample (N=3050)	Men (N=1292)	Women (N=1758)
Age, *M* years (*SD*)	73.01 (6.75)	73.00 (6.68)	73.01 (6.81)
% US born	55.9	55.2	56.4
% with heart disease	9.3	11.3	7.9
% with complications from CAD (of those with CAD)	62.9	60.1	65.9
% with diabetes	22.6	22.5	22.7
% with DM complications (of those with DM)	60.6	59.5	61.4
CES-D Total, mean score (*SD*)	9.94 (9.60)	8.10 (8.56)	11.23 (10.06)
% CES-D ≥16	23.9	17.3	28.6
% Smokers, Ever	41.5	61.1	27.1
% Smokers, Current	12.5	18.3	8.2
% Drinkers, Ever	45.7	74.4	24.6
% Drinkers, Current	15.9	29.1	6.2
CAGE score (%):			
No indicators	25.0	31.7	20.0
One indicator	8.1	1.9	16.6
Two indicators	4.2	9.1	0.6
Three indicators	2.4	5.2	0.4
Four indicators	2.3	5.0	0.3
% Current problem drinker (Current drinker with CAGE≥2)	40.2	48.7	11.0
BMI:			
Below 18.5 (Underweight)	1.9	1.9	1.9
18.5-24.99 (Normal)	28.6	30.1	27.5
25.0-29.99 (Overweight)	39.6	45.1	35.7
30.0 and Above (Obese)	29.9	22.9	34.9

At Time 2, the total sample was made up of 1682 subjects. The results at Time 2 are summarized in Table 2. The percentage of the sample that was female at Time 2 was 57.6% compared to 42.4% male, identical to Time 1. As shown in table 2, the mean age of the total sample at Time 2 was 79.12 years. The average age for men was 79.40 and

for women it was 78.91. This difference was not significant ($F(1, 1680) = 3.10, p > .05$). The percentage of U.S. born subjects at Time 2 was 57.6%, slightly higher than at Time 1. Sixty percent of the women at Time 2 were U.S. born while 54% of the men were born in the U.S. The chi-square for nativity by gender at Time 2 was not significant ($x^2(1) = 1.60, p > .05$).

Table 2
Sample characteristics at Time 2

Characteristic	Total Sample (N=1682)	Men (N=713)	Women (N=969)
Age, *M* years (*SD*)	79.12 (5.73)	79.40 (5.91)	78.91 (5.60)
% US born	57.6	54.4	59.6
% with Heart disease	22.8	23.8	22.1
% with Complications from CAD	--	--	--
% with Diabetes	26.1	26.3	26.0
% with DM Complications (of those with DM)	55.4	54.5	56.0
CES-D Total, mean score (*SD*)	7.12 (7.57)	6.03 (6.99)	7.79 (7.83)
% CES-D $\geq$16	12.9	9.7	14.8
% Smokers, Current	7.7	12.6	4.6
% Drinkers, Current	13.7	25.0	5.5
% Current Drinkers with CAGE score $\geq$2 (at time 1)	14.7	14.6	15.1
BMI:			
Below 18.5 (Underweight)	2.1	2.2	2.0
18.5-24.99 (Normal)	28.2	30.6	26.7
25.0-29.99 (Overweight)	37.7	42.2	34.7
30.0 and Above (Obese)	32.0	24.9	36.6

*CAD complications were not assessed at Time 2

DESCRIPTIVE SUMMARY OF PRIMARY STUDY VARIABLES

Physical Illness

Coronary Artery Disease. A history of CAD was reported by 9.3% of the sample at Time 1. Males (11.3%) had a slightly higher prevalence of CAD at Time 1 compared to females (7.9%). This difference in the prevalence of CAD between men and women was significant ($x^2(1) = 10.02, p < .01$).

At Time 2, 22.8% of the total sample reported having CAD. Men at this time had 23.8% prevalence rate of CAD while 22.1% of the women had CAD. At Time 2, the difference in the prevalence of CAD between genders was not significant ($x^2(1) = 1.14$, $p > .05$).

Coronary Artery Disease Complications. At Time 1, 62.9% of those with CAD had one or more CAD complications. More women than men with CAD said they had CAD complications (65.9% compared to 60.1%, respectively). However, this difference was not significant ($x^2(1) = .10, p > .05$). Of the total sample, there were 974 subjects who had one or more complications. The mean number of complications was 2.73 (*sd* =1.93). Men had an average of 2.55 complications (*sd* = 1.92) while women had 2.82 (*sd* = 1.93). This difference was significant ($F(1, 972) = 4.20, p < .05$). Therefore, women with CAD had significantly more complications than men with CAD.

The questions about CAD complications were not asked at the Time 2 interview, therefore, there are no results regarding Time 2 CAD complications.

Diabetic Illness Diagnosis. The prevalence of self-reported diabetes at Time 1 for the full sample was 22.6%. These results do not include affirmative answers to questions

regarding whether subjects had been diagnosed with "borderline" diabetes. The diabetes prevalence rate at Time 1 for males was 22.5% compared to 22.7% for women. There were no significant differences between the diabetes prevalence rates by gender ($x^2(1) = .01, p > .05$) at Time 1.

The percentage of subjects diagnosed with diabetes at Time 2 was 26.1. The percentages at Time 2 were very similar for both men and women (26.3 compared to 26.0, respectively) and were not significantly different ($x^2(1) = .15, p > .05$).

Diabetic Illness Complications. The prevalence of complications from diabetes among those with diabetes was 60.6% for the full sample at Time 1. The percentage of men with diabetes who reported one or more complications was 59.5% compared to 61.4% of women with diabetes. A chi-square was calculated to compare the difference between these rates for men and women and was not significant ($x^2(1) = .27, p > .05$). The average number of complications for the full sample was 1.78 ($sd = .88$). The average number of complications for men was 1.78 ($sd = .94$) while the mean for women was 1.77 ($sd = .84$). The difference in the average number of complications between the two genders was not significant ($F(1, 416) = .05, p = .83$).

At Time 2, 55.4% of those with diabetes had one or more complications. The percentage of women with one or more diabetes complications was slightly higher than the percentage for men (56.0 compared to 54.5, respectively). A chi-square calculated to examine this difference was not found to be significant ($x^2(1) = 3.74, p > .05$). The mean number of complications was 1.83 with no significant differences between men ($m = 1.85, sd = .94$) and women ($m = 1.82, sd = .93$) ($F(1, 250) = 3.77, p >. 05$).

Mortality

The mortality rate at Time 2 was 30.8%. The mortality rate for men was 36.2% and 26.8% for women. This gender difference was significant ($x^2(1) = 30.69, p < .01$).

Depressive Symptoms

The average score at Time 1 for the full sample on the CES-D was 9.94 (*sd* = 9.60). The average score for men was 8.10 (*sd* = 8.56) and 11.23 for women (*sd* = 10.06). A one-way ANOVA was calculated in order to examine the differences between men and women and was found to be significant ($F(1, 2821) = 74.91, p < .01$). Women had significantly higher average depressive symptom scores than men.

At Time 2, the mean CES-D score was 7.12 (sd = 7.57). Men had an average score of 6.03 (*sd*= 6.99) while the average score for women was 7.79 (*sd* = 7.83). A one-way ANOV calculated for these scores was significant ($F(1, 1560) = 20.13, p < .01$). Women also had more depressive symptoms at Time 2 than men.

Behavioral Health Risk Factors

Cigarette Smoking. At the first interview, 12.5% of the sample reported that they were current smokers. When broken down by gender, 18.3% of the men and 8.2% of the women were current smokers. Men were found to be significantly more likely than women to be current smokers at Time 1 ($x^2(1) = 70.22, p < .01$).

In response to the question about whether they had ever smoked in their lives, 41.4% of the total sample, 61.1% of men and 27.1% of women said that they had ever smoked. Men were significantly more likely to have ever smoked in their lifetimes than had women ($x^2(1) = 356.02, p < .01$).

At Time 2, the percentage of current smokers had dropped to 7.7%. The percentage of men who were smokers at this time was 12.6 while the percentage of women was 4.6. At Time 2, men were still significantly more likely to be smokers ($x^2(1) = 34.93$, $p < .01$).

Alcohol Use. Due to the relatively low use of alcohol in this sample, measures of quantity and frequency of beer, wine, and liquor were collapsed into one variable: current alcohol use. The percentage of current drinkers at Time 1 was 15.9. The percentage of men who were current drinkers was 29.1 while the percentage was only 6.2 for women. Men were significantly more likely to be current drinkers than women ($x^2(1) = 292.08$, $p < .01$).

The percentage of current problem drinkers (current drinkers who endorsed two or more items on the CAGE alcoholism screening scale) at Time 1 was 40.2%. The percentage of current problem drinkers was much higher for men than for women (48.7% compared to 11.0%). This difference between men and women was significant ($x^2(1) = 49.86$, $p < .01$).

At Time 2, the percentage of current drinkers had dropped to 13.7. The rate of current drinking among men was 25.0% compared to 5.5% for women. Men were significantly more likely than women to be drinkers at Time 2 ($x^2(1) = 168.47$, $p < .01$).

The percentage of current problem drinkers at Time 2 was 14.7. The rate was 14.6% for men and 15.1% for women. There were no significant differences in the current problem drinking prevalence rate between men and women ($x^2(1) = .01$, $p > .05$).

Body Mass Index. The percentage of those that were overweight or obese at Time 1 was 69.5. The percentage of men who were overweight or obese was 68.8 compared to 70.6 for women. Men were more likely than women to be overweight (45.1% for men vs. 35.7% for women), while women were more likely than men to fall into obese category (34.9% vs. 22.9%). These gender differences at Time 1 were significant ($x^2(3) = 49.32, p < .01$).

At Time 2, 69.7% of those for whom measurements were available ($n = 1293$) were overweight or obese. When broken down by gender, the percentage was 67.1% for men and 71.3% for women, not much different from Time 1. Men were still more likely to be in the overweight category when compared to women (42.2% vs. 34.7%, respectively), while women were more likely to be obese (36.6% for women vs. 24.9% for men). The gender differences at Time 2 were also significant ($x^2(3) = 19.52, p < .01$).

Exploratory Variables

Acculturation. Possible scores on the measure of acculturation ranged from 0 to 52. Higher scores were indicative of higher levels of acculturation. The average score on the acculturation scale was 15.97. For males, the average was 16.46 and for females it was 15.61. A one-way ANOVA found the difference between men and women to be significant ($F(1, 3018) = 15.02, p < .01$). Men were more acculturated than women.

Assimilation. Possible scores on the assimilation measure ranged for 0 to 9, with lower scores indicating less assimilation. The average score on this measure was 3.75. Men's scores were higher than women's with an average of 4.13 compared to 3.46 for

women. A one-way ANOVA found these to be significant differences ($F(1, 3039) = 92.39, p < .01$). Men also had significantly higher assimilation scores than women.

Self-rated health. The average score given by respondents for self-rated health was 2.36 (*sd* =.89) on a scale from 1 to 4. The average score for men was 2.44 (*sd* = .87), while for women it was 2.29 (*sd* = .91). This difference was found to be significant when an ANOVA was conducted for self-rated health by gender ($F(1, 2730) = 18.43, p < .01$). Men had significantly higher self-rated health than women.

Health locus of control. The mean score for the locus of control inventory was 4.23 (*sd* = 1.09) with possible scores ranging from 0 to 6. Higher scores indicated a more internal locus of control orientation. The mean score for men was 4.18 (*sd* = 1.12) and for women it was 4.26 (*sd* = 1.08). An ANOVA calculated to examine gender differences was not significant ($F(1, 2729) = 3.40, p > .05$). There were no differences in locus of control orientation between men and women.

OVERVIEW OF RELATIONSHIPS AMONG PRIMARY VARIABLES

Time 1

Analyses were run cross-sectionally at Time 1 in order to examine the relationships among the primary variables. Table 3 shows the correlations between Time 1 illness variables and Time 1 depressive symptoms. All the correlations were significant ($p<.01$).

Table 3: Cross-sectional relationships between depressive symptoms and illness at Time 1

	T1 CES-D
T1 CAD	.06**
T1 CAD complications	.23**
T1 DM	.09**
T1 DM complications	.15**

**p < .01

Time 2

Cross-sectional analyses at Time 2 examined the relationships between illness and depression. The results of these analyses are shown in Table 4. The correlations between CAD and depression and between diabetes complications and depressive symptoms were significant. The correlation between diabetes and depression at Time 2 was not significant.

Table 4: Cross-sectional relationships at Time 2

	T2 CES-D
T2 CAD	.06*
T2 CAD complications	--
T2 DM	.01
T2 DM complications	.12**

*p < .05, **p < .01, CAD complications not assessed at Time 2

Longitudinal relationships between Time 1 illness and Time 2 depressive symptoms

The relationships between Time 1 illness and Time 2 depression were also examined. The results of these analyses are shown in Table 5. None of these correlations were significant.

Table 5: Time 1 illness with Time 2 depressive symptoms

	T2 CES-D
T1 CAD	-.01
T1 CAD complications	.03
T1 DM	-.04
T1 DM complications	-.02

Longitudinal Relationships between Time 1 Depressive Symptoms and Time 2 Illness and Mortality

The relationships between Time 1 depressive symptoms and Time 2 outcomes were also examined. These results are shown in Table 6. The correlation between depressive symptoms at Time 1 and CAD at Time 2 was significant ($r(2821) = .076$, $p < .01$). The correlation between depressive symptoms at Time 1 and diabetes at Time 2 was significant ($r(2821) = .065$, $p < .01$) while the correlation between Time 1 depressive symptoms and Time 2 diabetes complications was not significant ($r(2821) = .007$, $p > .05$). The correlation between Time 1 depressive symptoms and mortality at Time 2 was found to be significant ($r(2821) = .119$, $p < .01$).

Table 6: Time 1 depressive symptoms and Time 2 illness/mortality

	T1 CES-D
T2 CAD	.08**
T2 CAD complications	--
T2 DM	.07**
T2 DM complications	.01
T2 Mortality	.12**

**p < .01

Longitudinal Relationships between Time 1 Behavioral Health Risk Factors and Time 2 Illness and Mortality

The correlations between Time 1 behavioral health risk factors and Time 2 physical illness and mortality are shown in Table 7. The correlation between alcohol use and diabetes at Time 2 was significant ($r(3050) = -.06, p < .01$). The correlation between alcohol use and mortality was also significant ($r(3050) = -.05, p < .01$). Body mass index at Time 1was significantly correlated with Time 2 diabetes ($r(3050) = .04, p < .05$) and with mortality at Time 2 ($r(2769) = -.10, p < .01$).

Table 7: Time 1 behavioral health risk factors and Time 2 illness/mortality

	T1 Smoking	T1 Alcohol Use	T1 BMI
T2 CAD	-.02	.01	-.03
T2 DM	-.02	-.06**	.04*
T2 DM complications	-.01	-.01	.01
T2 Mortality	.03	-.05**	-.10**

*p < .05, **p < .01

TESTS OF HYPOTHESES

Physical Illness as a Predictor of Depressive Symptoms

Hypotheses 1-5 examine physical illness at Time 1 as a predictor of depressive symptoms at Time 2.

