THE CARDIAC SELF-BLAME ATTRIBUTIONS SCALE AS A PREDICTOR OF PHYSICAL AND MENTAL HEALTH OUTCOMES IN UNDERREPRESENTED PATIENTS WITH CARDIOVASCULAR DISEASE

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ABSTRACT

Following a cardiovascular event, most patients engage in a causal search to understand why it occurred. One way of distinguishing attributions is through the construct of self-blame. There is a difference between patients blaming their illnesses on their own behaviors, referred to as behavioral self-blame (BSB), and the tendency to attribute their conditions to their dispositions, classified as characterological self-blame (CSB). BSB is predicted to result in positive outcomes, whereas CSB is predicted to result in negative outcomes. However, self-blame attributions have been associated with both positive and negative health outcomes in patients with cardiovascular disease (CVD). One possible reason for the discrepant findings is the lack of a validated, multiple-item measure of the construct. Thus, the 11-item Cardiac Self-Blame Attributions (CSBA) scale was developed to fill this gap. Preliminary analyses showed that this scale is a reliable and valid measure of self-blame, but it was not known whether the CSBA scale is *predictive* of physical and

mental health outcomes. The purpose of this study was to examine if the CSBA scale is associated with physical and mental health outcomes among underrepresented patients with CVD before and after cardiac rehabilitation (CR). Health outcomes included depressive symptoms, health-related quality of life (HRQoL), heart-healthy diet, and functional capacity. A secondary aim was to assess whether the relationships between both types of self-blame and health outcomes are mediated by control appraisals.

Self-reported, questionnaire data were collected from 95 patients at the beginning of CR and after they completed CR (n = 50). Results confirmed the two-factor structure of the CSBA scale, and indicated good internal validity and test-retest reliability. Both BSB and CSB were significantly positively related to depressive symptoms and negatively related to mental HRQoL cross-sectionally, but BSB was not related to any of the health outcomes longitudinally. CSB was significantly negatively related to physical HRQoL at the end of CR, but was not related to any other health outcomes longitudinally. Control appraisals at the beginning of CR did not mediate the association between either type of self-blame and health outcomes at the end of CR. Clinical and theoretical implications are discussed.

APPROVAL PAGE

The faculty listed below, appointed by the Dean of the College of Arts and Sciences have examined a dissertation titled "The Cardiac Self-Blame Attributions Scale as a Predictor of Physical and Mental Health Outcomes in Underrepresented Patients with Cardiovascular Disease," presented by Kadie McGowan Harry, candidate for the Doctor of Philosophy degree, and certify that in their opinion it is worthy of acceptance.

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LIST OF ABBREVIATIONS

Cardiac Rehabilitation = CR

Cardiovascular Disease = CVD

Truman Medical Center = TMC

Cardiac Self-Blame Attributions Scale = CSBA

Behavioral self-blame = BSB

Characterological self-blame = CSB

Health-related quality of life = HRQoL

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CHAPTER 1

INTRODUCTION

Identifying a potential cause, or creating an attribution, is a salient part of the adjustment process to serious illnesses, especially when etiologic factors can be identified. In cardiovascular disease (CVD), a disease for which specific health behaviors have been linked to onset (Roger et al., 2011), patients readily search for a cause. One way of distinguishing attributions is through the construct of self-blame. There is a difference between patients blaming their illnesses on their own behaviors, referred to as behavioral self-blame (BSB), and the tendency to attribute their conditions to their dispositions, referred to as characterological self-blame (CSB; Janoff-Bulman, 1979).

It is hypothesized that behavioral self-blame is positively related to control appraisals which leads to less distress and better health outcomes. In contrast, characterological self-blame is hypothesized to be negatively related to control appraisals and thus related to more distress and worse health outcomes (Janoff-Bulman, 1979). Previous research has shown mixed findings, in that both types of self-blame have been associated with positive and negative health outcomes in patients with CVD (Bennett, Howarter, & Clark, 2013; Harry, Bennett, Clark, Howarter, & Eways, 2015; Plaufcan, Wamboldt, & Holm, 2012). One possible reason for the discrepant findings is the lack of a validated, multiple-item measure. Thus, the 11-item Cardiac Self-Blame Attribution (CSBA) scale was developed to fill this gap in the literature. Preliminary analyses (based on data from a previous study) showed that the scale is a reliable and valid measure of self-blame attributions among patients with CVD, and represents two factors: behavioral self-blame and characterological self-blame.

The next step of the scale validation process is to examine if the CSBA scale is *predictive* of physical and mental health recovery outcomes among underrepresented patients with CVD. Thus, the present study aimed to further validate the CBSA scale to assess whether it differentiates between the two types of self-blame attributions (Hypothesis #1), and to examine whether it predicts physical and mental health recovery outcomes before and after cardiac rehabilitation (CR). Health outcomes included depressive symptoms, mental and physical health-related quality of life (HRQoL), heart-healthy diet, and functional capacity. We hypothesized that BSB would be positively related to health outcomes cross-sectionally (Hypothesis #2) and longitudinally (Hypothesis #3), but that CSB would be negatively related to health outcomes cross-sectionally (Hypothesis #4) and longitudinally (Hypothesis #5). Additionally, a secondary aim was to assess whether the relationships between both types of self-blame attributions (behavioral and characterological) and health outcomes were mediated by control appraisals (Hypotheses #6 and #7, respectively).

Self-reported, questionnaire data were collected from 95 patients at the beginning of CR and after they completed CR (n = 50). Results confirmed that Hypothesis #1 was supported; the factor analysis yielded a two-factor structure of the CSBA scale measuring BSB and CSB, and indicated good internal validity and good test-retest reliability. Hypothesis #2 was not supported because BSB at the beginning of CR was positively related to poor health outcomes (depressive symptoms), and negatively related to good health outcomes (mental HRQoL). Hypothesis #3 was not supported, as BSB was not related to any of the other health outcomes longitudinally. Hypothesis #4 was partially supported: baseline CSB was positively related to depressive symptoms and negatively related to mental HRQoL, cross-sectionally, but was unrelated to the other health outcomes. Hypothesis #5

was partially supported: baseline CSB was negatively related to physical HRQoL at Time 2, but was unrelated to the other health outcomes. Hypothesis #6 was not supported because control did not mediate any of the associations. However, results did support longitudinal effects of control on mental HRQoL. In addition, only in models controlling for CR sessions was BSB significantly negatively related to control appraisals. And, Hypothesis #7 was not supported because control did not mediate any of the associations. However, results did support longitudinal effects of control on mental HRQoL, and CSB on physical HRQoL.

Thus, the present study showed that blaming one's behavior for a cardiac event at the beginning of CR was related to more depressive symptoms and worse mental HRQoL at baseline, but not at the end of CR. Blaming your cardiac event on your character or personality at the beginning of CR was also related to more depressive symptoms and worse mental HRQoL at baseline, but not at the end of CR. Overall, findings imply that making any type of self-blame attribution is associated with poor concurrent mental health. In addition, CSB was related to worse physical HRQoL at the end of CR. Therefore, CR staff should identify patients' levels of BSB at the start of CR in order to design and implement interventions in hopes of affecting positive health outcomes, specifically related to psychological functioning. In addition, CR staff should identify patients' levels of CSB at the beginning of CR in order to implement strategies in hopes of ameliorating the negative effects on psychological distress and physical HRQoL. Findings do not imply a health benefit of BSB through increased control appraisals.

CHAPTER 2

REVIEW OF THE LITERATURE

Attributions

Identifying a potential cause, or creating an attribution, is a salient part of the adjustment process to any negative event, including serious diseases. Furthermore, previous research has identified a relationship between casual attributions and adjustment to chronic illness (Christensen et al., 1999; Malcarne, Compas, Epping-Jordan, & Howell, 1995). Taylor (1983) outlined a theory of cognitive adaptation that describes the process of adjustment after a traumatic or personally threatening event as involving three themes: a search for meaning, an attempt to regain mastery, and self-enhancement. More specifically, meaning involves a search to understand why the event happened and how it affects a person. This attributional search often includes an attempt to answer the question of why the event happened, what the significance is to the person, and how it may impact their life in the future. Mastery describes how a person attempts to gain control over the event and their life. Often this is centered on beliefs of personal control, including thoughts surrounding preventing a similar event in the future and how one can cope with the current incident. Self-enhancement includes the process of attempting to restore self-esteem lost as a result of the negative experience, regardless of their perceived control over the event. Thus, the process of regaining self-esteem may lead to self-enhancement that may help aid adjustment to cope with future negative occurrences. This may include social comparisons to assess how one may relate in coping compared to another person who has experienced a similar event, commonly in relation to downward social comparisons.

Taylor (1983) described how 78 recently diagnosed breast cancer patients engaged in a casual search to explain their diagnoses, which subsequently led to attempts to regain mastery and the restoration of self-esteem. The cancer patients' process of searching for meaning in relation to their diagnosis and treatment fit into two forms, including an analysis of a cause related to answering the "why" question and making life changes as a product of rethinking one's priorities and attitudes to meet the goal of having a more satisfying life. Patients' themes also demonstrated the emphasis on gaining personal control in order to feel like one can manage his/her cancer and try to keep it from happening again. Interestingly, the author found that patients' beliefs included personal control over cancer and the belief that the doctors or treatments could also control their cancer, both of which were related to overall positive adjustment. Taylor posited that the sense of mastery can be accomplished by actively engaging in steps that are perceived as directly related to the cancer or by assuming control over related features of one's cancer, such as treatment. More feelings of mastery and control over their chronic condition were then related to better short-term coping. Lastly, the third theme of self-enhancement embodied the process of enhancing the self to restore self-esteem. This included downward comparisons in the hopes of preserving a positive sense of self and better coping when comparing oneself to another cancer patient who they perceived as less adjusted. Therefore, the comparisons made the patients feel better about their coping process and thus enhanced their self-esteem. In sum, this cognitive process of transitioning through these three themes enabled patients to adapt following a negative event and hopefully led to successful adjustment.

Reformulated Learned Helplessness Model

Another model that examines the adjustment process to adverse experiences includes the learned helplessness hypothesis that postulates that learning that an outcome is uncontrollable can lead to deficits in three areas: motivational, cognitive, and emotional (Maier & Seligman, 1976). The cognitive portion encompasses the belief that mere exposure to uncontrollability is not sufficient to make a person feel helpless. Instead, the person must learn to expect that outcomes are uncontrollable in order to feel helpless. The motivational piece includes a lack of initiating responses due to expectations that the outcome is not controllable. Additionally, the emotional deficits consist of the idea that depressed affect or feelings of helplessness result after learning that outcomes are uncontrollable. This hypothesis did not fully encapsulate the idea of helplessness, because it did not distinguish between situations in which outcomes were uncontrollable for all individuals and those that were only uncontrollable for some individuals, deemed universal versus personal helplessness. In addition, it did not describe how to explain the difference between general and specific helplessness, or the discrepancy between chronic and acute, and thus the re-formulated model was created (Abramson, Seligman, & Teasdale, 1978). The reformulated learned helplessness model postulates that when a person feels helpless, he/she seeks to make an attribution as to why. This casual attribution subsequently determines the generality and chronicity of any resulting deficits and may affect self-esteem for handling future events.

