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Racial/Ethnic Differences in Depressive Symptoms and Treatment Effect among Patients with Myocardial Infarction from the Enhancing Recovery In Coronary Heart Disease (ENRICHD) Trial

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RACIAL/ETHNIC DIFFERENCES IN DEPRESSIVE SYMPTOMS AND
TREATMENT EFFECT AMONG PATIENTS WITH MYOCARDIAL INFARCTION
FROM THE ENHANCING RECOVERY IN CORONARY HEART DISEASE
(ENRICHED) TRIAL

By

Hsin-hua Lin

A DISSERTATION

Submitted to the Faculty
of the University of Miami
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UNIVERSITY OF MIAMI

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Racial/Ethnic Differences in Depressive
Symptoms and Treatment Effect
among Patients with Myocardial Infarction
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in Coronary Heart Disease (ENRICHD) Trial

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This study examined racial/ethnic group differences in depressive symptoms and treatment effect in a diverse clinical sample of post myocardial infarction (MI) patients. Specific aims were to test group measurement equivalence of the Beck Depression Inventory (BDI) across non-Hispanic Blacks, non-Hispanic Whites, and Hispanic cardiac patients and to test stability of a BDI measurement model over time from baseline to six-months post-treatment both in the treatment and the usual care groups. The participants included 2370 diverse post-MI patients (467 non-Hispanic Blacks, 1647 non-Hispanic Whites, and 256 Hispanics), a subgroup of the participants who were clinically depressed and/or socially isolated from the Enhancing Recovery in Coronary Heart Disease (ENRICHD) trial. Depression was measured using the BDI at baseline and six-months post-treatment. A between-group analysis of variance (ANOVA) of the baseline BDI total scores, a series of confirmatory factor analysis of the BDI items, and structural equation modeling of treatment effect on depressive symptoms were conducted to investigate the study aims. Gender, baseline depression levels, baseline antidepressant medication use, education, income,

and employment were included as covariates in the model testing for racial/ethnic differences in baseline depression levels and treatment effect on depression symptoms. Findings suggested that racial/ethnic cardiac patients exhibited different cognitive yet similar somatic depression symptoms and that treatment effect on the reduction of depressive symptoms were comparable across racial/ethnic groups. It is essential to distinguish cognitive and somatic depression symptoms among cardiac patients and to develop intervention programs targeted on specific subtypes of depression for treatment. Future investigations should consider the predictive validity and relevance of the BDI subscales with respect to underlying symptoms, treatment aims, and clinical outcomes among cardiac patients and other clinical populations.

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CHAPTER 1

INTRODUCTION

Depression is a common psychiatric disorder among both non-clinical and clinical populations. Prevalence estimates reported in the general population indicate that 6.6 to 10% of individuals have been affected by depression in the United States (U.S.) (Kessler et al., 2003; Robins & Regier, 1990). Medical populations, especially patients with myocardial infarction (MI), reportedly have a higher prevalence rate, in the range of 20 to 25% (Frasure-Smith & Lesperance, 2003b; Frasure-Smith, Lesperance, & Talajic, 1993). Myocardial infarction, known as a heart attack, is a failure or shortage of coronary circulation to supply adequate blood to the heart (i.e., cardiac muscle and surrounding tissue). Furthermore, racial/ethnic minorities report higher levels of depression compared to non-Hispanic Whites (Dunlop, Song, Lyons, Manheim, & Chang, 2003).

Individuals diagnosed with depression may experience, manifest, and present depression in a different manner across racial/ethnic groups (Mossakowski, 2008; 2006). Racial/ethnic disparities in depression may be partially explained by differences in cultural knowledge and perspectives about depression (Mezzich et al., 2008) and/or socioeconomic disadvantages

(Mossakowski, 2008), with racial/ethnic minorities reporting more somatic depressive symptoms than non-Hispanic Whites (Ayalon & Young, 2003; Brown, Schulberg, & Madonia, 1996; Myers et al., 2002).

The significant role that depression plays in coronary heart disease (CHD) patients is well-documented in the literature (Burg & Abrams, 2001). In particular, depression has been shown to be a risk factor not only for poor cardiac prognosis, but also for increased subsequent negative cardiac outcomes among post-MI patients (Alboni, Favaron, Paparella, Sciammarella, & Pedaci, 2008; Carney et al., 2008). Research has continued to show health disparities between racial/ethnic minority groups (i.e., Hispanics and non-Hispanic Blacks) and non-Hispanic Whites in the U.S., with racial/ethnic minority groups experiencing higher prevalence rates of depression and poorer health outcomes. Because mental health is an ineradicable component of overall health, addressing health disparity across racial/ethnic groups requires a critical examination of not only the measurement instruments but also the underlying models of depression used to provide culturally equivalent measures of depressive symptoms across racial/ethnic groups. Otherwise, research findings of depression and health outcomes across racial/ethnic groups can be hard to interpret.

The current study primarily investigated whether there were group differences in clinical depressive symptoms among non-Hispanic Black, non-Hispanic White, and Hispanic cardiac patients. Specifically, the first aim was to examine whether the three racial/ethnic groups differ significantly in the baseline Beck Depression Inventory (BDI) levels. The second purpose was to determine

if the BDI measures depressive symptoms comparably among 467 non-Hispanic Black, 1647 non-Hispanic White, and 256 Hispanic cardiac patients from a randomized clinical trial of treatment for depression. The third objective was to test the treatment effect of specific depressive symptoms across the three racial/ethnic groups. Last aim of this study was to test the stability of the baseline BDI model across a six-month time frame separately in the Treatment and Usual Care groups, only in non-Hispanic Whites.

CHAPTER 2

REVIEW OF LITERATURE

This chapter provides a summary of the literature from studies concerning clinical depression, including prevalence rates, depression in the context of CHD, and associations with adverse CHD outcomes. Because of the significant role of depression in CHD, current research on large clinical treatment trials for depression among cardiac patients were reviewed. In addition, because of racial/ethnic differences in manifestations of depression symptoms, specifying measurement equivalence for underlying depression symptoms is essential. Studies on utilization of the commonly used the BDI across racial/ethnic groups were reviewed and summarized. Finally, four study aims were proposed to examine racial/ethnic differences in manifestations of depression among non-Hispanic Black, non-Hispanic White, and Hispanic cardiac patients, as well as treatment effect on depressive symptoms across racial/ethnic groups.

According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)(American Psychiatric Association, 2000), clinical depression, known as Major Depressive Disorder (MDD), is defined as a psychiatric disorder with predictable symptoms and treatment response. MDD is characterized by a combination of symptoms that interfere with a person's ability to perform daily routines and enjoy pleasurable activities that the person used to do. For instance, according to the DSM-IV-TR (2000),

common MDD symptoms include depressed mood, and/or loss of interest or pleasure in activities, in addition to weight loss, insomnia, psychomotor agitation/retardation, fatigue, feelings of worthlessness or inappropriate guilt, and recurrent thoughts of death most of the day or every day for at least two weeks. Most individuals diagnosed with MDD experience recurring episodes over their lifetimes. The likelihood of recurrence increases with a previous history of depressive episodes. The average duration of an episode of depression is approximately six months, but some individuals may suffer from depression for several years.

Prevalence of Depression

Depending on the population studied, depression prevalence rates vary. Notably, these rates have varied considerably in non-clinical populations in the past three decades. In the Epidemiologic Catchment Area (ECA) study from 1980 to 1985, prevalence rates of MDD were 3.0% for a current depression and 5.2% for a lifetime depression (Weissman, Bruce, Leaf, Florio, & Holzer, 1991). The U.S. National Institute of Mental Health (NIMH) cites evidence that approximately 10% of individuals in the U.S. experience some variety of depressive symptoms in any one-year period (Robins & Regier, 1990). The National Comorbidity Survey Replication (NCS-R), a large national epidemiology study from 2001 to 2002, reported that the 12-month prevalence of MDD was

6.6% and the lifetime prevalence of MDD was 16.2% (Kessler et al., 2003).

These studies concluded that depression is a common psychiatric disorder in the general population.

Higher prevalence rates have been reported in the cardiac patient population as compared to the general population (Frasure-Smith & Lesperance, 2008; Frasure-Smith et al., 2000; Goldston & Baillie, 2008). In contrast to what has been found in the general population, the prevalence of MDD in patients with CHD is approximately 20% (Rudisch & Nemeroff, 2003; Thombs et al., 2006a). Especially for post-MI patients, as many as 65% of these individuals are at least mildly depressed or have experienced depressive symptoms (Frasure-Smith, Lesperance, & Talajic, 1995). Depression before and after MI was also found in a study showing that 27.5% of patients had at least one episode of MDD before their MI and 7.7% were depressed at some point during the year preceding their MI (Lesperance, Frasure-Smith, & Talajic, 1996). The study further asserted that approximately 31.5% of patients experienced depression in the hospital or during the year after being discharged. In comparison to healthy individuals, cardiac patients showed significantly more depressive symptoms at baseline and up to one to two years later (Frasure-Smith & Lesperance, 2008; Strik, Lousberg, Cheriex, & Honig, 2004). These studies suggested that post-MI patients had significantly higher levels of depression over the first follow-up year, more total symptoms of depression in the second follow-up year, and more depressive symptoms at the end of each follow-up year than those without MI.

Depression in Coronary Heart Disease (CHD)

Accumulating evidence indicates that depression is an independent risk factor for the onset of CHD in healthy individuals (Van der Kooy et al., 2007; Wulsin & Singal, 2003) with an independent and gradient association between depression and incident CHD (Rowan, Haas, Campbell, Maclean, & Davidson, 2005; Van Melle et al., 2004). Furthermore, research evidence suggests the associations between depression and future CHD risk work through two possible mediating mechanisms: 1) health-related behaviors such as diet, smoking, alcohol consumption, and physical inactivity (Knox et al., 2006; Sheps, Frasure-Smith, Freedland, & Carney, 2004); and/or 2) neuroendocrine changes due to chronic stress in the sympathetic nervous system and the hypothalamic–pituitary–adrenal cortical axis (Dawood, Lambert, Barton, & Lambert, 2008) or elevated levels of C-reactive protein (CRP), interleukin (IL)-1, and IL-6 (Howren, Lamkin, & Suls, 2009). In addition, research has shown that at-risk individuals with elevated depression symptoms and a prior treatment history reported higher rates of smoking, hypertension, and an increased incidence of death and cardiac events (Rutledge et al., 2006). Depressed individuals may have a higher risk of developing CHD than those who are not depressed.

Depression is also a predictor for increased cardiac mortality and morbidity in patients with existing CHD. Higher levels of depression were found associated with greater CHD severity (Barefoot, Brummett, Helms, et al., 2000; Van der Kooy et al., 2007). Patients with CHD frequently experience depression because post-MI survivors often face the challenge of many psychosocial

adjustments (Carney, Freedland, & Steptoe, 2007). For instance, Carney et al. (2007) suggested that many CHD patients suffering from MI must learn to make adjustments and live their lives with the limitations caused by their CHD. Furthermore, post-MI patients are confronted with the issues concerning morbidity, as manifested by their fear of having another heart attack or dying. Given the psychosocial issues with which CHD patients often have to cope, it is understandable that depression is prevalent in this clinical population. Thus, it is common to observe depression lasting several years among patients recovering from MI (Carney et al., 2008; Rugulies, 2002). Some patients return to their premorbid mood state within a few days or weeks following a MI. Many patients will develop a more serious or persistent presentation of clinical depression.

The co-occurrence of depression and post MI is well-documented. Prior research by Martens et al. (2010) and Meijer et al. (2011) suggested that post MI is associated with increased depressive symptoms and amplification of cardiac symptoms and events. Additionally, research suggests that the likelihood of depression dramatically increases at approximately 28 days before the onset of cardiac symptoms, and that it can increase morbidity and mortality (Berkman et al., 2003). Approximately 20 to 25% of MI patients develop MDD or experience depressive symptoms within a year after MI (Frasure-Smith et al., 1993). Watkins et al. (2003) showed in their study that clinical depression is significantly associated with high levels of medical co-morbidity, and this association remained significant after adjusting for CHD severity. Furthermore, depression predicts poor survival and prognosis after MI (Frasure-Smith & Lesperance,

2003a, 2008; Frasure-Smith et al., 1993; Frasure-Smith et al., 1995). These findings indicate that depressive symptoms play a significant role affecting the prognosis of CHD, particularly following a MI.

To date, several studies have investigated the relationship between specific types of depression symptoms and cardiac outcomes. A study by de Jonge et al. (2006) investigated the relationship between types of post-MI depressive symptoms and overall health prognosis. Their findings indicated that using the BDI, somatic but not cognitive BDI symptoms were positively associated with poor health status such as previous history of MI, and that they predicted subsequent cardiovascular mortality and cardiac events. Similarly, another recent study found that somatic but not cognitive BDI symptoms, predicted worse cardiovascular prognosis (Linke et al., 2009). The findings of the multicenter STAR*D trial showed a similar association between depression subtypes and greater morbidity among cardiac patients (Fraguas et al., 2007). After adjustments for gender, age, ethnicity, education, and employment status, sympathetic arousal and early-morning insomnia were significantly associated with the onset of CHD. Altogether, these studies indicated underlying depressive symptoms (i.e., somatic) are significant predictors for poor prognosis and adverse health outcomes among cardiac patients. However, these studies did not address the adequacy of current measurement of depressive symptoms in CHD across racial/ethnic groups.

Although MDD and depressive symptoms are common in medical populations, they are frequently under-diagnosed and thus undertreated in CHD

patients (Musselman, Evans, & Nemeroff, 1998). A prior study has found that compared to 38% of depressed minority patients with acute coronary syndrome, only 24.5% of non-Hispanic depressed White cardiac patients have their depressive symptoms recognized and that race/ethnicity was associated with unrecognized depressive symptoms (odds ratio [OR] = 6.73, 95% confidence interval [CI] 2.62-19.33) (Amin, Jones, Nugent, Rumsfeld, & Spertus, 2006). In particular, some CHD sickness symptoms may overlap with depressive symptoms (i.e., fatigue and negative mood). Consequently, these undiagnosed depressive symptoms persist throughout the course of CHD. Consequences of undiagnosed depression may lead to adverse cardiac outcomes and increased subsequent cardiac events. Thus, cardiac patients who may be at high risk for depression are in need of adequate assessment for their depressive symptoms.

In sum, depression is one of the most common psychiatric disorders affecting post-MI patients. Previous studies by Carney et al. (2001) and Watkins et al. (2003) have attempted to determine the development of clinical depression in relation with CHD progression, especially following an MI. Carney et al. (2008) suggested that depression is an independent and significant risk factor of death for up to five years after an acute MI, and that minor depression is associated with an increased risk for adverse cardiac outcomes. In addition, Carney and Freeland (2012) suggested that somatic depression is more common than cognitive depression in cardiac patients. While substantial research has provided strong evidence supporting the relationship between depression and CHD (i.e., post-MI), the literature suggests that somatic, but not cognitive, aspect

of depression predicts subsequent cardiac events among patients with existing CHD. Thus, it is important to further investigate the subtypes of depression, either somatic or cognitive, among cardiac patients.

Interventions for Depression in CHD Patients

Treating depression in the context of CHD is particularly important because it aids in the process of recovering to normal functioning after having a cardiac event. Understanding post-MI depression, including cognitive and somatic symptoms, is essential for identifying high risk groups for interventions. In the literature, a number of clinical trials, including the Danish Cardiac Rehabilitation (DANREHAB) trial, the Support, Education, and Research in Chronic Heart Failure Study (SEARCH), and the Sertraline Antidepressant Heart Attack Trial (SADHART) (Joynt & O'Connor, 2005; Sheps, Freedland, Golden, & McMahon, 2003; Sullivan et al., 2009), have shown the benefits of treatment interventions for cardiac patients. The findings of these clinical trials suggested that comprehensive treatment facilitated cardiac recovery and improved psychosocial functioning.

Other research findings have demonstrated the effectiveness of psychosocial interventions for depression. One study suggested that psychosocial treatment interventions for CHD reduced psychological distress and post-MI recurrence in recovery (Linden, Stossel, & Maurice, 1996). Similarly, the SEARCH project reported that psychosocial interventions reduced depression among cardiac patients (Sullivan et al., 2009). A meta-analytic review (Welton,

Caldwell, Adamopoulos, & Vedhara, 2009) indicated that education, cognitive and behavioral therapy, relaxation, and support or a combination of these components were more effective than usual care, in which physicians provide standard patient care practices (e.g., routine medical examinations).

This review further suggested that psychological interventions were particularly effective in reducing the level of anxiety and that behavioral interventions were effective in reducing the odds of all-cause mortality and nonfatal MI. Furthermore, it also indicated that behavioral and cognitive interventions were associated with reduced levels of depression.

Nevertheless, treating depression in cardiac patients raises challenges for clinicians because cardiac patients may have different depression profiles (i.e., symptomatology, severity, and history). Differences in depression profiles may account for differential treatment effect. The findings of large randomized clinical trials (i.e., SADHART and ENRICHD) have suggested that patients with severe depression and prior history may achieve better depression outcomes after treatment (Carney et al., 2004; Glassman et al., 2002). In particular, racial/ethnic differences in treatment outcomes were observed in the CHD research (Casale, Auster, Wolf, Pei, & Devereux, 2007; Iribarren et al., 2005; Jha et al., 2003; Palmeri et al., 2005). Therefore, treatment effectiveness may vary between majority group (i.e., non-Hispanic White men) and racial/ethnic minority groups (Schneiderman et al., 2004). The limited number of research studies on racial/ethnic differences in treatment outcomes in the context of CHD prevented a firm conclusion about the findings.

Research is lacking on racial/ethnic differences in treatment outcomes of depression in CHD. The challenge and limitation may be accounted by the fact that only a few clinical trials included sufficient numbers of racial/ethnic minority groups and female cardiac patients because these under-represented populations are often difficult to recruit (Mak, Law, Alvidrez, & Pérez-Stable, 2007; U.S. Department of Health & Human Services, 2001). The difficulty of recruitment and retention for racial/ethnic minority groups may be associated with language and cultural differences that can potentially impede effective communication of information and thus attenuate participation rates (Suarez-Morales et al., 2007).

However, despite the difficulty of recruiting racial/ethnic minority groups, it is important to learn the course of depression presentation and its relation to the progression and prognosis of CHD in racial/ethnic minority groups compared to non-Hispanic Whites. Specifically, it is important to study the depression symptoms and their relation to treatment effect across racial/ethnic groups. A better understanding can help to identify high risk racial/ethnic groups with specific depression symptoms and lead to more effective treatment interventions. This is particularly salient when developing interventions to eliminate or reduce health disparities for the control of CHD in the U.S. in the future.