Hypothesis 1

This hypothesis stated that CAD at Time 1 would predict depressive symptoms at Time 2. A multiple linear regression was calculated, predicting depressive symptoms at Time 2 from CAD at Time 1, controlling for age and gender. The regression coefficient

for CAD diagnosis was not significant (β = -.003, t = -.109, p > .05). Therefore, the presence of a CAD at Time 1 did not significantly predict depressive symptoms at Time 2.

Hypothesis 1 was also tested prospectively. A multiple linear regression was calculated predicting depressive symptoms at Time 2 from CAD at Time 1, controlling for age, gender, and Time 1 depressive symptoms. The regression coefficient for CAD was not significant (β = .007, t = .280, p > .05) when examined prospectively. After controlling for depressive symptoms at Time 1, CAD at Time 1 did not significantly predict depressive symptoms at Time 2.

Hypothesis 2

This hypothesis stated that CAD complications at Time 1 would add unique variance, beyond that predicted by a CAD alone, in predicting depressive symptoms at Time 2. A multiple linear regression was calculated predicting Time 2 depressive symptoms from CAD complications at Time 1, controlling for age, gender, and CAD. The regression coefficient for CAD complications was not significant (β = -.013, t = -.483, p > .05). Hence, CAD complications at Time 1 did not contribute unique variance beyond that provided by CAD alone, in predicting Time 2 depressive symptoms.

Hypothesis 2 was also examined prospectively. A multiple linear regression was calculated predicting Time 2 depressive symptoms from CAD complications at Time 1, controlling for age, gender, Time 1 CAD, and Time 1 depressive symptoms. The regression coefficient for CAD complications was not significant (β = -.007, t = -.261,

$p > .05$) when examined prospectively. Thus, after controlling for depressive symptoms at Time 1, CAD complications at Time 1 did not contribute unique variance beyond that provided by CAD alone, in predicting Time 2 depressive symptoms.

Hypothesis 3

This hypothesis stated that diabetic illness at Time 1would predict depressive symptoms at Time 2. A multiple linear regression was calculated predicting depressive symptoms at Time 2 from diabetes at Time 1, controlling for age and gender. The regression coefficient for diabetes was not significant ($\beta = -.046$, $t = -1.821$, $p > .05$). Therefore, a diagnosis of diabetes at Time 1 was not a significant predictor of depressive symptoms at Time 2.

Hypothesis 3 was also tested prospectively. A multiple linear regression was calculated predicting depressive symptoms at Time 2 from diabetes at Time 1, controlling for age, gender, and Time 1 depressive symptoms. The regression coefficient for diabetes was significant ($\beta = -.052$, $t = -1.978$, $p < .05$) when examined prospectively. After controlling for depressive symptoms at Time 1, diabetes at Time 1 was slightly significantly predictive of depressive symptoms at Time 2, however, the relationship was opposite to that which was predicted.

Hypothesis 4

This hypothesis stated that the presence of complications due to diabetes at Time 1 would add unique variance, beyond that predicted by diabetes alone, in predicting depressive symptoms at Time 2. A multiple linear regression was calculated predicting Time 2 depressive symptoms from diabetes complications at Time 1, controlling for age,

gender, and Time 1 diabetes. The regression coefficient for diabetes complications was not significant ($\beta = .046, t = 1.215, p > .05$). Therefore, the presence of diabetes complications at Time 1 did not contribute unique variance, beyond that provided by diabetes at Time 1, in predicting depressive symptoms at Time 2.

Hypothesis 4 was also tested prospectively. A multiple linear regression was calculated predicting Time 2 depressive symptoms from diabetes complications at Time 1, controlling for age, gender, Time 1diabetes, and Time 1 depressive symptoms. The regression coefficient for diabetes complications was not significant ($\beta = .058, t = 1.497, p > .05$) when examined prospectively. After controlling for depressive symptoms at Time 1, the presence of diabetes complications at Time 1 did not contribute unique variance, beyond that provided by diabetes at Time 1, in predicting depressive symptoms at Time 2.

Hypothesis 5

This hypothesis stated that the co-morbidity of CAD and diabetes at Time 1 would add unique variance, beyond that predicted by either disease alone, in predicting depressive symptoms at Time 2. A multiple linear regression was calculated predicting Time 2 depressive symptoms from CAD and diabetes at Time 1, and their interaction (computed as the multiplicative product term for CAD and diabetes), controlling for age and gender. The regression coefficient for the multiplicative product term for CAD and diabetes was not significant ($\beta = -.028, t = -.834, p > .05$). Hence, the co-occurrence of CAD and diabetes at Time 1 did not significantly predict depressive symptoms at Time 2.

Hypothesis 5 was also tested prospectively. A multiple linear regression was

calculated predicting Time 2 depressive symptoms from CAD and diabetes at Time 1, and their interaction (computed as the multiplicative product term for Time 1 CAD and diabetes), controlling for age, gender, and Time 1 depressive symptoms. The regression coefficient for the multiplicative product term for CAD and diabetes was not significant (β = -.022, t = -.614, p > .05) when examined prospectively. After controlling for Time 1 depressive symptoms, the co-occurrence of CAD and diabetes at Time 1 did not significantly predict depressive symptoms at Time 2.

Depressive Symptomatology as a Predictor of Physical Illness and Mortality

Hypotheses 6 to 8 examine depressive symptomatology as a predictor of physical illness and mortality.

Hypothesis 6

This hypothesis stated that depressive symptoms at Time 1 would be positively related to CAD at Time 2. A multiple logistic regression analysis was calculated predicting CAD at Time 2 from depressive symptoms at Time 1, controlling for age and gender. Depressive symptoms at Time 1 significantly predicted CAD at Time 2 (*OR* = 1.20, p < .01, 95% *CI* = 1.10, 1.31).

Hypothesis 6 was also tested prospectively. A multiple logistic regression analysis was calculated predicting CAD at Time 2 from depressive symptoms at Time 1, controlling for age, gender, and Time 1 CAD. Depressive symptoms significantly predicted CAD at Time 2 (*OR* = 1.16, p < .01, 95% *CI* = 1.05, 1.29) when examined prospectively.

Complications for CAD were not assessed at Time 2, therefore analyses predicting CAD complications at Time 2 from depressive symptoms at Time 1 were not calculated.

Hypothesis 7

This hypothesis stated that depressive symptoms at Time 1 would be positively related to both diabetes and diabetes complications at Time 2. First, a multiple logistic regression analysis was calculated predicting Time 2 diabetes from depressive symptoms at Time 1, controlling for age and gender. Depressive symptoms at Time 1 significantly predicted diabetes at Time 2 ($OR = 1.14, p < .01, 95\%\ CI = 1.06, 1.23$).

Then, a multiple logistic regression analysis was calculated predicting Time 2 diabetes complications from Time 1 depressive symptoms, controlling for age and gender. Depressive symptoms at Time 1 significantly predicted diabetes complications at Time 2 ($OR = 1.04, p < .01, 95\%\ CI = 1.10, 1.31$).

In order to test hypothesis 7 prospectively, a multiple logistic regression analysis was first calculated predicting Time 2 diabetes from depressive symptoms at Time 1, controlling for age, gender, and Time 1 diabetes. When examined prospectively, depressive symptoms at Time 1 no longer significantly predicted diabetes at Time 2 ($OR = 1.06, p > .05, 95\%\ CI = .97, 1.16$).

Then, a multiple logistic regression analysis was calculated predicting Time 2 diabetes complications from Time 1 depressive symptoms, controlling for age, gender, and Time 1 diabetes complications. When examined prospectively, depressive symptoms

at Time 1 no longer significantly predicted diabetes complications at Time 2 (*OR* = 1.04, $p > .05$, 95% *CI* = .91, 1.19).

Hypothesis 8

This hypothesis stated that depressive symptoms at Time 1 would be positively related to mortality at Time 2. A multiple logistic regression analysis was calculated predicting mortality from Time 1 depressive symptoms, controlling for age and gender. Depressive symptoms at Time 1 significantly predicted mortality at Time 2 (*OR* = 1.36, $p < .01$, 95% *CI* = 1.25, 1.49).

The Relationship between Depressive Symptoms and Behavioral Health Risk Factors

Hypotheses 9-11 examined the relationship between depressive symptoms and behavioral risk factors at Time 1. The results of the correlational analyses are shown in Table 8.

Table 8: Time 1 behavioral factors with time 1 depression

	T1 CES-D
T1 current smoker	.008
T1 current alcohol use	-.083**
T1 BMI	.033

$p < .01$

Hypothesis 9

This hypothesis stated that depressive symptoms at Time 1 would be positively associated with cigarette smoking at Time 1. The correlation between Time 1 depressive symptoms and **current** smokers at Time 1 is shown in Table 7 and was not significant

($r(2821) = .008, p > .05$). The correlation between depressive symptoms and **ever** smokers at Time 1 was also analyzed. The results of this correlation were also not significant ($r(2821) = -.030, p > .05$). Depressive symptoms at Time 1 were not significantly related to either current or ever smoking at Time 1.

Hypothesis 10

This hypothesis stated that depressive symptoms at Time 1 would be positively associated with alcohol use at Time 1. As seen in Table 8, the correlation between Time 1 depressive symptoms and Time 1 alcohol was significant ($r(2821) = -.083$ $p < .01$), but in the direction opposite than expected. Therefore, a second analysis was calculated for Time 1 depressive symptoms and Time 1 problem drinking (current drinkers with two or more indicators on the CAGE inventory). The results of this correlation were not significant ($r(2821) = -.020, p > .05$) but in the same direction as the initial correlation. Hence, there was a significant negative relationship between depressive symptoms and current alcohol use at Time 1.

Hypothesis 11

This hypothesis stated that depressive symptoms at Time 1 would be positively associated with body mass index at Time 1. As shown in Table 8, this correlation was not significant ($r(2821) = .033, p > .05$). There was not a significant relationship between Time 1 depressive symptoms and Time 1 BMI.

Behavioral Health Risk Factors as Mediators between Depressive Symptomatology and Outcomes

Hypotheses 12-14 proposed to examine behavioral risk factors as mediators between depressive symptomatology at Time 1 and physical illness and mortality measured at Time 2. These three mediational hypotheses could not be tested because the underlying relationships were not consistent with the hypotheses.

Hypothesis 12

This hypothesis integrated hypothesis 6 with hypotheses 9 through 11. It was hypothesized that the relationship between depressive symptoms at Time 1 and CAD at Time 2 would be partially mediated by behavioral health risk factors at Time 1.

Hypothesis 13

This hypothesis integrated hypothesis 7 with hypotheses 9 through 11. It was hypothesized that the relationship between depressive symptoms at Time 1 and diabetes at Time 2 would be partially mediated by behavioral health risk factors at Time 1.

Hypothesis 14

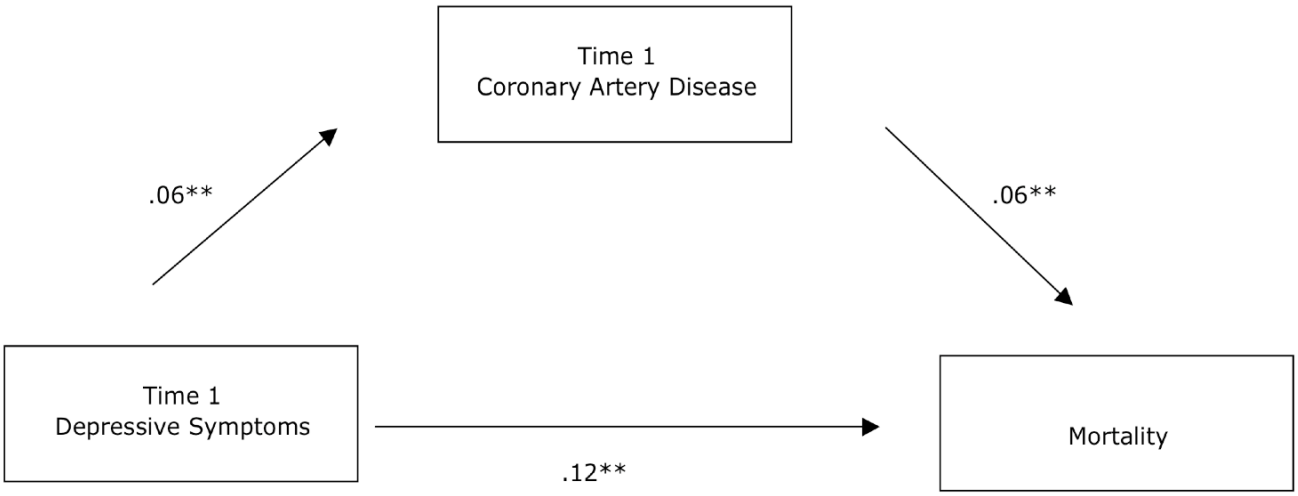
This hypothesis integrated hypothesis 8 with hypotheses 9 through 11. It was hypothesized that the relationship between depressive symptoms at Time 1 and mortality at Time 2 would partially mediated by behavioral health risk factors at Time 1.

Relationships Among the Variables

Two revised mediational models, which are consistent with the pattern of empirical findings, were tested. Both models used a multiple regression approach to test the mediational path models.

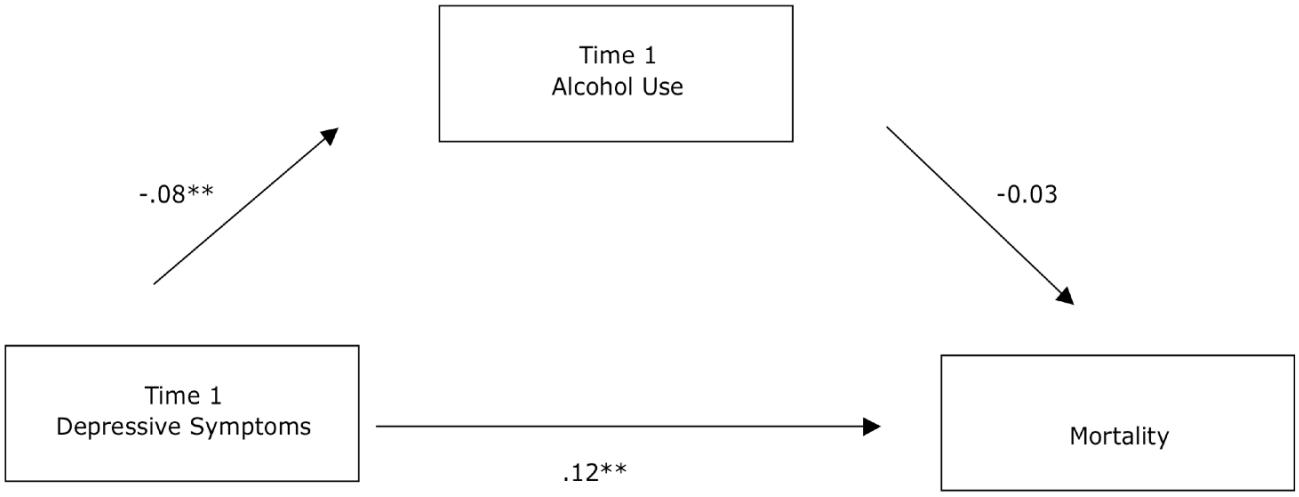
One mediational model (Figure 4) examined the role of CAD at Time 1 as a partial mediator of the relationship between depressive symptoms at Time 1 and subsequent mortality. The results of this path analysis are consistent with a mediational model whereby Time 1 depressive symptoms predict mortality in part through their relationship with CAD.