In relation to universal versus personal helplessness in the reformulated model, Abramson and colleagues (1978) also made a distinction between internal versus external locus of control. It was noted that individuals can fall under either category of universal versus personal helplessness and also differ on their perceived locus of control. More

specifically, in relation to failure, universally helpless people make external attributions, whereas personally helpless people make internal attributions. In relation to generality and chronicity of deficits, the model characterizes deficits as general, occurring in a broad range of situations, or specific, occurring in a narrow range of situations. Moreover, the course of helplessness can range from chronic, recurrent or long-standing, to transient, when deficits occur over a short period of time. The reformulated model was thus able to make more specific predictions on how making an attribution after feeling helpless can determine the subsequent course of outcomes. In addition, it adds the element of determining severity, which encompasses how strong a deficit is any one time in a given situation. Research on deficits produced by learned helplessness paradigms showed that non-depressed students given inescapable noise or unsolvable problems failed to escape the noise (Glass, Reim, & Singer, 1971; Hiroto & Seligman, 1975; Klein & Seligman, 1976)), failed to solve anagrams (Benson & Kennelly, 1976; Gatchel & Proctor, 1976; Hiroto & Seligman, 1975), and failed to see patterns in the anagram task (Hiroto & Seligman, 1975). Both learned helplessness models are able to describe the motivational and cognitive deficits, but the reformulated model explains why the expectation for not completing the task or failing must be global to generalize to other situations and stable enough to sustain the brief time between test intervals (Abramson, Seligman, & Teasdale, 1978).

Related to depression, the reformulation of learned helplessness explains how learning that an outcome is uncontrollable can produce motivational, cognitive, and emotional dimensions associated with depression (Abramson et al., 1978). Abramson and colleagues argue that the original model failed to explain the relationship between helplessness and depression because expectation of uncontrollability is not sufficient to

describe depressed affect, failed to explain how lowered self-esteem and internal attributions play a role, and did not describe how variations in generality, chronicity, and intensity can affect depression. Perceived lack of control may lead to a heightened vulnerability to depression, but it is not sufficient to cause depressed feelings. In relation to self-esteem, Abramson and co-authors found that those who perceived their helplessness as personal showed lower self-esteem than those who perceived their helplessness as universal, which corresponded to feelings of internal versus external locus of control. Additionally, depressed individuals tend to make more stable and global attributions for negative occurrences. In relation to making attributions for success and failure, depressed individuals tend to make internal, global, and stable attributions for failure, and often make external, specific, and less stable attributions for success (Klein, Fencil-Morse, & Seligman, 1976; Rizley, 1978). The intensity of the three deficits related to depression depends on the strength or certainty of the perceived controllability, and self-esteem and affective components depend on the importance of the outcome (Abramson, Seligman, & Teasdale, 1978). The deficits resulting from learned helplessness may lead to a person developing a negative attributional style, whereby a person is more likely to expect future negative events to occur and to develop depressed feelings. More recently, these relationships have been examined among patients with multiple sclerosis (Vargas & Amett, 2013), chronic pain (Samwel, Evers, Crul, & Kraaimaat, 2006), and acute myocardial infarction (Smallheer, 2011), showing how the model may predict depression and other adjustment processes in multiple chronic disease populations.

Self-Blame Attributions

Another way of describing the attribution process is the concept of self-blame. Janoff-Bulman (1979) made a distinction between the tendency for some individuals to attribute their illnesses to their own behaviors, referred to as behavioral self-blame, and the tendency for some individuals to attribute their conditions to their dispositions, referred to as characterological self-blame. Behavioral self-blame is deemed to encompass an effort attribution, whereas characterological self-blame is deemed to describe an ability attribution. The concept of self-esteem deficits outlined by the reformulated learned helplessness model (Abramson, Seligman, & Teasdale, 1978) when making an attribution of failure to factors that are controllable aligns with behavioral self-blame and the attribution made to uncontrollable events corresponds with characterological self-blame. Self-blame departs from the reformulated learned helplessness model because Janoff-Bulman (1979) predicts that certain types of internal attributions can aid adjustment.

Janoff-Bulman (1979) outlined how behavioral and characterological self-blame attributions may affect adjustment processes. Janoff-Bulman hypothesized that characterological self-blame is maladaptive for adjustment because blame is attributed to one's personality that is presumed to be non-modifiable. This type of internal attribution is similar to what was described in the reformulated learned helplessness model (Abramson, Seligman, & Teasdale, 1978), and is akin to what Heider (1958) suggested social actors create following an event. In contrast, Janoff-Bulman (1979) posited that behavioral self-blame is adaptive for adjustment because blame is attributed to modifiable factors, thereby increasing control appraisals. Enhancements in control appraisals are predicted to be the mechanism that links behavioral self-blame attributions to improved adjustment. It is

noteworthy that Janoff-Bulman's predictions (1979; 1992) about the benefits of behavioral self-blame attributions rest on the presumption that behavior is controllable. In reality, it is likely that the controllability of any given behavior ranges along a continuum of high to low, and that cognitive and motivational processes affect where one views a behavior along that continuum.

Early work on self-blame attributions examined how self-blame affects perceptions of events and distress. Janoff-Bulman's (1979) original work on attributions assessed the relationship between both types of self-blame attributions and depression among 129 undergraduate women who were classified into depressed versus non-depressed groups. Participants were asked to react to four scenarios as if they were happening to them, including a car accident on a snowy winter day, a social invitation being rejected by a new friend, an urgent call from a roommate results in taking down the wrong number and the roommate cannot return the call, and an intense love relationship is ended when the person's boyfriend leaves her and immediately gets involved with another woman. The participants subsequently responded to questions assessing blame for each of the four cases, including questions that specifically examined behavioral and characterological self-blame. Results indicated a significant difference between depressed and non-depressed college students on their endorsement of characterological self-blame, but not behavioral self-blame: depressed students showed higher characterological self-blame attributions towards the scenarios versus the non-depressed students. Additionally, characterological self-blame followed from attributions to uncontrollable factors. Janoff-Bulman's second study examined self-blame attributions among rape victims based on responses by counselors at 38 rape crisis centers. Results indicated that among those who blamed themselves, behavioral self-blame was

reported significantly more than characterological self-blame. Moreover, those who endorsed characterological self-blame blamed themselves significantly more for their rape than did those who endorsement behavioral self-blame.

Timko and Janoff-Bulman (1985) examined attributions among 42 women who underwent a mastectomy as treatment for breast cancer, and found that characterological self-blame, as measured by attributions to one's personality, was negatively associated with good psychological adjustment, whereas behavioral self-blame was positively associated with good adjustment. In addition, the relationship between behavioral self-blame and coping was mediated by perception of vulnerability: future avoidability of cancer was strongly associated with perceived past avoidability, and past avoidability was positively predicted by behavioral self-blame attributions. Interestingly, they also found that those who made personality attributions were less likely to believe that their mastectomy treatment was successful in removing all their breast cancer.

Self-Blame Attributions and Adjustment

Despite Janoff-Bulman's (1979) predictions, behavioral and characterological self-blame attributions have been associated with mostly negative adjustment outcomes in samples of patients diagnosed with various forms of cancer. For example, both characterological and behavioral self-blame have been associated with poor psychological functioning in women with breast cancer (Glinder & Compas, 1999). More specifically, the authors found that behavioral self-blame was related to poor adjustment near the time of a breast cancer diagnosis, and that characterological self-blame was related to poor adjustment six and 12 months post-diagnosis. Likewise, Bennett, Compas, Beckjord, and Glinder (2005) investigated relationships between self-blame and symptoms of anxiety and depression

among breast cancer patients. Results indicated that both behavioral and characterological self-blame were associated with distress at four, seven, and 12 months post-diagnosis. Malcarne et al. (1995) reported that characterological self-blame was unrelated to psychological distress immediately post-diagnosis, but was related to greater distress four months post-diagnosis in a sample of adult male and female cancer patients. Additionally, they found that behavioral self-blame was related to distress only when patients concurrently experienced characterological self-blame.

A cross-sectional study examined relationships between self-blame, self-forgiving attitude, mood disturbance (positive and negative emotional states), and quality of life among 123 women with breast cancer (Friedman et al., 2007). The authors used a somewhat different conceptualization of self-blame, which included questions about 11 factors that women may attribute to the development of their breast cancer and/or could affect the course of their cancer in the future. Although they used a different measurement variation on behavioral self-blame, results revealed a positive relationship between self-blame and mood disturbance and a negative relationship between self-blame and quality of life. In relation to a specific dimension of health-related quality of life, Scharloo and colleges (2005) examined behavioral blame among 68 patients with head and neck cancer. The authors also used a different variation in their assessment of behavioral self-blame, summing endorsement to items representing illness attributions to smoking, alcohol, and own behavior; higher scores represented stronger beliefs in one's own behavior causing their cancer. The dimension of health-related quality of life represented social functioning. Similarly, results indicated that behavioral attributions were negatively related to social functioning: greater blame on behavioral illness attributions was associated with worse social functioning.

More recently, researchers have examined effects of self-blame attributions in patient samples with other chronic health conditions. For example, among individuals with genital herpes, results showed that characterological self-blame, but not behavioral self-blame, was a significant predictor of maladjustment (Manne & Sandler, 1984). Plaufcan, Wamboldt, and Holm (2012) examined both types of self-blame attributions and their relations to psychological (e.g., depression and anxiety) and clinical outcomes (e.g., health-related quality of life and breathlessness) among patients with chronic obstructive pulmonary disease. Results showed that behavioral self-blame was negatively associated with symptoms of depression, whereas characterological self-blame was positively associated with symptoms of depression. Furthermore, participants who endorsed the maximum possible score for behavioral self-blame reported fewer symptoms of depression and less impairment in health-related quality of life than their counterparts who scored lower on the measure of behavioral self-blame. Among patients with inflammatory bowel disease,Voth and Sirois (2009) found a positive relationship between behavioral self-blame and adjustment between nine and 11 years post diagnosis.

Only a handful of studies have examined Janoff-Bulman's (1979) predictions about the role of perceived control in the adjustment effects of self-blame. For example, Malcarne, Compas, Epping-Jordan, and Howell (1995) assessed perceptions of control over cancer reoccurrence as a mediator of the relationship between both behavioral and characterological self-blame and psychological distress from baseline to four-month follow-up among 72 cancer patients. Perceptions of control were not related to either type of initial self-blame or Time 2 psychological distress, and control beliefs did not mediate the relationship between baseline self-blame and psychological distress at follow-up. Moreover, Bennett, Compas,

Beckjord, and Glinder (2005) examined perceptions of control as a mediator between both types of self-blame attributions and distress (symptoms of anxiety and depression) among 115 breast cancer patients at baseline, seven, and 12 months post-diagnosis. They also found that perceptions of control did not mediate the relationships between self-blame and both types of psychological distress.

Additionally, Bennett, Howarter, and Clark (2013) examined the relationship between self-blame and perceptions of control among 129 cardiac rehabilitation patients. Results indicated that characterological self-blame was negatively associated with general control over one's cardiac health and control over recovery; these associations are consistent with predictions outlined by Janoff-Bulman (1979). Results also indicated that behavioral self-blame attributions specific to diet and exercise were positively related to perceived control over recurrence, again consistent with theory. However, perceived control was un-related to psychological distress. Overall, then, very few studies have tested the theorized mediation by perceived control. From the three studies identified, though, evidence is mixed regarding whether perceived control acts as a mediator of the relationship between self-blame and adjustment.