ENRICH

One of the largest clinical trials investigating the effectiveness of interventions on cardiac patients with respect to reducing CHD mortality and

morbidity was the Enhancing Recovery in Coronary Heart Disease (ENRICHD) trial (The ENRICHD investigators, 2000, 2001), funded by the United States National Heart, Lung and Blood Institute in 1996. The ENRICHD trial was originally designed to test the relative efficacy of a Cognitive Behavioral Therapy (CBT) intervention in improving cardiac treatment for post-MI patients suffering from clinical depression. Its research focus was to specifically target common psychosocial problems (e.g., depression and low social support) among cardiac patients. ENRICHD applied the CBT intervention for a large diverse sample of cardiac patients with depression and/or low social support. However, only limited effects of the treatment for post-MI depression in the treatment group were observed. This finding may be explained, in part, by a spontaneous reduction in depression observed in a considerable number of the patients from the usual care group. It is plausible that the illness-related behaviors (e.g., fatigue, negative mood, or inactivity) caused by cytokine activations post-MI share features with depression (Maier & Watkins, 1998). Illness-related behaviors were associated with increased levels of C-reactive protein (CRP) (Clearfield, 2005), a nonspecific acute-phase protein synthesized in the liver in response to stimulation from proinflammatory cytokines (i.e., Interleukin (IL)-6 and IL-1). Once the elevated neuroendocrine system returns to the premorbid or normal state, patients may no longer exhibit the sickness behaviors (e.g., fatigue or negative mood) (Howren et al., 2009). This may explain the spontaneous alleviation of depression in the Usual Care group. Because the substantial improvement of depression was observed in the Usual Care group, it is crucial to

further investigate whether depressive symptoms are similar over time from baseline to six months post-treatment as a function of treatment group.

Although ENRICHHD did not significantly reduce mortality (i.e., overall death and subsequent non-fatal MI events), the effects of ENRICHHD on psychosocial factors (i.e., depression and social support) were beneficial and significant for some post-MI patients. Because the ENRICHHD treatment was based on a cognitive-behavioral therapy model, researchers suggested that this intervention for depression may alleviate negative affect or the cognitive aspects of depression, but these effects may not be strong enough to reverse post MI physical symptoms that share features with somatic aspects of depression (Frasure-Smith & Lespérance, 2005; Watkins et al., 2003). These findings of ENRICHHD suggest that a detailed analysis of depressive symptoms may help to distinguish cardiac patients with depression, who may be at higher risk for adverse outcomes. Also, a detailed analysis of treatment effect on post-MI depressive symptoms may help to understand how ENRICHHD reduced specific depressive symptoms among cardiac patients. To answer these questions, additional analyses will be required to examine the assumption that current measurement for depressive symptoms is adequate and generalizable across the racial/ethnic groups represented in the ENRICHHD study.

Disparities in Depression

Mental health is an essential component of overall health. While addressing health disparity issues in CHD, one should also examine health

disparity issues in depression across all groups. In particular, the number of racial/ethnic minority groups residing in the United States has increased dramatically in recent years. Based on the 2010 population census in United States (U.S. Census Bureau, 2011), Hispanics grew by 43% in the last decade, accounting for 16.3% of the total population in 2010. By 2050, non-Hispanic Whites will no longer be the majority group because Hispanics are projected to continue increasing to 25% (U.S. Department of Health & Human Services, 2000). A projected change of the U.S. racial/ethnic population census in 2050 calls attention to eliminating and reducing the existing racial/ethnic health disparities. A number of studies have investigated differences in depression prevalence rates, sociodemographic factors, and symptom presentations in various settings (Boutin-Foster, 2008; Cuellar & Roberts, 1997; Riolo, Nguyen, Greden, & King, 2005; Waite, 2006). The majority of the studies did not examine the prevalence and severity of depression in the context of racial/ethnic differences. The few studies that concentrated on different cultural groups have reported racial/ethnic differences in depression prevalence. Some studies have suggested that racial/ethnic minorities are more likely to experience depression than non-Hispanic Whites (Boutin-Foster et al., 2008; Hernandez & Sachs-Ericsson, 2006; Holahan, Moerkbak, & Suzuki, 2006; Munson, 2002). According to these studies, this finding may be related to the psychosocial differences in years of education, employment status, stressful life events, emotional social support, or interpersonal functioning between Hispanics and non-Hispanic Whites. One study that examined variations in the total scores of depressive

symptomatology across the three largest racial/ethnic groups (i.e., non-Hispanic Blacks, Hispanics born in the US, and Hispanics born outside the US) observed high scorers on total depression measured by the Center for Epidemiological Studies Depression Scale (CES-D) within the Hispanic immigrant group (Iwata, Turner, & Lloyd, 2002). Blacks and Hispanic immigrants were more likely to score higher on CES-D than Whites. Hispanic immigrants were more likely to endorse the item of “low positive affect” compared to U.S. born Hispanics and Whites. A study by Myers et al. (2002) reported a similar finding that racial/ethnic minority women such as non-Hispanic Black and Hispanic women were rated as significantly more depressed than non-Hispanic Whites, after controlling for differences in socioeconomic status. Furthermore, a recent meta-analysis indicated that more depressive symptoms were reported among Hispanics than non-Hispanic Whites (Mendelson, Rehkopf, & Kubzansky, 2008).

Higher prevalence rates of depression were reported in Hispanics, compared to other racial/ethnic groups. The first population-based comprehensive assessment of the mental health status of Hispanics, known as the Hispanic Health and Nutrition Examination Survey (HHANES), indicated increased lifetime depression prevalence rates (Narrow, Rae, Moscicki, Locke, & Regier, 1990). Ten percent of the Hispanic population reported high levels of depression measured by the CES-D (i.e., a total score of 16 and higher). This regional survey also indicated that females (23.5%) were twice as likely to be depressed as males (12.9%). Similarly, another study investigating racial/ethnic differences in rates of MDD across three racial/ethnic groups found that

Hispanics had the highest 12-month prevalence rate of 10.8% compared to non-Hispanic Blacks (8.9%) and non-Hispanic Whites (7.8) (Dunlop et al., 2003). Racial/ethnic minority women were rated significantly more depressed than non-Hispanic Whites, after controlling for differences in socioeconomic status (Myers, et al., 2002). An analytic review found that more depressive symptoms were reported among Hispanics than non-Hispanic Whites (Mendelson et al., 2008). These studies suggest that depression levels are highest among Hispanics across all groups, indicating Hispanics as a high risk population for poor mental health outcomes.

However, a few studies reported contradictory or insignificant findings with respect to racial/ethnic differences in depression prevalence rates. From the National Health and Nutrition Examination Survey III, the results showed that the prevalence of MDD was highest among non-Hispanic Whites (10.40%) compared to Hispanics (8.00%) and non-Hispanic Blacks (7.50%); whereas the opposite order was observed for Dysthymic Disorder (i.e., 7.50% non-Hispanic Blacks, 7.40% Hispanics, and 5.70% non-Hispanic Whites) (Riolo et al., 2005). Similarly, the National Epidemiologic Survey on Alcoholism and Related Conditions reported the prevalence rate of 12-month and lifetime MDD to be 5.28% for Hispanics and 13.23% for non-Hispanic Whites respectively (Hasin, Goodwin, Stinson, & Grant, 2005). Being Hispanics reduced the risk, with 4.27% and 9.64% respectively for 12-month and lifetime MDD prevalence; whereas non-Hispanic Whites reported 5.53% and 14.58% otherwise. Furthermore, a few

earlier studies did not find any racial/ethnic group differences in the prevalence of depression (Perl, Bagne, & Gurevich, 1989; Zung, MacDonald, & Zung, 1988).

Inconsistent research results revealed the complexity in the relationship between race/ethnicity and depression manifestations. These racial/ethnic differences in depression may be related to specific cultural or racial/ethnic characteristics, which contribute to variations in depressive symptoms, expressions, and responses (Mezzich & Caracci, 2008; Vega & Rumbaut, 1991). In other words, it suggests that depression presentations may be “shaped by cultural values and norms governing perception, interpretation, and meaning of the emotional experience” (Mezzich et al., 1999, p. 458). Such speculation posits that factors associated with cultural values, beliefs, and perspectives (e.g., being a member of a racial/ethnic group) can therefore contribute to the psychological functioning of an individual from a particular racial/ethnic group. For instance, cultural influence of fatalism (i.e., a belief that events are determined by fate) on poor cardiac health and social functioning were observed in Hispanics (Urizar & Sears, 2006). In addition, compared with non-Hispanic White women, most depressed racial/ethnic minority women were less likely to perceive a need for mental health care (Nadeem, Lange, & Miranda, 2009). These plausible racial/ethnic differences in depression may also be attributed to socioeconomic disadvantages that racial/ethnic minorities are more likely to experience early in life than non-Hispanic Whites (Pearlin, Menaghan, Lieberman, & Mullan, 1981). Compared to non-Hispanic Whites, non-Hispanic Blacks have limited access to health services and often receive poor quality of care (U.S.

Department of Health & Human Services, 2001). Socioeconomic disadvantages are associated with increased stress and restricted coping resources (Meyer, Schwartz, & Frost, 2008), which may lead to higher depression among racial/ethnic minorities (Mossakowski, 2008). Although the influence of race/ethnicity on depression manifestations is unclear, racial/ethnic disparities in depression warrant further investigation.

A review of a number of research studies revealed that some racial or ethnic groups are more likely than others to exhibit somatic symptoms of depression. Research has suggested that non-Hispanic Blacks (Ayalon & Young, 2003; Brown et al., 1996) or Hispanics (Myers, 2002) are more likely to exhibit somatic symptoms than non-Hispanic Whites. In particular, compared to non-Hispanic Blacks, evidence has suggested that Hispanics have a tendency of somatization in depression (Escobar, Burnam, Karno, Forsythe, & Golding, 1987; Lewis-Fernandez, Das, Alfonso, Weissman, & Olfson, 2005; Ruiz, 1998). Somatization, commonly observed among Hispanics at primary care settings, is conceptualized as a form of distress through a presentation of physical symptoms without the presence of organic pathology (Gureje, Simon, Ustun, & Goldberg, 1997; Gureje, Simon, & Von Korff, 2001). It was speculated that somatization of depression serves as a stress-coping mechanism displayed within the Hispanic culture and norms (Ruiz, 1998; Vega & Rumbaut, 1991). Clinical depression may be associated with stigma and thus somatization of depression among Hispanics may be more acceptable within the culture

(Nadeem et al., 2009). These findings, although limited, have revealed plausible racial/ethnic differences in the manifestations of depressive symptoms.

The limited literature studies indicate that more research is needed to investigate the racial/ethnic differences in the manifestations of depression. Given the research that shows a higher prevalence of depression in ethnic minorities, it is important to test whether depression is measured comparably across racial/ethnic groups.

In addition to racial/ethnic differences in depression, gender differences in depression are reported in the literature. Epidemiological studies suggest that women are approximately 1.7 times as likely as men to report a lifetime history of MDD (Kessler, McGonagle, Nelson, & Hughes, 1994; Kessler, McGonagle, Swartz, & Blazer, 1993). Research data also indicate that women have a higher prevalence of somatic depression compared to men (Silverstein, 1999, 2002) and that women are more likely than men to endorse somatic symptoms (Wenzel, Steer, & Beck, 2005). Researchers have suggested that gender differences in the presentations of somatic symptoms may be due to biological or hormonal differences (Wenzel et al., 2005) or differences in social roles or cultural norms between men and women (Silverstein & Lynch, 1998). However, inconsistent findings with respect to gender differences in somatic symptoms were found (Salokangas, Vaahtera, Pacriev, Sohlman, & Lehtinen, 2002), suggesting that the observed gender differences in somatic symptoms may be an artifact of the measurement instruments that have been used. Therefore, although women

may be particularly prone to depression, evidence suggesting gender differences in somatic symptoms of depression has not emerged. In sum, inconsistent research results indicate that further investigation is needed to show whether there is a disparity in the measurement of depression among different racial/ethnic groups.

Measurement Models for Depression

Specific manifestations of depressive symptoms have implications for both assessment and treatment. Presentations of clinical depression or MDD can be heterogeneous, varying greatly from patient to patient. Thus, it is conceivable that patients who have different symptom profiles are likely to have different prognoses and may require different treatment, indicating the potential significance of further examining underlying depression symptoms. For instance, several studies showed that specific depressive symptoms have a strong association with other health outcome variables such as prognosis and survival in CHD (Barefoot, Brummett, Clapp-Channing, et al., 2000; Barefoot, Brummett, Helms, et al., 2000; Holahan et al., 2006). Also, a few studies showed that racial/ethnic groups have reported different mean levels of depression based on specific depressive symptom factors (Devins, Orme, Costello, & Binik, 1988; Martens et al., 2006). Research has also demonstrated that treatment can affect some specific depressive symptoms but not others (Sullivan et al., 2009). These research findings suggest that proper assessment and diagnosis of clinical depression involves consideration of differences in racial/ethnic groups

with their associated differences in manifestations of depressive symptoms. The assumption that depressive symptoms are measured similarly across racial/ethnic groups needs to be evaluated.

Several instruments have been developed to screen individuals for the presence of depressive symptoms (Beck et al., 1961; Brink et al., 1982; Hamilton, 1960). The Beck Depression Inventory (BDI) (Beck et al., 1961) is one of the most widely used inventories that assesses self-reported depressive symptoms. Specifically, it is commonly used for assessing post-MI depression (Thombs et al., 2006b; Thombs et al., 2008). Over the years, psychometric properties of the BDI have been established not only for assessing the intensity of depression in psychiatric populations but also for detecting the symptoms of depression in general populations (Beck, Steer, & Garbin, 1988). The 21 items in the BDI were developed based on attitudes and symptoms that Beck found common among the depressed patients and uncommon among the non-depressed individuals. These items of the BDI were found to cover primarily cognitive and somatic symptoms (Byrne, 2005; Carmody, 2005; Contreras, Fernandez, Malcarne, Ingram, & Vaccarino, 2004; Wiebe & Penley, 2005), and thus it has been recommended to use for epidemiological studies of depression and CHD (Davidson et al., 2006).

An extensive literature provides support for the psychometric properties of the BDI (Beck et al., 1988; Carmody, 2005; Wiebe & Penley, 2005). In a review by Beck, et al. (1988), the BDI psychometric properties are shown to be well established and to reflect good to excellent reliability and validity for both clinical

and general populations. Good internal consistency and test-retest reliabilities were also reported, as well as significant correlations between the BDI and other measures used to assess depression (Beck et al., 1988). Furthermore, the psychometric aspects of the BDI translated in Spanish were validated and have been used in prior research (Contreras et al., 2004). The English and Spanish versions of the BDI have comparable validity and reliability in various samples (Bonicatto, Dew, & Soria, 1998; Bonilla, Bernal, Santos, & Santos, 2004; Nuevo et al., 2009).

Several studies have conducted a confirmatory factor analysis of the BDI in clinical populations (Dunkel, Froehlich, Antretter, & Haring, 2002; Johnson, DeLuca, & Natelson, 1996; Miles et al., 2001; Morley, Williams, & Black, 2002), and a general two-factor model has emerged from these studies. The first fourteen BDI (1-14) items represent cognitive-affective symptoms of depression. The second factor, which included the last seven BDI items (15-21) represent somatic or vegetative symptoms of depression in all studies. Overall, the results provided evidence of a strong general factor, with two inter-correlated specific factors: cognitive and somatic. In light of these studies, the two factors of the cognitive and somatic BDI model were used as indicators of an underlying disorder of clinical depression in the present study. Specifically, the primary purpose of the current study was to utilize the two-factor BDI model (i.e., cognitive and somatic) derived from the literature to test whether the BDI items measure the two depressive factors or symptoms similarly in a large sample, including Hispanic, non-Hispanic Black, and non-Hispanic White post-MI patients.

Establishing measurement invariance of depression is necessary for racial/ethnic group comparisons. Measurement invariance refers to the same factor structure and items onto the factor (i.e., configural invariance), similar item thresholds (i.e., scalar invariance), and same item loadings (i.e., metric invariance) in the measurement model across groups. Research studying measurement invariance of the frequently-used BDI across racial/ethnic groups is limited. Two studies have compared the English and Spanish versions of the BDI measure and analyzed the BDI items for bias between Spanish and English-speaking patients to determine the measurement equivalence (Azocar, Areal, Miranda, & Munoz, 2001; Penley, Wiebe, & Nwosu, 2003). Their results supported measurement equivalence of the BDI model, but one study (Azocar et al., 2001) indicated that compared to non-Hispanic Whites, Hispanics are more likely to endorse items associated with tearfulness and punishment, and less likely to endorse the item reflecting inability to work. Another study evaluated the psychometric properties of the BDI in a sample of low-income non-Hispanic Blacks and supported measurement invariance of the two-factor (i.e., cognitive and somatic) BDI model (Grothe et al., 2005). Their results also showed that non-Hispanic Blacks were less likely to endorse the item reflecting suicidal thoughts than non-Hispanic Whites.

In sum, research examining racial/ethnic group differences in depression manifestations is limited. Studies that reported various prevalence rates of depression across racial/ethnic groups suggest that racial/ethnic groups may manifest depression or exhibit depressive symptoms differently. However, these

studies that compare racial/ethnic groups are also limited and do not include large numbers of non-Hispanic Blacks and Hispanics. The current study addresses this limitation by including a diverse sample of individuals with a range of depression levels.

Rationale

Accurate assessment of depression among cardiac patients has significant implications for understanding the commonalities and distinctions of depression among non-Hispanic Blacks, non-Hispanic Whites, and Hispanics and may also be crucial for treatment considerations. It is important for researchers and clinicians to fully understand the relationship between racial/ethnic differences and depression symptoms in order to consider different treatment options. Before researchers can make valid inferences about racial/ethnic differences in depression, measurement invariance across racial/ethnic groups must be established. Most studies of race/ethnicity and depression assessment address differences in two groups, either non-Hispanic Blacks compared to non-Hispanic Whites (Cornelius, Fabrega, Cornelius, Mezzich, & Maher, 1996; Neighbors, Njai, & Jackson, 2007; Perl et al., 1989) or Hispanics compared to non-Hispanic Whites (Golding & Aneshensel, 1989; Golding, Aneshensel, & Hough, 1991). These studies have provided some evidence supporting measurement invariance of various depression scales such as BDI, CES-D, and Geriatric Depression Scale between Hispanic and/or non-Hispanic Black and non-Hispanic White community samples.