A second mediational model (Figure 5) examined the role of current alcohol use in partially mediating the relationship between depressive symptoms at Time 1 and subsequent mortality. The results of this path analysis are not consistent with a mediational model. Time 1 alcohol use does not relate to mortality after controlling for Time 1 depressive symptoms.



$^{**}p < .01$

Figure 4: Time 1 Depressive Symptoms, Time 1 CAD, and Mortality



$^{**}p < .01$

Figure 5: Time 1 Depressive Symptoms, Time 1 Alcohol Use, and Mortality

EXPLORATORY ANALYSES

Exploratory Question 1: Gender as a Moderator

The role of gender as a moderator of key relationships examined in hypotheses 1 through 5 was explored. These hypotheses pertained to physical illness as a predictor of depressive symptoms.

The moderating role of gender on the relationship between Time 1 CAD and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD and gender, the gender by Time 1 CAD interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 CAD ($\beta = .026, t = .318, p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of gender on the relationship between Time 1 CAD complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD complications and gender, the gender by Time 1 CAD complications interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 CAD complications ($\beta = -.006, t = -.064, p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of gender on the relationship between Time 1 diabetes and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes and gender, the gender by Time 1 diabetes interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 diabetes ($\beta = -.040, t = -.473, p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of gender on the relationship between Time 1 diabetes complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes complications and gender, the gender by Time 1 diabetes complications interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 diabetes complications (β = -.069, t = -.823, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of gender on the interaction between Time 1 CAD and diabetes in predicting Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD, Time 1 diabetes, and gender, the interaction between gender and the multiplicative product term for Time 1 CAD and diabetes, and age as a covariate. There was not a significant effect for the interaction between gender and the multiplicative product term for Time 1 CAD and diabetes (β = -.024, t = -.713, p > .05) in predicting Time 2 depressive symptoms.

The role of gender as a moderator of key relationships examined in hypotheses 6 through 8 was also explored. These hypotheses pertained to depressive symptomatology as a predictor of physical illness and mortality.

The moderating role of gender on the relationship between Time 1 depressive symptoms and Time 2 CAD was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and gender, the gender by Time 1 depressive symptomatology interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 depressive symptoms (OR = .961, p > .05, 95% CI = .803, 1.151) in predicting Time 2 CAD.

The moderating role of gender on the relationship between Time 1 depressive symptoms and Time 2 diabetes was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and gender, the gender by

Time 1 depressive symptomatology interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 depressive symptoms ($OR = .901, p > .05, 95\%\ CI = .765, 1.06$) in predicting Time 2 diabetes.

The moderating role of gender on the relationship between Time 1 depressive symptoms and Time 2 diabetes complications was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and gender, the gender by Time 1 depressive symptomatology interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 depressive symptoms ($OR = 1.260, p > .05, 95\%\ CI = .937, 1.695$) in predicting Time 2 diabetes complications.

The moderating role of gender on the relationship between Time 1 depressive symptoms and mortality up to Time 2 was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and gender, the gender by Time 1 depressive symptomatology interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 depressive symptoms ($OR = 1.022, p > .05, 95\%\ CI = .856, 1.220$) in predicting mortality up to Time 2.

The role of gender as a moderator of key relationships examined in hypotheses 9 through 11 was also explored. These hypotheses pertained to the relationship between depressive symptoms and behavioral risk factors at Time 1.

The moderating role of gender on the relationship between Time 1 depressive symptoms and Time 1 cigarette smoking was examined in a multiple regression analysis that included the main effects for Time 1 cigarette smoking and gender, the gender by Time 1 cigarette smoking interaction, and age as a covariate. There was not a significant

effect for the interaction between gender and Time 1 cigarette smoking ($\beta = .080, t = 1.419, p > .05$).

The moderating role of gender on the relationship between Time 1 depressive symptoms and Time 1 alcohol use was examined in a multiple regression analysis that included the main effects for Time 1 alcohol use and gender, the gender by Time 1 alcohol use interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 alcohol use ($\beta = .089, t = 1.558, p > .05$).

The moderating role of gender on the relationship between Time 1 depressive symptoms and Time 1 BMI was examined in a multiple regression analysis that included the main effects for Time 1 BMI and gender, the gender by Time 1 BMI interaction, and age as a covariate. There was not a significant effect for the interaction between gender and Time 1 BMI ($\beta = .252, t = 1.853, p > .05$).

Exploratory Question 2: Acculturation as a Moderator

The role of acculturation as of moderator of key relationships in hypotheses 1 through 5 was explored. These hypotheses pertained to physical illness as a predictor of depressive symptoms.

The moderating role of acculturation on the relationship between Time 1 CAD and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD and acculturation, the acculturation by Time 1 CAD interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 CAD ($\beta = .020, t = .738, p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of acculturation on the relationship between Time 1 CAD complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD complications and acculturation,

the acculturation by Time 1 CAD complications interaction, and age and gender as covariates. The interaction between acculturation and Time 1 CAD complications approached significance (β = -.063, t = -1.952, p = .051) in predicting Time 2 depressive symptoms.

The moderating role of acculturation on the relationship between Time 1 diabetes and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes and acculturation, the acculturation by Time 1 diabetes interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 diabetes (β = -.020, t = -.716, $p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of acculturation on the relationship between Time 1 diabetes complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes complications and acculturation, the acculturation by Time 1 diabetes complications interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 diabetes complications (β = -.006, t = -.236, $p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of acculturation on the interaction between Time 1 CAD and diabetes in predicting Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD, Time 1 diabetes, and acculturation, the interaction between acculturation and the multiplicative product term for Time 1 CAD and diabetes, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and the multiplicative product term for CAD and diabetes (β = -.028, t = -1.092, $p > .05$) in predicting Time 2 depressive symptoms.

The role of acculturation as a moderator of key relationships in hypotheses 6 through 8 was also explored. These hypotheses pertained to depressive symptomatology as a predictor of physical illness and mortality.

The moderating role of acculturation on the relationship between Time 1 depressive symptoms and Time 2 CAD was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and acculturation, the acculturation by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 depressive symptoms ($OR = .999$, $p > .05$, 95% $CI = .990, 1.009$) in predicting Time 2 CAD.

The moderating role of acculturation on the relationship between Time 1 depressive symptoms and Time 2 diabetes was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and acculturation, the acculturation by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 depressive symptoms ($OR = 1.009$, $p > .05$, 95% $CI = 1.000, 1.018$) in predicting Time 2 diabetes.

The moderating role of acculturation on the relationship between Time 1 depressive symptoms and Time 2 diabetes complications was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and acculturation, the acculturation by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 depressive symptoms ($OR = .999$, $p > .05$, 95% $CI = .983, 1.015$) in predicting Time 2 diabetes complications.

The moderating role of acculturation on the relationship between Time 1 depressive symptoms and mortality up to Time 2 was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and acculturation, the acculturation by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 depressive symptoms ($OR = 1.001, p > .05, 95\%\ CI = .991, 1.011$) in predicting mortality up to Time 2.

The role of acculturation as a moderator of key relationships in hypotheses 9 through 11 was also explored. These hypotheses pertained to the relationship between depressive symptoms and behavioral risk factors at Time 1.

The moderating role of acculturation on the relationship between Time 1 depressive symptoms and Time 1 cigarette smoking was examined in a multiple regression analysis that included the main effects for Time 1 cigarette smoking and acculturation, the acculturation by Time 1 cigarette smoking interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 cigarette smoking ($\beta = .006, t = .317, p > .05$).

The moderating role of acculturation on the relationship between Time 1 depressive symptoms and Time 1 alcohol use was examined in a multiple regression analysis that included the main effects for Time 1 alcohol use and acculturation, the acculturation by Time 1 alcohol use interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 alcohol use ($\beta = -.017, t = -.794, p > .05$).

The moderating role of acculturation on the relationship between Time 1 depressive symptoms and Time 1 BMI was examined in a multiple regression analysis that included the main effects for Time 1 BMI and acculturation, the acculturation by

Time 1 BMI interaction, and age and gender as covariates. There was not a significant effect for the interaction between acculturation and Time 1 BMI (β = -.011, t = -.109, p > .05).

Exploratory Question 3: Assimilation as a Moderator

The role of assimilation as of moderator of key relationships in hypotheses 1 through 5 was explored. These hypotheses pertained to physical illness as a predictor of depressive symptoms.

The moderating role of assimilation on the relationship between Time 1 CAD and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD and assimilation, the assimilation by Time 1 CAD interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and Time 1 CAD (β = .026, t = .952, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of assimilation on the relationship between Time 1 CAD complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD complications and assimilation, the assimilation by Time 1 CAD complications interaction, and age and gender as covariates. There was no significant effect for the interaction between assimilation and Time 1 CAD complications (β = -.028, t = -.846, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of assimilation on the relationship between Time 1 diabetes and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes and assimilation, the assimilation by Time 1 diabetes interaction, and age and gender as covariates. There was not a significant

effect for the interaction between assimilation and Time 1 diabetes ($\beta = .014, t = .490, p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of assimilation on the relationship between Time 1 diabetes complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes complications and assimilation, the assimilation by Time 1 diabetes complications interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and Time 1 diabetes complications ($\beta = .024, t = .892, p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of assimilation on the interaction between Time 1 CAD and diabetes in predicting Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD, Time 1 diabetes, and assimilation, the interaction between assimilation and the multiplicative product term for Time 1 CAD and diabetes, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and the multiplicative product term for CAD and diabetes ($\beta = .001, t = .035, p > .05$) in predicting Time 2 depressive symptoms.

The role of assimilation as a moderator of key relationships in hypotheses 6 through 8 was also explored. These hypotheses pertained to depressive symptomatology as a predictor of physical illness and mortality.

The moderating role of assimilation on the relationship between Time 1 depressive symptoms and Time 2 CAD was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and assimilation, the assimilation by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and

Time 1 depressive symptoms (*OR* = 1.026, $p > .05$, 95% *CI* = .968, 1.087) in predicting Time 2 CAD.

The moderating role of assimilation on the relationship between Time 1 depressive symptoms and Time 2 diabetes was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and assimilation, the assimilation by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and Time 1 depressive symptoms (*OR* = 1.038, $p > .05$, 95% *CI* = .983, 1.097) in predicting Time 2 diabetes.

The moderating role of assimilation on the relationship between Time 1 depressive symptoms and Time 2 diabetes complications was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and assimilation, the assimilation by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and Time 1 depressive symptoms (*OR* = .976, $p > .05$, 95% *CI* = .893, 1.068) in predicting Time 2 diabetes complications.

The moderating role of assimilation on the relationship between Time 1 depressive symptoms and mortality up to Time 2 was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and assimilation, the assimilation by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and Time 1 depressive symptoms (*OR* = .962, $p > .05$, 95% *CI* = .907, 1.021) in predicting mortality up to Time 2.

The role of assimilation as a moderator of key relationships in hypotheses 9 through 11 was also explored. These hypotheses pertained to the relationship between depressive symptoms and behavioral risk factors at Time 1.

The moderating role of assimilation on the relationship between Time 1 depressive symptoms and Time 1 cigarette smoking was examined in a multiple regression analysis that included the main effects for Time 1 cigarette smoking and assimilation, the assimilation by Time 1 cigarette smoking interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and Time 1 cigarette smoking ($\beta = .001, t = -.071, p > .05$).

The moderating role of assimilation on the relationship between Time 1 depressive symptoms and Time 1 alcohol use was examined in a multiple regression analysis that included the main effects for Time 1 alcohol use and assimilation, the assimilation by Time 1 alcohol use interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and Time 1 alcohol use ($\beta = -.025, t = -1.171, p > .05$).

The moderating role of assimilation on the relationship between Time 1 depressive symptoms and Time 1 BMI was examined in a multiple regression analysis that included the main effects for Time 1 BMI and assimilation, the assimilation by Time 1 BMI interaction, and age and gender as covariates. There was not a significant effect for the interaction between assimilation and Time 1 BMI ($\beta = .123, t = 1.201, p > .05$).

Exploratory Question 4: Nativity as a Moderator

The role of nativity as of moderator of key relationships in hypotheses 1 through 5 was explored. These hypotheses pertained to physical illness as a predictor of depressive symptoms.

The moderating role of nativity on the relationship between Time 1 CAD and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD and nativity, the nativity by Time 1 CAD interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 CAD (β = -.047, t = -1.135, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of nativity on the relationship between Time 1 CAD complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD complications and nativity, the nativity by Time 1 CAD complications interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 CAD complications (β = -.048, t = -1.115, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of nativity on the relationship between Time 1 diabetes and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes and nativity, the nativity by Time 1 diabetes interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 diabetes (β = -.047, t = -1.104, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of nativity on the relationship between Time 1 diabetes complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes complications and nativity, the nativity by Time 1 diabetes complications interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 diabetes complications (β = .001, t = .021, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of nativity on the interaction between Time 1 CAD and diabetes in predicting Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD, Time 1 diabetes, and nativity, the interaction between nativity and the multiplicative product term for Time 1 CAD and diabetes, and age and gender as covariates. There was not a significant effect for the interaction between nativity and the multiplicative product term for CAD and diabetes ($\beta = -.036, t = -1.178, p > .05$) in predicting Time 2 depressive symptoms.

The role of nativity as a moderator of key relationships in hypotheses 6 through 8 was also explored. These hypotheses pertained to depressive symptomatology as a predictor of physical illness and mortality.

The moderating role of nativity on the relationship between Time 1 depressive symptoms and Time 2 CAD was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and nativity, the nativity by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 depressive symptoms ($OR = 1.054, p > .05, 95\%\ CI = .888, 1.253$) in predicting Time 2 CAD.

The moderating role of nativity on the relationship between Time 1 depressive symptoms and Time 2 diabetes was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and nativity, the nativity by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was a significant effect for the interaction between nativity and Time 1 depressive symptoms ($OR = 1.199, p < .05, 95\%\ CI = 1.030, 1.396$) in predicting Time 2 diabetes, with the effect stronger for those born in the U.S.

The moderating role of nativity on the relationship between Time 1 depressive symptoms and Time 2 diabetes complications was examined in a multiple logistic

regression analysis that included the main effects for Time 1 depressive symptoms and nativity, the nativity by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 depressive symptoms ($OR = 1.156, p > .05, 95\%\ CI = .881, 1.516$) in predicting Time 2 diabetes complications.

The moderating role of nativity on the relationship between Time 1 depressive symptoms and mortality up to Time 2 was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and nativity, the nativity by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was a significant effect for the interaction between nativity and Time 1 depressive symptoms ($OR = 1.156, p < .05, 95\%\ CI = 1.027, 1.441$) in predicting mortality up to Time 2, with the effect stronger for those born in the U.S.