Together, results suggest negative adjustment outcomes among cancer patients engaging in both forms of self-blame. Findings are mixed using patient samples with other chronic illnesses; most suggest negative outcomes following characterological self-blame, but are mixed linking behavioral self-blame to adjustment. Additionally, many of the studies cited above examine causal origins of various forms of cancer, which are complex in etiology. More recently, researchers have begun to test the hypotheses outlined by Janoff-Bulman (1979) regarding self-blame and control in predicting *physical* health outcomes in

the face of a chronic disease diagnosis. For example, Costanzo, Lutgendorf, Bradley, Rose, and Anderson (2005) examined associations between beliefs about cancer, health practices, and distress among 134 gynecologic cancer survivors. Results showed that participants who made positive behavior changes and obtained regular cancer screenings were likely to attribute their cancer to controllable causes (e.g., stress, unhealthy lifestyle) or environmental toxins. Furthermore, believing that an unhealthy lifestyle or diet caused one's cancer was related to distress only among women who had not made positive changes in their diet and exercise patterns since their diagnosis.

Additionally, Rabin and Pinto (2006) examined the association between cancer-related beliefs and factors that could prevent recurrence among 65 breast cancer survivors. They found that participants who believed that insufficient exercise, unhealthy diet, or alcohol consumption contributed to their cancer were more likely to make healthy changes than their counterparts who did not attribute blame to those behaviors. Consistent with the abovementioned studies, Lebel and colleagues (2013) found that behavioral self-blame (measured by beliefs that cancer was caused by tobacco or alcohol use) predicted positive health changes, including changes to diet, exercise, and smoking behavior, among 206 survivors of head and neck cancer. Thus, these studies indicate the potential impact that self-blame (specifically behavioral self-blame) may have on physical health outcomes, although these ideas have not been widely tested among cardiovascular disease (CVD) patients.

Cardiovascular Disease

Following a cardiovascular event, most patients engage in a casual search to understand why it occurred (i.e., why did this happen to me?). Creating an attribution, thus, is a salient part of the adjustment process, especially when etiologic factors can be identified.

In CVD, a disease for which specific health behaviors (Roger et al., 2011) have been linked to onset, patients readily search for a cause. CVD is the number one cause of death in the U.S., accounting for an average of 1 death every 39 seconds (Roger et al., 2011). Risk for CVD has etiologic links among behavioral and psychological risk factors. A majority of the risk factors is associated with modifiable behaviors that include tobacco use, hypertension, physical inactivity, and unhealthy diet. Psychosocial factors contribute independently to the risk of CVD, and include depression and anxiety (Rosengren et al., 2004). Because most of the risk factors for CVD are modifiable, cardiac rehabilitation is recommended as a secondary prevention program to decrease rates of recurrence of future cardiac events by targeting healthy lifestyle change (Balady, et al., 2007).

Cardiac rehabilitation programs have demonstrated numerous positive health outcomes among patients with CVD. Reviews and meta-analyses show reduced risk of all-cause mortality (OR = .80) and cardiac mortality (OR = .74), reduction in modifiable risk factors (cholesterol, triglyceride level, systolic blood pressure), and lower rates of self-reported smoking (Barzi et al., 2003) following participation in cardiac rehabilitation. Additionally, research determined that adherence to a lifestyle modification program, which combined consumption of a heart healthy diet, regular exercise, and stress management, reduced the likelihood of mortality and a secondary cardiac event (Lisspers et al, 2005). A meta-analysis examining 23 randomized controlled trials evaluating the additional impact of psychosocial intervention of rehabilitation for individuals with coronary artery disease showed these treatments also reduced mortality and morbidity (Linden, Stossel, & Maurice, 1996). The investigators also found that the addition of a psychosocial intervention was associated with less psychological distress, and improvement of some biological risk factors.

Studies portraying the importance of psychosocial intervention among patients with CVD highlight the impact that psychosocial variables have on adjustment among patients recovering from CVD.

Self-Blame and Cardiovascular Disease

Cardiac-related self-blame attributions have been associated with both positive and negative health outcomes. For example, Bennett, Howarter, and Clark (2013) investigated associations between self-blame attributions and distress among patients with CVD who participated in a cardiac rehabilitation (CR) program. Results showed behavioral self-blame was positively associated with symptoms of anxiety and depression at the beginning of CR, as well as at the end of the program. Contrary to the majority of the abovementioned studies, Bennett et al. found that characterological self-blame was not significantly predictive of distress. Additionally, Harry, Bennett, Clark, Howarter, and Eways (2015) examined both types of self-blame attributions as predictors of cardiac symptom experiences in 93 patients with CVD at the beginning of CR and 21 months later. Results indicated that both baseline behavioral and characterological self-blame were positively associated with cardiac symptom experiences cross-sectionally, but only characterological self-blame predicted cardiac symptom experiences 21 months later. These studies demonstrate how both types of self-blame may affect psychological adjustment and physical health consequences among patients with CVD.

Overall, findings have been mixed on how behavioral and characterological self-blame relate to physical and mental health outcomes in chronic illness, and specifically in patients with CVD. Of great importance, most previous studies examining the effects of self-blame attributions have used a one-item measure for each type of self-blame adapted for each

chronic illness. For example, Bennett, Howarter, and Clark (2013) used the following questions adapted from studies examining patients with cancer (e.g., Glinder & Compas, 1999): for behavioral self-blame, "In general, how much do you blame yourself for your past behaviors? In other words, how much do you blame yourself for engaging in behaviors that contributed to your cardiac event?" and for characterological self-blame, "How much do you blame the type of person you are (your personal characteristics) for your cardiac event? In other words, do you blame yourself for being the type of person who has bad things like a cardiac event happen to them?" Responses to both questions were assessed on a 4-point scale, ranging from 1 (*not at all*) to 4 (*extremely*), with higher scores representing more self-blame. One-item measures for each of the self-blame constructs likely do not accurately capture self-blame attributions in their conceptual complexity. Furthermore, the psychometric properties of single-item measures of psychological constructs have been widely criticized. For instance, the internal consistency (i.e., reliability) statistic cannot be computed (Clark & Watson, 1995). Additionally, single items are more susceptible to measurement error. Moreover, multiple-item scales are designed to sample a broader range of meaning to cover the full range of a construct (Hoeppner, Kelly, Urbanoski, & Slaymaker, 2011).

Thus, one possible reason for the discrepant findings documented above is the lack of a validated, multiple-item measure of self-blame. Therefore, the 11-item Cardiac Self-Blame Attributions (CSBA) scale was created to fill this gap in the literature. After feedback and input from two subject matter experts, as well as pilot-testing with CR patients, the original CSBA scale consisted of 14 items. The scale was administered individually to eligible CVD patients in the Intensive Care Unit at Truman Medical Center, while they were recovering

from a cardiac procedure pre-hospital discharge. A test of the scale with a sample of 107 patients with CVD supported the two-factor structure. Results yielded an 11-item, 2-factor structure explaining 70.55% of the variance. Two reverse-worded items were removed due to problems with process validity and reliability. Pattern coefficients ranged from .60 to .92. These two factors represented behavioral self-blame (with 6 items) and characterological self-blame (with 5 items). Internal consistency for the 2 factors indicated good reliability (α =.93, and α = .88, respectively). The CSBA also showed good discriminant validity with Exercise Self-Efficacy (r = -.15, p > .05). These findings suggest that the CSBA scale is a reliable and valid measurement tool among patients with CVD (Harry et al., 2018). However, it has not been investigated whether the CSBA scale is *predictive* of physical and mental health recovery outcomes among CVD patients, and this empirical question is the focus of the proposed study.

Gaps in the Literature and Hypotheses

Given previous research linking self-blame to psychological distress and physical health outcomes among chronic illness populations, it is important to better understand these relationships among patients with CVD. And, the CSBA scale has not been examined in relation to health outcomes among CVD patients. Therefore, to fill these gaps in the literature, the project aimed to: (1) further validate the CBSA scale (and the factor structure of the two proposed sub-scales), and (2) examine whether the CBSA predicts mental and physical health recovery outcomes (e.g., depressive symptoms, health-related quality of life, heart-healthy diet, and functional capacity) before and after CR. Data were collected from CVD patients at the beginning of CR, and when they graduated from their CR program. Additionally, a secondary aim was to assess whether the relationship between both types of

self-blame attributions and health outcomes were mediated by control appraisals, as predicted by Janoff-Bulman (1979). Generally, it was predicted that the behavioral self-blame sub-scale would predict positive cardiac outcomes cross-sectionally and prospectively, and that the characterological self-blame sub-scale would predict negative cardiac outcomes cross-sectionally and prospectively. Thus, the proposed project tested the following hypotheses:

Hypothesis for Psychometric Properties of the CSBA scale

Hypothesis #1: Exploratory factor analysis of the CSBA at the beginning of CR will yield a two-factor structure representing behavioral and characterological self-blame. Those sub-scale scores will be calculated and used in subsequent analyses.

Hypotheses for Behavioral Self-Blame and Outcomes

Hypothesis #2: Behavioral self-blame at the beginning of CR will be positively related to good health outcomes (e.g., health-related quality of life, adherence to heart healthy diet, and functional capacity) and negatively related to poor health outcomes (e.g., depressive symptoms) cross-sectionally.

Hypothesis #3: Behavioral self-blame at the beginning of CR will be positively related to good health outcomes (e.g., health-related quality of life, adherence to heart healthy diet, and functional capacity) and negatively related to poor health outcomes (e.g., depressive symptoms) at the end of CR (controlling for baseline levels of the health outcomes).

Hypotheses for Characterological Self-Blame and Outcomes

Hypothesis #4: Characterological self-blame at the beginning of CR will be negatively related to good health outcomes (e.g., health-related quality of life, adherence to

heart healthy diet, and functional capacity) and positively related to poor health outcomes (e.g., depressive symptoms) cross-sectionally.

Hypothesis #5: Characterological self-blame at the beginning of CR will be negatively related to good health outcomes (e.g., health-related quality of life, adherence to heart healthy diet, and functional capacity) and positively related to poor health outcomes (e.g., depressive symptoms) at the end of CR (controlling for baseline levels of the health outcomes).

Mediation Hypotheses with Behavioral Self-Blame, Control, and Outcomes

Hypothesis #6: Control appraisals at the beginning of CR will mediate the association between baseline behavioral self-blame and health outcomes at the end of CR. That is, baseline behavioral self-blame will be positively related to baseline control appraisals, which, in turn, will be positively related to good health outcomes (e.g., health-related quality of life, adherence to heart healthy diet, and functional capacity) and negatively related to poor health outcomes (e.g., depressive symptoms; controlling for baseline levels of health outcomes).

Mediation Hypotheses with Characterological Self-Blame, Control, and Outcomes

Hypothesis #7: Control appraisals at the beginning of CR will mediate the association between baseline characterological self-blame and health outcomes at the end of CR. That is, baseline characterological self-blame will be negatively related to baseline control appraisals, which, in turn, will be positively related to good health outcomes (e.g., health-related quality of life, adherence to heart healthy diet, and functional capacity) and negatively related to poor health outcomes (e.g., depressive symptoms; controlling for baseline levels of health outcomes).