However, the literature of measurement invariance of depression across racial/ethnic groups has substantial limitations. Racial/ethnic minorities groups are under-represented in the research, especially Hispanics. Much of the research on depression or depression and treatment effect has focused on non-Hispanic Whites (de Jonge et al., 2006). By incorporating Hispanics as well as non-Hispanic Blacks in our sample and testing racial/ethnic group differences in depression presentations, this present study aimed to contribute more information about this emerging multi-ethnic population. The present study compared non-Hispanic Black, Non-Hispanic White, and Hispanic cardiac patients to assess measurement invariance of a commonly-used inventory (i.e., BDI) for post-MI depression, focusing specifically upon depressive symptomatology. The present study aimed to extend the literature on race/ethnicity differences in depression symptoms.

Limited studies have attempted to evaluate the adequacy of current depression measures for cross-cultural comparisons in depression. In particular, little is known about specific depression presentations among the post-MI survivors. Only a few studies have examined symptom dimensions of post-MI depression (de Jonge et al., 2006; Fraguas et al., 2007; Linke et al., 2009; Martens et al., 2010) , but none has tested the BDI depression model among racial/ethnic minority MI patients. In order to distinguish high risk groups for adverse cardiac outcomes, it is important to investigate depressive symptoms expressed in the context of CHD and measured equally across different racial/ethnic groups. A better understanding of the psychometric quality of the

BDI aids in the examination of racial/ethnic differences in the manifestations and presentations of depression in CHD and the identifications of high risk subgroups.

In addition, this present study aimed to investigate the similarity of the item loadings, thresholds, and scales for the depressive symptoms across a six-month time frame, from the baseline to post-treatment. This project utilized a longitudinal design that began in the critical period after MI diagnosis and followed participants up to six months post-treatment in order to effectively control for baseline depression. The design was used to help in the interpretation of specific depressive symptoms on cardiac prognosis and possibly improve depressive symptoms in CHD. Health disparities exist between non-Hispanic Whites and racial/ethnic minority groups in depression prevalence rates and treatment effect on depression (Kessler, et al., 2003; Ohayon, 2007; Schneiderman, et al., 2004; Waldman et al., 2009; Williams et al., 2007), and thus, the present study also aimed to investigate the treatment effect on depressive symptoms across three racial/ethnic groups.

Overall, this current project aimed to address aforementioned limitations and to bridge gaps in the literature. Although the overarching aim of the present study was to examine racial/ethnic differences in depression presentation and treatment effect on depression, gender was included as a factor in the depression model in light of well-established findings on gender variations in prevalence rates of depression. In addition, interactive effects of race/ethnicity

and gender were examined. Additional covariates including baseline depression levels, baseline antidepressant medication use, education, income, and employment were also controlled in the analyses.

Current Study

This study assessed whether there were group differences in the BDI depression symptoms and treatment effect on the BDI depressive symptoms in a large clinical sample of non-Hispanic Black, non-Hispanic White, and Hispanic cardiac patients with MI. This study has four aims.

Aim 1. To compare the baseline BDI total scores among non-Hispanic Black, non-Hispanic White, and Hispanic MI patients in order to examine potential group differences in levels of depression.

Hypothesis 1a: It was predicted that at baseline, there would be significant differences in baseline depression levels across the three racial/ethnic groups.

Hypothesis 1b: It was expected that non-Hispanic Whites would have baseline BDI scores that were significantly lower than the other two racial/ethnic minority groups.

Hypothesis 1c: It was expected that Hispanics would have baseline BDI scores that were significantly higher than the other two racial/ethnic groups.

Hypothesis 1d: It was expected that women would have baseline BDI scores that were significantly higher than men and that gender would have a significant effect on the association between race/ethnicity and the baseline BDI total scores.

Hypothesis 1e: It was expected that there would be a significant interaction between gender and race/ethnicity and that racial/ethnic minority women would have baseline BDI scores that were significantly higher than White men.

Aim 2. To test a two-factor BDI model in order to determine whether the cognitive and somatic items measured the two factors of depression comparably among the three racial/ethnic groups of MI patients.

Hypothesis 2a: It was predicted that at baseline, the cognitive and somatic items would measure the two BDI factors comparably across the three racial/ethnic groups. Factor loadings and thresholds of the cognitive and somatic items for the cognitive and somatic BDI factor at baseline would be comparable across the three racial/ethnic groups.

Hypothesis 2b: It was expected that Hispanics would have a significantly higher differentiation of the somatic from cognitive symptoms than non-Hispanic Whites or non-Hispanic Blacks do at baseline. The correlation between baseline somatic and cognitive factor would be significantly different across the three racial/ethnic groups.

Aim 3. To address if there was a differential treatment effect on cognitive and somatic symptoms at six months after treatment across the three racial/ethnic groups. In effect, it was to investigate whether ENRICHD treatment improved cognitive and somatic symptoms across racial/ethnic groups.

Hypothesis 3a: It was predicted that there would be significant group differences in treatment effect on the cognitive depressive symptoms across the three racial/ethnic groups at six months post-treatment after controlling for baseline depression, gender, baseline antidepressant use, education, income, and employment.

Hypothesis 3b: It was expected that there would be stronger treatment effect on reducing cognitive symptoms at six months post-treatment among non-Hispanic Whites than the other two racial/ethnic groups and that there would be stronger treatment effect on reducing somatic symptoms at six months post-treatment among Hispanics than the other two racial/ethnic groups after controlling for baseline depression, baseline antidepressant use, gender, education, income, and employment.

Hypothesis 3c: At six-months post treatment, non-Hispanic Whites would show greater treatment effect on overall depression than non-Hispanic Blacks or Hispanics. Non-Hispanic White Males would show the greatest treatment effect on overall depression.

Aim 4. To test the stability of the two-factor BDI model across time from baseline to six months post-treatment.

Hypothesis 4a: It was expected that in the usual care group, the cognitive and somatic items measured the two BDI factors comparably over time between the baseline and at six-months post-treatment. Measurement invariance of the two-factor BDI model over time was expected between the two time points.

Hypothesis 4b: It was expected that in the treatment group, the cognitive and somatic items measured the two BDI factors comparably over time between the baseline and at six-months post-treatment. Measurement invariance of the two-factor BDI model over time was expected between the two time points.

CHAPTER 3

METHODS

Participants

The current study included a subgroup (N=2370) of all patients participating in the ENRICH trial. Figure 1 summarizes the flow of patients through the screening process in ENRICH.

[Insert Figure 1 here]

Overall, the ENRICH screened a total of 33,780 patients, but 31,299 individuals were excluded. For those patients who were excluded, 22,967 patients were not eligible for logistic and/or medical reasons, 1534 patients did not meet the MI eligibility criteria, and 6798 patients did not meet the psychosocial eligibility criteria of clinically-depressed and/or socially-isolated. In the end, a total of 2481 patients met eligibility criteria and were randomized, with 1243 assigned to the usual care and 1238 assigned to the psychosocial treatment group. Among the 2481 patients, a total of 2370 participants (i.e., 467 non-Hispanic Blacks, 1647 non-Hispanic Whites, and 256 Hispanics) were used for the current study, and other ethnic groups were excluded. Out of the 2370 participants included in this current study, 226 non-Hispanic Black, 829 non-Hispanic White, and 126

Hispanic patients were randomized to the Treatment intervention group; whereas 241 non-Hispanic Black, 818 non-Hispanic White, and 130 Hispanic patients were randomized to the Usual Care group (Berkman et al., 2003).

Screening and Recruitment for ENRICHD (The ENRICHD investigators, 2000)

This trial was a multicenter clinical trial of cognitive behavior therapy (CBT) for treating symptoms of depression and perceived low social support in patients that recently suffered an acute MI (The ENRICHD investigators, 2000, 2001). All ENRICHD research protocols and procedures were approved by the Institutional Review Board at each institution and followed the guidelines by the Health Insurance Portability and Accountability Act (HIPAA). The eight participating research centers for the trial were Duke University, Rush-Presbyterian-St. Lukes Medical Center, Stanford University, University of Alabama at Birmingham, University of Miami, University of Washington, Washington University, and Yale/Harvard Universities. The original ENRICHD trial ran from October 1996 to October 1999 and included a total of 2481 patients with an acute MI. In order to be medically eligible for participation in ENRICHD, patients had to have more than two times the upper normal limit for cardiac enzyme measurements. Also, all eligible patients had evolving ST-T changes and/or new Q waves on the electrocardiography (ECG) or symptoms similar to an acute MI. Patients were excluded from participation if they had any one of the following: a) life-threatening non-cardiac conditions, b) physical or logistical limitations, c) major psychiatric co-morbid illness(s) other than depression, d)

currently enrolled in a conflicting research protocol, e) MI symptoms subsequent to a procedure such as percutaneous transluminal coronary angioplasty (PTCA), coronary artery bypass grafting (CABG), f) declined informed consent, g) incomplete screening instruments or visits, h) currently on antidepressant medications or highly suicidal, and i) unapproachable for treatment and/or follow-up visits (i.e., relocation or death). One thing to note is that from October 1996 to April 1998, patients on antidepressant medications were excluded from participation (ENRICHD, 2003). After April 1998, the protocol was changed to allow enrollment of patients who took an antidepressant medication for longer than 14 days but remained clinically depressed and met eligibility criteria prior to being randomized for the trial (ENRICHD, 2003). Having met the medical eligibility for participation in ENRICHD within the 28 days of the onset of MI, patients were further screened for depression and/or low social support using the Depression Interview and Structured Hamilton (DISH), a semistructured interview based on the items from the Ham-D and the National Institute of Mental Health Diagnostic Interview Schedule developed for patients with CHD in ENRICHD (Freeland, et al., 2002). All patients must have met modified criteria for depression (i.e., symptoms present for at least one week for those patients with a prior depression history and two weeks for those patients without a prior depression history) of the Diagnostic and Statistical Manual of Mental Disorder-IV (DSM-IV) (APA, 2002).

Procedures

The critical period of time for cardiac reinfarction and/or death is during the first six months after an acute MI, and therefore, the majority of the patients with an acute MI were screened for medical eligibility. Patients meeting medical eligibility were provided informed consent for participation in the study. After obtaining patients' consent, patients with medical eligibility were further screened for psychosocial eligibility (i.e., depression and/or low social support). Patients who met both medical and psychosocial eligibility were randomly assigned to either a Cognitive Behavioral Treatment (CBT) group or a usual care (UC) group. The CBT aims to change participants' distorted thoughts and maladaptive behaviors. Patients in both the CBT and UC groups received the American Heart Association's Active Partnership health education booklet for reference.

Individual Therapy. The CBT treatment was aimed at improving the patients' clinical depression and increasing their low levels of social support. Participating cardiac patient in the treatment group received a psychosocial intervention that was based on Beck's CBT (Beck, Rush, Shaw, & Emery, 1979; Beck, 1995). Individual CBT treatment that targeted specific needs of patients began within a week after randomization, whereas group therapy started after the patient had completed at least three sessions of individual CBT. In usual circumstances, when participants were unable to complete at least three sessions within six months, they were enrolled in a group as soon as one was available. Some patients continued in individual CBT while in group therapy;

others discontinued individual therapy after starting group therapy. For most of the participants, the maximum length of the CBT was six months. However, the adjunctive pharmacotherapy might have continued for one year. Patients who required maintenance pharmacotherapy beyond one year were referred to their physicians.

Tailoring to depressed participants, individual therapists employed a combination of behavioral, cognitive, and social techniques. In treatment, individual therapists began by establishing a supportive therapeutic relationship with the participants to provide empathy and emotional support. Through a clinical diagnosis of participant's social support deficits, therapists determined whether the participants were associated with maladaptive cognition, poor communication skills, social deficits, or actual social isolation. Thus, treatment for patients with low social support was tailored to match a participant's needs. For instance, participants may have brought a family member or friend along to individual treatment. For participants suffering from both depression and low perceived social support, a combination of various therapeutic techniques was in use for treatment.

Adjunctive Pharmacotherapy Treatment. After being randomly assigned to the treatment intervention group, participants completed a clinical evaluation when the interventionist re-administered the Depression Interview and Structured Hamilton (DISH). If patients were found to exhibit severe depression or to be unresponsive to CBT, adjunctive pharmacotherapy (i.e., Sertraline) was also

initiated unless contraindicated. In particular, clinically depressed patients were required to consult with psychiatrists for medication after five weeks of receiving CBT, especially those who scored higher than 24 on the 17-item Hamilton Depression Rating Scale (HAM- D), or who showed less than 50% reduction on the BDI after 5 weeks, or who scored greater than 20 on the HAM-D within 12 months of randomization. Implementation of the ENRICHD pharmacotherapy protocol was initiated for severely depressed post-MI patients.

Group therapy. Participants assigned to the treatment intervention group were also referred to group therapy as soon as they had completed a minimum of 3 individual sessions. Groups consisted of at least 3 participants. The group therapy protocol included not only the CBT techniques but also didactic instructional and supportive or expressive therapy. In group therapy sessions, participants acquired skills for relapse prevention, problem solving, anger management, and relaxation. The goal of the group therapy was to continue to reduce depression and to increase perceived social support beyond levels achieved during the individual therapy.

Criteria for treatment termination

The CBT intervention was terminated only under the following conditions: 1) after patients met the ENRICHD criteria for successful termination, or 2) after six months of treatment. Specifically, in order to terminate the treatment, participants must have completed at least six individual or group CBT sessions and demonstrated adequate self-therapy skills for maintaining the gains made in

treatment and preventing and/or coping with relapse. For patients enrolled on the basis of low social support, they must have additionally reported at least a sustainable, supportive relationship and adequate social support on a short form of the Perceived Social Support Scale (PSSS). In addition, patients must have had a total score of seven or less on two consecutive BDI measures, and/or a total score of four or more on two items of the PSSS.

Usual Care (UC). The UC patients received health education on information of CHD and its management. They also received standard medical treatment from their physicians such as routine examination. However, the UC patients received no further contact from study personnel except for follow-up.

Psychosocial Measures

At the baseline visit after enrollment, eligible participants completed questionnaires regarding their demographic information and medical history, and underwent physical examinations. These participants also filled out psychometric questionnaires assessing depressed affect and levels of social support. Follow-up assessments began six months after treatment and annually thereafter, which included a medical history, physical examination, resting ECG to detect the possibility of an acute MI, the BDI and HAM-D to assess depressive symptoms, as well as the PSSS evaluate perceived social support. The assessments and interviews of the DISH were administered by trained research nurses, who were evaluated by trial psychiatrists and psychologists who

administered the Structured Clinical Interview for DSM-IV (SCID) (Freedland et al., 2002). In ENRICH, the correlation between the clinicians and research nurses' diagnoses was 93% in agreement.

Depression. The original BDI (Beck et al., 1961) was administered to all participants to assess depressive symptomatology. The BDI is a standardized inventory with strong reliability and validity established in the literature (Beck et al., 1988; Contreras et al., 2004; Davidson et al., 2006). It is a 21-item questionnaire with a 4-point scale based on the patient's self-report of the mood over the past week. Each item has a value of zero to three for its answer. The total score is summed up to compare to a scoring key to determine the severity of depression levels. Higher total scores are indicative of more depressive symptoms. The standard cut-offs include a total score of 0 to 9 (not depressed), a total score of 10 to 18 (mild to moderate depression), a total score of 19 to 29 (moderate to severe depression), and a total score of 30 to 63 (severe depression). Reliability estimates of the BDI range from .48 to .90 (Beck, Steer, Garbin, 1988; Lightfoot and Oliver, 1985; Zimmerman, 1986), showing higher coefficient alphas were reported among clinical populations. The Chronbach's Alpha for the BDI at baseline is .82 in our sample. Appendix 1 displays the 21 BDI items.

For the Hispanic participants, all inventories were translated into Spanish and back-translated into English to ensure an accurate translation. Patients

participated in their preferred language, mostly English. The interviewers and therapists for Hispanic patients were bilingual.

Follow-up Visits

All participants were followed six months and 18 months after randomization and annually thereafter up to three years through the duration of the original study. However, only data from baseline and six month post-treatment will be used in the current analysis because the intervention ended after six months of treatment. A brief medical history, physical examination, and resting electrocardiogram to detect potentially unrecognized cardiac events were conducted during a follow-up assessment. All psychosocial assessments, including the BDI, measured at baseline were repeated at all follow-up visits. Potential end points were identified primarily during all follow-up visits and phone calls. Cardiac events were also identified during all routine hospital examinations or by physicians.

Statistical Analyses

Statistical Analysis Systems (SAS Institute Inc., 2000-2004) version 9.1 was used to perform descriptive statistics for demographic and other baseline variables, including age, gender, treatment group, marital status, education, living arrangement, psychosocial risk factors, and antidepressant medication prescribed at baseline. The first aim of this current study was to examine group differences in depression at baseline, specifically the BDI total scores, among

non-Hispanic Black, non-Hispanic White, and Hispanic MI patients. The second aim was to investigate if the two factor BDI model is comparable for non-Hispanic Black, non-Hispanic White, and Hispanic patients at baseline as well as six months post-treatment. The third aim was to test the stability of the baseline BDI model across time within the treatment and usual care groups. The last aim was to examine racial/ethnic differences in treatment effect on cognitive and somatic symptoms six months post-treatment after controlling for baseline depression.

Aim 1: Group Differences in the Baseline BDI Scores

For the first aim of group differences in depression, a 2 X 3 factorial analysis of variance (ANOVA) was performed using SAS version 9.1 to test whether the baseline BDI means were equal across the three racial/ethnic groups. Gender and race/ethnicity were used as two independent variables and the baseline BDI total score was included as the dependent variable. The inclusion of gender increased statistical power because it accounted for some of the variability in the BDI scores and allowed for a test of interaction. In the presence of a significant interaction of gender and race/ethnicity, simple main effect tests were performed as follow-up tests for gender and for race/ethnicity separately. Tukey's Honestly Significant Difference (HSD) comparison tests were conducted to assess the nature of the significant interaction.

General Approach for Aims 2, 3, and 4

To investigate the second, third, and fourth aims, confirmatory factor analysis (CFA) models and structural equation modeling were performed using Mplus version 6 (Muthen & Muthen, 2010), explicitly modeling the BDI items as categorical data for all three aims and with an intent-to-treat approach for aim 4. Specifically, the analyses for Aim 2 involved the following statistical procedures: 1) establishing a well-fitting factor model of the BDI at baseline separately for non-Hispanic Blacks, non-Hispanic Whites, and Hispanics, and 2) testing for measurement invariance of the cognitive and somatic latent factors among the three racial/ethnic groups. When measurement invariance across the three racial/ethnic groups was not supported, additional statistical analyses were conducted to test metric and scalar invariance between pairs of racial/ethnic groups. In addition, analyses were conducted to test invariance in the variances and covariance of the two latent factors between pairs of racial/ethnic groups. For Aim 3, analyses were performed to test treatment effect on the cognitive and somatic depressive symptoms at six months post-treatment while controlling for baseline depression across the three racial/ethnic groups. For Aim 4, specific statistical analyses tested measurement invariance of the well-fitting BDI model over time from the baseline to six months post-treatment for the treatment and usual care groups only in non-Hispanic White patients.