The role of nativity as a moderator of key relationships in hypotheses 9 through 11was also explored. These hypotheses pertained to the relationship between depressive symptoms and behavioral risk factors at Time 1.

The moderating role of nativity on the relationship between Time 1 depressive symptoms and Time 1 cigarette smoking was examined in a multiple regression analysis that included the main effects for Time 1 cigarette smoking and nativity, the nativity by Time 1 cigarette smoking interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 cigarette smoking ($\beta = .011, t = .360, p > .05$).

The moderating role of nativity on the relationship between Time 1 depressive symptoms and Time 1 alcohol use was examined in a multiple regression analysis that included the main effects for Time 1 alcohol use and nativity, the nativity by Time 1

alcohol use interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 alcohol use (β = -.010, t = -.319, $p > .05$).

The moderating role of nativity on the relationship between Time 1 depressive symptoms and Time 1 BMI was examined in a multiple regression analysis that included the main effects for Time 1 BMI and nativity, the nativity by Time 1 BMI interaction, and age and gender as covariates. There was not a significant effect for the interaction between nativity and Time 1 BMI (β = -.081, t = -.766, $p > .05$).

Exploratory Question 5: Locus of Control as a Moderator

The role of locus of control as of moderator of key relationships in hypotheses 1 through 5 was explored. These hypotheses pertained to physical illness as a predictor of depressive symptoms.

The moderating role of locus of control on the relationship between Time 1 CAD and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD and locus of control, the locus of control by Time 1 CAD interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 CAD (β = -.026, t = -.947, $p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of locus of control on the relationship between Time 1 CAD complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD complications and locus of control, the locus of control by Time 1 CAD complications interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 CAD complications (β = .014, t = .376, $p > .05$) in predicting Time 2 depressive symptoms.

The moderating role of locus of control on the relationship between Time 1 diabetes and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes and locus of control, the locus of control by Time 1 diabetes interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 diabetes (β = -.036, t = -1.182, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of locus of control on the relationship between Time 1 diabetes complications and Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 diabetes complications and locus of control, the locus of control by Time 1 diabetes complications interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 diabetes complications (β = -.029, t = -1.023, p > .05) in predicting Time 2 depressive symptoms.

The moderating role of locus of control on the interaction between Time 1 CAD and diabetes in predicting Time 2 depressive symptoms was examined in a multiple regression analysis that included the main effects for Time 1 CAD, Time 1 diabetes, and locus of control, the interaction between locus of control and the multiplicative product term for Time 1 CAD and diabetes, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and the multiplicative product term for CAD and diabetes (β = -.024, t = -.870, p > .05) in predicting Time 2 depressive symptoms.

The role of locus of control as a moderator of key relationships in hypotheses 6 through 8 was also explored. These hypotheses pertained to depressive symptomatology as a predictor of physical illness and mortality.

The moderating role of locus of control on the relationship between Time 1 depressive symptoms and Time 2 CAD was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and locus of control, the locus of control by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 depressive symptoms ($OR = 1.043, p > .05, 95\%\ CI = .929, 1.172$) in predicting Time 2 CAD.

The moderating role of locus of control on the relationship between Time 1 depressive symptoms and Time 2 diabetes was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and locus of control, the locus of control by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 depressive symptoms ($OR = .951, p > .05, 95\%\ CI = .858, 1.054$) in predicting Time 2 diabetes.

The moderating role of locus of control on the relationship between Time 1 depressive symptoms and Time 2 diabetes complications was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and locus of control, the locus of control by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 depressive symptoms ($OR = .877, p > .05, 95\%\ CI = .744, 1.033$) in predicting Time 2 diabetes complications.

The moderating role of locus of control on the relationship between Time 1 depressive symptoms and mortality up to Time 2 was examined in a multiple logistic regression analysis that included the main effects for Time 1 depressive symptoms and

locus of control, the locus of control by Time 1 depressive symptomatology interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 depressive symptoms ($OR = .964, p > .05, 95\%\ CI = .861, 1.079$) in predicting mortality up to Time 2.

The role of locus of control as a moderator of key relationships in hypotheses 9 through 11was also explored. These hypotheses pertained to the relationship between depressive symptoms and behavioral risk factors at Time 1.

The moderating role of locus of control on the relationship between Time 1 depressive symptoms and Time 1 cigarette smoking was examined in a multiple regression analysis that included the main effects for Time 1 cigarette smoking and locus of control, the locus of control by Time 1 cigarette smoking interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 cigarette smoking ($\beta = .011, t = .570, p > .05$).

The moderating role of locus of control on the relationship between Time 1 depressive symptoms and Time 1 alcohol use was examined in a multiple regression analysis that included the main effects for Time 1 alcohol use and locus of control, the locus of control by Time 1 alcohol use interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 alcohol use ($\beta = -.016, t = -.771, p > .05$).

The moderating role of locus of control on the relationship between Time 1 depressive symptoms and Time 1 BMI was examined in a multiple regression analysis that included the main effects for Time 1 BMI and locus of control, the locus of control by Time 1 BMI interaction, and age and gender as covariates. There was not a significant effect for the interaction between locus of control and Time 1 BMI ($\beta = .031, t = .312, p > .05$).

Exploratory Question 6: Threshold Depression as an Alternative to Continuous Depressive Symptoms

The study will explore a dichotomous measure of threshold depression (high versus low) in contrast to a continuous measure of depressive symptoms in examining the relationships in hypotheses 1 through 5. The first five hypotheses pertained to physical illness as a predictor of high depression.

The relationship between Time 1 CAD and high depression at Time 2 was examined. A multiple logistic regression analysis was calculated predicting high depression at Time 2 from CAD at Time 1, controlling for age and gender. CAD at Time 1 did not predict high depression at Time 2 ($OR = .905$, $p > .05$, 95% $CI = .532, 1.539$).

The relationship between Time 1 CAD complications and high depression at Time 2 was examined. A multiple logistic regression analysis was calculated predicting high depression at Time 2 from CAD complications at Time 1, controlling for age, gender, and Time 1 CAD. CAD complications at Time 1 did not add unique variance beyond that provided by Time 1 CAD in predicting high depression at Time 2 ($OR = .871$ $p > .05$, 95% $CI = .635, 1.194$).

The relationship between Time 1 diabetes and high depression at Time 2 was examined. A multiple logistic regression analysis was calculated predicting high depression at Time 2 from diabetes at Time 1, controlling for age and gender. Diabetes at Time 1 did not predict high depression at Time 2 ($OR = .721$, $p > .05$, 95% $CI = .490$, 1.062).

The relationship between Time 1 diabetes complications and high depression at Time 2 was examined. A multiple logistic regression analysis was calculated predicting high depression at Time 2 from diabetes complications at Time 1, controlling for age, gender, and Time 1 diabetes. Diabetes complications at Time 1 did not add unique

variance beyond that provided by Time 1 diabetes in predicting high depression at Time 2 (*OR* = 1.981 $p > .05$, 95% *CI* = .898, 4.374).

The relationship between the co-morbidity of Time 1 CAD and diabetes in predicting high depression at Time 2 was examined. A multiple logistic regression analysis was calculated predicting high depression at Time 2 from CAD and diabetes at Time 1, and their interaction (computed as the multiplicative product term for CAD and diabetes), controlling for age and gender. The interaction between CAD and diabetes at Time 1 did not predict high depression at Time 2 (*OR* = .559, $p > .05$, 95% *CI* = .161, 1.941).

The study also explored a dichotomous measure of threshold depression (high versus low) in contrast to a continuous measure of depressive symptoms in predicting the relationships in hypotheses 6 through 8. These hypotheses pertained to high depression as a predictor of physical illness and mortality.

The relationship between high depression at Time 1 and CAD at Time 2 was examined. A multiple logistic regression analysis was calculated predicting Time 2 CAD from high depression at Time 1, controlling for age and gender. High depression at Time 1 did not predict CAD at Time 2 (*OR* = .834, $p > .05$, 95% *CI* = .679, 1.025).

The relationship between high depression at Time 1 and diabetes at Time 2 was examined. A multiple logistic regression analysis was calculated predicting Time 2 diabetes from high depression at Time 1, controlling for age and gender. High depression at Time 1 did not predict diabetes at Time 2 (*OR* = .842, $p > .05$, 95% *CI* = .706, 1.004).

The relationship between high depression at Time 1 and diabetes complications at Time 2 was examined. A multiple logistic regression analysis was calculated predicting Time 2 diabetes complications from high depression at Time 1, controlling for age and

gender. High depression at Time 1 did not predict diabetes complications at Time 2 (*OR* = 1.122, $p > .05$, 95% *CI* = .810, 1.555).

The relationship between high depression Time 1 and mortality up to Time 2 was examined. A multiple logistic regression analysis was calculated predicting mortality from Time 1 high depression, controlling for age and gender. High depression at Time 1 significantly predicted mortality up to Time 2 (*OR* = .585, $p < .01$, 95% *CI* = .479, .714).

The study also explored a dichotomous measure of threshold depression (high versus low) in contrast to a continuous measure of depressive symptoms in predicting the relationships in hypotheses 9 through 11. These hypotheses pertained to the relationships between high depression at Time 1 and behavioral risk factors at Time 1.

The correlation between high depression at Time 1 and current smoking at Time 1 was not significant ($r(2823) = -.001$, $p > .05$). The correlation between high depression at Time 1 and alcohol use at Time 1 was significant ($r(2823) = -.057$, $p < .01$). And, the correlation between high depression at Time 1 and BMI at Time 1 was not significant ($r(2823) = .021$, $p > .05$).

Exploratory Question 7: Self-rated Health as an Alternative to Physical Illness Indicators in Predicting Depressive Symptomatology

The study explored self-rated health in contrast to physical illness indicators at Time 1 in predicting depressive symptomatology at Time 2. The relationship between self-rated health at Time 1 and depressive symptoms at Time 2 was examined in a multiple regression analysis, controlling for age and gender. The regression coefficient for self-rated health approached significance ($\beta = .049$ $t = 1.803$, $p = .072$).

Exploratory Question 8: Self-rated Health as a Predictor of Mortality

The relationship between self-rated health at Time 1 and mortality up to Time 2 was examined. A multiple logistic regression analysis was calculated predicting

mortality from self-rated health at Time 1, controlling for age and gender. Self-rated health at Time 1 significantly predicted mortality up to Time 2 ($OR = .642, p < .01, 95\%$ $CI = .577, .713$). To examine if these results would hold up after controlling for physical illness, a multiple logistic regression analysis was calculated, controlling for age, gender, CAD, and diabetes. The results of these additional analyses found that self-rated health still significantly predicted mortality independent after still controlling for both diseases.

CHAPTER 5: DISCUSSION

The purpose of the current study was to examine the relationship between physical illness and depressive symptoms in elderly Mexican Americans at baseline and seven years later. In addition, the relationship between depressive symptoms and behavioral health risk factors was examined at baseline. The two physical illnesses studied were CAD and type 2 diabetes mellitus. The behavioral health risk factors studied were alcohol use, cigarette smoking, and BMI as a proxy for physical inactivity. The longitudinal nature of the data allowed for the investigation of the relationship between physical illness and depressive symptoms in both directions.

This chapter is divided into six sections. The first section contains an overview of the findings of the study. After the overview, an elaboration of what relates the findings related to the literature is provided. Broader implications of the findings are then discussed, followed by the practical implications of the study. These are followed by a discussion of the limitations of the study and directions for future research. Concluding remarks are presented in the final section.

SUMMARY OF FINDINGS

Major Findings Related to Subject Characteristics

The percentage of individuals in the current study with CES-D scores indicative of probable depression (a score greater than or equal to 16) was 23.9%. This was significantly greater than that found in other community samples of the elderly. Using

the same cutpoint as in the current study, the prevalence rate of depressive caseness for participants in a comparable study was 16% among White participants and 14.4% among Black participants (Kim, Chiriboga, & Yang, 2009).

The rates of physical illness for the H-EPESE participants in this study were considerably different than those found in comparable community based studies of the elderly. The rates of CAD in the present study were 11.3% for men and 7.9% for women. Rates of CAD reported by subjects in the Cardiovascular Health Study were much higher: 14.7% and 26.2% for Caucasian men and women, respectively (Arnold et al., 2005). Conversely, rates of diabetes in the present study were much higher than those in the Cardiovascular Health Study. Prevalence rates of diabetes in the present study were 22.5% for men and 22.7% for women compared to 12.3% for Caucasian men and 17.9% for Caucasian women in the Cardiovascular Health Study (Arnold et al., 2005).

The smoking rates in the current study were much lower, 18.3% for men and 8.2% for women, than for those in another EPESE sample, which included only White and Black participants. Current smoking rates in that study were 22% for men and 15% for women (Salive & Blazer, 1993). Likewise, the current drinking rate was much lower in this study, 15.9%, than the 53.7% prevalence rate in the Cardiovascular Health Study sample (Arnold et al., 2005). With respect to BMI, the subjects in this study were less likely to be overweight but were more likely to be obese compared to the subjects in the Cardiovascular Health Study. In this study, 39.6% of participants fell into the overweight category and 29.9% in the obese category. The percentage of participants in the

Cardiovascular Health Study who were overweight was 44.3% and the percentage in the obese category was 18.9% (Janssen, 2007).

Major Findings Associated with Relationships among Primary Variables

The results of this study showed significant correlations between physical illness and depressive symptoms at baseline. Both CAD and diabetes at Time 1 were significantly related to depressive symptoms at Time 1. In addition, complications associated with each disease at Time 1 were also significantly related to the presence of depressive symptoms at Time 1.

When the relationships were examined at Time 2, seven years later, only the diagnosis of CAD at Time 2 and the presence of diabetes complications at Time 2 were significantly related to depressive symptoms at Time 2. Diabetes at Time 2 was not significantly correlated with depressive symptoms at Time 2. The relationship between CAD complications at Time 2 and depressive symptoms was not included because CAD complications were not assessed at Time 2.

When the relationships between the variables were examined longitudinally, neither CAD nor diabetes at Time 1 was related to depressive symptomatology at Time 2. The complications of each disease at Time 1 were also not related to depressive symptoms at Time 2. However, when the longitudinal relationships between Time 1 depressive symptoms and Time 2 physical outcomes were examined, Time 1 depressive symptoms were significantly related to CAD, diabetes, and mortality at Time 2.

There were only a few significant findings when the relationships between behavioral health risk factors at Time 1 and physical outcomes at Time 2 were examined.

Alcohol use at Time 1 was inversely related to diabetes at Time 2. Alcohol use at Time 1 also had an inverse association with mortality. Additionally, there was a positive association between body mass index at Time 1 and subsequent diabetes. However, body mass index at baseline was found to have an inverse relationship with mortality.

Major Findings Related to Hypotheses

In examining physical illness at Time 1 as a predictor of depressive symptoms at Time 2, none of the current findings supported the hypotheses. Contrary to expectation, neither CAD nor diabetes predicted depressive symptoms seven years later. When examined prospectively, the relationship between Time 1 diabetes and Time 2 depressive symptoms was significant. However, this relationship was in the opposite direction than predicted.