CHAPTER 3

METHODOLOGY

Participants

Institutional Review Board approval was obtained prior to study initiation. Participants were recruited through a Phase II CR program at Truman Medical Center (TMC), an urban safety-net hospital serving predominantly low-income, largely uninsured, ethnically-diverse patients with the following eligibility criteria: 1) Enrolled in CR at TMC, 2) English speaking, 3) at least 18 years of age, and 4) lack of physical or cognitive impairments that would inhibit their ability to complete study materials. CVD patients are referred to CR for a variety of CVD-related diagnoses and procedures, including coronary artery disease, acute coronary event, percutaneous coronary intervention, heart surgery, and chronic heart failure (Balady et al., 2007). Phase II CR programs typically include monitored exercise classes (i.e., patients' heart rates are monitored while using treadmills, exercise bikes, etc.), and additional life-style change classes focusing on diet, stress management, and pharmacology. These programs usually consist of exercise sessions three times per week for 12 weeks, for a total of 36 sessions. The lapsed time between hospitalization (Phase I) to Phase II varies by the severity of the cardiac event, with entry into Phase II when exercise is not a risk. Trained cardiac rehabilitation nurses and staff lead CR sessions. For insured patients, most CVD diagnoses come with 36 sessions covered at little to no out-of-pocket expense. Participants received $10 at baseline and at follow-up, for a total of $20.

Recruitment efforts lasted from August 2016 to February 2018. During this time, 140 eligible participants were approached for this study, and 45 declined to participate (32%). Figure 1 outlines the reasons cited for declining to participate. Thus, the sample consisted of

95 patients at Time 1 and 50 patients at Time 2. Of the 45 participants who did not complete Time 2, 26 dropped out of CR, 12 are still enrolled in Phase II and will not graduate for at least six weeks, and seven lost their medical data and health outcomes do to a medical record archive. Reasons cited for attrition are also listed below in Figure 1.

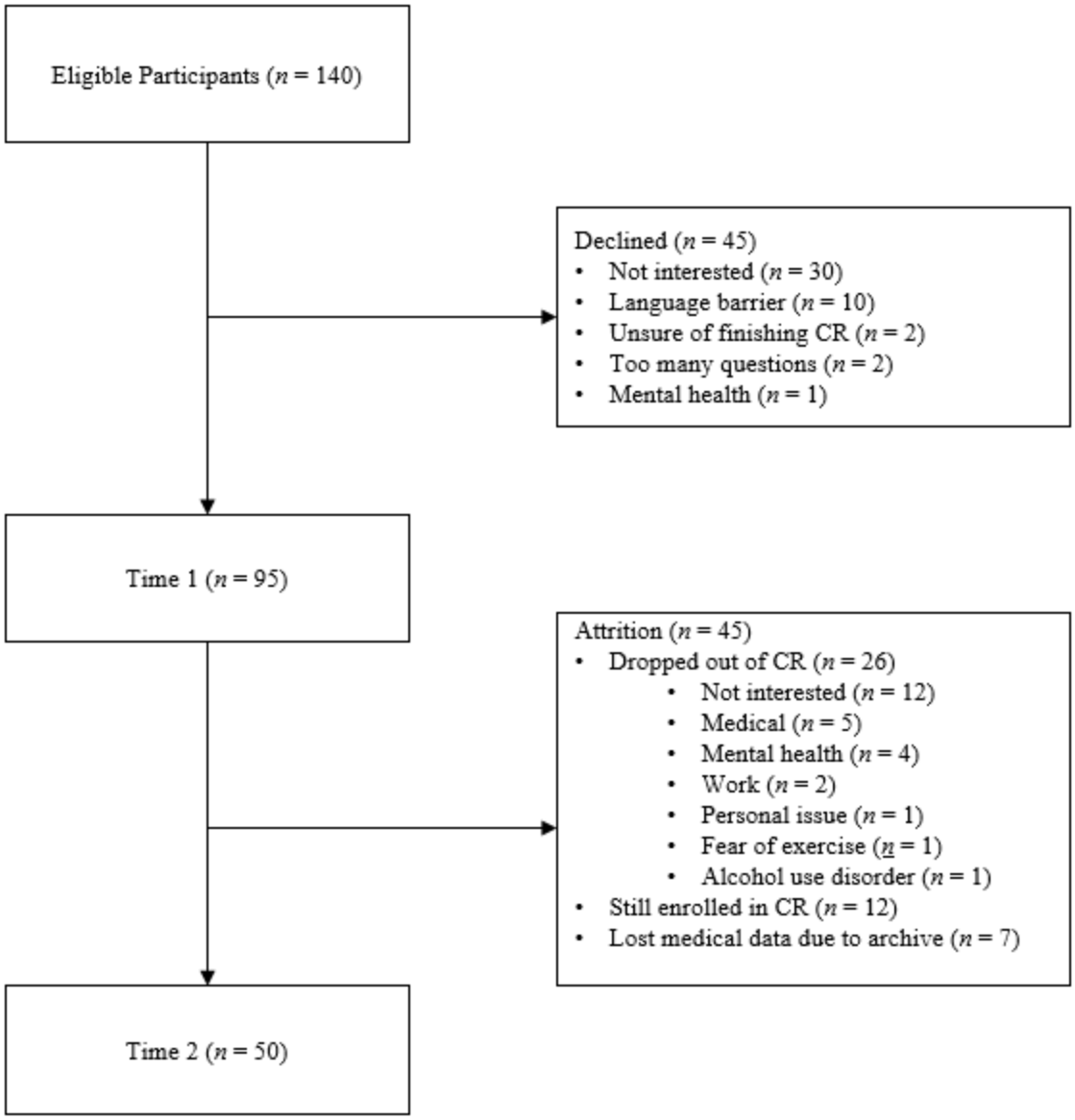

Figure 1. Flow diagram of participant recruitment

Procedures

Baseline (Time 1). The purpose of the study was introduced to eligible patients during their intake/orientation appointments at TMC's CR by one of the CR staff members. If patients expressed interest in the study, the research staff obtained the patient's signature on the informed consent document, and a baseline questionnaire and envelope was provided. Participants completed the brief questionnaire on-site at the CR. Participants placed completed questionnaires in sealed envelopes in a locked box at the CR program. At that time, CR staff members provided participants with a $10 TMC payment voucher that could be taken immediately to the cashier's office for cashing. Granting informed consent allowed us access to participants' responses on several health outcome measures used by TMC's CR with all its patients (see below for more details). Participants also completed this study's questionnaire that contained demographic questions, the CSBA scale, and a measure of perceived control. Thus, data from this study were abstracted from participants' medical charts at TMC's CR and from the self-report questionnaire administered at Time 1 and Time 2.

Follow-up (Time 2). After participants completed their final session of CR (typically three to six months after their initial appointment) they participated in an exit-interview-type appointment. CR staff members reviewed their exercise progress during the program, and developed a plan to sustain those behavioral changes post-CR. During this final appointment, participants were asked to complete the study's follow-up questionnaire. Participants left their completed questionnaires in the locked box, and CR staff members provided them with their $10 payment vouchers. Time 2 health outcome measures were

collected from participants' medical records at TMC's CR and reflect their self-reported physical and mental health at the time of their last exercise session.

Measures

Questionnaire packets including demographic items and validated measures were used at Time 1 and Time 2. In addition, a number of validated and commonly-used health outcome measures were abstracted from participants' files at TMC's CR at Time 1 and Time 2. These measures are ones already collected by TMC's CR at the beginning and end of their program. These data are used to track patients' progress during the program, and for accreditation purposes to the CR's governing body.

Demographics. The following demographic data were extracted from patients' medical records: age, sex, race, insurance status, education, employment status, diagnosis, risk stratification (a measure of risk for disease progression), and number of CR sessions completed. The following demographic variables were added to the questionnaire: marital status and income.

Cardiac Self-Blame Attributions (in questionnaire). The Cardiac Self-Blame Attributions (CSBA) Scale (Harry et al., 2018) is an 11-item self-report measure assessing self-blame attributions. The scale consists of two subscales, including behavioral self-blame (6 items; BSB) and characterological self-blame (5 items; CSB). Scores are assessed on a five-point scale ranging from zero (*not at all*) to four (*completely*). Scores were summed for each subscale, ranging from 0 to 24 for BSB and 0-20 for CSB, with higher scores indicating more self-blame for each dimension. Both behavioral and characterological scales have shown good to excellent reliability in a sample of underrepresented patients with CVD (α =.93, and α = .88, respectively; Harry, et al., 2018), and both subscales showed excellent

reliability in the present sample (α = .93 and α = .90, respectively), See Appendix A-1 for a copy of this measure.

Control Appraisals (in questionnaire). Control appraisals were measured by the Control Attitudes Scale-Revised (CAS-R; Moser et al., 2009). The CAS-R is an 8-item self-report measure that assesses perceived control over cardiac-related symptoms and behavior related to a heart condition. Questions are measured on a five-point scale, ranging from one (*totally disagree*) to five (*totally agree*). Two items were reverse-scored and then summed with the remaining six items, with scores ranging from eight to 40; higher scores indicate more perceived control. The CAS-R has shown acceptable reliability in a previous sample of CVD patients, including patients with acute myocardial infarction and heart failure (α = .72, α = .76, respectively; Moser et al., 2009), and showed good reliability in the present sample (α = .80). See Appendix A-2 for a copy of this measure.

Depressive Symptoms (abstracted from medical file). Depressive symptoms were measured with the Patient Health Questionnaire (PHQ-9; Kroenke & Spitzer, 2007). The PHQ-9 is a 9-item self-report measure that assesses the frequency of depressive symptoms over the past two weeks. Questions are measured on a four-point scale ranging from zero (*not at all*) to three (*nearly every day*). Scores were summed and range from 0 to 27, with higher scores indicating more depressive symptoms. The PHQ-9 has shown excellent reliability in a cardiac sample (α = .90; Stafford, Berk, & Jackson, 2007). The total score for each patient was extracted from patients' medical records, so it was not possible to calculate reliability coefficients for this variable. However, this is a widely-used measure that has demonstrated reliability in cardiac patients (e.g., Bennett, Buchanan, Jones, & Spertus, 2015; Stafford, Berk, & Jackson, 2007).

Health-related Quality of Life (abstracted from medical file). Health-related quality of life (HRQoL) was measured with the Short Form-12 Health Survey (SF-12; Ware, Kosinski, & Keller, 1996). The SF-12 is a 12-item self-report measure assessing health-related quality of life. The 12 items include one or two items from each of the eight health concepts including: physical functioning, physical role limitations, bodily pain, general health, vitality (energy/fatigue), social functioning, emotional role limitation, and mental health (psychological distress and psychological well-being). The 12 items comprising the eight quality of life domains are calculated into two summary measures covering physical (Physical Component Summary, PCS) and mental (Mental Component Summary, MCS) health. The PCS score (physical HRQoL) primarily represents general health, mobility, amount accomplished during physical activity, ability to climb stairs, and work limitations resulting from physical problems or pain. The MCS (mental HRQoL) score primarily measures feelings of depression and anxiety, social activity, carelessness, and impact of feelings on amount accomplished. Each scale has a possible range from 0-100; higher scores indicate better physical and mental health. Both the PCS and MCS have been shown to be reliable among cardiac patients (α = .84, α = .81, respectively; Lim & Fisher, 1999). The total score for each patient was extracted from their medical record, so it was not possible to calculate reliability coefficients for this variable.