Assessing the adequacy of the measurement models. Chi-square tests were performed to assess model fit. However, obtaining a non-significant chi-

square test becomes increasingly unlikely with a large sample size (Kline, 1998). Thus, other multiple commonly-used fit index measures were used to assess model fit in the analyses. For instance, the comparative fit index (CFI) and the Tucker-Lewis index (TLI) (Gottlieb et al., 2004), also known as the non-normed fit indices, assess fit relative to a null model using non-centrality parameters (Bentler, 1988). The range of the CFI and TLI indices is between zero and one. The CFI and TLI values of 0.95 or greater indicate well fitting models (Hu & Bentler, 1999), whereas the values from 0.90 to 0.94 indicate acceptable model fit. The root mean square error of approximation (RMSEA) is a standardized measure of lack of fit of the hypothesized model in the population (Browne & Cudeck, 1993). RMSEA shows fit per degree of freedom of the model, with a cut-off value of 0.08 indicating acceptable fit and values of 0.05 or lower indicating well-fitting models (Hu & Bentler, 1999). The weighted root mean square residual (WRMR) refers to the (weighted) average differences of the sample and model estimated variances and covariances. WRMR is used with categorical data, with a cutoff value close to 1.0 indicating well fitting models (Yu, 2002).

Aim 2: Two Factor Baseline Depression Model

It is important to establish a well-fitting baseline model separately for each group before conducting the test for measurement invariance (Byrne, Shavelson, & Muthen, 1989). Statistical analysis used a CFA to assess the goodness of fit of the original two factor BDI model (Beck et al, 1996; Contreras, et al., 2004) in

each racial/ethnic group, explicitly modeling all 21 items as categorical data (Thombs, et al., 2008). Based on the literature (Beck, et al., 1988; Contreras, et al., 2004), the first factor contained mostly cognitive items, whereas the second factor contained mostly somatic items. In particular, 14 items (i.e., Item 1 through 14) were specified to load on the cognitive factor, and seven items (i.e., Item 15 through 21) were specified to load on the somatic factor. All 21 items from the BDI were specified as categorical variables in model testing.

To improve the goodness of fit for the data, this original 21-item BDI model was first modified in the non-Hispanic Whites based on theory-driven model modifications, guided by previous BDI research (Beck, Steer, & Garbin, 1988; Contreras, Fernandez, Malcarne, Ingram, & Vaccarino, 2004). After modification, the two-factor BDI model that best fit non-Hispanic Whites was tested in non-Hispanic Black and Hispanic groups separately. If the two-factor BDI model showed acceptable fit in each group, factorial invariance was considered.

A multiple-group CFA was conducted to test further for metric and scalar invariance across all groups. In one model, the item factor loadings and thresholds were freely estimated across all groups for all but the first item. In the other model, all item factor loadings and thresholds were constrained to be equal across all groups. A chi-square difference (χ^2 diff) test was used to determine whether equality constraints for the factor loadings and thresholds resulted in a significant increase in chi-square. The chi-square value and degrees of freedom of the freely estimated model were subtracted from the chi-square value and

degrees of freedom of the constrained model. If the χ^2 diff test value was significant, the model constraining factor loadings and thresholds to be equal significantly worsened the fit of the model.

Lastly, group differences in variability and covariability of the two latent factors specified in the BDI model were examined. Means, variances, and covariance of the latent cognitive and somatic BDI factors at baseline were also compared between groups. In addition, factor loadings, intercepts, or thresholds of the items were inspected in each group. Furthermore, gender effect was investigated as a covariate in its associations with the two BDI latent factors at baseline.

Aim 3: Treatment effect on Depressive symptoms

To test the third aim that examined whether there were differential effects of treatment on depressive symptoms across the three racial/ethnic groups, a structural equation modeling analysis was performed (Kline, 2005). Figure 2 shows the SEM testing the treatment effect on depressive symptoms controlling for baseline depression, gender, income, education, employment 3 months before MI, and antidepressant use at baseline.

[Insert Figure 2 Here]

A well-fitting treatment model was initially estimated and tested separately for each racial/ethnic group. To compare the treatment effect on depression at six-months post-treatment across racial/ethnic groups, chi-square difference testing

for multiple group comparisons was performed to test the model with an equally constrained path for each of the two latent cognitive and somatic factors at six month post-treatment and treatment in all pairs of the groups. In these analyses, the best-fitting model was included where invariant items were constrained equally and variant items were freely-estimated across all pairs of the groups. In the SEM model testing, each of the two latent factors at six month post-treatment was regressed on treatment, the two latent depression factors at baseline, use of antidepressants at baseline, gender, education, income, and employment in the SEM model. Measurement errors of the indicators at baseline and six months were also correlated in the model. Follow-up tests were also conducted to examine whether there were differential effects of treatment on the BDI total score at six-months post-treatment across the three racial/ethnic groups, after controlling for the baseline BDI score, use of antidepressants at baseline, gender, education, income, and employment three-months prior to MI.

Aim 4: Stability of the BDI Model over Time (Only Non-Hispanic Whites)

To test the hypotheses of the last aim examining measurement invariance of the BDI model over time, between baseline and six-months post-treatment, the best fitting model obtained from Aim 2 was used. To test the stability of the BDI model over time, the baseline model was replicated at six months post-treatment, separately for Usual Care and Treatment groups. If the model at baseline replicated at six months, a CFA that included the baseline and six-months post-treatment BDI models was then conducted to test whether the cognitive and

somatic indicators had similar loadings and thresholds over time. Measurement errors of the indicators at baseline and six months were also correlated in the model. Furthermore, the chi-square difference statistic was performed to compare the unconstrained and constrained models for factor loadings and thresholds between the baseline and six-month depression models, separately for Usual Care and Treatment groups.

CHAPTER 4

RESULTS

Sample Characteristics

A total of 2370 participants (44% female) were included in this study.

Table 1 presents the key demographic characteristics by racial/ethnic groups of the sample, including age, gender, treatment, marital status, education, income, employment 3 months before MI, living arrangement, psychosocial risk factors, and antidepressant medication prescribed at baseline.

[Insert Table 1 Here]

Overall, non-Hispanic Blacks were approximately five years younger than non-Hispanic Whites or Hispanics. The non-Hispanic Black group had significantly more female (60%) than Hispanics (38%) or non-Hispanic Whites (41%). Non-Hispanic Whites (58%) were more likely to be married than non-Hispanic Blacks or Hispanics (both 49%). More non-Hispanic Whites (77%) completed high school or college than non-Hispanic Blacks (59%) or Hispanics (57%) did. Out of the 2370 participants included in this current study, 143 non-Hispanic Blacks, 408 non-Hispanic Whites, and 60 Hispanics were not clinically depressed but socially isolated; whereas 156 non-Hispanic Blacks, 536 non-Hispanic Whites, and 125 Hispanics were both clinically depressed and socially isolated.

Approximately 7% of the non-Hispanic Whites were prescribed antidepressant medication at baseline, compared to 4% of the Hispanics and 2.4% of the non-Hispanic Blacks. Due to attrition (i.e., death, cancellation, or inability to contact) after randomization, a total of 39 participants (10 non-Hispanic Blacks, 3 Hispanics, and 26 non-Hispanic Whites) had missing data on baseline BDI. Considering the intention to treat analysis, complete data on all randomized participants including these 39 participants were included in the analyses for Aims 2, 3, and 4.

Aim 1: Examine Group Differences in the BDI Total Scores

BDI Total Scores. A 2 X 3 factorial Analysis of Variance (ANOVA) was performed to examine the gender effect and its interaction with race/ethnicity on the baseline BDI total score. Table 2 displays the cell means of the baseline BDI total scores by gender and racial/ethnic subgroups and the ANOVA table.

[Insert Table 2 Here]

The results indicated that there was a significant main effect for race/ethnicity [$F(2, 2325) = 34.10, p < .001$]. A significant main effect was also observed for gender. Females ($M = 17.29$) scored significantly higher on the baseline BDI total scores than males ($M = 14.52$), [$F(1, 2325) = 68.69, p < .001$]. A significant gender by race/ethnicity interaction was shown [$F(2, 2325) = 5.93, p < .01$].

Figure 3 displays the interaction of gender and race/ethnicity on the BDI total scores at baseline.

[Insert Figure 3 Here]

For follow-up tests, simple main effect tests of race/ethnicity and gender were conducted separately to explore the nature of the significant interaction. The result of ANOVA for ethnicity indicated that the BDI scores at baseline were significantly different across all three racial/ethnicity groups for women [$F(2, 1041) = 25.63, p < .001$] and for men [$F(2, 1288) = 8.15, p < .001$], at the $p < .01$ level. To further determine how the three racial/ethnic groups differed from one another, a Tukey's HSD Post Hoc Analysis was conducted to compare pairs of racial/ethnic groups for each gender separately. The post-hoc comparisons indicated that for men, Hispanics ($M = 16.88, SD = 8.73$) had a significantly higher BDI score at baseline than non-Hispanic Blacks ($M = 14.28, SD = 8.94$) or Whites ($M = 14.17, SD = 7.42$). However, no significant difference was observed in the baseline BDI scores between the non-Hispanic Black and White men. Similarly, for women, Hispanics ($M = 23.18, SD = 9.77$) had a significantly higher BDI score at baseline than non-Hispanic Black ($M = 16.83, SD = 8.39$) or White ($M = 16.65, SD = 8.22$). However, no significant difference was observed in the baseline BDI scores between the non-Hispanic Black and White women.

In addition, the simple main effect of gender on the baseline BDI score was tested for each racial/ethnic group separately. Within each racial/ethnic group, women had a significantly higher BDI score at baseline than men for non-Hispanic Blacks, [$t(455) = 3.09, p < .01$], Hispanics [$t(251) = 5.32, p < .001$], and

non-Hispanic Whites [$t(1619) = 6.32, p < .001$], at the $p < .01$ level. Based on the magnitude of the means, the gender difference was largest for the Hispanic subgroup.

Aim 2: Establish a Two-Factor Baseline Depression Model

Confirmatory Factor Analysis (CFA)

Starting with non-Hispanic Whites in a single group analysis, the CFA replicating the original two-factor BDI model (i.e., 14 cognitive items and 7 somatic items) indicated a poor fit at baseline [$\chi^2(131) = 957.51, p < .0001, CFI = .85, TLI = .92, RMSEA = .06, \text{ and } WRMR = 1.90$]. Guided by previous BDI research (Beck, et al., 1988; Contreras et al., 2004), multiple sets of CFAs were conducted by eliminating or adding items that could be loaded on either the cognitive or somatic factor in order to obtain desirable model fit for the non-Hispanic Whites. The best model fit among the CFA results for non-Hispanic Whites indicated that a total of nine items (e.g., Items 1, 2, 4, 10, 11, 12, 13, 20 and 21) should be cross-loaded onto both the cognitive and somatic factors and that Item 19 (i.e., Weight loss) should be removed from the model. Among all tested CFA models, this best-fitting two-factor BDI model was further applied to non-Hispanic Blacks and Hispanics separately. The model fit was improved in a single group analysis, separately for non-Hispanic Whites [$\chi^2(160) = 670.16, p < .001, CFI = .95/ TLI = .94, RMSEA = .05, \text{ and } WRMR = 1.42$], non-Hispanic Blacks [$\chi^2(160) = 298.74, p < .001, CFI = .96/ TLI = .95, RMSEA = .04, \text{ and } WRMR = .93$],

or Hispanics [$\chi^2(160) = 301.61, p < .001, CFI = .91/ TLI = .89, RMSEA = .06,$ and $WRMR = 1.00$]. The final model indicated good model fit for non-Hispanic Whites and Blacks, but acceptable model fit for Hispanics. Table 3 displays the unstandardized factor loadings for the two latent factors in each group separately.

[Insert Table 3 Here]

Measurement Invariance in Multiple Group Analysis

Three Group Analysis

After a best-fitting two-factor BDI model was established for each group separately, multiple-group analysis was performed to test for measurement invariance across all three racial/ethnic groups. However, the result of the Chi Square difference test with the equality constraints on all factor loadings and thresholds did not support complete scalar and metric invariance across three racial/ethnic groups [$\Delta\chi^2 (54) = 144.43, p < .001$].

To further examine racial/ethnic group invariance, follow-up analyses were conducted to compare two groups at a time. Thus, a series of CFA and Chi Square difference tests were performed constraining the factor loadings and thresholds equal between pairs of groups. The results of two group analyses also failed to support complete measurement invariance. Complete measurement invariance was not supported between Hispanics and

Non-Hispanic Whites [$\Delta\chi^2(27) = 62.38, p <.001$], between Blacks and Non-Hispanic White [$\Delta\chi^2(27) = 72.87, p <.001$], or between Blacks and Hispanics [$\Delta\chi^2(27) = 105.03, p <.001$].

Therefore, models with and without equality constraints for specific groups of items were tested separately in multiple group analysis, including only pure cognitive items (i.e., Item 3, 5, 6, 7, 8, 9, and 14), only pure somatic items (i.e., Item 15, 16, 17, and 18), or cross-loaded items (i.e., Item 1, 2, 4, 10, 11, 12, 13, 20, and 21). Table 4 summarizes unstandardized factor loadings of invariant and variant items between Hispanics and non-Hispanic Whites. Table 5 shows unstandardized factor loadings of invariant and variant items between non-Hispanic Blacks and Whites. Table 6 displays unstandardized factor loadings of invariant and variant items between non-Hispanic Blacks and Hispanics.

[Insert Table 4, 5, and 6 Here]

Two Groups: Hispanics and Non-Hispanic Whites

Pure Cognitive Items. With equally constraining factor loadings and thresholds for only pure cognitive items (i.e., Item 3, 5, 6, 7, 8, 9, 14), the Chi Square difference test result indicated that these pure cognitive item factor loadings between the two racial/ethnic groups were not comparable [$\Delta\chi^2(6) = 25.02, p <.001$]. However, when the thresholds and factor loading of Item 14 (Body Image) was freely estimated, metric and scalar invariance was reached

[$\Delta\chi^2(5) = 10.69, p = .06$]. Because only one of the items was different between groups, the Cognitive factor was considered to have the same meaning for Hispanics and non-Hispanic Whites.

Pure Somatic Items. While equally constraining factor loadings and thresholds for only pure somatic items (i.e., Item 15, 16, 17, 18), the Chi Square difference test result indicated that these pure somatic item factor loadings and thresholds between the two racial/ethnic groups were not comparable [$\Delta\chi^2(3) = 12.01, p < .01$]. However, when the thresholds and factor loading of Item 18 (Loss of appetite) was freely estimated, metric and scalar invariance was reached [$\Delta\chi^2(2) = 2.84, p = .24$]. Because only one of the items was different between groups, the Somatic factor was considered to have the same meaning for Hispanics and non-Hispanic Whites.

Cross-loaded Items. Furthermore, while equally constraining the factor loadings and thresholds for the cross-loaded items (1, 2, 4, 10, 11, 12, 13, and 20), the Chi Square difference test result confirmed metric and scalar invariance, showing that these cross-loaded item factor loadings and thresholds between these two groups were comparable [$\Delta\chi^2(18) = 29.26, p = .05$].

Two Groups: Non-Hispanic Blacks and Non-Hispanic Whites

Pure Cognitive Items. While equally constraining factor loadings and thresholds for only pure cognitive items (i.e., Item 3, 5, 6, 7, 8, 9, 14), the Chi Square difference test indicated that these pure cognitive item factor loadings between the two racial/ethnic groups were not comparable [$\Delta\chi^2(6)=36.58,$

$p < .001$]. However, when the thresholds and factor loading of Item 6 (Sense of punishment), 9 (Self Punitive Wishes) and 14 (Body Image) were freely estimated, metric and scalar invariance was reached [$\Delta\chi^2(3) = 7.29, p = .06$]. Because three of the seven cognitive items were different between groups, the Cognitive factor was considered to have a different meaning for non-Hispanic Blacks and non-Hispanic Whites.

Pure Somatic Items. While equally constraining factor loadings and thresholds for only pure somatic items (i.e., Item 15, 16, 17, 18), the Chi Square difference test result indicated that these pure somatic item factor loadings and thresholds between the two racial/ethnic groups were not comparable [$\Delta\chi^2(3) = 14.57, p < .001$]. However, when the thresholds and factor loading of Item 16 (Sleep disturbance) was freely estimated, metric and scalar invariance was reached [$\Delta\chi^2(2) = 4.02, p = .13$]. Because only one of the items was different between groups, the Somatic factor was considered to have the same meaning for non-Hispanic Blacks and Whites.

Cross-loaded Items. Furthermore, with equally constraining the factor loadings and thresholds for the cross-loaded items (1, 2, 4, 10, 11, 12, 13, and 20), the Chi Square difference test result confirmed metric and scalar invariance, showing that these cross-loaded item factor loadings and thresholds between these two groups were comparable [$\Delta\chi^2(18) = 21.95, p = .23$].

Two Groups: Non-Hispanic Blacks and Hispanics

Pure Cognitive Items. With equally constraining factor loadings and thresholds for only pure cognitive items (i.e., Item 3, 5, 6, 7, 8, 9, 14), the Chi Square difference test indicated that these pure cognitive item factor loadings between the two racial/ethnic groups were not comparable [$\Delta\chi^2(6) = 54.42$, $p < .001$]. However, when the thresholds and factor loading of Item 6 (Sense of punishment), 8 (Self accusations), and 14 (Body Image) were freely estimated, metric and scalar invariance was reached [$\Delta\chi^2(3) = 2.79$, $p = .43$]. Because three of the seven cognitive items were different between groups, the Cognitive factor was considered to have a different meaning for Hispanics and non-Hispanic Blacks.

Pure Somatic Items. While equally constraining factor loadings and thresholds for only pure somatic items (i.e., Item 15, 16, 17, 18), the Chi Square difference test result indicated that these pure somatic item factor loadings and thresholds between the two racial/ethnic groups were comparable [$\Delta\chi^2(3) = 2.67$, $p = .44$]. This suggested that the Somatic factor has the same meaning across Hispanics and non-Hispanic Blacks.