Also, contrary to expectation, the hypothesis that the addition of complications associated with each illness would contribute unique variance beyond that provided by each illness alone in predicting depressive symptoms was not supported for either disease. Another unexpected finding was that the comorbidity of CAD and diabetes did not add unique variance beyond that provided by each disease alone in predicting depressive symptoms at Time 2.

When depressive symptomatology as a predictor of physical illness and mortality was examined, the findings supported hypotheses 6 through 8. This second set of hypotheses examined the relationships between depressive symptoms at Time 1 and physical illness and mortality measured at Time 2. As expected, depressive symptoms at baseline significantly predicted the presence of CAD seven years later. In addition,

depressive symptoms at Time 1 predicted the presence of diabetes and diabetes complications at Time 2. Also, as hypothesized, baseline depressive symptoms predicted mortality assessed at Time 2.

When the relationship between depressive symptoms and behavioral health risk factors was examined, none of the findings supported hypotheses 9 through 11. These hypotheses examined the relationship between Time 1 depressive symptoms and behavioral health risk factors. Contrary to expectation, there was no significant relationship between depressive symptoms and cigarette smoking. There was a significant relationship between depressive symptoms and alcohol use; however, it was in the opposite direction than expected. Also, contrary to expectation, there was not a significant relationship between depressive symptoms and BMI.

In examining the relationships among the variables, hypotheses 12 through 14 proposed to examine behavioral risk factors as mediators between depressive symptomatology at Time 1 and physical illness and mortality at Time 2. However, the proposed mediational models could not be examined because the underlying relationships between depressive symptoms and the outcome behavioral health risk factors studied were not supported by the findings. Instead, two revised models were tested.

The results of the revised model that explored Time 1 CAD as a mediator of the relationship between Time 1 depressive symptoms and mortality seven years later, were consistent with the proposed mediation. The results of the second revised model, which examined Time 1 alcohol use as a mediator of the relationship between Time 1

depressive symptoms and mortality assessed at Time 2, were not consistent with the proposed mediation.

Major Findings Related to Exploratory Questions

The first exploratory question concerned the role of gender as a moderator of the key relationships set forth in the hypotheses. There were no significant findings concerning the role of gender as a moderator of any of the relationships studied. Another exploratory question involved acculturation as a moderator of the key relationships examined in the hypotheses. The results for acculturation as a moderator of the relationship predicting Time 2 depressive symptoms from Time 1 CAD complications approached significance. The relationship between Time 1 CAD and Time 2 depressive symptoms was more robust for individuals with higher acculturation scores. Assimilation was also examined as a moderator, however, there were no significant findings on assimilation as a moderator of any of the relationships studied. Another moderator that was tested in an exploratory question was nativity. Nativity was identified as a significant moderator of the predictive relationship between Time 1 depressive symptoms and Time 2 diabetes and also for the predictive relationship between Time 1 depressive symptoms and mortality. The associations between Time 1 depressive symptoms and Time 2 diabetes and mortality were stronger for those individuals born in the United States than for those born in Mexico. Locus of control was the final moderator studied in the exploratory questions. Locus of control was not found to be a significant moderator of any of the relationships examined.

An additional exploratory question that was examined concerned the effect of a dichotomous measure of threshold depressive symptomatology as an alternative to a continuous measure of depressive symptoms on the relationships examined in the hypotheses. As with the continuous measure, high depression at Time 1 significantly predicted mortality. In addition, high depression at Time 1, like the continuous measure, had a significant negative relationship with Time 1 alcohol use.

The final exploratory questions involved self-rated health at Time 1. First, self-rated health at Time 1 was examined as an alternative to physical illness indicators in predicting depressive symptoms at Time 2. Self-rated health did not significantly predict Time 2 depressive symptoms. Self-rated health at Time 1 was also examined as an alternative to physical illness as a predictor of mortality. Self-rated health was a significant predictor of mortality.

RELATIONSHIP OF FINDINGS TO THE LITERATURE

Although previous research has found a relationship between CAD and subsequent depression (Hance et al., 1996; Schleifer et al., 1989), this relationship was not demonstrated in the present study. And, though there is evidence that an increased number of CAD complications are associated with a higher likelihood of depressive symptoms (Whooley et al., 2008), this expectation was also not supported in the present study. Previous studies have found a relationship between diabetes and later depression, (Peyrot & Rubin, 1997; Kawakami et al., 1999), however, this relationship was not demonstrated in the present study. In addition, although more diabetes complications have been found to be associated with a higher likelihood of major depression (Katon et

al., 2005), the hypothesis that the addition of diabetes complications would add unique variance in predicting depressive symptoms at Time 2 was also not supported in the present findings. Although previous research has found that the presence of co-morbid medical conditions is related to an increased number of depressive symptoms (Weyerer et al., 2008), this expectation was not supported by the current findings.

The lack of an association between baseline physical illness and later depressive symptoms may be due to several factors. Those who were most ill at baseline may have also been the same individuals with the highest number of depressive symptoms. The mean number of baseline depressive symptoms was significantly higher for those who had died by follow-up compared to those who did not ($t(2821) = -6.39$, $p < .01$). This rationale is also supported by the fact that the mean CES-D scores dropped considerably from baseline to Time 2 (from 9.94 to 7.12). Previous research has established that those with comorbid physical illness and depressive symptoms are at highest risk of mortality (Black, Markides, & Ray, 2003; Egede, Nietart, & Zheng, 2005). Alternatively, other researchers have also found that increased age in the elderly is associated with lower levels of depressive symptoms (Blazer et al, 1991).

Another explanation for the lack of an association between physical illness at Time 1 and depressive symptoms at Time 2 may be that the time between the baseline assessment and the follow-up seven years later is too long to capture emotional reactions to illness diagnoses. In fact, recent research suggests that even dramatic life events do not produce enduring emotional change because individuals may drift back to an established emotional "set point" (Wilson & Gilbert, 2008). The studies reviewed were

primarily cross-sectional in nature (Katon et al., 2005) or had shorter follow-up times (Hance et al., 1996; Frasure-Smith et al., 1993) than the time period in the present study, and may therefore have been more able to capture emotional reactions to chronic disease diagnosis.

With respect to the lack of variance added by the illness complications and co-morbidity of the two illnesses, it may be that factors such as functional limitation and cognitive impairment are more important than medical co-morbidity (Weyerer, 2008). For example, Blazer et al. (1991) found that physical disability and cognitive impairment were among the variables associated with increased depressive symptomatology in the elderly. Other researchers have established that acute and chronic economic stressors such as low-income and financial strain are significantly associated with high levels of depressive symptomatology in elderly Mexican Americans (Chiriboga et al., 2002).

Consistent with the current finding that depressive symptoms at Time 1 predicted CAD at Time 2, previous longitudinal research has shown that the presence of depressive symptoms predicted an increased incidence of subsequent CAD (Ariyo et al., 2000; Agatisa et al., 2005). One explanation for this finding may be that depression impacts the progression of underlying atherosclerotic disease, which progresses to CAD (Ford et al., 1998). Additionally, Barefoot and Schroll (1996) suggest that heightened reactivity of the sympathetic nervous system in those who are depressed may be responsible for the association between baseline depression and later CAD.

The present findings also showed that depressive symptoms at Time 1 predicted diabetes at Time 2, was also supported by the findings of the study. Some mechanisms

that are suggested as explanations for this relationship are that depression may be a risk factor for noncompliance with medical treatment recommendations (Gross et al., 2005). Another explanation is that depressed individuals may have increased serotonin concentrations and catecholamine levels which adversely affect glucose regulation and contribute to the development of diabetes (Gross et al., 2005).

In addition, the current study found that depressive symptoms measured at baseline were associated with subsequent diabetes complications, although not significantly so after controlling for Time 1 diabetes complications. This finding is consistent with that of Lin et al. (2010) who studied adults with type 2 diabetes over a five-year period and found that those who were depressed were more likely to develop diabetes complications. Lin et al. (2010) propose that biological processes such as increased cortisol secretion or inflammatory responses in those with depression may play a role in the progression of complications in those with type 2 diabetes.

The current finding that depressive symptoms at Time 1 predicted mortality is consistent with previous research by Schulz et al. (2000) who found depressive symptomatology as measured by the CES-D to be an independent risk factor for mortality in a large sample of elderly individuals. Again, it may be that people who are depressed are less likely to comply with medical treatment recommendations. In addition, depressed individuals have also been found to suffer from impaired functional status, which interferes with the ability to carry out daily activities related to treatment regimens (St. John & Montgomery, 2009). An alternative explanation for the association between Time 1 depression and mortality is that psychological distress causes adverse biological

changes in these individuals that are neurohormonal or inflammatory in nature and contribute to disease progression and ultimately to death (Egede et al., 2005; Lin et al., 2010).

Although previous research has found a relationship between depression and cigarette smoking in adults (Glassman et al., 1990; Escobedo, Kirch, & Anda, 1996), there was no significant relationship between depressive symptoms and current smoking in this sample. One reason for this finding may be that the present study assessed only whether subjects were "current smokers" or "not current smokers." Therefore, those who may have been previous smokers and quit would have fallen in the "not current smokers" category. This may have obscured the relationship between depression and current smoking. However, other studies have failed to find a relationship between smoking and depressive symptoms in community samples (Frerichs, Aneshensel, Clark, & Yokopenic, 1981). Moreover, other researchers have found depression to be related to smoking only through its relationship with other types of substance abuse (Black, Zimmerman, & Coryell, 1999; Kenney, Holahan, North, & Holahan, 2006).

Although a positive relationship between depression and alcohol abuse has been well established (Kessler et al., 1997; Golding et al., 1992), a negative relationship between alcohol use and depressive symptoms was found in this study. A possible explanation for this finding is that current alcohol use was assessed only as "current drinker" or "not a current drinker" for these analyses. Previous research has found that a relationship exists between depression and alcohol abuse, not alcohol use (Kessler et al., 1997; Golding et al., 1992). In fact, the level of alcohol use in the current sample was

low. To further elucidate the association between depressive symptoms and alcohol use in the study sample, the relationship between problem drinkers and depressive symptoms was examined. However, this relationship was also not found to be significant. Again, the number of problem drinkers in the study sample also was quite low.

In the current study, there was no significant relationship found between BMI and depressive symptoms at Time 1 even though other researchers have observed a positive correlation between these two variables (Galper et al., 2006; Goodman & Whitaker, 2002). The negative finding in this study is consistent with that of Janssen (2007) who found elderly individuals with a BMI in the overweight range to have a lower mortality risk than those who were not overweight. Additionally, Weiss et al. (2007), in a study on very elderly patients, found those in the lowest BMI quartile to have the highest rate of mortality. This finding may reflect the fact that, although an elevated BMI is associated with increased mortality at younger ages, it may instead serve as a protective factor in older ages. A higher BMI could protect older individuals from poor health outcomes and functional limitations associated with depression (Al Snih et al., 2007).

Two models were proposed to examine possible mediational pathways between the variables. In the first meditational model, in addition to relating directly to mortality, depressive symptoms at Time 1 were found to relate to mortality indirectly, through their relationship with CAD. These results are consistent with prior research that found depressive symptoms to be an independent risk factor after six years for the development of coronary heart disease and total mortality in an elderly sample free of any cardiovascular disease at baseline (Ariyo et al., 2000). The mechanisms proposed by the

authors as a rationale for why depressive symptoms predispose individuals to an increased risk of heart disease are behavioral, such as increased behavioral health risks, and biological, such as increased levels of circulating platelets caused by increases in autonomic sympathetic activation (Ariyo et al., 2000).

In the second meditational model, although alcohol use was not found to be a mediator of the relationship between depressive symptoms and mortality, it is possible that other behavioral health risk factors could mediate this relationship. For example, it has been suggested that non-compliance with prescribed medical regimens may be one behavioral factor that mediates the link between depressive symptoms and mortality (Katon et al., 2005; Kuo et al., 2003).

In addition to mediators examined, there were several variables in the exploratory analyses that were studied as potential moderators of the key relationships proposed in the study hypotheses. The results with respect to acculturation as a moderator of the relationship between CAD complications at baseline and subsequent depressive symptoms approached significance, with the relationship more robust for individuals with higher acculturation scores. One reason for this finding may be due to the fact that those with higher acculturation scores have less familial support (Ramirez, 1983). Another variable examined as a potential moderator was nativity, or country of birth. Nativity was found to be a significant moderator of the predictive relationship between Time 1 depressive symptoms and diabetes at Time 2. Specifically, the relationship between depressive symptoms at Time 1 and diabetes at Time 2 was stronger for Mexican Americans born in the United States than for those born in Mexico. Nativity was also

found to be a significant moderator of the predictive relationship between Time 1 depressive symptoms and later mortality. That is, the relationship between Time 1 depression and mortality was more pronounced for Mexican Americans born in the United States than for those born in Mexico. This is broadly consistent with research that finds that advantages in Hispanic health and mortality are due to social and lifestyle behaviors from the native country (Palloni & Arias, 2004). These advantages seem to decrease with length of stay in the U.S. and in subsequent generations. For example, the Hispanic mortality advantage has been attributed in part to Mexico-born vs. U.S.-born Mexican Americans (Palloni & Arias, 2004). The other moderators examined, gender and locus of control, were not found to be significant moderators of any of the relationships examined in this study.

An additional exploratory question explored self-rated health as an alternative to physical illness indicators in predicting depressive symptoms. Self-rated health at Time 1 did not predict depressive symptoms at Time 2. Self-rated health was also examined as a predictor of mortality in the exploratory analyses. The results of the study showed that self-rated health at Time 1 significantly predicted mortality. These results remained significant even after controlling for CAD and diabetes at Time 1. The results on self-rated health as a significant predictor of mortality are consistent with other studies of self-rated health in the elderly. For example, Idler and Kasl (1991) found that individuals who perceived their health as "poor" were six times as likely to die than those who rated their health as "excellent." Likewise, Jylhä et al. (2006) found that those who reported fair or poor self-rated health were more likely to die than those who reported excellent

health. In their study, these researchers examined the association between self-rated health and several biological measurements, including albumin concentration, hemoglobin, and white blood cell counts. Their results showed that these poorer indexes on these "biomarkers" were significantly related to self-rated health, suggesting that lower self-rated health has a biological basis (Jylhä et al., 2006).

BROADER IMPLICATIONS OF FINDINGS

The results of this study are consistent with the view that the presence of depressive symptomatology is an independent risk factor for the development of physical illness (Ariyo et al., 2000; Kawakami et al., 1999). Previous research has found that depressive symptoms are associated with an increased risk of developing both CAD and type 2 diabetes in those free of disease at baseline (Ford et al., 1998; Kawakami et al., 1999). In this study, prospective analyses showed that those with elevated depressive symptoms at baseline were more likely to have CAD seven years later. However, the relationship between high depressive symptoms at baseline and later diabetes was not supported when examined prospectively.