Healthy Diet (abstracted from medical file). A heart-healthy diet was assessed with Rate Your Plate (RYP; Gans et al., 1993). RYP is a 24-item scale that covers 24 food categories that are formatted into three columns. Column A includes the most "heart-healthy" choices, Column C includes the least "heart-healthy" choices, and Column B is a middle ground between Columns A and C. Responses in column A receive 3 points, column

B receive 2 points, and column C receive 1 point. Scores were summed and ranged from 24 to 72, with higher scores indicating a more heart-healthy diet. RYP has shown good reliability in previous research (Gans, Hixson, Eaton, & Lasater, 2000), but no Chronbach's alphas were listed in the literature. The total score for each patient was extracted from their medical record, so it was not possible to calculate reliability coefficients for this variable.

Functional Capacity (abstracted from medical file). Functional capacity was assessed by the Duke Activity Status Index (DASI; Hlatky et al., 1989). The DASI is a 12-item quality of life questionnaire validated for CVD (Hlatky, 1989; Nelson, Herndon, Mark, Pryor, Califf, & Hlatky, 1991) and measures major activities of daily living, such as person care, ambulation, sexual function, household tasks, and recreational activities. Each item is weighted on the known metabolic cost of each activity, and weights of positive terms are summed to create a total score for each patient. Scores are measured on a semi-continuous scale, ranging from 0 to 58.2; higher scores indicate better physical functioning, and a score of zero reflects an inability to perform basic activities of daily living. The DASI has shown good reliability in previous samples of cardiac patients (α = .82; Pressler et al., 2010). The total score for each patient was extracted from their medical record, so it was not possible to calculate reliability coefficients for this variable.

Data Analysis

Preliminary Statistics. Preliminary data analyses were conducted using SPSS 24.0 (IBM Corp, 2016). To assess for missing data, Little's test (Little, 1988) was conducted to identify if data are missing at a random pattern. The Little's MCAR test resulted in a chi-square = 23.442 (df = 24, p > .05), which indicates that the data were missing at random and no identifiable pattern exists. As such, Expectation-Maximization (EM) imputation was used

to account for missing data. Next, assumptions of multivariate normality were tested. Normality distributions were examined using skewness and kurtosis statistics and histogram plots. Levene's statistic was utilized to examine homogeneity of variance. *T*-tests and chi-square analyses were used to examine demographic (e.g., age, gender, race, income, education, employment status, and insurance status) differences between participants who completed both time points of the study and those who did not. Correlation analyses and *t*-tests were conducted between participant characteristics and outcome variables in order to identify potential covariates. Sociodemographic variables found to be significantly correlated with the outcome variable were included as covariates in the analyses.

Hypothesis Testing. Hypothesis #1 was tested through exploratory factor analysis (EFA). After employing Parallel Analysis (PA) and Velicer's Minimum Average Partial (MAP) test to assess the number of factors that should be extracted, a correlation matrix was calculated to determine if the CSBA items were related. PA and MAP approaches were used to determine the number of factors because previous research has shown that they are more accurate and show little variation when compared to the scree test and retaining eigenvalues greater than 1.0 (Henson & Roberts, 2006; Zwick & Wllicer, 1986). Next, an EFA was conducted with Principal-Axis Factoring (PAF) extraction method with direct oblimin rotation with the set number of factors yielded from the PA and MAP test. Pattern coefficients were reviewed to ensure high factor loadings (greater than .30) on either behavioral self-blame or characterological self-blame. Total variance explained by the extracted factors was examined, with higher percentages of total variance explained indicating that a strong relationship exists among the CSBA scale. Internal consistency for each of the factors was calculated to assess the scale reliability. Test-retest reliability was

also examined to assess if scores on both types of self-blame at Time 1 were related to self-blame scores at Time 2; a significant and high correlation would indicate good test-retest reliability.

Hierarchical linear regression was used to test the cross-sectional hypotheses (2 and 4) by entering control variables as the first block of predictors, followed by Time 1 behavioral self-blame or characterological self-blame as the second block; the four outcome variables at Time 1 were the dependent variables (DVs) in these models. The longitudinal hypotheses (3 and 5) were tested by entering control variables and Time 1 outcomes as the first block of predictors, and Time 1 behavioral self-blame or characterological self-blame as the second block; the four outcome variables at Time 2 were the DVs in these models.

The mediation hypotheses (6 and 7) were tested using the PROCESS add-on for SPSS (Hayes, 2013). All models were run using Model 4 of Hayes' macro. Estimates of the indirect effects utilized bias-corrected bootstrap confidence intervals at the 95% level based on 1,000 bootstrap samples. Separate models were run for each type of self-blame at Time 1 as the predictor, control appraisals at Time 1 as the mediator, and each of the four health outcomes at Time 2, controlling for Time 1 health outcomes and significant covariates.

CHAPTER 4

RESULTS

Completers and Non-completers

Those who completed both time points of the study (n = 50) did not significantly differ from those who did not complete time two (n = 45) by age [t (95) = 1.09, *n.s.*], race [χ^2 (1) = 2.11, *n.s.*], gender [χ^2 (1) = 1.83, n.s.], insurance status [χ^2 (1) = .00, *n.s.*], income [t(93) = .13, *n.s.*], education [t(94) = -.83, p = *n.s.*], marital status [χ^2 (1) = .49, *n.s.*], or employment status [χ^2 (1) = 1.98, *n.s.*].

Additionally, those who completed both time points of the study (n = 50) did not significantly differ from those who dropped out of CR (n = 26) by age [t(76) = -1.63, *n.s.*], race [χ^2 (1) = 3.41, *n.s.*], gender [χ^2 (1) = 1.31, *n.s.*], insurance status [χ^2 (1) = .00, *n.s.*], income [t(75) = .17, *n.s.*], education [t(75) = -.78, *n.s.*], marital status [χ^2 (1) = .06, *n.s.*], or employment status [χ^2 (1) = 1.31, *n.s.*].

Participant Characteristics

Table 1 provides descriptive statistics for the sample. Ages ranged from 32 to 78 years, with a mean age of 57. The majority of participants was male (61%) and Non-White (51%). Most individuals were married or living with a partner (56%), but approximately 30% reported being single, separated, divorced, or widowed. Of note, 24% did not complete high school or a GED, but 39% completed high school or GED and 25% completed some college or trade school. One-third of the participants earned an annual income of less than $10,000 (33%), and only 24% earned over $30,000. In regards to health variables, the most common diagnoses were percutaneous coronary intervention (36%), myocardial infarction (21%), and coronary artery bypass graph (13%). All participants were stratified by risk for

cardiac disease progression according to the American Association of Cardiovascular and Pulmonary Rehabilitation (2004) guidelines. The CR staff assigned risk stratifications (i.e., low, moderate, or high) based on participants' diagnoses, prior cardiac events, and current risk factors (e.g., comorbid disease, exercise, diet, etc.). Over one-third of the participants were stratified as high risk for disease progression (38%). Of the 36 available sessions, participants completed an average of 28 CR sessions (range: 0 – 36 sessions) over the 12-week Phase II program.

Table 1

Descriptive Statistics of Participant Characteristics

	N = 95
Participant Characteristics	*M* (*SD*)
Age (years)	56.6 (9.3)
CR Sessions	28.0 (*12.5*)
	%
Gender	
Male	60.8
Female	39.2
Race	
White	49.0
African American	44.8
Hispanic	3.1
Other	2.1
Asian	1.0
Marital Status	
Married	35.1
Single, never married	23.4
Divorced	23.4
Living with a partner	8.5
Widowed	5.3
Separated	4.3

Participant Characteristics	*N = 95* *M* (*SD*)
Education	
Less than 9th grade	1.0
Some high school	22.9
High School/GED	38.5
Some college/trade school	25.0
2-year college degree	4.2
4-year college degree	7.3
Graduate degree	1.0
Income	
<$10,000	32.6
$10,000-19,999	21.1
$20,000-29,999	17.9
$30,000-39,999	8.4
$40,000-49,999	6.3
$50,000-59,999	3.2
$60,000-69,999	1.1
$70,000-79,999	1.1
$80,000-89,999	1.1
$90,000-99,999	2.1
≥$100,000	5.3
Employment Status	
Not Employed	62.9
Employed	37.1
Diagnoses	
PCI	35.8
MI	21.1
CABG	12.6
Heart Valve	10.5
CHF	10.5
MI & PCI	5.3
Angina	4.2
Insurance Status	
Private	35.1
Medicaid	29.9
Medicare	20.6
TMC Discount	11.3
Both Medicaid & Medicare	2.1
Risk stratification	
Low	33.3
Moderate	29.2
High	37.5

Note. PCI = percutaneous coronary intervention; MI = myocardial infarction; CABG = coronary artery bypass graph; CHF = congestive heart failure

Preliminary Analysis and Descriptive Statistics

Preliminary data screening and descriptive statistics indicated that the data violated the assumption of normality required by OLS regression: Time 2 RYP (heart-healthy diet) scores were significantly negatively skewed and kurtotic. The bootstrapping procedure employed by conditional process analysis theoretically addresses non-normality, and Hayes (2013) contends that this assumption is one of the least important in OLS regression. In addition, all other assumptions required by Hayes' approach using OLS regression were met.

Analyses assessing for possible covariates (see Tables 2 and 3) revealed that age was significantly related to Time 1 depressive symptoms (r = -.25, p <.05) and employment status was significantly related to depressive symptoms at Time 2 [$t(48)$ = -2.52, p < .05]. Additionally, age was significantly related to Time 1 physical HRQoL (r = .30, p < .05). Income was significantly related to Time 1 heart-healthy diet (r = .23, p <.05) and CR sessions were significantly related to heart-healthy diet at Time 2 (r = .59, p <.05). The following variables were significantly related to Time 1 functional capacity: education (r = .22, p < .05), income (r = .42, p < .05), sex [$t(94)$ = -2.52, p < .05], and employment status [$t(94)$ = 2.94, p < .01]. In addition, the following variables were significantly related to Time 2 functional capacity: CR sessions (r = .35, p <.05), education (r = .35, p <.05), income (r = .40, p <.05), and employment status [$t(47)$ = 2.89, p < .05]. Thus, all significant covariates were included in each relevant analysis. Descriptive statistics for each study variable are presented in Table 4, and correlations among all model variables are presented in Table 5.

Table 2

Correlation Results for Assessing Possible Covariates

	Possible covariate				
Outcome	Age	CR sessions	Education	Income	Risk stratification
Depressive symptoms, T1	**-.25***	.56	-.10	-.09	.03
Mental HRQoL, T1	.08	-.06	.04	.10	.05
Physical HRQoL, T1	**.30***	.11	.03	.11	-.08
Heart healthy diet, T1	-.08	.09	.16	**.23***	.08
Functional capacity, T1	-.07	-.16	**.22***	**.42***	-.06
Depressive symptoms, T2	.12	.13	-.25	-.20	.10
Mental HRQoL, T2	-.01	.07	-.05	-.06	-.02
Physical HRQoL, T2	.04	.17	.25	.16	-.03
Heart healthy diet, T2	-.10	**.59***	.10	.23	.01
Functional capacity, T2	-.21	**.35***	**.35***	**.40***	-.23

*at least $p < .05$
Note. CR = cardiac rehabilitation; T1 = Time 1; T2 = Time 2; HRQoL = health-related quality of life.