Cross-loaded Items. Furthermore, with equally constraining the factor loadings and thresholds for the cross-loaded items (1, 2, 4, 10, 11, 12, 13, and 20), the Chi Square difference test result showed that these cross-loaded item factor loadings and thresholds between these two groups were not comparable [$\Delta\chi^2(18) = 47.13$, $p < .001$]. However, when the thresholds and factor loading of Item 2 (Pessimism), 4 (Lack of satisfaction), and 12 (Social withdrawal) were

freely estimated for both latent factors, metric and scalar invariance was reached [$\Delta\chi^2(12) = 17.08, p = .15$]. Three cross-loaded items were considered to have different meanings for Hispanics and non-Hispanic Blacks.

In short, the results of the Chi Square difference testing for measurement invariance for the two-factor BDI model indicated that the BDI model was not completely invariant across the three racial/ethnic groups. Findings from additional Chi Square difference testing for measurement invariance of the two-factor BDI model between pairs of racial/ethnic groups showed partial measurement invariance. Specifically, under the cognitive factor, all pure cognitive items measure cognitive depression similarly between Hispanics and non-Hispanic Whites, except for Item 14 (Body image). The cognitive factor was considered different due to variant Items 6 (Sense of punishment), 9 (Self punitive wishes), and 14 (Body image) between non-Hispanic Whites and Blacks. Similarly, the cognitive factor was different because of variant Items 6 (Sense of punishment), 8 (Self accusations), and 14 (Body image) between Hispanics and non-Hispanic Blacks. Under the somatic factor, all four pure somatic items measure depressive symptoms comparably across the groups, except for Item 18 (Loss of appetite) between Hispanics and non-Hispanic Whites and Item 16 (Sleep disturbance) between non-Hispanic Blacks and non-Hispanic Whites. Consistently, all nine cross-loaded items measured both cognitive and somatic symptoms similarly between non-Hispanic Whites and Hispanics, as well as

between non-Hispanic Whites and Blacks, except for Items 2 (Pessimism), 4 (Lack of satisfaction), and 12 (Social withdrawal) between non-Hispanic Blacks and Hispanics.

Baseline Cognitive and Somatic Factor Means

Cognitive and somatic latent factor means at baseline were also compared between all pairs of racial/ethnic groups in multiple group analyses when the loadings and thresholds of the variant items were freely estimated. These results confirmed the hypothesis that there were significant group differences in cognitive factor mean at baseline across groups. However, a significant group difference in somatic factor mean at baseline was only shown between Hispanics and non-Hispanic Whites. Hispanics had a baseline cognitive factor mean that was on average .25 standard deviation units greater than non-Hispanic Whites ($Z = 3.89, p < .001$). Non-Hispanic Blacks had a baseline cognitive factor mean that was on average .11 standard deviation points lower than non-Hispanic Whites ($Z = -2.04, p < .05$). Similarly, Hispanics had baseline cognitive factor mean that was on average .40 standard deviation units greater than non-Hispanic Blacks ($Z = 5.31, p < .001$). The results supported the hypothesis that Hispanics had the highest levels of cognitive symptoms across all three racial/ethnic groups.

With respect to the group differences in baseline somatic depression, the somatic factor mean at baseline among Hispanics was on average .16 standard units greater than that among non-Hispanic Whites ($Z = 2.06, p < .05$). However,

no significant difference was found between non-Hispanic Whites and Blacks ($Z = 1.37, p = .17$), or between non-Hispanic Blacks and Hispanics ($Z = .62, p = .53$). The results did not support the hypothesis that Hispanics had the highest baseline depression level, among all three racial/ethnic groups.

Latent Variable Correlations

Based on multiple group analyses while constraining latent variable covariances to be equal, the results of Chi-Square difference tests showed that the three groups differed in the two latent factor correlations. All correlations between cognitive and somatic factors were positive and small to moderate, ranging from .14 to .46. Contrary to the hypotheses, racial/ethnic minority groups had significantly greater correlations between the two factors than non-Hispanic Whites. The result of the Chi Square difference test [$\Delta\chi^2(1) = 10.75, p < .001$] showed that Hispanics ($\beta = .46, SE = 5.81, p < .001$) had a significantly higher correlation between the somatic and cognitive factors than non-Hispanic Whites ($\beta = .23, SE = 13.28, p < .001$). The Chi Square difference test result [$\Delta\chi^2(1) = 4.32, p < .05$] also revealed that non-Hispanic Blacks ($\beta = .20, SE = 6.08, p < .001$) had a significantly greater factor correlation than non-Hispanic Whites ($\beta = .14, SE = 11.15, p < .001$). However, Hispanics and non-Hispanic Blacks did not significantly differ in the association between the cognitive and somatic factors [$\Delta\chi^2(1) = 2.53, p = .11$].

Gender Effect on Latent Factors at Baseline

To examine gender effects on depressive factors, the two latent cognitive and somatic factors were regressed on gender. Significant gender differences on the baseline cognitive and somatic depression levels were observed. In terms of the cognitive depression level at baseline, Hispanic females reported on average .29 standard deviation units significantly higher than Hispanic males ($Z = 2.56, p < .05$). No gender difference in the baseline cognitive depression levels was found among non-Hispanic Blacks and Whites ($p = .59$ and $p = .16$, respectively). With respect to the somatic depression level at baseline, White women reported on average .43 standard points significantly higher than White men ($Z = 9.11, p < .001$); non-Hispanic Black women reported on average .38 standard deviation units significantly higher than non-Hispanic Black men ($Z = 4.74, p < .001$); and Hispanic women reported on average .69 standard units significantly higher than Hispanic men ($Z = 5.62, p < .001$).

Aim 3: Examine Treatment effect on Depressive symptoms

For non-Hispanic Whites, treatment had a significant effect on reducing the cognitive symptoms ($\beta = -.23, SE = -3.07, p < .01$) and somatic symptoms ($\beta = -.28, SE = -5.27, p < .001$) at six-months post-treatment. Antidepressant use at baseline also had a significant effect on post-treatment symptoms; participants on medication had greater levels of cognitive symptoms ($\beta = .23, SE = 2.05, p < .05$) and somatic symptoms ($\beta = .29, SE = 3.01, p < .01$) than those without medication. Men and women did not differ significantly in cognitive or somatic

symptoms at six-months post-treatment ($p > .05$). Education and employment three months before MI did not significantly affect cognitive or somatic symptoms at six-months post-treatment ($p > .05$). However, income had a significant impact on both cognitive and somatic symptoms at six-months post-treatment. Patients with lower incomes reported greater levels of cognitive symptoms ($\beta = -.07$, $SE = -2.44$, $p < .05$) and somatic symptoms ($\beta = -.19$, $SE = -2.20$, $p < .01$) than those with higher incomes. From the results of the follow-up analyses, treatment did not have a significant effect on reducing the BDI total depression at six-months post-treatment in non-Hispanic Whites ($p > .05$). Gender, income, education, and antidepressant use at baseline did not significantly affect overall depression at six-month post-treatment ($p > .05$). The results did not support the interaction of gender and treatment in non-Hispanic Whites ($p > .05$). However, employment three months before MI had a significant impact on the BDI overall depression at six-months post-treatment. Patients who were not employed three months prior to MI reported greater levels of the BDI total score at six-months post-treatment ($\beta = -2.16$, $SE = -4.22$, $p < .001$) than those who were employed three-months prior to MI.

For non-Hispanic Blacks, treatment had a significant effect on reducing cognitive symptoms ($\beta = -.38$, $SE = -3.11$, $p < .01$) but not somatic symptoms ($p > .05$). Antidepressant use at baseline did not have a significant effect on post-treatment cognitive or somatic symptoms ($p > .05$). Compared to men, women endorsed significantly greater somatic symptoms six-months post-treatment ($\beta = -.28$, $SE = -2.56$, $p < .05$) but not cognitive symptoms ($p = .78$).

Socioeconomic status (i.e., education, employment, and income) did not have an effect on cognitive or somatic symptoms at six-months post-treatment ($p > .05$). From the results of the follow-up analyses, treatment did not have a significant effect on reducing the BDI total depression at six-months post-treatment in non-Hispanic Blacks ($p > .05$). Gender, income, education, employment three months before MI, and antidepressant use at baseline did not significantly affect overall depression at six-month post-treatment ($p > .05$). The results did not support the interaction of gender and treatment in non-Hispanic Blacks ($p > .05$). However, the BDI total score at baseline had a significant impact on the BDI overall depression at six-months post-treatment. Patients who had a higher BDI total score at baseline reported greater levels of the BDI total score at six-months post-treatment ($\beta = .13$, $SE = 2.12$, $p < .05$) than those who had a lower BDI baseline score.

For Hispanics, treatment had a significant effect on reducing cognitive symptoms ($\beta = -.57$, $SE = -3.70$, $p < .001$) and somatic symptoms ($\beta = -.28$, $SE = -2.04$, $p < .05$). Antidepressant use at baseline also had a significant effect on post-treatment symptoms; participants on medication had greater levels of cognitive symptoms ($\beta = .88$, $SE = 2.48$, $p < .05$) and somatic symptoms ($\beta = .98$, $SE = 2.80$, $p < .01$) than those without medication. Men and women did not differ significantly in cognitive or somatic symptoms at six-month-post-treatment ($p > .05$). Socioeconomic status (i.e., education, employment, and income) did not significantly affect cognitive or somatic symptoms at six-months post-treatment ($p > .05$). From the results of the follow-up analyses, treatment did not

have a significant effect on reducing the BDI total depression at six-months post-treatment in Hispanics ($p > .05$). Gender, income, education, employment three months before MI, antidepressant use at baseline, or the baseline BDI total score did not significantly affect overall depression at six-month post-treatment ($p > .05$). The results did not support the interaction of gender and treatment in Hispanics ($p > .05$).

Group Comparisons for Treatment effect

Cognitive Symptoms. The results of Chi-Square different tests indicated that there was a significant group difference in treatment effect on the cognitive depressive symptoms at six months post-treatment between Hispanics and non-Hispanic Whites [$\Delta\chi^2(1) = 4.82, p < .05$], after controlling for baseline depression, gender, and antidepressant use, and socioeconomic status. However, treatment effects on the reduction of cognitive symptoms were not significantly different between non-Hispanic Blacks and Hispanics [$\Delta\chi^2(1) = 1.70, p = .19$] or between non-Hispanic Blacks and Whites [$\Delta\chi^2(1) = 2.50, p = .11$].

Somatic Symptoms. The Chi Square difference tests also supported significant group differences in treatment effect on reducing somatic symptoms at six-months post-treatment between non-Hispanic Blacks and Whites [$\Delta\chi^2(1) = 5.26, p < .02$], after controlling for baseline depression, gender, antidepressant use, and socioeconomic status. However, no significant treatment effect on reducing somatic symptoms at six-months post-treatment was observed between Hispanic and non-Hispanic Whites [$\Delta\chi^2(1) = 0.04, p = .84$], or non-Hispanic Blacks and Hispanics [$\Delta\chi^2(1) = 1.26, p = .26$].

Total BDI Symptoms. The results of Chi-Square different tests indicated that there was no significant group difference in treatment effect on the BDI total depression at six months post-treatment across racial/ethnic groups, between Hispanics and non-Hispanic Whites [$\Delta\chi^2(1) = 1.31, p = .25$], Hispanics and non-Hispanic Blacks [$\Delta\chi^2(1) = .54, p = .46$], or between non-Hispanic Blacks and Whites [$\Delta\chi^2(1) = .20, p = .66$], after controlling for baseline depression, gender, and antidepressant use, and socioeconomic status (i.e., education, income, and employment three months prior to MI). The results did not support the hypothesis that at six-months post treatment, non-Hispanic Whites would show greater treatment effect on overall depression than non-Hispanic Blacks or Hispanics nor that non-Hispanic White Males would show the greatest treatment effect on overall depression. The results did not support a three-way interaction of gender, race/ethnicity, and treatment ($p > .05$).

Aim 4: Test of Stability of the BDI Model over Time (Non-Hispanic Whites)

Based on the findings of partial measurement invariance from Aim 2 and a small sample size for the racial/ethnic minority groups, the test of stability of the BDI model over time was only limited to non-Hispanic White patients. The results of CFA analyses confirmed measurement invariance of the two-factor BDI model over time in the Treatment group, but only partial measurement invariance in the Usual Care group. Table 7 summarizes the factor loadings of the two-factor BDI model that contains data over a six-month time frame in the Usual

Care group. Table 8 displays the factor loadings of the two-factor BDI model that contains data over a six-month time frame in the Treatment group.

[Insert Table 7 and 8 Here]

Usual Care Group (across time). The best-fitting BDI model established at baseline fit the data in the Usual Care group [$\chi^2(160) = 369.82, p < .001, CFI = .95, TLI = .95, RMSEA = .04, \text{ and } WRMR = 1.07$], which was then applied to the six-month data, indicating desirable model fit indices [$\chi^2(160) = 286.22, p < .001, CFI = .98, TLI = .98, RMSEA = .04, \text{ and } WRMR = .88$]. While correlating measurement errors for each item from baseline to six-months, the best fitted two-factor BDI model including both the baseline and six-month data demonstrated configural invariance over a six-month time frame in the Usual Care group. Desirable model fit indices were obtained [$\chi^2(696) = 986.07, p < .001, CFI = .97, TLI = .97, RMSEA = .02, \text{ and } WRMR = .99$].

However, metric and scalar invariance the BDI model with the baseline and six-months post-treatment data was not supported in the Usual Care group, when the loadings and thresholds of all BDI items were constrained equally between the two time points from baseline and six-months post-treatment [$\Delta\chi^2(27) = 61.28, p < .001$]. Therefore, models with and without equality constraints for specific groups of items were tested separately in the following analyses, including only pure cognitive items (i.e., Items 3, 5, 6, 7, 8, 9, and 14), only pure somatic items (i.e., Items 15, 16, 17, and 18), or cross-loaded items (i.e., Items 1, 2, 4, 10, 11, 12, 13, 20, and 21). The Chi Square difference test

results did not support metric and scalar invariance across time when the loadings for pure cognitive items were equally constrained [$\Delta\chi^2(6)=15.29, p <.05$]. However, when the factor loadings were freely estimated for pure cognitive Item 3 (Sense of failure), 8 (Self accusations), and 9 (Self punitive wishes), metric and scalar invariance across time was supported [$\Delta\chi^2(3)=2.79, p =.42$]. On the other hand, the results supported metric and scalar invariance while the factor loadings were equally constrained for pure somatic items [$\Delta\chi^2(3)=2.84, p =.42$].

In addition, the Chi Square difference test result did not support metric and scalar invariance across time when the loadings for cross-loaded items were equally constrained [$\Delta\chi^2(18) = 39.46, p <.01$]. However, when the factor loadings were freely estimated for cross-loaded Item 4 (Lack of satisfaction), 10 (Crying spells), 12 (Social withdrawal), 20 (Somatic preoccupation), and 21 (Loss of libido) on both cognitive and somatic factors, metric and scalar invariance across time was supported [$\Delta\chi^2(9)=15.47, p=.08$].

Treatment Group (across time). The two-factor BDI model used in the Usual Care group was also applied to the non-Hispanic Whites in the Treatment group. At baseline, the BDI model obtained desirable goodness of model fit [$\chi^2(160)= 458.56, p <.001, CFI= .94, TLI= .93, RMSEA= .05, and WRMR= 1.19$]. Similarly, the same BDI model established at baseline was applied to the BDI model at six-months post-treatment, and desirable model fit indices were obtained [$\chi^2(160)= 345.09, p <.001, CFI= .98, TLI= .97, RMSEA= .04, and WRMR= .93$]. While correlating measurement errors for each item from baseline to six-months post-treatment, the two-factor BDI model fit the data across time

[$\chi^2(172) = 448.63, p < .001, CFI = .97, TLI = .96, RMSEA = .03, \text{ and } WRMR = 1.03$].

The Chi Square difference test results confirmed metric and scalar invariance when all 20 item factor loadings were constrained equally across time [$\Delta\chi^2(27) = 38.09, p = .08$].

In short, analysis for BDI model stability testing across time was conducted among non-Hispanic White patients, separately in Usual Care or Treatment group. In the Usual Care group, the cognitive factor was considered having partial invariance across time because three out of seven pure cognitive items (i.e., Items 3, 8, and 9) were variant from baseline to six-months post-treatment. In turn, the somatic factor was considered having the similar meaning across time because all four pure somatic items were consistent over six month period of time. However, in the Treatment group, both the cognitive and somatic factors were considered having the consistent meaning across time, supporting measurement invariance.

CHAPTER 5

DISCUSSION

The present study investigated cross-cultural differences in depressive symptoms and CBT treatment effect on depressive symptoms among non-Hispanic White, non-Hispanic Black, and Hispanic post-MI patients who participated in ENRICHED. The four aims of this study were to examine 1) racial/ethnic differences in the Beck Depression Inventory (BDI) total score at baseline, 2) measurement invariance of the two-factor BDI model including cognitive and somatic symptoms of depression among racial/ethnic groups, 3) racial/ethnic differences in the CBT treatment effect on cognitive and somatic symptoms of depression at six-months post-treatment, after controlling for covariates including baseline depression, baseline antidepressant use, gender, education, income, and employment in the model, and 4) the stability of the two-factor BDI model from the baseline to six-months post-treatment in the Usual Care and Treatment groups (only non-Hispanic Whites). Furthermore, this study tested gender differences in the total BDI scores and depressive symptoms, both at baseline and six-months post-treatment.

The present study was based on the assumption that race/ethnicity considerations are essential to the assessment and treatment of mental health problems (U.S. Department of Health and Human Services, 2001). Past

research suggests that racial/ethnic groups may manifest dissimilar depression symptoms and that current assessment may not measure the underlying symptoms equally across various racial/ethnic groups. These measurement issues may account for existing health disparities and differential treatment outcomes across racial/ethnic groups. The present study included two racial/ethnic minority groups (Hispanic and non-Hispanic Blacks) of post-MI patients, comparing them to non-Hispanic White patients in order to test differences in BDI measurement for depressive symptoms and to assess treatment effect across groups.

The primary objective of this study was to explore one plausible explanation for existing health disparities across racial/ethnic groups by investigating differences in depressive symptoms and treatment effect among cardiac patients of different racial/ethnic groups. In particular, the main body of this chapter discusses the racial/ethnic differences in depression symptoms, using measurement invariance of the BDI model. Distinct and overlapping depressive symptoms that are pertinent to each racial/ethnic group are summarized and discussed. Treatment effect on depression across groups is also discussed. Furthermore, implications for future research and clinical practice based on the findings of the study are addressed. This chapter concludes with the strengths and limitations of the present study.