The findings of the present study are also consistent with research that suggests that depression is a risk factor for increased mortality (Blazer et al., 2001; St. John & Montgomery, 2009). Depressive symptoms in this sample of elderly Mexican Americans were associated with an increased risk of mortality. The mechanisms that have been proposed as predisposing depressed individuals to an increased risk of disease and mortality are believed to be both behavioral and biological in nature. One behavioral factor that has been proposed as a mediator of the relationship between depression and

physical outcomes is functional decline. Depression may cause functional impairment, which leads to changes in lifestyle and activity levels. These lifestyle changes could then lead to an increased risk of mortality (St. John & Montgomery, 2009). Biological mechanisms have also been implicated as mediators of this relationship. For example, Kawakami et al. (1999) studied the development of diabetes in those with depressive symptoms. The authors suggest that those with chronic depressive symptoms have an impaired ability to break down carbohydrates due to an endocrine disturbance or a release of counterregulatory hormones. Further, Ariyo et al. (2000) believe that depressive symptoms are related to increased levels of platelet and fibrinogen production, both of which have been implicated in the development of CAD.

With respect to behavioral health risk factors, previous research has found a relationship between depressive symptoms and increased alcohol use (Crum, Brown, Liang, & Eaton, 2001), increased cigarette smoking (Salive & Blazer, 1993), and elevated BMI (Goodman & Whitaker, 2002). However, the results of the present study were not consistent with previous research that established associations between depressive symptoms and these behavioral health risk factors. There was no relationship found between depressive symptoms and cigarette smoking or depressive symptoms and BMI in the current study. And although there was a relationship between depressive symptoms and alcohol use in this study, it was an inverse association: those who reported fewer depressive symptoms reported more alcohol use. This finding is similar to that of Bots, Tijhuis, Giampaoli, Kromhout, and Nissinen (2007) who found that moderate alcohol consumption served as a protective factor against symptoms of

depression in men aged 70-89. These researchers suggest that moderate alcohol intake may be part of a healthy lifestyle or that it may act as a buffer for stressful life events. Further, Holahan et al. (2010) found that those individuals who abstained from alcohol had significantly higher depressive symptomatology than moderate drinkers.

The results of the mediational model with CAD as a mediator of the relationship between depressive symptoms at Time 1 and mortality have important theoretical implications. The mediational model indicated that the pathway from depressive symptoms to mortality is partially explained through CAD. The present findings are consistent with and extend previous research that found depressive symptomatology to be an independent risk factor in the development of coronary heart disease and also of total mortality in the elderly (Ariyo et al., 2000).

The current finding also highlights the importance of nativity in the relationship between depressive symptoms and diabetes. Although Mexican Americans born in Mexico have been shown to be more likely to have high depressive symptomatology (Gerst, Al-Ghatrif, Beard, Samper-Ternent, & Markides, 2010), the interaction between depressive symptoms and diabetes was more pronounced for U.S. born Mexican Americans in the present study. Likewise, the present findings call attention to the role of nativity in the relationship between depressive symptoms and mortality. The results of the present study showed the association between depression and mortality to be stronger for Mexican Americans born in the United States than for those born in Mexico. Therefore, it would be advantageous to promote the protective health and lifestyle factors associated with Mexican culture. This could be accomplished by having social scientists

promote a diverse, multicultural view of psychology, rather than one based on cultural superiority (Ramirez, 1983).

The exploratory analyses on self-rated health extend previous research conducted with a predominantly White sample on the relationship between self-rated health and mortality measured after four years (Idler & Kasl, 1991). In the present study, self-rated health was a strong predictor of mortality seven years later, even after adjusting for the presence of CAD and diabetes. Self-rated health has been found to be a consistent predictor of mortality and it has been suggested that it can even be utilized in the place of objective health status as an indicator of future longevity (Idler & Kasl, 1991). Mackenbach, Simon, Looman, and Joung (2002) describe self-rated health as "a very inclusive measure of health reflecting health aspects relevant to survival which are not covered by other health indicators" (p. 1162). Jylhä (2009) suggests that the strength of the association between self-rated health and mortality may reflect the assessment of biological or physiological components by the individual, such as underlying inflammatory processes related to infections, fatigue, and undiagnosed illnesses.

In addition to highlighting the increased mental and physical health disadvantages present among elderly Mexican Americans, the findings of the present study also call attention to the resilience of this sample of elderly individuals. Although the study participants were very poor (close to 50% percent had household incomes of less than $10,000 in 1993) and suffered from chronic financial strains (almost 50% reported having either "some" or "great" difficulty meeting monthly payments on their bills) (Chiriboga, 2003), this sample of study participants is a sample of survivors. As

mentioned earlier, there is some evidence that the Hispanic mortality advantage is due in fact to lower mortality among Mexico-born Mexican Americans (Palloni & Arias, 2004). It has been proposed that part of the mortality advantage found among individuals born in Mexico is due to a robustness among those who survived childhoods in a developing country compared to those who grew up in America (Wong, Gerst, & Michaels-Obregon, 2010). Close to 50% of participants in the current study were born in Mexico, therefore it would be useful to further study the factors associated with survival differences in those born in Mexico compared to those born in the U.S.

PRACTICAL IMPLICATIONS OF FINDINGS

The findings of the current study have several practical implications. One implication is that there should be more efforts to improve awareness in the general public of depression and its role in disease progression. Dumesnil and Verger (2009) studied several public awareness campaigns on depression and concluded that there are several characteristics associated with successful programs. They recommend that campaigns be organized at a local level and targeted at homogenous populations. In addition, these researchers suggest that rather than target mental illness as a whole, targeting one or two mental health problems would be more feasible. Those with CAD or diabetes, especially, should be made aware of the increased risks associated with co-morbid depression.

Primary care physicians, particularly those who treat elderly Mexican Americans, would also benefit from interventions related to depression awareness. Although depression has been established as a common problem, especially among the elderly,

most physicians under-recognize the existence of depressive symptoms in their elderly patients (Lamberg, 1996). There exists a need to increase awareness among primary care providers of the potential for increased depressive symptoms in their elderly patients and of the link between depressive symptoms and illness progression and reduced longevity.

The finding regarding the strength of the relationship between self-rated health and mortality in this elderly Mexican American sample may also be of relevance to primary care physicians. Some researchers have argued that an individual's self-rating of their own health represents a unique measure of health status and that a patient's health status should not be addressed without it (Idler & Benyamini, 1997). A change for the worse in these ratings could prove a useful screening tool to identify those older adults at a higher risk of negative health outcomes (Sargent-Cox, Anstey, & Luszcz, 2010).

Even those primary care physicians who treat younger Mexican Americans should be made aware of the need for depression screening in this population. Disparities in depression care among Latinos have been found predominantly among Mexican American individuals (Gonzalez et al., 2010). In addition, because Mexican Americans are genetically predisposed to type 2 diabetes and obesity, special efforts should be made to screen for depressive symptoms in this group. The development of culturally appropriate depression screening tools could facilitate the identification of depression in Mexican Americans.

LIMITATIONS OF THE PRESENT STUDY

There are several limitations associated with the present study. One of the limitations is that most of the key variables were gathered via self-report. The physical

illness diagnoses and complications were self-reported and not verified against medical records. It is possible that this led to an underestimation of the diseases that were studied. It is also possible that there may have been some individuals in the study who were unaware they had the physical illnesses they were asked about. For example, it is estimated that 6.3 million people in the U.S. had undiagnosed diabetes in 2007 (Zhang et al., 2009). In addition, the diagnosis of CAD was made based on the response to only one question: "Have you had a heart attack?" Therefore, it is possible that there is an underestimation of the prevalence of CAD in this sample because CAD is commonly present in those who have not had a heart attack. An underestimation of these illnesses would weaken any prospective relationship between depressive symptoms and physical illness.

A second limitation of the study has to do with the fact that the CES-D is a measure of depressive symptomatology and not a clinical diagnosis of depression. Research has suggested that scales such as the CES-D may overestimate the prevalence of depression because they reflect underlying physical disorders and somatic symptoms (Turk & Okifuji, 1994). This may be particularly true for Mexican Americans who are more likely to somatize their psychological distress as somatic symptoms (Angel & Angel, 1995; Beltran, 2005). For example, Kim et al. (2009) reported that Mexican Americans appeared to report somatic symptoms differently than did Whites or Blacks. Specifically, Mexican Americans were more likely to endorse four of the seven items in the CES-D associated with somatic symptoms. Another issue with respect to the CES-D is that there may be a tendency towards a response bias for Mexican Americans. In one

study, elderly Mexican Americans were more likely to endorse all of sixteen symptoms on the CES-D, even the positive affect item, than similarly aged Whites and Blacks (Kim et al., 2009).

It has also been argued that the CES-D is a measure of emotional distress rather than depressive symptoms. Fisher et al. (2007) utilized the Composite International Diagnostic Interview (CIDI) and the CES-D to study depression in patients with diabetes. They found that more than 70% of those who received a diagnosis of likely depression on the CES-D did not receive a diagnosis of major depressive disorder utilizing the CIDI. They suggest that the CES-D may more generally and heterogeneously capture emotional distress than specifically depressive affect. For all these reasons, the prevalence of depressive symptomatology may be overestimated in this study sample.

A third limitation of the current study is that the study questions were not planned a priori because this study is a secondary examination of longitudinal data. For example, if the variable about the presence of CAD had been planned prior to this study, it would have included more questions than just the one about the history of a heart attack. Likewise, physical activity at baseline would have been assessed. This would have avoided the need for BMI as a proxy for physical activity. Physical activity may have been a more appropriate variable to include, as a lack of exercise has been associated with depression in those with diabetes (Lin et al., 2004). Alternatively, caloric intake may have been a better variable to include than physical inactivity, as increased caloric intake has been associated with increased depressive symptoms (Grossniklaus et al., 2010).

A fourth limitation associated with the study findings concerns the sample. The sample is made up of elderly individuals, so the results may not be generalizable to younger Mexican Americans. This may be especially true regarding the lack of an association between depressive symptoms and behavioral health risk factors. Relationships between these behavioral health risk factors have been established in previous research examining younger individuals. For example, an association between current smoking and depression was found among Latino adults (Escobedo et al., 1996) and among Latino adolescents (Pesa et al., 2001).

A final limitation is that although several relationships were statistically significant, the absolute size of these associations was not large. These significant relationships should be interpreted in the context of the relatively high power provided by sample sizes of between two- and three-thousand for these analyses (Freedman, 2010). Nevertheless, even relatively small effect sizes, when they pertain to serious physical illnesses and mortality cannot be ignored.

DIRECTIONS FOR FUTURE RESEARCH

Including objective health measures such as Hemoglobin A1c for the diagnosis of diabetes and electrocardiograms to detect the presence of CAD would strengthen future research. In addition, the physical health histories of those who participate could be validated by reviewing their medical charts. These methods would provide more objective physical illness diagnoses.

Another way to improve the current study would be to utilize a structured clinical interview instead of a symptoms checklist to diagnose the presence of depression. If a

scale such as the CES-D needs to be utilized to measure depressive symptoms, then culturally appropriate adjustments, such as changing the cutoff scores used to diagnose depression, could be made (Kim et al., 2009).

Another way of extending the current research would be to examine the study relationships in a different age group. The relationships between depressive symptoms and behavioral health risk factors might be more relevant in a younger population because the prevalence of at risk behaviors is higher at younger ages. In addition, middle age is a time when many illnesses begin to manifest themselves.

CONCLUSION

The present study examined the directionality of the relationship between physical illness and depressive symptoms in elderly Mexican Americans, a group that has an increased vulnerability to emotional distress and to certain chronic diseases including type 2 diabetes mellitus. The findings from the current study demonstrate that the relationship from depressive symptoms to physical illness across seven years is more robust than that from physical illness to depression across seven years. As noted earlier, the relatively long 7-year time-frame examined in the present study is more appropriate for detecting the more slowly evolving relationship between emotional distress and illness progression than for examining more immediate emotional reactions to illness.

Another finding worth noting highlights the importance of country of birth when studying the health of elderly Mexican Americans. The relationship between depressive symptomatology at Time 1 and diabetes at Time 2 was stronger for U.S. born participants than for those who immigrated from Mexico. Likewise, the relationship between

depressive symptoms and mortality was also more robust for those born in the U.S. than for those born in Mexico. These findings demonstrate the salience of country of birth when studying the health of elderly Mexican Americans.

The current study underscores the plight of a population in need of increased medical and mental health services. Mexican American elderly are becoming a significant proportion of the elderly population. In addition, the health status of this population is worsening as estimates of diabetes among elderly Mexican Americans grew to 37% in recent years (Beard, Al Ghatrif, Samper-Ternent, Gerst, & Markides, 2009). The importance of preventive efforts directed toward this vulnerable population is essential given their lack of economic resources. This study contributes to the literature in that it identifies a specific, viable target for preventive efforts by drawing attention to the role of depressive symptomatology as a precursor to chronic physical illness and higher mortality in this population.

The associations between the key variables in the present study also have important implications for health service providers. Health care workers should be alerted to the increased risk of depressive symptoms in this population. They should also be made aware of the importance of screening for depression so that those who need it receive proper treatment. In addition, the relevance and utility of measures of self-rated health should be promoted as a means for primary care physicians and other health care workers to more fully assess the well-being of their elderly patients.

Appendix A: Selected H-EPESE Questionnaire Items

Sociodemographic Characteristics

LOUIS HARRIS AND ASSOCIATES, INC.
630 Fifth Avenue
New York, New York 10111

/ FOR OFFICE USE ONLY:
/
/ Questionnaire No.:_____
/
/_____1-2-3-4-5

Study No. 922027 (Mexican-American Elderly)
(8-13)
July 13, 1993

CARD NUMBER (6-7)

Time Started:_____A.M./P.M.

Interviewer:_____ Date:_____

Area Code:_____ Telephone No.:_____
(14-16) (17-23)

Hello, I'm _____ from Louis Harris and Associates, the national survey research firm in New York. We are conducting a study about health in your community and we'd like to speak to an adult in your household.

To begin, I would like to ask some very general questions about your household.

S1. How many people in this household are in each of the following age categories: (READ LIST)

Under 18.....(24(/___/
18 to 24.....(25(/___/
25 to 49.....(26(/___/
50 to 64.....(27(/___/
65 or older.....(28(/___/

INTERVIEWER:

* IF NO ONE IN HOUSEHOLD 65 OR OLDER THANK AND TERMINATE — SCREEN OUT Q.S1.
* IF ONE PERSON 65 OR OLDER ASK TO SPEAK TO THAT PERSON.
* IF MORE THAN ONE PERSON 65 OLDER MAY SCREEN ALL FOR FURTHER ELIGIBILITY REQUIREMENTS.

S2.P Are you of Mexican, Mexican-American, Chicano or Hispanic origin or descent, or not?

Yes, Mexican, Mexican American, Chicano..(29(____-1
Yes, Hispano.....____-2 } (GO TO Q.S3)

Yes, Puerto Rican.....____-3
Yes, Cuban.....____-4
Yes, other Spanish/Hispanic.....____-5 } (SCREEN OUT Q.S2)
Not, not Spanish/Hispanic.....____-6

We are particularly interested in speaking to older Mexican American about their health and health care experiences. (As you know) we are conducting this study for the University of Texas.)