Table 3

T-test Results for Assessing Possible Covariates

	Possible covariate									
	Sex		Race		Marital status		Employment status		Insurance status	
Outcome	Female	Male	White	Non-White	Yes	No	Yes	No	Yes	No
Depressive symptoms, T1	6.1 (4.5)	4.6 (4.1)	4.6 (4.0)	5.9 (4.6)	5.0 (4.0)	5.2 (4.1)	4.3 (5.8)	5.8 (4.5)	5.1 (4.3)	6.5 (4.2)
Mental HRQoL, T1	48.7 (12.4)	50.7 (11.0)	50.9 (11.0)	48.9 (12.6)	50.4 (12.6)	50.2 (10.1)	52.6 (12.0)	48.3 (11.1)	49.9 (11.9)	50.4 (8.6)
Physical HRQoL, T1	32.9 (9.1)	35.4 (9.4)	34.7 (9.7)	34.3 (9.1)	34.7 (10.6)	33.8 (8.3)	35.4 (10.4)	33.9 (8.7)	34.9 (9.5)	31.2 (7.5)
Heart healthy diet, T1	45.5 (8.3)	44.9 (11.4)	44.8 (11.1)	45.3 (9.4)	45.3 (9.1)	45.5 (11.2)	47.8 (10.6)	43.6 (9.8)	45.7 (10.5)	41.3 (7.8)
Functional capacity, T1	**17.3 (11.0)**	**23.2 (11.4)***	20.4 (11.4)	21.2 (11.9)	22.5 (12.9)	19.4 (10.1)	**25.2 (12.8)**	**18.3 (9.3)***	20.6 (11.4)	23.4 (12.7)
Depressive symptoms, T2	5.8 (4.6)	6.7 (5.3)	5.8 (3.9)	6.6 (5.6)	5.6 (3.8)	6.3 (5.1)	**4.4 (4.2)**	**7.7 (4.9)***	5.9 (5.0)	8.2 (3.3)
Mental HRQoL, T2	47.2 (10.5)	48.5 (10.7)	48.2 (11.4)	47.5 (10.1)	46.3 (10.0)	49.3 (10.9)	49.7 (9.8)	46.5 (10.9)	48.7 (10.7)	42.0 (6.2)
Physical HRQoL, T2	42.4 (13.2)	43.3 (11.1)	41.4 (9.6)	44.0 (13.7)	40.6 (11.5)	44.8 (12.4)	45.0 (11.0)	41.1 (12.7)	42.6 (10.1)	44.9 (22.9)
Heart healthy diet, T2	51.1 (11.7)	54.9 (9.0)	51.9 (7.2)	54.1 (12.5)	53.6 (12.9)	52.5 (8.3)	55.3 (13.5)	51.6 (7.1)	52.8 (10.8)	55.5 (7.0)
Functional capacity, T2	31.2 (16.8)	35.4 (15.3)	29.4 (13.1)	35.7 (17.1)	34.0 (17.3)	33.8 (15.4)	**40.4 (16.7)**	**28.7 (13.2)***	33.4 (16.0)	34.8 (17.2)

*at least $p < .05$; Mean (SD)

Note. T1 = Time 1; T2 = Time 2; HRQoL = health-related quality of life.

Table 4

Descriptive Statistics for Study Variables

	M	SD	Range	Skew	Kurtosis
T1 BSB	13.97	6.27	0-24	-.57	-.51
T2 BSB	13.45	6.20	0-24	-.34	-.57
T1 CSB	6.97	5.08	0-20	.41	-.53
T2 CSB	7.27	5.06	0-20	.30	-.69
T1 Control Appraisals	20.97	4.99	8-32	-.22	-.24
T2 Control Appraisals	21.41	4.18	8-32	-.01	.33
T1 Depressive Symptoms	5.23	4.34	0-22	1.66	.25
T2 Depressive Symptoms*	6.22	4.86	0-22	.89	1.18
T1 Mental HRQOL	49.94	11.57	20-78	-.43	.10
T2 Mental HRQOL	47.92	10.49	22-64	-.41	-.60
T1 Physical HRQOL	34.44	9.31	13-58	.25	-.31
T2 Physical HRQOL*	42.88	11.99	18-85	.68	1.78
T1 Heart-healthy diet	45.15	10.26	30-87	.97	1.44
T2 Heart-healthy diet*	53.16	10.36	6-71	-2.06	7.89
T1 Functional Capacity	20.88	11.53	2.7-50.7	.76	-.14
T2 Functional Capacity*	33.61	15.95	12.45-85.2	.46	-1.36

*Health outcomes that significantly ($p < .05$) differed between Times 1 & 2

Note. BSB = behavioral self-blame; CSB = characterological self-blame; T1 = Time 1; T2 = Time 2; HRQoL = health-related quality of life.

Table 5

Correlations among Study Variables

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1.	T1 BSB	--	.73**	.74**	.42**	-.07	-.10	.30**	.16	-.33**	-.32*	-.17	-.21	-.15	-.05	.06	.01
2.	T2 BSB		--	.61**	.73**	-.14	-.05	.16	.09	-.22	-.27	-.09	-.18	-.26	.06	.11	.03
3.	T1 CSB			--	.49**	-.05	.07	.31**	.34*	-.35**	-.09	-.05	-.28	-.15	.18	-.05	-.12
4.	T2 CSB				--	-.09	-.04	.33*	.32*	-.31*	-.32*	-.11	-.16	-.28	.05	-.06	-.19
5.	T1 CAS-R					--	.91**	-.38**	-.28*	.40**	.45**	.11	-.04	.09	.04	.06	.01
6.	T2 CAS-R						--	-.35*	-.39**	.37**	.46**	-.01	-.04	.02	.05	-.18	-.02
7.	T1 PHQ-9							--	.77**	-.66**	-.36*	-.16	-.16	-.01*	-.02	-.09	-.15
8.	T2 PHQ-9								--	-.66**	-.50**	.09	-.25	-.01	.06	.03	-.38**
9.	T1 MCS									--	.47**	-.25*	.03	.33**	-.14	-.08	.13
10.	T2 MCS										--	-.03	-.13	.02	-.08	-.16	.13
11.	T1 PCS											--	.46**	-.02	.35*	.39**	.26
12.	T2 PCS												--	.21	.19	.38**	.66**
13.	T1 RYP													--	.12	.08	.23
14.	T2 RYP														--	.19	.32*
15.	T1 DASI															--	.55*
16.	T2 DASI																--

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

Note. T1 = Time 1; T2 = Time 2; BSB = behavioral self-blame; CSB = characterological self-blame; CAS-R = Control Attitudes Scale- Revised; PHQ-9 = Patient Health Questionnaire; MCS = Mental Component Scale; PCS = Physical Component Summary; RYP = Rate Your Plate; DASI = Duke Activity Status Index.

Hypothesized Models

Hypothesis #1 for Psychometric Properties of the CSBA scale

PAF using an oblimin rotation yielded an 11-item, two-factor structure explaining 69.9% of the variance. The final factor solution yielded primary pattern coefficients ranging from .56 to .99, with no cross-loadings greater than .37. These two factors represented BSB (six items) and CSB (five items), and were strongly correlated ($r = .74$, $p < .01$). Internal consistency for the subscales formed in accord with the two factors was excellent ($\alpha = .93$ and .90, respectively), as well as excellent reliability for the total CSBA scale ($\alpha = .94$). Items 1-6 loaded on Factor 1 (reflecting BSB), and Items 7-11 on Factor 2 (reflecting CSB). Table 6 lists the item pattern and structure coefficients.

Results showed that both BSB and CSB had good test-retest reliability in the form of strong, significant associations with both types of self-blame at Time 2 ($r = .73$, $p < .01$; $r = .49$, $p < .01$, respectively). Thus, the CSBA was shown to be a reliable and valid measure in the present sample. Therefore, results supported Hypothesis #1 because the EFA of the CSBA scale yielded a two-factor structure representing BSB and CSB.

Table 6

CSBA Descriptives and Factor Pattern/Factor Matrix Rotated to Oblimin Criterion

			Factors		
	Item stem	*M (SD)*	*1*	*2*	h^2
1.	How much do you think your past behaviors contributed to your cardiac event?	1.97 (1.25)	**.85 (.82)**	.002 (-.23)	.70
2.	To what extent do you accept fault for behaviors that may have caused your cardiac event?	2.41 (1.21)	**.87 (.75)**	-.10 (-.29)	.75
3.	How much do you think your past behaviors contributed to your cardiac event?	2.41 (1.17)	**.86 (.82)**	-.005 (-.23)	.74
4.	To what extent do you believe that a change in your behavior could have prevented your cardiac event?	2.52 (1.19)	**.69 (.73)**	.07 (-.15)	.56
5.	To what extent do you feel accountable when thinking about past behaviors that may have caused your cardiac event?	2.48 (1.19)	**.84 (.81)**	-.002 (-.23)	.73
6.	When discussing possible causes of your cardiac event with important people in your life, to what extent have you blamed your past behavior?	2.16 (1.24)	**.89 (.88)**	.02 (-.22)	.70
7.	How much do you blame the type of person you are for your cardiac event?	1.74 (1.26)	.19 (-.06)	**.62 (.77)**	.45
8.	To what extent do you believe that a change in the type of person you are could have prevented your cardiac event?	1.79 (1.16)	.37 (.74)	**.57 (.20)**	.62
9.	How much do you blame your personality for your cardiac event?	1.13 (1.25)	.02 (.74)	**.86 (.46)**	.70
10.	How much do you blame yourself for being the type of person who has bad things, like a cardiac event, happen to them?	1.14 (1.18)	.09 (.65)	**.67 (.34)**	.46
11.	When discussing possible causes of your cardiac event with important people in your life, to what extent have you blame your personality?	1.15 (1.19)	-.09 (.74)	**.99 (.56)**	.71

Note. Factor 1 = Behavioral self-blame. Factor 2 = Characterological self-blame. Bold indicates items retained on each factor. Pattern coefficients followed by factor coefficients in parentheses. h^2 are the communalities.

Hypotheses for Behavioral Self-Blame and Outcomes

Hypothesis #2: All regression results are listed in Table 7 (See models 1-5). Time 1 BSB was significantly positively related to baseline depressive symptoms, controlling for age, $\beta = .24$, $t(93) = 2.36$, $p < .05$, and BSB also explained a significant proportion of the variance in baseline depressive symptoms, $R^2 = .06$, $F(2,93) = 6.07$, $p < .05$. Baseline BSB was also significantly negatively related to baseline mental HRQoL, $\beta = -.33$, $t(94) = -3.36$, $p < .05$, and BSB also explained a significant proportion of the variance in baseline mental HRQoL, $R^2 = .11$, $F(1,94) = 11.29$, $p < .05$. BSB was not significantly related to PCS, $\beta = -.09$, $t(93) = -.84$, *n.s.* (controlling for age), heart-healthy diet, $\beta = -.12$, $t(91) = -1.17$, *n.s.* (controlling for income), or functional capacity, $\beta = .10$, $t(87) = 1.09$, *n.s.* (controlling for education, income, sex, and employment status), cross-sectionally. Thus, Hypothesis #2 was not supported because BSB at the beginning of CR was positively related to poor health outcomes (depressive symptoms), and negatively related to good health outcomes (mental HRQoL). Additionally, BSB was not related to any of the other health outcomes cross-sectionally.