Racial/Ethnic Differences in Depressive Symptoms

Measurement invariance of the BDI was systematically examined across three racial/ethnic groups of post-MI patients in the present study. Similar to previous research which suggested that somatic and cognitive factors would appear (de Jonge et al., 2006; Dunkel et al., 2002; Miles et al., 2001; Morley et al., 2002), the results from the present study confirmed a two-factor BDI model emerged consistently and had a good model fit among three racial/ethnic groups of post-MI patients. The model fit results indicated that a total of nine items had both cognitive and somatic components and that Item 19 (i.e., Weight loss) should be removed from the model. Specifically, items 1 (Feeling of sadness), 2 (Pessimism), 4 (Lack of satisfaction), 10 (Crying spells), 11 (Irritability), 12 (Social withdrawal), and 13 (Indecision), which were the Cognitive items in the original Beck's model (Beck, et al., 1988), loaded on both Cognitive and Somatic factors. In addition, items 20 (Somatic preoccupation) and 21 (Loss of libido), which were the Somatic items in the original Beck's model (Beck et al., 1988), loaded on both Cognitive and Somatic factors. In the following paragraphs, distinct depressive symptoms presented by each racial/ethnic group and differences between groups are discussed.

Hispanic Patient Group

In Comparison with Non-Hispanic Whites. As predicted, the results demonstrated that Hispanics exhibited significantly higher levels of baseline depression than non-Hispanic Whites. The findings from the current study were

consistent with prior research (Azocar et al., 2001; Holahan et al., 2006; Kim, Chiriboga, & Jang, 2009), which suggested that Hispanics with poor physical health are more likely to report a higher level of depressive symptoms than non-Hispanic Whites. In particular, Holanhan et al. (2006) indicated that having a cardiac illness may cause greater physical limitations and family burdens on Hispanic patients compared to non-Hispanic Whites. In the present study, the Hispanic sample also contained fewer people who were married; they were less educated and had less annual income than their non-Hispanic White counterparts. Hispanic cardiac patients who reported a higher level of depression may deal with a broader spectrum of life challenges and stressors associated with a disadvantaged socioeconomic status than non-Hispanic Whites (Castro, Baezconde-Garbanati, & Beltran, 1985; Williams & Rucker, 2000). However, when controlling for education, income, employment, and marital status at baseline in the relationship between race/ethnicity and depression, the differences in baseline depression remained significant, suggesting that other factors associated with race/ethnicity can play a role in contributing to different levels of depression across groups. In addition, Hispanics showed higher levels of somatic symptoms but similar cognitive depression levels than non-Hispanic Whites. Hispanics also reported a stronger relation between the somatic and cognitive factors than non-Hispanic Whites. This suggests that Hispanics are less likely to differentiate the cognitive from the somatic symptoms than non-Hispanic Whites.

The majority of the items were invariant within the core features of Cognitive and Somatic factors in the BDI model between Hispanics and non-Hispanic Whites, except for the two items concerning “body image” (cognitive) and “loss of appetite” (somatic). The item of “body image” is a more salient cognitive symptom in Hispanics, whereas the item of “loss of appetite” is a more unique somatic symptom in non-Hispanic Whites, which will be discussed in a later section focusing on non-Hispanic Whites. Hispanics who experience depression tend to express concerns about body image which suggests that physical appearance is an important factor to the core belief of Hispanic identity (Rinderle & Montoya, 2008). Part of Hispanic identity may develop through different social experiences related to physical appearance. In addition, disadvantaged social experiences such as prejudice may increase their awareness of physical appearance among Hispanics, and this concern of physical appearance increases especially when they feel depressed. Nevertheless, all the somatic symptoms and cross-loaded items were comparable between Hispanics and non-Hispanic White patients.

In Comparison with Non-Hispanic Blacks. The lack of significant differences in depression levels between Hispanics and non-Hispanic Blacks was not anticipated. In the present study, Hispanics had less annual income than non-Hispanic Blacks, but the two groups did not differ in education, employment, or marital status. It was speculated that low income or financial burdens may be associated with increased depression levels. When controlling for education, income, employment, and marital status in the relationship between

race/ethnicity and depression, the differences in baseline depression remained significant for Hispanics and non-Hispanic Whites, but not for Hispanics and non-Hispanic Blacks, suggesting that other factor (i.e., acculturation levels in minority groups) besides socioeconomic status may also play a role in contributing to different levels of depression among Hispanics.

In addition, Hispanics had greater baseline cognitive depression but reported similar somatic depression, when compared to non-Hispanic Blacks. However, no significant group difference was observed between Hispanics and non-Hispanic Blacks in the association between the Cognitive and Somatic factors, suggesting the link between the Cognitive and Somatic depression is similar for these two groups.

In terms of unique depressive symptoms, comparing to non-Hispanic Blacks, approximately one third of the total items were found variant among Hispanics. Except for the items concerning “sense of punishment” (cognitive), “self accusations” (cognitive), and “body image” (cognitive), four of the seven cognitive items were invariant within the core features of cognitive factor in the BDI model between Hispanics and non-Hispanic Blacks. Again, the cognitive item of “body image” is prominent in Hispanics. Depressed Hispanics are concerned about their body image, which may attribute to the fact that physical appearance and skin tone contributes significantly to their cultural identity (Rinderle & Montoya, 2008). In addition, all somatic items were comparable within the core features of somatic factor in the model. Similarly, four out of nine cross-loaded items were found comparable between these two groups. In

particular, the cross-loaded items of “lack of satisfaction” and “social withdrawal” had stronger correlations with the Somatic factor than the Cognitive factor in Hispanics, whereas these items had greater correlations with the Cognitive factor than the Somatic factor in non-Hispanic Blacks. This finding suggested that Hispanics and non-Hispanic Blacks with high levels of depression were likely to endorse a few different depressive symptoms that were unique to each group.

Non-Hispanic Black Patient Group

Non-Hispanic Blacks did not exhibit differences in baseline depression compared to non-Hispanic Whites. The lack of significant finding was unexpected in the present study because increased depression is usually associated with low income and education. One could argue that Black patients are likely to under-report depression while they feel stigmatized with depression (Bradford, Newkirk, & Holden, 2009; Cooper-Patrick et al., 1997). However, a lack of significant differences in depression between non-Hispanic Black and White cardiac patients has been previously observed (Gavin et al., 2010; Waldman et al., 2009).

Compared to non-Hispanic Whites, a stronger relationship between the somatic and cognitive factors at baseline was found in non-Hispanic Blacks. A significant group difference in the links between the somatic and cognitive factors was found between non-Hispanics Blacks and Whites, but not between non-Hispanic Blacks and Hispanics. In addition, the findings revealed that

racial/ethnic minority groups, both non-Hispanic Blacks and Hispanics, reported comparable levels of depression.

In addition, approximately 16 out of the total 20 items were found invariant non-Hispanic Blacks and Whites. The majority of the items were comparable within the core features of Cognitive and Somatic factors in the BDI model in non-Hispanic Blacks, except for the four items concerning “sense of punishment” (cognitive), “self punitive wishes” (cognitive), “body image” (cognitive) and “sleep disturbance” (somatic). Specifically, in comparison with non-Hispanic Whites, the cognition of a “sense of punishment” in depression plays a crucial role to non-Hispanic Blacks. Cognitive items, such as “sense of punishment” and “self-critical”, are more salient in non-Hispanic Blacks, possibly related to the perception of social discrimination. Discrimination and resulting social disadvantages, unique to non-Hispanic Blacks, may account for their tendency to report feeling punished stemming from their internalized negative identity (Carter & Reynolds, 2011; Chae et al., 2010). Research suggests that compared to non-Hispanic Whites, Blacks are more likely to internalize other’s negative attitude and report feeling punished as a result of unpleasant social situations and discrimination experiences (Chae, Lincoln, Adler, & Syme, 2010). In addition, the somatic item, “sleep disturbance”, is more salient to non-Hispanic Blacks than it is to non-Hispanic Whites. An early study indicated that non-Hispanic Blacks are more likely to report sporadic sleep patterns and durations (i.e., both prolonged and reduced time) when compared to non-Hispanic Whites (Ayalon & Young, 2003).

Furthermore, cross-loaded items such as “pessimism”, “lack of satisfaction”, and “social withdrawal” had greater association with the Cognitive than the Somatic factor among non-Hispanic Blacks, compared to non-Hispanic Whites.

Non-Hispanic White Patient Group

As predicted, non-Hispanic Whites displayed the lowest levels of depression among all three racial/ethnic groups. In particular, non-Hispanic Whites reported a lower cognitive depression at baseline than the two racial/ethnic minority groups. However, it was not expected that non-Hispanic Whites would report a weaker relation between the Somatic and Cognitive factors than the racial/ethnic minorities did at baseline. Significant group differences in the links between the Somatic and Cognitive factors were found between non-Hispanic Whites and Blacks, as well as between non-Hispanic Whites and Hispanics. For non-Hispanic Whites, both cognitive and somatic aspects of depression are important factors attributing to their depression. Compared to the racial/ethnic minorities, the correlation of the two BDI latent factors was relatively small in non-Hispanic Whites, suggesting that the cognitive and somatic factors are two separate constructs. Hence, non-Hispanic Whites are more likely to differentiate cognitive from somatic symptoms than the other two racial/ethnic groups. In comparison to Hispanics, the item concerning “loss of appetite” is a more salient somatic symptom in non-Hispanic Whites who experience depression. It was speculated that non-Hispanic Whites who are

depressed are likely to report loss in appetite, possibly due to reduced food variety and dietary restrictions after having MI.

In comparison with non-Hispanic Blacks, the cognitive items of “feeling suicidal” and “body image” associated with depression are more prominent in non-Hispanic Whites. Non-Hispanic Whites who are depressed were more likely to freely report thoughts of suicide, whereas non-Hispanic Blacks may be less likely to report suicidal thoughts due to stigma. The literature suggests Blacks have a lower suicide rate compared to other racial/ethnic groups in the U.S. (Castle, Conner, Kaukeinen, & Tu, 2011; Spicer & Miller, 2000). Furthermore, non-Hispanic Whites who are depressed may be more likely to endorse the item concerning “body image” than non-Hispanic Blacks. It may be because non-Hispanic Blacks may have different cultural beauty ideals that protect them against disordered eating or concerns about body image (Shuttlesworth & Zotter, 2011).

As discussed earlier, 18 out of the total 20 items were found invariant within the core features of Cognitive and Somatic factors in the BDI model in non-Hispanic Whites and Hispanics, except for the two items concerning “body image” (cognitive) and “loss of appetite” (somatic). Non-Hispanic Whites and Hispanics reported comparable cross-loaded symptoms on the BDI. Compared to non-Hispanic Blacks, sixteen items were found invariant in non-Hispanic Whites. The majority of the items were invariant within the core features of Cognitive and Somatic factors in the BDI model, except for the four items concerning “sense of punishment” (cognitive), “self punitive wishes” (cognitive), “body image”

(cognitive) and “sleep disturbance” (somatic). All three groups reported similar cross-loaded symptoms on the BDI. While the majority of the items were overlapping across groups, few significant differences in the depression symptoms between racial/ethnic minority groups and non-Hispanic Whites were observed predominately in cognitive depression, suggesting that racial/ethnic groups conceptualize and manifest depression comparably with minimal variations.

In short, findings from the present study indicated that a few depressive symptoms were not identical across all three racial/ethnic groups. Variant items were identified within the core features of the underlying Cognitive and Somatic factors in the BDI across racial/ethnic groups, primarily limited to cognitive symptoms such as sense of punishment, self-accusations, self-punitive wishes, and body image. All four pure somatic items measure depressive symptoms comparably across the racial/ethnic groups, except for sleep problems. Although several items are cross-loaded onto both Cognitive and Somatic factors, these cross-loaded items were similar across racial/ethnic groups. These cross-loaded items reflect that some cognitive and somatic symptoms may overlap in the context of post-MI. Thus, evaluation of depressive symptoms among cardiac patients is complicated by that fact that nine items were cross-loaded onto both factors in the current study. It is possible that while screening depression for cardiac patients, there are overlapping features of depression that cannot be categorized as cognitive or somatic on the BDI. The challenge of screening for depression for racial/ethnic minority cardiac patients is to differentiate somatic

from cognitive symptoms of depression. The observed differences across racial/ethnic groups may be partly explained by the fact that because this is a sample of post-MI patients. Some somatic symptoms that post-MI patients experience overlap with the somatic depressive items on the BDI. Thus, somatic depression symptoms are similar across these racial/ethnic groups, whereas few cognitive depressive symptoms distinguish these patients. However, although post-MI patients may experience similar somatic complaints that overlap with somatic depressive symptoms, they may perceive severity and impact of the illness very differently, which contributes to cognitive symptoms.

Implications. Although the BDI has been extensively studied and widely cited in the literature and widely used in clinical settings, a critical gap in this research is that these studies have consisted of a limited number of racial/ethnic minorities (Beck et al., 1988). Studies that examined depression across racial/ethnic groups using the BDI often combine individuals with different racial/ethnic backgrounds into one group because of a small sample size. Implications of the present study raise questions about measuring depression symptoms adequately and appropriately among different racial/ethnic groups of post-MI patients. Partial measurement invariance in the BDI depression model for the present findings can be interpreted as evidence that racial/ethnic minorities have fairly comparable concepts of depression than non-Hispanic Whites, given that the differences are relatively minor compared to the similarities. In a sample of post-MI patients, somatic depressive symptoms may be similar across racial/ethnic groups, but a few cognitive depressive symptoms may be

more prominent and unique to a specific racial/ethnic group. Nevertheless, the majority of the cognitive items on the BDI are comparable across groups.

Findings from the present study also suggest that caution should be exercised when interpreting data gathered from the BDI as it might over-estimate or under-estimate specific depressive symptoms for ethnic/racial minorities. The current findings indicate that compared to depressed non-Hispanic Whites who are likely to report poor appetite, Hispanic patients are likely to report concerns about body image when they are feeling depressed, whereas depressed non-Hispanic Blacks are likely to report feelings of punishment, self-punitive wishes, and sleep disturbance. Racial/ethnic differences in depressive symptoms would be consistent with the notion that concepts of mental health and illness are culturally derived (James & Prilleltensky, 2002). For instance, non-Hispanic Whites who experience depression are likely to report concerns about poor appetite. A speculation may be that the poor appetite was related to decreased food variety as a result of dietary restriction post MI among non-Hispanic Whites. Some studies indicated that low motivation to eat in the elderly was linked to depression, decreased commitment in activities, or dietary change (Donini, Savina, & Cannella, 2003; Engel et al., 2011). Meanwhile, Hispanics who experience depression tend to express concerns about body image which may be linked to physical appearance being an important factor of Hispanic identity (Rinderle & Montoya, 2008). Among non-Hispanic Blacks, stigma about mental health is particularly worse because of discrimination that has deep-rooted effects on their culture and social factors. In the present study, non-Hispanic

Black patients with greater levels of depression were more likely to report feelings of self-punishment or self-punitive thoughts. Discrimination against Blacks and resulting disadvantaged social environment may induce negative feelings and thoughts, which further cause sleep disturbance among non-Hispanic Blacks.

Nevertheless, the BDI is still a very useful tool for screening underlying depression symptoms among post-MI patients, including presence of cognitive symptoms (e.g., negative mood, loss of interest, worthlessness, concentration problems, and suicidal ideation) and somatic symptoms (e.g., fatigue, sleep problems, appetite problems, and psychomotor changes) that vary considerably across racial/ethnic groups. Therefore, screening depression using the BDI requires careful consideration as it may reveal more than merely a total score. Clinicians should continue to screen, assess, and monitor underlying depression symptoms, the cognitive and somatic, among post-MI patients while in the process of treatment.

Stability of the BDI. Furthermore, because current findings suggest racial/ethnic differences in a few depressive symptoms (Aim 2) and because the minority groups have a small sample size, the reliability testing of the BDI model across time in the Usual Care and Treatment groups was limited to non-Hispanic White patients only (Aim 4). The results supported partial measurement invariance of the two-factor BDI model in the Usual Care group, indicating that the cognitive items measure the Cognitive factor differently whereas the somatic items measure the somatic factor comparably across time. In particular, pure

Cognitive items 3 (Sense of failure), 8 (Self accusations), and 9 (Self punitive wishes), as well as cross-loaded items 4 (Lack of satisfaction), 10 (Cry spells), 12 (Social withdrawal), 20 (Somatic preoccupation), and 21 (Loss of libido) were variant across time. On the other hand, in the Treatment group, findings from the present study confirmed measurement invariance of the two-factor BDI model over a six-month time frame. This indicated that the cognitive and somatic items measure depression symptoms similarly between pre- and post-treatment for the participants in the Treatment group.

However, measurement instability of the depression model over time in Usual Care group was not expected. It may be related to substantial improvement in depression found in the Usual Care group, where individuals could spontaneously moderate their experience of depressive symptoms and the manifestation of depression over six months, compared to those in Treatment.

ENRICHD and Treatment Effect

The primary results of ENRICHD indicated that CBT significantly reduced the levels of depression at post-treatment for the depressed participants (Berkman et al., 2003). Previous ENRICHD findings also showed that the association of somatic depression with medical comorbidity is stronger than that is for cognitive depression (Watkins et al., 2003). Similar findings were also observed in prior research studies, showing that somatic depressive symptoms

assessed by the BDI may be strongly related to disease prognosis than cognitive depressive symptoms (de Jonge, 2006; Linke et al., 2009). In addition, White male patients, compared to other subgroups (i.e., female patients and minorities), might have benefited more from the treatment that aimed to improve cardiac mortality and non-fatal MI (Schneiderman et al., 2004). The results suggest a differential treatment effect across racial/ethnic and gender groups from ENRICHHD, in terms of morbidity and mortality. Thus, in the current project, we investigated further whether the ENRICHHD treatment intervention improved cognitive and somatic depressive symptoms across racial/ethnic groups.

With respect to treatment effect on overall depression across racial/ethnic groups, current findings indicated that treatment did not improve the BDI total depression score at six-months post-treatment across racial/ethnic majority and minority groups. It may be that the analyses of the current project included not only the depressed patients but also those who were not depressed but socially-isolated; thus, the treatment effect on depression six-months post-treatment may have been attenuated in patients who were not depressed at baseline. However, while examining the treatment effect on specific depressive symptoms at six-months post-treatment, the results showed different and significant findings. Specifically, the treatment significantly improved cognitive symptoms in all racial/ethnic groups. However, it was effective in alleviating somatic symptoms only for both non-Hispanic Whites and Hispanics, but not for non-Hispanic Blacks. In addition, significant group differences in treatment effect on cognitive depressive symptoms at six-months post-treatment was only observed between

non-Hispanic Blacks and Hispanics, after controlling for baseline depression, antidepressant use, gender, education, annual income, and employment status. Furthermore, significant group differences in treatment effect on improving somatic depressive symptoms at six-months post-treatment was only found between non-Hispanic Blacks and Whites, after controlling for baseline depression, gender, antidepressant use, annual income and employment status.