S3.P Respondent name:
_____(30-80)

S4.P Respondent's Birth date: /__/__/ - /__/__/ - /__/__/
2*(08-13)

P Age: /__/__/ Years
(14-15)

FROM OBSERVATION: P Respondent Gender

Male.....(16(____-1
Female.....____-2

INTERVIEWER: WHEN CONDUCTING CP PROXY INTERVIEW, QUESTIONS MARKED "P" SHOULD BE ASKED OF PROXY; "P-R" QUESTIONS, WHEN POSSIBLE, SHOULD BE ASKED OF RESPONDENT. DURING A PROXY INTERVIEW, ALL QUESTIONS REFER TO THE RESPONDENT.

Questionnaire No. / / / / / / (17-21)

A. ETHNIC ALGORITHM

I would like to ask you some questions about your background and your parents' backgrounds.

A1.P Where were you born?

City: _____ (22-67)

State: _____ (68-69) 70-80Z

Country: _____ 3* (08-53)

Unknown.....(54 (____-8
Refused to answer.....____-9

IF RESPONDENT WAS BORN IN SOME COUNTRY OTHER THAN THE UNITED STATES, ASK Q.A2. ALL OTHERS SKIP TO Q.A3.

A2.P How old were you when you came to the United States to stay?

/ / / Age
(55-56)

Unknown.....(57 (____-8
Refused to answer.....____-9

58-79Z

A3.P What is your father's last name? (RECORD ACCURATELY; VERIFY THE SPELLING)

_____ 4* (08-53)

Unknown.....(54 (____-8
Refused to answer.....____-9

A4. P And where was your father born?

Arizona.....(55-56)____-01
California.....____-02
Colorado.....____-03
Mexico.....____-04
New Mexico.....____-05
Texas.....____-06
Other United States.....____-07
Spain (or one of its Provinces).....____-08
Central American Country.....____-09
South American Country (except Brazil).....____-10
Caribbean Island.....____-11
Asian Country (Philippines included).....____-12
Canada, Australia, Brazil, or any European Country (except Spain)....____-13
Mediterranean or Middle Eastern Country.....____-14
African Country.....____-15
Unknown.....____-98
Refused to answer.....____-99

57-80Z

A5.P What is your mother's maiden name? (RECORD ACCURATELY; VERIFY THE SPELLING)

5*(08-53)

Unknown.....(54(____-8
Refused to answer.....____-9

A6.P And where was your mother born?

Arizona.....(55-56)____-01
California.....____-02
Colorado.....____-03
Mexico.....____-04
New Mexico.....____-05
Texas.....____-06
Other United States.....____-07
Spain (or one of its Provinces).....____-08
Central American Country.....____-09
South American Country (except Brazil).....____-10
Caribbean Island.....____-11
Asian Country (Philippines included).....____-12
Canada, Australia, Brazil, or any European Country (except Spain)....____-13
Mediterranean or Middle Eastern Country.....____-14
African Country.....____-15
Unknown.....____-98
Refused to answer.....____-99

A7.P What is the ethnic background or national origin of each of your grandparents? (IF NECESSARY, SAY: Think back about your father's and your mother's parents. What country did their ancestors live in before any of them first came to the United States?) RECORD VERBATIM

Father's father: _____

Father's mother: _____

Mother's father: _____

Mother's mother: _____

FOR OFFICE USE ONLY:	Father's Father	Father's Mother	Mother's Father	Mother's Mother
Mexico (57-58) (59-60) (61-62) (63-64)	___-01	___-01	___-01	___-01
Texas	___-02	___-02	___-02	___-02
New Mexico	___-03	___-03	___-03	___-03
Arizona	___-04	___-04	___-04	___-04
California	___-05	___-05	___-05	___-05
Colorado	___-06	___-06	___-06	___-06
Other United States	___-07	___-07	___-07	___-07
Spain (or one of its Provinces)	___-08	___-08	___-08	___-08
Central American Country	___-09	___-09	___-09	___-09
South American Country (except Brazil)	___-10	___-10	___-10	___-10
Caribbean Island	___-11	___-11	___-11	___-11
Asian Country (Philippines included)	___-12	___-12	___-12	___-12
Canada, Australia, Brazil, or any European Country (except Spain)	___-13	___-13	___-13	___-13
Mediterranean or Middle Eastern Country	___-14	___-14	___-14	___-14
African Country	___-15	___-15	___-15	___-15
Don't know	___-98	___-98	___-98	___-98
Refused	___-99	___-99	___-99	___-99

IF DON'T MENTION MEXICO IN Q.A1, A4, A6 OR A7 CONFIRM RESPONSES AND ASK Q.S2 AGAIN.

A8.P Some people in the United States call themselves Mexican-American, Italian-American, Anglo, and so on. What do you prefer to call yourself? RECORD VERBATIM

_____ (65-66)

_____ (67-68)

A9.P What is the highest grade or year of regular school that you have completed?
(RECORD HIGHEST GRADE)

/___/___/
(69-70)

12 = H.S. Graduate/G.E.D
16 = College Graduate - 4 Yr.
17 = Any Graduate Education

Don't know.....(71(___-8
Refused.....___-9

A10.P Are you married, separated, divorced, widowed, or never married? (INCLUDE COMMON LAW MARRIAGES UNDER MARRIED)

Married.....(72(___-1
Separated.....___-2
Divorced.....___-3
Widowed.....___-4

(ASK Q.A11)

Never married.....___-5
Not Applicable.....___-6
Don't know.....___-8
Refused.....___-9

(SKIP TO Q.B1)

A11.P How long have you been (married/separated/divorced/widowed)?

/___/___/ Years IF LESS THAN A YEAR, CODE 01
(73-74)

Unknown.....(75(___-8
Refused to answer.....___-9

76-80Z

B. LIVING ARRANGEMENTS

B1.P How many people live in this household? (CONFIRM WITH TOTAL IN Q.S1)

/__/__/
6*(08-09)

Refused.....(10(___-9

B2.P Who is the head of this household and what is their relationship to you?

Who:_____(11-78)

Relationship Code: /__/__/
(79-80)

ASK Q.B3 IF MARRIED IN Q.A10 — ALL OTHERS SKIP TO Q.B4
B3.P What is the name of your spouse, if any, who lives in this household?

_____7*(08-53)

IF ONLY 1 PERSON IN Q.B1, SKIP TO Q.C1 — ALL OTHERS ASK Q.B4 /54-80Z/
B4.P We would like to know how the other people who live here with you are related to you.

Relationship Code (SEE BELOW)	Name (LAST, FIRST)	Sex	Age	
/__/__/ 8*(08-09)	_____ (10-45)	Male..(46(___-1 Female....___-2	/__/__/ (47-48)	49-80Z
/__/__/ 9*(08-09)	_____ (10-45)	Male..(46(___-1 Female....___-2	/__/__/ (47-48)	49-80Z
/__/__/ 10*(08-09)	_____ (10-45)	Male..(46(___-1 Female....___-2	/__/__/ (47-48)	49-80Z
/__/__/ 11*(08-09)	_____ (10-45)	Male..(46(___-1 Female....___-2	/__/__/ (47-48)	49-80Z
/__/__/ 12*(08-09)	_____ (10-45)	Male..(46(___-1 Female....___-2	/__/__/ (47-48)	49-80Z
/__/__/ 13*(08-09)	_____ (10-45)	Male..(46(___-1 Female....___-2	/__/__/ (47-48)	49-80Z

CODES FOR RELATIONSHIPS:
01 = Respondent is head of household
02 = Spouse
03 = Son/Daughter (including Stepchildren)
04 = Son-In-Law/Daughter-In-Law
05 = Grandchild
06 = Parent
07 = Brother or Sister
08 = Nephew or Niece
09 = Cousin
10 = Aunt/Uncle
11 = Great Grandchild
12 = Other Relative (SPECIFY):
_____
13 = Friend
14 = Boarder or Roomer
15 = Paid Employee
16 = Other Non-Relative (SPECIFY):
_____
98 = Don't Know
99 = Refused

Acculturation

F. ACCULTURATION

In this next part of the interview, I will be asking some more questions about your background, attitudes, and beliefs. First, I'm going to ask you about your use of language, in particular, English and Spanish, in various situations.

F1.P In your opinion, how well do you (READ EACH ITEM) -- very well, pretty well, not too well or not at all? USE SHOW CARD

	Very Well	Pretty Well	Not Too Well	Not At All	Don't Know	Refused
a. Understand spoken English.....(45	-4	-3	-2	-1	-8	-9
b. Speak English.....(46	-4	-3	-2	-1	-8	-9
c. Read English.....(47	-4	-3	-2	-1	-8	-9

F2.P What language do you usually use [READ EACH APPLICABLE ITEM]? Would you say only English, mostly English, Spanish and English equally, mostly Spanish or only Spanish? USE SHOW CARD

	Only English	Mostly English	Spanish & English Equally	Mostly Spanish	Only Spanish	Not Applicable	Don't Know	Refused
a. With your (spouse/partner).....(48	-1	-2	-3	-4	-5	-6	-8	-9
b. With your children.....(49	-1	-2	-3	-4	-5	-6	-8	-9
c. With your parents.....(50	-1	-2	-3	-4	-5	-6	-8	-9
d. With most of your friends.....(51	-1	-2	-3	-4	-5	-6	-8	-9
e. With most of your neighbors.....(52	-1	-2	-3	-4	-5	-6	-8	-9
f. With most of the people at work.....(53	-1	-2	-3	-4	-5	-6	-8	-9
g. At family gatherings, such as Christmas or other holidays.....(54	-1	-2	-3	-4	-5	-6	-8	-9

F3.P In what language are the [READ EACH ITEM]? Are they (READ CHOICES)? USE SHOW CARD

DO NOT ROTATE	Only English	Mostly English	Spanish & English Equally	Mostly Spanish	Only Spanish	Not Applicable	Don't Know	Refused
a. TV programs you watch.....(55	-1	-2	-3	-4	-5	-6	-8	-9
b. Radio stations you listen to....(56	-1	-2	-3	-4	-5	-6	-8	-9
c. Books and magazines you read....(57	-1	-2	-3	-4	-5	-6	-8	-9

Assimilation

-14- CARD 14 922027

F4.P Throughout your adult life, have your neighbors been mostly Mexican-Americans, mostly Anglo, or about equal numbers of each? USE SHOW CARD

Mostly Mexican-American.....(58(___-1
Mostly Anglo.....___-2
About equal numbers of each.....___-3
Other (SPECIFY): (RECORD VERBATIM)

_____..___-4
Don't know.....___-8
Refused.....___-9

IF "NEVER WORKED" IN Q.E1, SKIP TO Q.F6 -- ALL OTHERS ASK Q.F5

F5.P (Are/Were) the people with whom you work closely on [the job/your last job] mostly Mexican-Americans, mostly Anglo, or about equal numbers of each? USE SHOW CARD

Mostly Mexican-American.....(59(___-1
Mostly Anglo.....___-2
About equal numbers of each.....___-3
Other (SPECIFY): (RECORD VERBATIM)

_____..___-4
Don't know.....___-8
Refused.....___-9

F6.P Throughout your adult life, have your close personal friends been mostly Mexican-Americans, mostly Anglo, or about equal numbers of each? USE SHOW CARD

Mostly Mexican-American.....(60(___-1
Mostly Anglo.....___-2
About equal numbers of each.....___-3
Other (SPECIFY): (RECORD VERBATIM)

_____..___-4
Don't know.....___-8
Refused.....___-9

Global Health Rating

G. GLOBAL HEALTH RATING

G1. Now I would like to ask you some questions about your health. Overall, how would you rate your health — excellent, good, fair, or poor? USE SHOW CARD

Excellent.....(61(____-1
Good.....____-2
Fair.....____-3
Poor.....____-4
Don't know.....____-8
Refused.....____-9

G2.P During the past 3 months, did you ever have to cut down on things you usually do because of illness or injury, not counting day[s] in bed?

Yes.....(62(____-1
No.....____-2
Don't know.....____-8
Refused.....____-9

G3.P During the past 3 months, did you ever stay in bed all or most of the day because of illness or injury? (INTERVIEWER: INCLUDE DAYS IN THE HOSPITAL.)

Yes.....(63(____-1
No.....____-2
Refused.....____-9

H. CHEST PAIN

H1. Have you ever had pain or discomfort in your chest?

Yes.....(64(___-1 } (SKIP TO Q.H3)

No.....___-2
Don't know.....___-8 } (ASK Q.H2)
Refused.....___-9

H2. Have you ever had any pressure or heaviness in your chest?

Yes.....(65(___-1 } (ASK Q.H3)

No.....___-2
Don't know.....___-8 } (SKIP TO Q.H5)
Refused.....___-9

H3. Do you get this pain (or discomfort) when you walk uphill or hurry?

Yes.....(66(___-1 } (ASK Q.H4)

Cannot walk (vol.).___-2 } (SKIP TO Q.H8)

No.....___-3
Don't know.....___-8 } (SKIP TO Q.H5)
Refused.....___-9

H4. Do you get this pain or discomfort when you walk at an ordinary pace on level ground?

No.....(67(___-1
Yes.....___-2
Don't know.....___-8
Refused.....___-9

(Shortness of Breath)

H5. Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?

Yes.....(68(___-1 } (ASK Q.H6)

Cannot walk (vol.).___-2 } (SKIP TO Q.H8)

No.....___-3
Don't know.....___-8 } (SKIP TO Q.I1)
Refused.....___-9

H6. Do you get short of breath walking with other people of your own age on level ground?

Yes.....(69(____-1 } (ASK Q.H7)

No.....____-2
Don't know.....____-8 } (SKIP TO Q.I1)
Refused.....____-9

H7. Do you have to stop for breath when walking at your own pace on level ground?

Yes.....(70(____-1 } (ASK Q.H8)

No.....____-2
Don't know.....____-8 } (SKIP TO Q.I1)
Refused.....____-9

H8. Do you get short of breath when bathing or dressing?

Yes.....(71(____-1
No.....____-2
Don't know.....____-8
Refused.....____-9

I. CARDIOVASCULAR

I1.P Has a doctor ever told you that you had a heart attack, or coronary, or myocardial infarction, or coronary thrombosis?

Yes.....(72(___-1 } (ASK Q.I2)
Suspect or possible.....___-2

No.....___-3
Don't know.....___-8 } (SKIP TO Q.J1)
Refused.....___-9

I2.P Did you have only one, or more than one?

One.....(73(___-1
More than one.....___-2
Don't know.....___-8
Refused.....___-9

I3.P When was your first one? (PROBE) In what year? RECORD NUMBER OF YEARS

/___/___/
(74-75)

Don't know.....(76(___-8
Refused.....___-9

IF ONLY 1 IN Q.I2, SKIP TO Q.I5 — ALL OTHERS ASK Q.I4.
I4.P How many years ago was the last one? (PROBE) In what year? RECORD NUMBER OF YEARS

/___/___/
(77-78)

Don't know.....(79(___-8
Refused.....___-9

I5.P Were you hospitalized overnight or longer for this (last one)?