Hypothesis #3: All results are listed in Table 7 (See models 6-10). Controlling for baseline scores, BSB was not significantly related to Time 2 depressive symptoms (controlling for employment status), $\beta = -.09$, $t(46) = -.95$, *n.s.*, mental HRQoL, $\beta = -.18$, $t(47) = -1.31$, *n.s.*, physical HRQoL, $\beta = -.13$, $t(46) = -1.01$, *n.s.*, heart-healthy diet (controlling for CR sessions), $\beta = .01$, $t(37) = .06$, *n.s.*, or functional capacity (controlling for education, income, sex, and employment status), $\beta = .01$, $t(32) = .08$, *n.s.*, longitudinally.

Thus, Hypothesis #3 was not supported, in that BSB was not significantly related to any of the health outcomes longitudinally.

Table 7

Regression Analyses Predicting Health Outcomes from BSB

Regression Model	*F(df)*	R^2	ΔR^2	β	*SE*
Model 1: Predicting depressive symptoms, T1					
Step 1	6.23(1,94)*	.06*	--		
Age				-.25*	.05
Step 2	6.1(2,93)*	.12*	.05*		
BSB				.24*	.07
Model 2: Predicting mental HRQoL, T1					
Step 1	11.29(1,94)	.11*	--		
BSB				-.33*	.18
Model 3: Predicting physical HRQoL, T1					
Step 1	9.06(1,94)*	.09*	--		
Age				.30*	.10
Step 2	4.88(2,93)	.08	.01		
BSB				-.09	.15
Model 4: Predicting heart-healthy diet, T1					
Step 1	5.25(1,92)*	.05*	--		
Income				.23*	.38
Step 2	3.32(2,91)*	.07	.01		
BSB				-.12	.17
Model 5: Predicting functional capacity, T1					
Step 1	6.71(4,88)*	.23*	--		
Education				.05	1.02
Income				.23*	.48
Sex				-.19*	2.21
Employment Status				.17	2.44
Step 2	5.62(5,87)	.24	.01		
BSB				.10	.17

Regression Model	*F(df)*	R^2	ΔR^2	β	*SE*
Model 6: Predicting depressive symptoms, T2					
Step 1	37.52(2,47)*	.62*	--		
Employment Status				-.17	.90
T1 depressive symptoms				.73*	.09
Step 2	25.26(3,26)*	.62	.01		
BSB				-.09	.07
Model 7: Predicting mental HRQoL, T2					
Step 1	13.34(1,48)*	.22*	--		
T1 mental HRQoL				.47*	.11
Step 2	7.63(2,47)*	.25	.03		
BSB				-.18	.22
Model 8: Predicting physical HRQoL, T2					
Step 1	12.82(1,47)*	.21*	--		
T1 physical HRQoL				.46*	.16
Step 2	6.93(2,46)*	.23	.02		
BSB				-.13	.24
Model 9: Predicting heart-healthy diet, T2					
Step 1	10.75(2,38)*	.36*	--		
CR Sessions				.59*	.86
T1 heart-healthy diet				.11	.17
Step 2	6.98(3,37)*	.36	.00		
BSB				.01	.25
Model 10: Predicting functional capacity, T2					
Step 1	5.99(5,33)*	.48*	--		
Education				.20	2.1
Income				.02	1.19
CR Sessions				.34*	1.27
Employment Status				.26	4.84
T1 functional capacity				.34*	.22
Step 2	4.85(6,32)*	.48	.00		
BSB				.01	.36

*at least $p < .05$

Note. T1 = Time 1; T2 = Time 2; BSB = behavioral self-blame; CSB = characterological self-blame; HRQOL = health-related quality of life; CR = cardiac rehabilitation.

Hypotheses for Characterological Self-Blame and Outcomes

Hypothesis #4: All regression results are listed in Table 8 (See models 1-5). Time 1 CSB was significantly positively related to baseline depressive symptoms, controlling for age, $\beta = .28$, $t(93) = 2.90$, $p < .05$, and CSB also explained a significant proportion of variance in baseline depressive symptoms, $R^2 = .06$, $F(2,93) = 6.24$, $p < .05$. Baseline CSB was also significantly negatively related to baseline mental HRQoL, $\beta = -.35$ $t(94) = -3.60$, $p < .05$. CSB was not significantly related to physical HRQoL (controlling for age), $\beta = -.001$, $t(93) = -.006$, *n.s.*, heart-healthy diet (controlling for income), $\beta = -.10$, $t(91) = -.97$, *n.s.*, or functional capacity (controlling for education, income, sex, and employment status), $\beta = .05$, $t(87) = .48$, *n.s.*, cross-sectionally. Thus, Hypothesis #4 was partially supported, in that baseline CSB was positively related to depressive symptoms and negatively related to mental HRQoL, cross-sectionally. CSB was not significantly related to the other health outcomes cross-sectionally.

Hypothesis #5: All results are listed in Table 8 (See models 6-10). Time 1 CSB was significantly negatively related to Time 2 physical HRQoL, controlling for Time 1 physical HRQoL, $\beta = -.25$, $t(46) = -2.02$, $p < .05$, and CSB also explained a significant proportion of the variance in Time 2 physical HRQoL, $R^2 = .25$, $F(2,46) = 8.88$, $p < .05$. Controlling for baseline scores, CSB was not significantly related to Time 2 depressive symptoms (controlling for employment status), $\beta = .05$, $t(46) = .05$, *n.s.*, mental HRQoL, $\beta = .08$, $t(47) = .57$, *n.s.*, heart-healthy diet (controlling for CR sessions), $\beta = .28$, $t(37) = .94$, *n.s.*, or functional capacity (controlling for education, income, sex, and employment status), $\beta = -.09$, $t(32) = -.59$, *n.s*, longitudinally. Thus, Hypothesis #5 was partially supported, in that

baseline CSB was negatively related to physical HRQoL at Time 2. CSB was not related to the other health outcomes longitudinally.

Table 8

Regression Analyses Predicting Health Outcomes from CSB

Regression Model	*F(df)*	R^2	ΔR^2	β	*SE*
Model 1: Predicting depressive symptoms, T1					
Step 1	6.24(1,94)*	.03*	--		
Age				-.25*	.05
Step 2	7.57(2,93)*	.14*	.08*		
CSB				.28*	.08
Model 2: Predicting mental HRQoL, T1					
Step 1	12.98(1,94)*	.12*	--		
CSB				-.35*	.22
Model 3: Predicting physical HRQoL, T1					
Step 1	9.06(1,94)*	.09*	--		
Age				.30*	.09
Step 2	4.48(2,93)*	.09	.00		
CSB				.00	.18
Model 4: Predicting heart-healthy diet, T1					
Step 1	5.25(1,92)*	.05*	--		
Income				.23*	.38
Step 2	3.10(2,91)	.06	.01		
CSB				-.10	.21

Table continued

Regression Model	*F(df)*	R^2	ΔR^2	β	*SE*
Model 5: Predicting functional capacity, T1					
Step 1	6.71(4,88)*	.23*	--		
Education				.06	1.02
Income				.29*	.48
Sex				-.19*	2.21
Employment Status				.17	2.44
Step 2	5.37(5,87)*	.24	.00		
CSB				.05	.23
Model 6: Predicting depressive symptoms, T2					
Step 1	37.51(2,47)*	.62*	--		
Employment Status				-.17	.90
T1 depressive symptoms				.73*	.09
Step 2	24.67(3,46)*	.62	.00		
CSB				.05	.09
Model 7: Predicting mental HRQoL, T2					
Step 1	13.34(1,48)*	.22*	--		
T1 mental HRQoL				.47*	.11
Step 2	6.74(2,47)*	.22	.00		
CSB				.08	.27
Model 8: Predicting physical HRQoL, T2					
Step 1	12.82(1,47)*	.21*	--		
T1 physical HRQoL				.46*	.16
Step 2	8.88(2,46)*	.28*	.06*		
CSB				-.25*	.29
Model 9: Predicting heart-healthy diet, T2					
Step 1	10.75(2,38)*	.36*	--		
CR Sessions				.59*	.86
T1 heart-healthy diet				.11	.17
Step 2	7.44(3,37)*	.38	.02		
CSB				.13	.29

Table continued

Regression Model	*F(df)*	R^2	ΔR^2	β	*SE*
Model 10: Predicting functional capacity, T2					
Step 1	5.99(5,33)*	.48*	--		
Education				.20	2.10
Income				.02	1.19
CR Sessions				.34*	1.27
Employment Status				.26	4.84
T1 functional capacity				.34*	.22
Step 2	4.95(6,32)*	.48	.01		
CSB				-.09	.47

*at least $p < .05$

Note. T1 = Time 1; T2 = Time 2; BSB = behavioral self-blame; CSB = characterological self-blame; HRQOL = health-related quality of life; CR = cardiac rehabilitation.

Mediation Hypotheses (#6) with Behavioral Self-Blame and Control

Depressive Symptoms: See Table 9 for model coefficients. Bootstrapped 95% confidence intervals [-.02,.10] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline BSB and depressive symptoms at the end of CR (Time 2). Neither BSB or control appraisals had significant direct effects on depressive symptoms, after controlling for Time 1 depressive symptoms and employment status.

Table 9

Model Coefficients for Hypothesis 6: BSB, Control, and Depressive Symptoms

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Depressive Symptoms)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 BSB)	*a*	-.13	.10	.21	*c´*	-.07	.07	.33
M (T1 Control)		---	---	---	*b*	-.06	.10	.56
T1 Depressive Symptoms	i_1	.78	.10	.00	i_1	.76	.11	.00
T1 Employment Status	i_2	-1.65	.90	.07	i_2	-1.72	.92	.07
		$R^2 = .79$				$R^2 = .63$		
		$F(3, 46) = 25.26, p < .001$				$F(4, 45) = 18.76, p < .001$		

Note. T1 = Time 1, T2 = Time 2, BSB = Behavioral self-blame

Mental HRQoL: See Table 10 for model coefficients. Bootstrapped 95% confidence intervals [-.47,.06] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline BSB and mental HRQoL at the end of CR (Time 2). Control appraisals had a significant direct effect on mental HRQoL, wherein control appraisals were positively related to mental HRQoL. BSB did not have a significant direct effect on mental HRQoL, after controlling for Time 1 mental HRQoL.

Table 10

Model Coefficients for Hypothesis 6: BSB, Control, and Mental HRQoL

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Mental HRQoL)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 BSB)	*a*	-0.13	.10	.21	*c´*	-.27	.21	.21
M (T1 Control)		---	---	---	*b*	.67	.29	.03
T1 Mental HRQoL	*i*	.34	.11	.00	*i*	.24	.12	.04
		$R^2 = .32$				$R^2 = .24$		
		$F(3, 46) = 7.31, p < .001$				$F(2, 47) = 7.63, p < .001$		

Note. T1 = Time 1, T2 = Time 2, BSB = Behavioral self-blame

Physical HRQoL: See Table 11 for model coefficients. Bootstrapped 95% confidence intervals [-.05,.36] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline BSB and physical HRQoL at the end of CR (Time 2). Neither BSB or control appraisals had significant direct effects on physical HRQoL, after controlling for Time 1 physical HRQoL.