While CBT treatment was generally effective in improving depressive symptoms, the present study showed that Hispanics showed less improvement in cognitive symptoms than non-Hispanic Whites and no difference in improvement of somatic symptoms than the other two groups. For Hispanic patients, this project indicated that Hispanic patients might have benefited less from the traditional CBT treatment compared to non-Hispanic Whites, given that the CBT-oriented treatment was initially developed for non-Hispanic Whites and thus might have been novel to the Hispanic minority patients.

Similarly, for non-Hispanic Blacks, this project also suggested that non-Hispanic Black patients might have benefited less from the traditional CBT treatment compared to the non-Hispanic Whites. One explanation may be that somatic rather than cognitive symptoms of depression are associated with MI severity and cardiovascular prognosis (Barefoot et al., 2000; Barefoot & Schroll, 1996; Carney & Freedland, 2012; Martens et al., 2010; Watkins et al., 2003) and non-response to treatment of post-MI depression may be associated with severity of cardiac events (de Jonge, Mangano, & Whooley, 2007). While there

is no difference in total numbers of previous MI and cardiac procedures (e.g., coronary artery bypass graft surgery and percutaneous transluminal carotid angioplasty and stenting) across groups in the current project, a speculation concerning that non-Hispanic Blacks' non-response to treatment may be related to their poor prognosis post-MI, compared to the other two racial/ethnic groups. Another speculation could be that Blacks are likely to delay the process of seeking treatment and thus have more severe and debilitating CHD symptoms than others.

In short, findings from the current project indicated that CBT was effective in alleviating predominantly cognitive symptoms across all three groups, but was ineffective in reducing underlying somatic symptoms among non-Hispanic Blacks. Group differences in treatment effect on the reduction of cognitive depression were only significant between Hispanic and non-Hispanic Whites. Significant treatment differences in somatic depression were only apparent between non-Hispanic Blacks and Whites.

Implications. Current findings indicated that CBT improved predominantly cognitive depression across all groups but did not alleviate somatic depression in non-Hispanic Blacks. Thus, interventions that include management of somatic depression symptoms should be considered. The distinction between somatic and cognitive symptoms supports the conceptualization of depression as a two-dimensional syndrome. Cognitive symptoms have been specific targets for intervention in psychotherapy treatment, and behavioral interventions include

exercise training that may improve somatic depressive symptoms. It may be possible that in order to maximize treatment outcomes for post-MI patients, especially non-Hispanic Blacks, management of somatic depression symptoms should also be incorporated into the traditional CBT. Depending on the aims and intensity of the CBT treatment, either cognitive (e.g., mood or negative thinking) or behavioral (e.g., exercise or muscle relaxation), clinical treatment outcomes may differ (Sheps, Freedland, Golden, & McMahon, 2003).

In fact, recent clinical trials or treatment interventions for depression that involve cardiac patients included behavioral components that reduce cardiac patients' somatic depression (Blumenthal et al., 2005; Sebregts, Falger, Appels, Kester, & Bär, 2005). For instance, interventions may focus on teaching patients about exercise or stress management in addition to promoting patients' cognitive depression functioning. Increasing evidence confirmed that somatic rather than cognitive symptoms are associated with MI severity and cardiovascular prognosis (Barefoot et al., 2000; Martens et al., 2010; Watkins et al., 2003). Nonresponse to treatment of post-MI depression may be associated with severity of cardiac events (de Jonge et al., 2007) or utilization of recommended cardiac treatment (Sanderson, Raczynski, Cornell, Hardin, & Taylor, 1998). Because depression symptomatology and cardiac outcomes are closely related, interventions must pay special attention on reducing somatic aspects of depression and aim for improving cardiac survival. Effort should be dedicated to developing more cost-effective treatment interventions for particular cardiac patients based on patients' underlying depression symptoms.

Gender Differences in Depression

As anticipated, female patients demonstrated significantly higher levels of baseline depression than male patients. This finding is similar to those shown among cardiac patients in other studies (Frasure-Smith, Lesperance, Juneau, Talajic, & Bourassa, 1999; Grace et al., 2005), confirming the well-established evidence of gender differences in depression in the literature. This finding may be explained by multiple roles performed by the female patients and stressors related to household and family duties (Kristofferzon, Löfmark, & Carlsson, 2003). The literature suggests that women with heart disease report different psychosocial stressors, use different coping styles, and experience lower quality of life and family support than male patients (Emery et al., 2004; Larsen, Vickers, Sampson, Netzel, & Hayes, 2006; Rosland, Heisler, Choi, Silveira, & Piette, 2010). Furthermore, a significant gender and race/ethnicity interaction was also found. Hispanic women had the highest levels of depression at baseline among all racial/ethnic men and women groups.

Expectations and conflicts in Hispanic gender role differentiation in work and marriage may explain greater depression levels among Hispanic females compared to non-Hispanic Whites (Golding et al., 1991; Golding & Karno, 1988). The findings also indicated that racial/ethnic minority female patients experienced greater baseline depression than non-Hispanic White male and female patients. In addition to the compelling evidence of gender differences in depression, the present study also suggested that the combination of being a woman and a

minority is associated with increased depression symptoms. While some researchers have emphasized the importance of identifying and modifying gender-specific psychosocial and behavioral risk factors of depression in CHD (Burnette, Mui, & Zodikoff, 2004; van Jaarsveld et al., 2006), predominately among non-Hispanic Whites, future research should further investigate psychosocial, and behavioral factors associated with greater depression among racial/ethnic minority female patients.

In terms of gender differences, as expected, women exhibited higher levels of cognitive and somatic symptoms at baseline than men. Similar findings were found in early studies (Silverstein, 1999, 2002; Silverstein & Lynch, 1998), which showed that disproportionate differences in depression symptoms may be related to differences in social roles or cultural norms between men and women. It may be more socially accepting for women to express somatic complaints or exhibit depression than it is for men. Because men are expected to be strong and self-reliant in the society, they may be less likely to report somatic complaints or depression. For men, feeling depressed is often seen as a personal weakness or vulnerability. Also, significant gender-specific findings in symptom levels may be reflective of biological or hormonal differences between men and women (Wenzel et al., 2005). Greater disturbances of sleep and poorer appetite are characteristic among depressed female patients, compared to male patients. The findings from the current study further confirmed gender differences in depression prevalence and symptoms found in the literature.

Implications. Gender specific issues may be incorporated into the traditional CBT for depression in cardiac patients to maximize treatment outcomes and improve treatment prognosis. The findings from the current study indicated that female patients reported higher levels of baseline depression and more somatic symptoms compared to male patients. Female and male cardiac patients may differ in their unique needs in emotional coping and family role issues (Claesson et al., 2005) as well as social isolation (Barth et al., 2009). Therefore, future clinical trials designed for treating depression in CHD should include gender-specific aims for treatment, which may yield gender-specific benefits for health outcome, psychosocial wellbeing, and role functioning.

Strengths and Limitations of the Current Study

Strengths

The current study has several strengths. A major strength is the use of three racial/ethnic groups of post-MI patients, Hispanics, non-Hispanic Blacks, and Whites, including underrepresented female patients in each group. Most of the prior studies in the literature investigated depression in a racial/ethnic minority group and compared their results to non-Hispanic Whites. The present study utilized a large sample of post-MI patients to investigate the core depression features across Hispanics, non-Hispanic Blacks, and Whites, allowing a better understanding of racial/ethnic differences in underlying BDI symptoms. In addition, it was the first study to systematically examine the underlying factor

structure of the BDI model and the CBT treatment effect on depressive symptoms across three racial/ethnic groups of post-MI patients.

With increasing diversity of the population in the U.S., screening for depression in clinical care among disparate groups is especially important, particularly for patients with heart disease, the number one cause of death in the U.S. The findings of the current study bridged the gaps between the literature findings through identifying specific racial/ethnic depressive symptoms on the BDI among cardiac patients, which may help to explain existing disparities in depression among racial/ethnic minority groups. The results of this study further shed some light to the extent in which racial/ethnic groups differ in depression manifestation and response treatment effect, suggesting that current BDI screening for depression may not capture all depressive symptoms across racial/ethnic groups and that current CBT treatment for depression may be limited in its efficacy to alleviate somatic depression among racial/ethnic minority patients.

The methodology and research design of the present study is another strength. In particular, the depression was measured at different time points, both at baseline and six-months post-treatment (Tomarken & Waller, 2005). SEM was conducted to establish and specify well fitting models for hypotheses testing, allowing a best representation of the data. The use of “intent-to-treat” analysis in this randomized control trial was an additional strength. Furthermore, all eligible participants were screened and interviewed by clinicians to determine

and confirm their diagnosis of clinical depression as specified in the Diagnostic Statistical Manual of Mental Disorders (DSM-IV) criteria (American Psychiatric Association, 2000).

Limitations

Interpretation of the findings from the present study should be considered within the context of several limitations. At least two limitations warrant further attention. One limitation concerns the generalizability of the findings from the study. Other racial/ethnic minority groups (i.e., Asians and American Indians) were excluded due to insufficient sample sizes. Also, Hispanics recruited in the present study were predominantly Cuban or of Cuban descent living in Miami, Florida. Thus, caution needs to be exercised in generalizing the findings of the present study to other racial/ethnic groups, except for non-Hispanic Whites, Blacks, and Hispanics. Also, participants included in the present study were post-MI patients. Therefore, the generalizability of these results is limited to depression among post-MI patients and should be replicated with patients with other types of heart disease as well as populations of differing medical illnesses. Another limitation is that the BDI used in the current study was not the updated version of the depression screening measure, the BDI-II (Beck, Steer, & Brown, 1996). Compared to the BDI, the BDI-II involved a clarification and modification of several items to include more depression symptoms, including Agitation, Worthlessness, Loss of Energy, and Concentration Difficulty. Some items were reworded, but the majority of the items remain similar with few variations in wording. Furthermore, the two versions were found to be highly correlated

(i.e., .93 in a clinical sample)(Beck, Steer, Ball, & Ranieri, 1996). Future research may test measurement invariance of the BDI-II.

Conclusions

Despite the limitations, findings of the current study have implications for clinical research and practice. In particular, the present study offered new insights to the extent in which racial/ethnic groups manifested depression differently. Current findings suggest that although the core cognitive and somatic depressive symptoms are similar across racial/ethnic groups, the presentation of unique depressive symptoms varies across the three racial/ethnic groups. In particular, body image issue is salient in Hispanic patients; poor appetite is important diagnostically among non-Hispanic Whites; and sleep disturbance is a critical factor among non-Hispanic Blacks.

This was the first study to systematically examine the depression symptom measure across the three racial/ethnic groups of post-MI patients. It revealed that a two-factor structure of the BDI model emerged consistently and provided the best model fit across a six-month time frame among a diverse and representative sample of post-MI patients. For the BDI, the underlying structure is composed of two-factors identified as cognitive and somatic. However, the results indicated some variation across racial/ethnic groups in the manifestation of the items, primarily limited to cognitive symptoms (e.g., sense of punishment, self-accusations, self-punitive wishes, and body image), on the BDI.

Despite strong empirical support of the BDI, validation in a medical sample is salient for making accurate assessment and treating depression among

diverse cardiac patients. Given observed partial measurement invariance of the BDI, the present study suggested that interpretations of the data using the BDI as a screening instrument across racial/ethnic post-MI patients require caution. Specifically, researchers should examine specific items that are pertinent to each racial/ethnic group rather than assuming all items are equivalent across cultures. In addition, instead of examining depression as a one-dimensional construct, both cognitive and somatic depression symptoms should be assessed. Based on the current findings, specific cognitive depressive items that distinguish racial/ethnic groups should be carefully monitored among cardiac patients. The findings of measurement non-invariance indicate that variant items across racial/ethnic groups in a medical sample should be mainly examined for racial/ethnic minorities and that a close examination at specific depression manifestations across groups may be necessary to further understand depression symptomatology among cardiac patients.

Moreover, findings from the present study may help to inform researchers and clinicians regarding targeted depression symptoms and treatment aims among post-MI patients for future application. The results informed that in a group of diverse cardiac patients, depression is conceptualized comparably. However, few variations in depression presentation were observed across groups. Given that nine items were cross-loaded onto both cognitive and somatic factors, it suggested the challenge of differentiating cognitive from somatic symptoms among cardiac patients. Because prior research suggested that somatic symptoms were highly associated with poor prognosis and cardiac

outcomes, it is especially critical to disentangle the interrelationship between the severity of cognitive and somatic depressive symptoms and the prognosis of treatment in order to identify a subgroup of cardiac patients who may benefit from psychological treatment for depression.

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APPENDIX 1.

THE BECK DEPRESSION INVENTORY (BDI) ITEM LIST

1. Feeling of sadness
2. Pessimism
3. Sense of failure
4. Lack of satisfaction
5. Guilty feelings
6. Sense of punishment
7. Self hate
8. Self accusations
9. Self punitive wishes
10. Crying spells
11. Irritability
12. Social withdrawal
13. Indecision
14. Body image
15. Work inhibition
16. Sleep disturbance
17. Fatigability
18. Loss of appetite
19. Weight loss
20. Somatic preoccupation
21. Loss of libido

FIGURE 1.

FLOW CHART ILLUSTRATING THE NUMBERS OF A SUBGROUP OF THE ENRICHD PATIENTS (N = 2370) THROUGH THE SCREENING PROCESS; MET MI CRITERIA, MET MEDICAL AND PSYCHOSOCIAL ELIGIBILITY, AND RANDOMIZED FOR COGNITIVE BEHAVIORAL INTERVENTION OR USUAL CARE

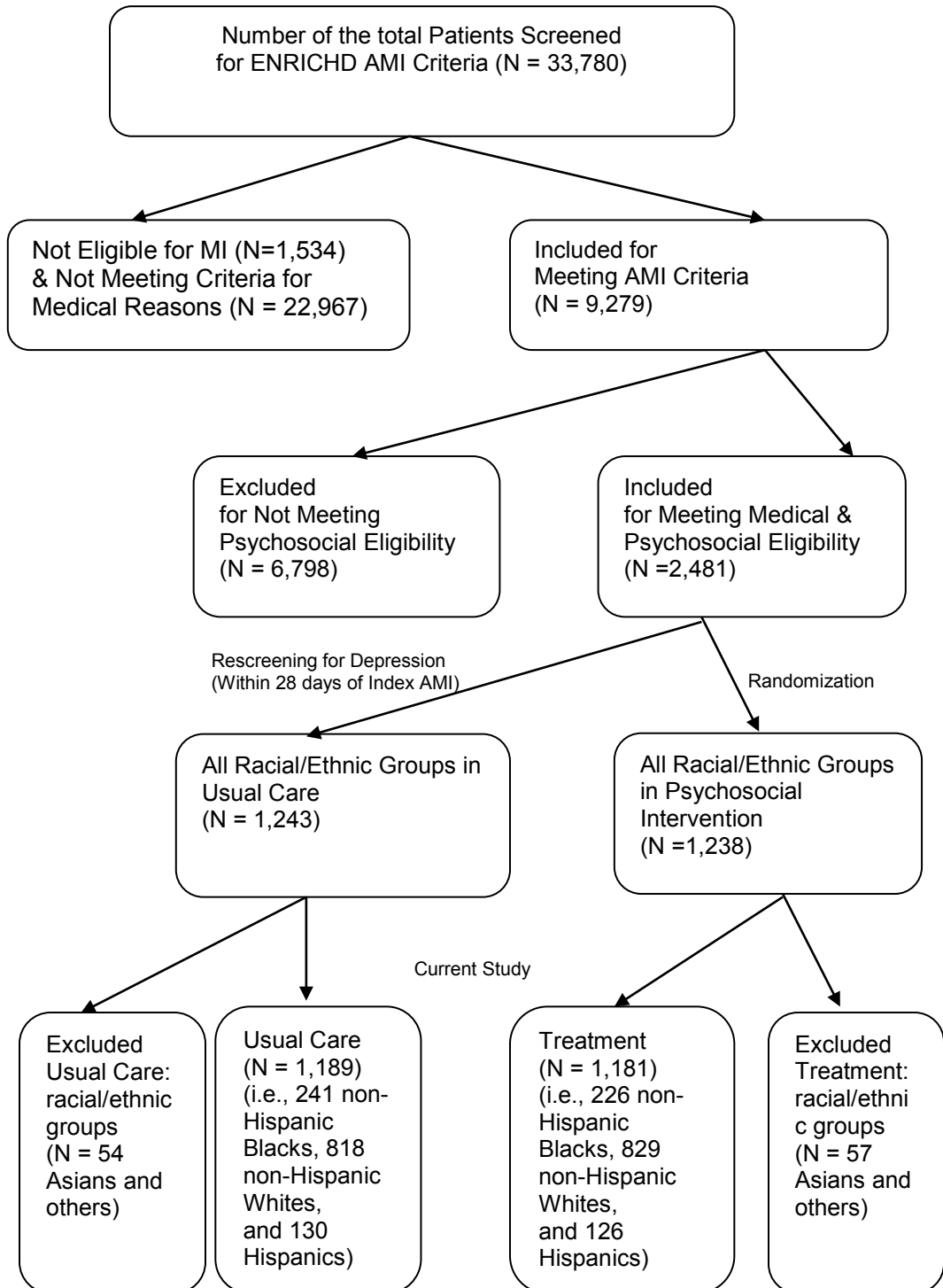


FIGURE 2.