Yes.....(80(___-1
No.....___-2
Don't know.....___-8
Refused.....___-9

J. STROKE

J1.P Did a doctor ever tell you that you had a stroke, a blood clot in the brain, or brain hemorrhage?

Yes.....15*(08(___-1 } (ASK Q.J2)
Suspect or possible.....___-2

No.....___-3
Don't know.....___-8 } (SKIP TO Q.K1)
Refused.....___-9

J2.P Did you have more than one?

One.....(09(___-1
More than one.....___-2
Don't know.....___-8
Refused.....___-9

J3.P How many years ago was your first one? (PROBE) In what year? RECORD NUMBER OF YEARS

/___/___/
(10-11)

Don't know.....(12(___-8
Refused.....___-9

IF ONLY 1 IN Q.J2, SKIP TO Q.J5 — ALL OTHERS ASK Q.J4
J4.P How many years ago was the last one? (PROBE) In what year?

/___/___/
(13-14)

Don't know.....(15(___-8
Refused.....___-9

J5.P Were you hospitalized overnight or longer for this?

Yes.....(16(___-1
No.....___-2
Don't know.....___-8
Refused.....___-9

J6.P Do you still have leftover troubles from your stroke?

Yes.....(17(____-1 } (ASK Q.J7)

No.....____-2
Don't know.....____-8 } (SKIP TO Q.K1)
Refused.....____-9

J7.P Are your leftover troubles (READ EACH ITEM)?

	Yes	No	Don't Know	Refused
a. Arm and/or leg still weak or hard to use...(18	____-1	____-2	____-8	____-9
b. Trouble walking.....(19	____-1	____-2	____-8	____-9
c. Trouble with speech.....(20	____-1	____-2	____-8	____-9
d. Other (SPECIFY)				
_____.....(21	____-1	____-2	____-8	____-9

K. HYPERTENSION

K1.P Has a doctor ever told you that you have high blood pressure?

Yes.....(22(____-1 } (ASK Q.K2)
Suspect or possible.....____-2

No.....____-3
Don't know.....____-8 } (SKIP TO Q.L1)
Refused.....____-9

K2.P How many years ago were you first told this? (PROBE) In what year? RECORD NUMBER OF YEARS

/___/___/
(23-24)

Don't know.....(25(____-8
Refused.....____-9

K3.P Have you ever taken medicine prescribed by a doctor for your high blood pressure?

Yes.....(26(____-1 } (ASK Q.K4)

No.....____-2
Don't know.....____-8 } (SKIP TO Q.L1)
Refused.....____-9

K4.P Are you currently taking any medication for high blood pressure?

Yes.....(27(____-1
No.....____-2
Don't know.....____-8
Refused.....____-9

M. DIABETES

M1.P Have you ever been told by a doctor that you have diabetes, sugar in your urine or high blood sugar?

Yes, definite.....(38(___-1 } (ASK Q.M2)

Yes, borderline.....___-2
No.....___-3 } (SKIP TO Q.M9)
Don't know.....___-8
Refused.....___-9

M2.P In what month and year did a doctor first tell you that you have diabetes? (IF NECESSARY: PROBE FOR AGE OR AGE DECADE AT DIAGNOSIS TO ESTIMATE YEAR OF DIAGNOSIS, AND/OR PROBE FOR SEASON OF YEAR TO ESTIMATE MONTH OF DIAGNOSIS.)

/ / / Month (39-40) / 1 / 9 / / / Year (41-42)

Don't know.....(43(___-8
Refused.....___-9

M3.P Are you taking any medicine for diabetes now?

Yes.....(44(___-1 } (SKIP TO Q.M5)

No.....___-2
Don't know....___-8 } (ASK Q.M4)
Refused.....___-9

M4.P Have you ever taken any medicine for diabetes?

Yes.....(45(___-1 } (ASK Q.M5)

No.....___-2
Don't know....___-8
Refused.....___-9 } (SKIP TO Q.M8a)

M5.P Did the doctor prescribe pills or insulin shots or both together? IF PILLS AND INSULIN SEPARATELY, PROBE FOR MOST RECENT

Pills.....(46(___-1 } (ASK Q.M6)

Insulin shots.....___-2
Both pills and insulin shots together..___-3 } (SKIP TO Q.M7)
Don't know.....___-8
Refused.....___-9

M6.P Have you ever taken insulin shots?

Yes.....(47(___-1 } (ASK Q.M7)

No.....___-2
Don't know....___-8 } (SKIP TO Q.M8a)
Refused.....___-9

M7.P For how many years altogether (have you taken/did you take) insulin shots?

/__/__/ Years (48-49) OR /__/__/ Months (50-51) OR /__/__/ Weeks (52-53)

Don't know...(54(___-8
Refused.....___-9

M8a.P As a result of your diabetes, have you ever had any problems with your kidneys, or not?

Yes.....(55(___-1 } (ASK Q.M8b)

No.....___-2
Don't know.....___-8 } (SKIP TO Q.M8d)
Refused.....___-9

M8b.P Are you currently receiving kidney dialysis or artificial kidney treatments, or not?

Yes.....(56(___-1
No.....___-2
Don't know....___-8
Refused.....___-9

M8c.P Have you ever had a kidney transplant, or not?

Yes.....(57(___-1
No.....___-2
Don't know....___-8
Refused.....___-9

M8d.P As a result of your diabetes, have you ever had any problem with your eyes, or not?

Yes.....(58(___-1 } (ASK Q.M8e)

No.....___-2
Don't know....___-8 } (SKIP TO Q.M8f)
Refused.....___-9

M8e.P Have you ever had laser treatment on your eyes, or not?

Yes.....(59(____-1
No.....____-2
Don't know....____-8
Refused.....____-9

M8f.P As a result of your diabetes, have you ever had any problems with the circulation in your legs or arms, or not?

Yes.....(60(____-1 } (ASK Q.M8g)

No.....____-2
Don't know....____-8 } (SKIP TO Q.M8h)
Refused.....____-9

M8g.P Have you ever had any part of your body amputated as a result of your diabetes, or not? (IF MORE THAN ONE, PROBE FOR MOST SERIOUS — IF YES, SPECIFY)

Fingers.....(61(____-1
Toes.....____-2
One foot.....____-3
Both feet.....____-4
Lower leg.....____-5
Both lower legs...____-6
Other (SPECIFY)

_____.____-7
No.....____-8
Don't know....(62(____-8
Refused.....____-9

M8h.P As a result of your diabetes, have you ever had any other medical complications? (IF MORE THAN ONE, PROBE FOR MOST SERIOUS COMPLICATION)

Yes (SPECIFY:)

_____.(63-64)
No.....(65(____-2
Don't know.....____-8
Refused.....____-9

ASK EVERYONE
M9.P Have either of your parents had diabetes? (SPECIFY WHETHER MOTHER ONLY, FATHER ONLY OR BOTH)

Yes, mother only.....(66(____-1
Yes, father only.....____-2
Yes, both.....____-3
Yes, mother/Don't know father.____-4
Yes, father/Don't know mother.____-5
No, neither.....____-6
Don't know.....____-8
Refused.....____-9

-47- CARD 21 922027

X. CESD

X.P-R Now I have some questions about your feelings during the past week. For each of the following statements, please tell me if you felt that way in the past week rarely or none of the time which is less than 1 day, some or a little of the time which is 1 to 2 days, occasionally or a moderate amount of time which is 3 to 4 days, most or all of the time which is 5 to 7 days? (READ IF RESPONDENT GIVES NUMBER OF DAYS RECORD APPROPRIATELY) USE SHOW CARD In the past week (READ EACH ITEM).

	Rarely or None Of The Time (Less Than 1 Day)	Some Or A Little Of The Time (1-2 Days)	Occasionally Or A Moderate Amount Of Time (3-4 Days)	Most Or All Of the Time (5-7 Days)	Not Sure
1. I was bothered by things that usually don't bother me.....(18	___-0	___-1	___-2	___-3	___-8
2. I did not feel like eating; my appetite was poor.....(19	___-0	___-1	___-2	___-3	___-8
3. I felt that I could not shake off the blues even with help from my family and friends.....(20	___-0	___-1	___-2	___-3	___-8
4. I felt that I was just as good as other people.....(21	___-0	___-1	___-2	___-3	___-8
5. I had trouble keeping my mind on what I was doing.....(22	___-0	___-1	___-2	___-3	___-8
6. I felt depressed.....(23	___-0	___-1	___-2	___-3	___-8
7. I felt that everything I did was an effort.....(24	___-0	___-1	___-2	___-3	___-8
8. I felt hopeful about the future..(25	___-0	___-1	___-2	___-3	___-8
9. I thought my life had been a failure.....(26	___-0	___-1	___-2	___-3	___-8
10. I felt fearful.....(27	___-0	___-1	___-2	___-3	___-8
11. My sleep was restless.....(28	___-0	___-1	___-2	___-3	___-8
12. I was happy.....(29	___-0	___-1	___-2	___-3	___-8
13. It seemed that I talked less than usual.....(30	___-0	___-1	___-2	___-3	___-8
14. I felt lonely.....(31	___-0	___-1	___-2	___-3	___-8
15. People were unfriendly.....(32	___-0	___-1	___-2	___-3	___-8
16. I enjoyed life.....(33	___-0	___-1	___-2	___-3	___-8
17. I had crying spells.....(34	___-0	___-1	___-2	___-3	___-8
18. I felt sad.....(35	___-0	___-1	___-2	___-3	___-8
19. I felt that people disliked me..(36	___-0	___-1	___-2	___-3	___-8
20. I could not get going.....(37	___-0	___-1	___-2	___-3	___-8

-48- CARD 21 922027

Y. SMOKING

Y1.P Have you ever smoked at least 100 cigarettes in your entire life?

Yes.....(38(___-1) (ASK Q.Y2)

No.....___-2) (SKIP TO Q.Za)

Don't know.....___-8
Refused.....___-9) (ASK Q.Y2)

Y2.P About how old were you when you first started smoking cigarettes?

___/___/___/ Age
(39-40)

Never smoked regularly (vol.)...(41(___-0
Don't know.....___-8
Refused.....___-9

Y3.P Do you smoke cigarettes now?

Yes.....(42(___-1) (SKIP TO Q.Y5)

No.....___-2
Refused.....___-9) (ASK Q.Y4)

Y4.P About how long has it been since you have smoked cigarettes?

___/___/___/ Years
(43-44)

Less than 1 year.....(45(___-0
Don't know.....___-8
Refused.....___-9

Y5.P We are interested in the actual # of cigarettes people smoke each day. How many cigarettes do/did you smoke (when you last smoked regularly)? (ONE PACK EQUALS 20 CIGARETTES.)

___/___/___/___/ Actual number
(46-48)

Less than 1 cigarette per day..(49(___-0
Don't know.....___-8
Refused.....___-9

Z. ALCOHOL CONSUMPTION

Za.P Have you ever drunk any type of alcohol (beer, wine, liquor) in your entire life? (DO NOT INCLUDE COMMUNION WINE)

Yes.....(50(___-1) (ASK Q.Z1)

No.....___-2
Don't know.....___-8) (SKIP TO Q.AA1)
Refused.....___-9

Z1.P Have you had any beer, wine or liquor (that is, things like whiskey or tequila) during the past year?

Yes.....(51(___-1) (ASK Q.Z2)

No.....___-2) (SKIP TO Q.Z5)

Don't know.....___-8
Refused.....___-9) (ASK Q.Z2)

Z2.P We are especially interested in recent times. In the past month, have you had any (READ EACH ITEM)?

	Yes	No	Don't Know	Refused
1. Beer.....(52	(___-1	___-2	___-8	___-9
2. Wine.....(53	(___-1	___-2	___-8	___-9
3. Liquor.....(54	(___-1	___-2	___-8	___-9

IF YES TO ANY ITEM IN Q.Z2, ASK Q.Z3 — ALL OTHERS SKIP TO Q.Z5. CODE ACTUAL NUMBER GIVEN, FOR EXAMPLE: 16 TIMES PER MONTH - "16".

Z3.P Over the last month how often have you had beer, wine or liquor?

/___/___/ Times Per Month
(55-56)

Don't know.....(57(___-8
Refused.....___-9

IF RESPONSE IS GIVEN IN TERMS OF PER WEEK OR PER DAY, USE GUIDE BELOW

3 OR MORE TIMES PER DAY = 90
2 TIMES PER DAY = 60
1 TIME PER DAY = 30
6 TIMES PER WEEK = 24
5 TIMES PER WEEK = 20
4 TIMES PER WEEK = 16
3 TIMES PER WEEK = 12
2 TIMES PER WEEK = 08
1 TIME PER WEEK = 04

Z4.P When you had beer, wine or liquor in the last month, how many cans or bottles, glasses or drinks did you usually have at one time?

/___/___/ Cans/Bottles
(58-59)

None.....(60(___-0
Don't know.....___-8
Refused.....___-9

61-80Z

Z5. Has there ever been a time when you drank quite a bit more than you drink now?

Yes.....22*(08(___-1
No.....___-2
Don't know.....___-8
Refused.....___-9

Z6. Has there ever been a time when you considered yourself to be a heavy drinker?

Yes.....(09(___-1
No.....___-2
Don't know.....___-8
Refused.....___-9

Z7. Have you ever felt you should cut down on your drinking?

Yes.....(10(___-1
No.....___-2
Don't know.....___-8
Refused.....___-9

Z8. Have people annoyed you by criticizing your drinking?

Yes.....(11(___-1
No.....___-2
Don't know.....___-8
Refused.....___-9

Z9. Have you ever felt bad or guilty about your drinking?

Yes.....(12(___-1
No.....___-2
Don't know.....___-8
Refused.....___-9

Z10. Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover? (Eye-opener)

Yes.....(13(___-1
No.....___-2
Don't know.....___-8
Refused.....___-9

II. HEIGHT AND WEIGHT

II1a.P-R Now we'd like to get your height and weight. Why don't you slip off your shoes and remove heavy jewelry or clothing. Now stand back against this door with your feet, heels together on the floor and with your heels, hips, back and head directly against the wall. Look straight ahead.

Height (to nearest quarter-inch) /__/__/__/ . /_/_/ Inches
(22-26)

Refused.....098.00

II1b.P-R Now let's get your weight.

Weight (to nearest pound) /__/__/__/ Pounds
(27-29)

Refused.....(30(___-9

II1c.P-R INTERVIEWER: TYPE OF SURFACE.

Uncarpeted.....(31(___-1
Low carpet.....___-2
Other (SPECIFY):

_____..___-3

DD. HEALTH LOCUS OF CONTROL

DD1. How concerned are you about being unable to be independent and take care of yourself and your affairs in the future? Would you say you are very concerned, somewhat concerned, or not concerned at all? USE SHOW CARD

Very concerned.....(66(____-1
Somewhat concerned.....____-2
Not concerned at all....____-3
Don't know.....____-8
Refused.....____-9

DD2. To what extent do you feel you can control the general state of your health through your own actions — a great deal, somewhat, or not at all? USE SHOW CARD

A great deal.....(67(____-1
Somewhat.....____-2
Not at all.....____-3
Don't know.....____-8
Refused.....____-9

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Vita

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