STRUCTURAL EQUATION MODELING TESTING TREATMENT EFFECT ON COGNITIVE AND SOMATIC DEPRESSION SYMPTOMS

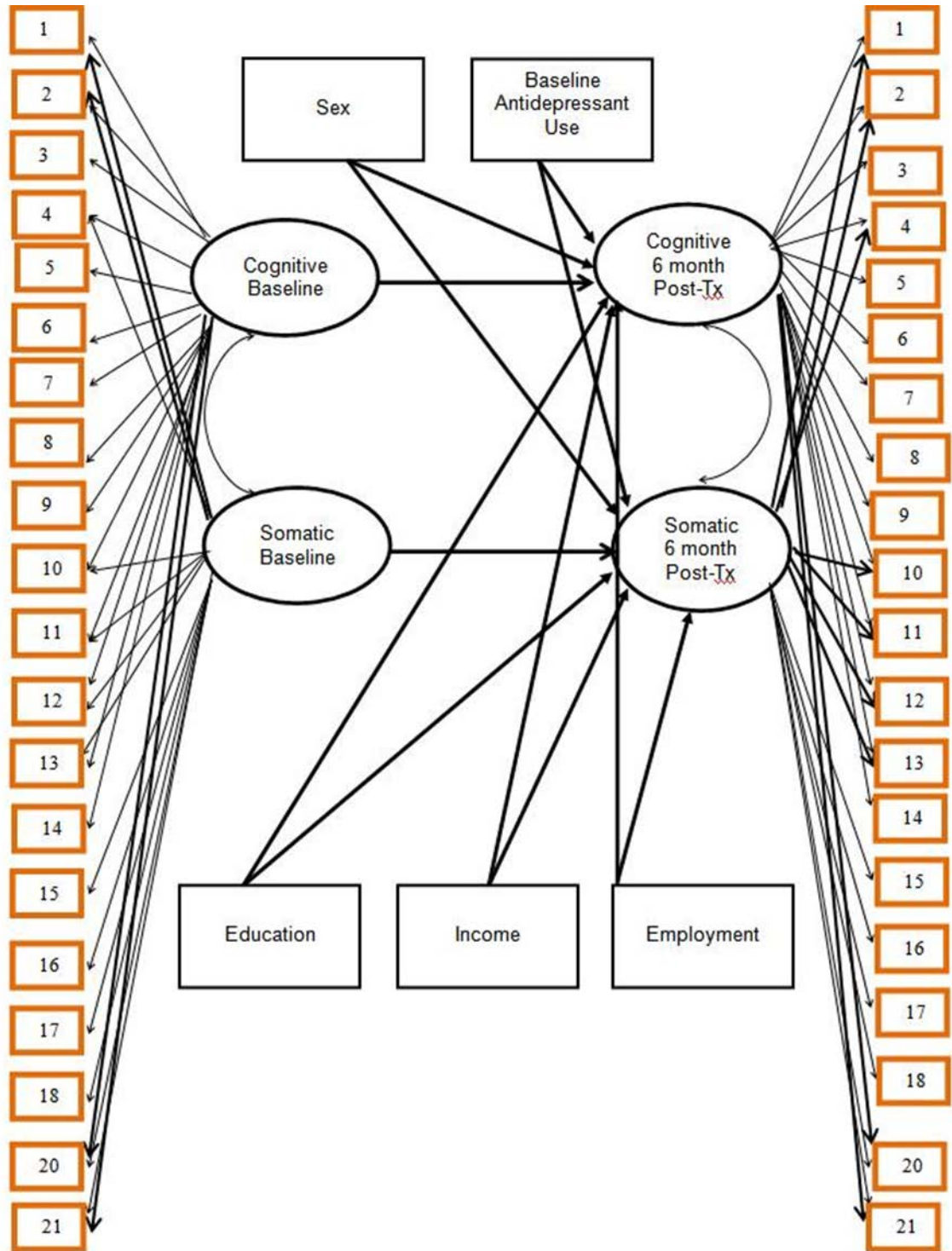


FIGURE 3.

A LINE GRAPH OF GENDER BY RACE/ETHNICITY ON THE BASELINE BDI TOTAL SCORES

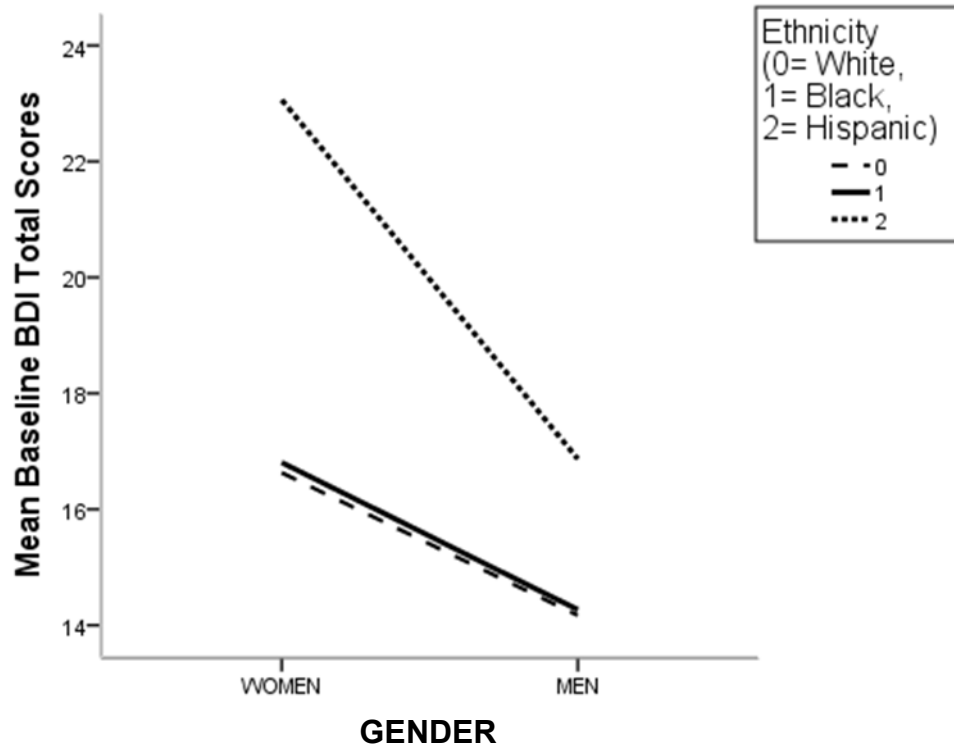


TABLE 1.

KEY DEMOGRAPHIC CHARACTERISTICS BY RACIAL/ETHNIC SUBGROUPS

Demographic Variables	Total (N =2370)	Non-Hispanic Blacks (n=467)	Hispanics (n=256)	Non-Hispanic Whites (n=1647)	p value (Chi-Square)
Age, Mean(SD), Year	60 (12.76)	56 (12.3)	61 (11.76)	61 (12.84)	N/A
Sex, Female	1048 (44%)	280 (60%)	96 (38%)	672 (41%)	< .0001*
Treatment, Intervention	1181 (50%)	226 (48%)	126 (49%)	829 (50%)	0.74
Marital Status, Married	1286 (54%)	209 (49%)	126 (49%)	951 (58%)	< .0001*
Education, High School or Higher	1697 (71%)	277 (59%)	135 (57%)	1275 (77%)	< .0001*
Employment 3 Mo. Before MI	1024 (44%)	113 (44%)	192 (41%)	719 (44%)	0.51
Income < \$15,000	437 (18%)	170 (36%)	152 (59%)	310 (19%)	< .05*
Living Arrangement, With Spouse (other person)	1630 (69%)	319 (68%)	182 (71%)	1129 (69%)	0.51
Psychosocial Risk Factors					
Depressed	942 (40%)	168 (36%)	71 (28%)	703 (43%)	< .0001*
Isolated	611 (26%)	143 (31%)	60 (23%)	408 (25%)	< .001*
Depresses & Isolated	817 (34%)	156 (33%)	125 (49%)	536 (33%)	< .0001*
Antidepressant Medication Prescribed, Baseline					
	137 (6%)	11 (2.4%)	10 (4%)	116 (7%)	< .001*

* Data are presented as frequencies (%), unless otherwise indicated.

TABLE 2.

DESCRIPTIVE STATISTIC AND THE TESTS OF BETWEEN-SUBJECTS EFFECTS IN A 2 X 3 FACTORIAL ANALYSIS OF VARIANCE (ANOVA)

Dependent Variable: Baseline BDI Total Score

Ethnicity	Gender	Mean	SD	N
Whites	Women	16.65	8.22	669
	Men	14.17	7.42	952
	Total	15.22	7.87	1621
Blacks	Women	16.83	8.39	278
	Men	14.28	8.94	179
	Total	15.83	8.69	457
Hispanics	Women	23.18	9.77	95
	Men	16.88	8.73	158
	Total	19.24	9.62	253
Total	Women	17.29	8.61	1042
	Man	14.52	7.86	1289
	Total	15.76	8.32	2331

Dependent Variable: Baseline BDI Total Score

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Race/ethnicity	4461.05	2	2230.53	34.10	<.001	.027
Gender	4493.05	1	4493.05	68.69	<.001	.027
Race * Gender	775.77	2	387.89	5.93	<.01	.005
Error	152073.72	2325	65.41			
	4461.05	2	2230.53	34.10	<.001	.027

* R Squared = .054 (Adjusted R Squared = .052)

TABLE 3.

UNSTANDARDIZED FACTOR LOADINGS OF THE BEST FITTING TWO-FACTOR BDI MODEL IN EACH RACIAL/ETHNIC GROUP

UNSTANDARDIZED FACTOR LOADINGS ON TWO LATENT FACTORS						
BDI- ITEM	WHITE		BLACK		HISPANIC	
	COG	SOM	COG	SOM	COG	SOM
1	0.66	0.32	0.51	0.57	0.50	0.42
2	0.82	0.18	0.61	0.40	0.68	0.16($p=.13$)
3	0.97	--	0.96	--	0.88	--
4	0.53	0.40	0.52	0.38	0.54	0.47
5	0.86	--	0.78	--	0.79	--
6	0.78	--	0.93	--	0.66	--
7	1.00	--	1.00	--	1.00	--
8	0.89	--	0.95	--	0.85	--
9	0.83	--	0.76	--	0.68	--
10	0.53	0.31	0.48	0.48	0.38	0.33
11	0.47	0.22	0.40	0.37	0.33	0.33
12	0.65	0.22	0.52	0.25	0.45	0.50
13	0.54	0.40	0.48	0.48	0.66	0.21
14	0.70	--	0.58	--	0.83	--
15	--	0.90	--	0.92	--	0.90
16	--	0.65	--	1.04	--	0.69
17	--	1.00	--	1.00	--	1.00
18	--	0.68	--	0.63	--	0.51
19	--	--	--	--	--	--
20	0.39	0.35	0.32	0.31	0.21($p=.05$)	0.46
21	0.24	0.39	0.14($p=.09$)	0.57	0.44	0.22

* Items 3, 5, 6, 7, 8, 9, and 14 loaded on pure Cognitive factor. Items 15, 16, 17, and 18 loaded on pure Somatic factor. Items 1, 2, 4, 10, 11, 12, 13, 20 and 21 cross-loaded on both Cognitive and Somatic Factors

** COG = BDI Cognitive Factor, SOM = BDI Somatic Factor

*** P value included in the parentheses indicates an insignificant factor loading. All the factor loadings are significant at $p < .05$.

TABLE 4.

**UNSTANDARDIZED FACTOR LOADINGS OF INVARIANT AND VARIANT
ITEMS ACROSS NON-HISPANIC WHITE AND HISPANIC GROUPS**

UNSTANDARDIZED FACTOR LOADINGS				
BDI ITEM	WHITE		HISPANIC	
	COG	SOM	COG	SOM
INVARIANT				
1	0.66	0.33	0.66	0.33
2	0.83	0.19	0.83	0.19
3	0.97	--	0.97	--
4	0.53	0.42	0.53	0.42
5	0.85	--	0.85	--
6	0.76	--	0.76	--
7	1.00	--	1.00	--
8	0.87	--	0.87	--
9	0.81	--	0.81	--
10	0.53	0.30	0.53	0.30
11	0.45	0.23	0.45	0.23
12	0.62	0.28	0.62	0.28
13	0.56	0.36	0.56	0.36
15	--	0.89	--	0.89
16	--	0.66	--	0.66
17	--	1.00	--	1.00
20	0.38	0.36	0.38	0.36
21	0.26	0.34	0.26	0.34
VARIANT				
14	0.70	--	1.16	--
18	--	0.68	--	0.40

* Items 3, 5, 6, 7, 8, 9, and 14 loaded on pure Cognitive factor. Items 15, 16, 17, and 18 loaded on pure Somatic factor. Items 1, 2, 4, 10, 11, 12, 13, 20 and 21 cross-loaded on both Cognitive and Somatic Factors

** COG = BDI Cognitive Factor, SOM = BDI Somatic Factor

*** All the factor loadings are significant at $p < .001$.

TABLE 5.

UNSTANDARDIZED FACTOR LOADINGS OF INVARIANT AND VARIANT ITEMS ACROSS NON-HISPANIC WHITE AND BLACK GROUPS

UNSTANDARDIZED FACTOR LOADINGS				
BDI ITEM	WHITE		BLACK	
	COG	SOM	COG	SOM
INVARIANT				
1	0.62	0.37	0.62	0.37
2	0.78	0.22	0.78	0.22
3	0.97	--	0.97	--
4	0.53	0.39	0.53	0.39
5	0.83	--	0.83	--
7	1.00	--	1.00	--
8	0.90	--	0.90	--
10	0.52	0.35	0.52	0.35
11	0.45	0.26	0.45	0.26
12	0.63	0.23	0.63	0.23
13	0.52	0.41	0.52	0.41
15	--	0.89	--	0.89
17	--	1.00	--	1.00
18	--	0.65	--	0.65
20	0.38	0.35	0.38	0.35
21	0.21	0.43	0.21	0.43
VARIANT				
6	0.77	--	1.26	--
9	0.82	--	0.59	--
14	0.70	--	0.54	--
16	--	0.65	--	1.01

* Items 3, 5, 6, 7, 8, 9, and 14 loaded on pure Cognitive factor. Items 15, 16, 17, and 18 loaded on pure Somatic factor. Items 1, 2, 4, 10, 11, 12, 13, 20 and 21 cross-loaded on both Cognitive and Somatic Factors

** COG = BDI Cognitive Factor, SOM = BDI Somatic Factor

*** All the factor loadings are significant at $p < .001$.

TABLE 6.

UNSTANDARDIZED FACTOR LOADINGS OF INVARIANT AND VARIANT ITEMS ACROSS NON-HISPANIC BLACK AND HISPANIC GROUPS

UNSTANDARDIZED FACTOR LOADINGS				
BDI ITEM	BLACK		HISPANIC	
	COG	SOM	COG	SOM
INVARIANT				
1	0.56	0.49	0.56	0.49
3	0.96	--	0.96	--
5	0.81	--	0.81	--
7	1.00	--	1.00	--
	0.79	--	0.79	--
10	0.51	0.41	0.51	0.41
11	0.38	0.36	0.38	0.36
13	0.56	--	0.56	--
15	--	0.91	--	0.91
16	--	1.02	--	1.02
17	--	1.00	--	1.00
18	--	0.60	--	0.60
20	0.30	0.35	0.30	0.35
21	0.22	0.44	0.22	0.44
VARIANT				
2	0.63	0.36	1.13	0.25
4	0.55	0.33	0.69	0.77
6	0.95	--	0.51	--
8	0.98	--	0.61	--
12	0.54	0.21	0.46	0.73
14	0.59	--	1.11	--

* Items 3, 5, 6, 7, 8, 9, and 14 loaded on pure Cognitive factor. Items 15, 16, 17, and 18 loaded on pure Somatic factor. Items 1, 2, 4, 10, 11, 12, 13, 20 and 21 cross-loaded on both Cognitive and Somatic Factors

** COG = BDI Cognitive Factor, SOM = BDI Somatic Factor

*** All the factor loadings are significant at $p < .001$.

TABLE 7.

**UNSTANDARDIZED FACTOR LOADINGS OF THE TWO-FACTORBDI MODEL
OVER A SIX MONTH TIME FRAME IN USUAL CARE GROUP**

UNSTANDARDIZED FACTOR LOADINGS					
BDI ITEM	BASELINE		6 MONTH POST-TREATMENT		
	COG	SOM	COG	SOM	
INVARIANT					
1	0.49	0.36	0.66		0.44
2	0.73	0.26	0.69		0.37
5	0.74	--	0.90		--
6	0.62	--	0.82		--
7	1.00	--	1.00		--
9	0.94	--	0.83		--
11	0.34	0.23	0.40		0.32
13	0.46	0.35	0.47		0.35
14	0.67	--	0.75		--
15	--	0.84	--		1.01
16	--	0.59	--		0.78
17	--	1.00	--		1.00
18	--	0.62	--		0.64
VARIANT					
3	1.05	--	0.96		--
4	0.56	0.40	0.49		0.59
8	0.98	--	0.86		--
9	1.01	--	0.79		--
10	0.41	0.44	0.61		0.27
12	0.69	0.21	0.51		0.45
20	0.24	0.40	0.37		0.44
21	0.20	0.36	0.20		0.54

* Items 3, 5, 6, 7, 8, 9, and 14 loaded on pure Cognitive factor. Items 15, 16, 17, and 18 loaded on pure Somatic factor. Items 1, 2, 4, 10, 11, 12, 13, 20 and 21 cross-loaded on both Cognitive and Somatic Factors

** COG = BDI Cognitive Factor, SOM = BDI Somatic Factor

*** All the factor loadings are significant at $p < .001$.

TABLE 8.

**UNSTANDARDIZED FACTOR LOADINGS OF THE TWO-FACTOR BDI
MODEL OVER A SIX MONTH TIME FRAME IN TREATMENT GROUP**

UNSTANDARDIZED FACTOR LOADINGS				
BDI ITEM	BASELINE		6 MONTH POST-TREATMENT	
	COG	SOM	COG	SOM
1	0.75	0.29	0.65	0.41
2	0.97	0.13	0.75	0.35
3	1.00	--	1.03	--
4	0.60	0.42	0.45	0.69
5	0.95	--	0.95	--
6	0.94	--	0.89	--
7	1.00	--	1.00	--
8	0.92	--	0.99	--
9	0.88	--	1.09	--
10	0.57	0.26	0.34	0.57
11	0.55	0.21	0.37	0.51
12	0.72	0.25	0.61	0.46
13	0.59	0.33	0.40	0.53
14	0.75	--	0.79	--
15	--	0.90	--	1.03
16	--	0.65	--	0.86
17	--	1.00	--	1.00
18	--	0.62	--	0.79
20	0.48	0.34	0.51	0.42
21	0.21	0.48	0.33	0.42

* Items 3, 5, 6, 7, 8, 9, and 14 loaded on pure Cognitive factor. Items 15, 16, 17, and 18 loaded on pure Somatic factor. Items 1, 2, 4, 10, 11, 12, 13, 20 and 21 cross-loaded on both Cognitive and Somatic Factors

** COG = BDI Cognitive Factor, SOM = BDI Somatic Factor

*** All the factor loadings are significant at $p < .001$.