Table 11

Model Coefficients for Hypothesis 6: BSB, Control, and Physical HRQoL

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Physical HRQoL)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 BSB)	*a*	-.17	.10	.11	*c´*	-.28	.25	.27
M (T1 Control)		---	---	---	*b*	-.22	.35	.52
T1 Physical HRQoL	*i*	.53	.16	.00	*i*	.53	.16	.00
		$R^2 = .24$				$R^2 = .23$		
		$F(3, 45) = 4.70, p < .05$				$F(2, 46) = 6.92, p < .001$		

Note. T1 = Time 1, T2 = Time 2, BSB = Behavioral self-blame

Heart-healthy diet: See Table 12 for model coefficients. Bootstrapped 95% confidence intervals [-.10,.36] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline BSB and a heart-healthy diet at the end of CR (Time 2). Neither BSB or control appraisals had significant direct effects on a heart healthy diet, after controlling for Time 1 heart healthy diet scores and CR sessions. When controlling for CR sessions, BSB was significantly negatively related to control appraisals.

Table 12

Model Coefficients for Hypothesis 6: BSB, Control, and Heart-healthy Diet

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Heart-healthy diet)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 BSB)	*a*	-.23	.11	.03	c'_1	-.02	.26	.95
M (T1 Control)		---	---	---	*B*	-.14	.36	.71
T1 Heart-healthy diet	i_1	.14	.19	.43	i_1	.14	.18	.43
T2 CR sessions	i_2	3.98	.88	.00	i_2	3.87	.89	.00
		$R^2 = .36$				$R^2 = .36$		
		$F(3, 37) = 6.98, p < .05$				$F(4, 36) = 5.15, p < .05$		

Note. T1 = Time 1, T2 = Time 2, BSB = Behavioral self-blame

Functional Capacity: See Table 13 for model coefficients. Bootstrapped 95% confidence intervals [-.34,.28] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline BSB and functional capacity at the end of CR (Time 2). Neither BSB or control appraisals had significant direct effects on functional capacity, after controlling for Time 1 functional capacity, CR sessions, education, income, and employment status. When controlling for CR sessions, BSB was significantly negatively related to control appraisals.

Table 13

Model Coefficients for Hypothesis 6: BSB, Control, and Functional Capacity

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Functional Capacity)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 BSB)	*a*	-.23	.11	.04	c'_1	.06	.38	.89
M (T1 Control)		---	---	---	*b*	.10	.53	.85
T1 Functional Capacity	i_1	.49	.23	.04	i_1	.50	.23	.04
T2 CR Sessions	i_2	3.28	1.32	.02	i_2	3.30	1.34	.02
Education	i_3	2.76	2.19	.22	i_3	2.78	2.22	.22
Income	i_4	.14	1.20	.91	i_4	.15	1.23	.90
Employment Status	i_5	8.52	4.95	.09	i_5	8.49	5.03	.10
		$R^2 = .48$				$R^2 = .48$		
		$F(6,32) = 4.84, p < .001$				$F(7, 31) = 4.03, p < .001$		

Note. T1 = Time 1, T2 = Time 2, BSB = Behavioral self-blame

Therefore, overall, Hypothesis #6 was not supported because control did not mediate any of the associations. However, results did support longitudinal effects of control on mental HRQoL, as well as a high degree of stability between times 1 and 2 in all of the health outcomes, except heart-healthy diet. In addition, only in models controlling for CR sessions, BSB was significantly negatively related to control appraisals.

Mediation Hypotheses (#7) with Characterological Self-Blame and Control

Depressive Symptoms: See Table 14 for model coefficients. Bootstrapped 95% confidence intervals [-.14,.24] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline CSB and

depressive symptoms at the end of CR (Time 2). Neither CSB or control appraisals had significant direct effects on depressive symptoms, after controlling for Time 1 depressive symptoms and employment status.

Table 14

Model Coefficients for Hypothesis 6: CSB, Control, and Depressive Symptoms

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Depressive Symptoms)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 CSB)	*a*	.05	.13	.69	*c´*	.05	.10	.56
M (T1 Control)		---	---	---	*b*	-.06	.10	.56
T1 Depressive Symptoms	i_1	.74	.10	.00	i_1	.72	.11	.00
T1 Employment Status	i_2	-1.53	.94	.11	i_2	-1.57	.95	.11
		$R^2 = .62$				$R^2 = .62$		
		$F(3, 46) = 24.67, p < .001$				$F(4, 45) = 18.32, p < .001$		

Note. T1 = Time 1, T2 = Time 2, CSB = Characterological self-blame

Mental HRQoL: See Table 15 for model coefficients. Bootstrapped 95% confidence intervals [-.15,.32] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline CSB and mental HRQoL at the end of CR (Time 2). Control appraisals had a significant direct effect on mental HRQoL, wherein control appraisals were positively related to mental HRQoL. CSB

did not have a significant direct effect on mental HRQoL, after controlling for Time 1 mental HRQoL.

Table 15

Model Coefficients for Hypothesis 7: CSB, Control, and Mental HRQoL

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Mental HRQ0L)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 CSB)	*a*	.05	.13	.69	*c´*	.02	.27	.94
M (T1 Control)		---	---	---	*b*	.68	.30	.03
T1 Mental HRQoL	*i*	.41	.12	.00	*i*	.29	.12	.02
		$R^2 = .22$				$R^2 = .30$		
		$F(2, 47) = 6.74, p < .001$				$F(3, 46) = 6.55, p < .001$		

Note. T1 = Time 1, T2 = Time 2, CSB = Characterological self-blame

Physical HRQoL: See Table 16 for model coefficients. Bootstrapped 95% confidence intervals [-.08,.12] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline CSB and physical HRQoL at the end of CR (Time 2). CSB had a significant direct effect on Time 2 physical HRQoL, wherein CSB was negatively related to physical HRQOL. Control appraisals did not have a significant direct effect on physical HRQoL, after controlling for Time 1 physical HRQoL.

Table 16

Model Coefficients for Hypothesis 7: BSB, Control, and Physical HRQoL

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Physical HRQoL)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 CSB)	*a*	.00	.13	.97	*c´*	-.59	.29	.00
M (T1 Control)		---	---	---	*b*	-.13	.33	.69
T1 Physical HRQoL	*i*	.55	.15	.00	*i*	.55	.15	.00
		$R^2 = .28$				$R^2 = .28$		
		$F(2, 46) = 8.88, p < .001$				$F(3, 45) = 5.86, p < .001$		

Note. T1 = Time 1, T2 = Time 2, CSB = Characterological self-blame

Heart-healthy diet: See Table 17 for model coefficients. Bootstrapped 95% confidence intervals [-14,.09] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline CSB and a heart-healthy diet at the end of CR (Time 2). Neither CSB or control appraisals had significant direct effects on a heart healthy diet, after controlling for Time 1 heart healthy diet scores and CR sessions.

Table 17

Model Coefficients for Hypothesis 7: CSB, Control, and Heart-healthy Diet

		Consequent						
		M (T1 Control Appraisals)				*Y* (T2 Heart-healthy diet)		
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 CSB)	*a*	.05	.13	.73	c'_1	.28	.29	.35
M (T1 Control)		---	---	---	*b*	-.14	.34	.68
T1 Heart-healthy diet	i_1	.18	.18	.31	i_1	.19	.18	.30
T2 CR Sessions	i_2	3.76	.87	.00	i_2	3.74	.89	.00
		$R^2 = .38$				$R^2 = .38$		
		$F(3, 37) = 7.44, p > .05$				$F(4, 36) = 5.50, p > .05$		

Note. T1 = Time 1, T2 = Time 2, CSB = Characterological self-blame

Functional Capacity: See Table 18 for model coefficients. Bootstrapped 95% confidence intervals [-.18,.23] for the indirect effect contained zero, showing that Time 1 control appraisals did not significantly mediate the relationship between baseline CSB and functional capacity at the end of CR (Time 2). Neither CSB or control appraisals had significant direct effects on functional capacity, after controlling for Time 1 functional capacity, CR sessions, education, income, and employment status.

Table 18

Model Coefficients for Hypothesis 7: CSB, Control, and Functional Capacity

		Consequent					
		M (T1 Control Appraisals)				*Y* (T2 Functional Capacity)	
Antecedent		Coeff	*SE*	*p*		Coeff	*SE*	*p*
X (T1 CSB)	*a*	.02	.14	.88	c'_1	-.28	.48	.57
M (T1 Control)		---	---	---	*b*	.09	.49	.86
T1 Functional Capacity	i_1	.49	.22	.03	i_1	.50	.23	.04
T2 CR Sessions	i_2	3.33	1.29	.01	i_2	3.33	1.31	.02
Education	i_3	2.22	2.29	.34	i_3	2.20	2.33	.35
Income	i_4	.23	1.21	.85	i_4	.25	1.23	.84
Employment Status	i_5	7.63	5.09	.14	i_5	7.57	5.18	.15
		$R^2 = .48$				$R^2 = .48$		
		$F(6,32) = 4.95, p < .001$				$F(7, 31) = 4.12, p < .001$		

Note. T1 = Time 1, T2 = Time 2, CSB = Characterological self-blame

Therefore, overall, Hypothesis #7 was not supported because control did not mediate any of the associations. However, results did support longitudinal effects of control on mental HRQoL, and CSB on physical HRQoL, as well as a high degree of stability between times 1 and 2 in health outcomes, except heart-healthy diet.

CHAPTER 5

DISCUSSION

The purpose of this study was to examine if the CSBA scale is associated with physical and mental health recovery outcomes among underrepresented patients with CVD before and after CR. Health outcomes included depressive symptoms, mental and physical HRQoL, heart-healthy diet, and functional capacity. Additionally, a secondary aim was to assess whether the relationship between both types of self-blame attributions (behavioral and characterological) and health outcomes were mediated by control appraisals.

Hypothesis for Psychometric Properties of the CSBA scale

Results confirmed the CSBA scale to be a reliable and valid measure in the present sample. The two subscales represented BSB and CSB with items congruent with the original validation study (Harry et al., 2018). This two-factor solution suggests there is a practical difference between making cardiac attributions to one's past behaviors compared to one's personality or character traits. Additionally, both BSB and CSB had good test-retest reliability at the beginning and end of CR. Average scores on BSB in the current sample at both time points (T1 $M = 13.97$; T2 $M = 13.45$) were similar and only slightly higher than those found in the underrepresented sample of CR patients ($M = 12.88$) in the original validation study (Harry et al., 2018). Additionally, average scores on CSB in the current sample at both time points (T1 $M = 6.97$; T2 $M = 7.27$) were similar and slightly higher than those found in the CR patients ($M = 5.46$) in the original study (Harry et al., 2018).

Hypotheses for Behavioral Self-Blame and Outcomes

Hypothesis #2 was not supported because BSB at the beginning of CR was positively related to poor health outcomes (depressive symptoms), and negatively related to good health

outcomes (mental HRQoL). Additionally, BSB was not related to any of the other health outcomes cross-sectionally. It appears that attributing your cardiac event to your behavior does not have protective effects on mental health outcomes among this sample of underrespresented patients with CVD. Janoff-Bulman (1979) posited that BSB is adaptive for adjustment because blame is attributed to modifiable factors, but this was not found in the present study. This rests on the presumption that behavior is controllable, but in reality, it is likely that the controllability of any given behavior ranges along a continuum of high to low, and that cognitive and motivational processes affect where one views a behavior along that continuum. In the present sample, blaming your behavior may not be perceived as controllable, and thus be related to higher depressive symptoms and worse HRQOL for patients at the start of CR.

Although previous studies used a one-item measure or different variations on the measurement of BSB, these results are congruent with a study of CR patients that found BSB to be positively associated with symptoms of anxiety and depression at the beginning and end of CR (Bennett, Howarter, & Clark, 2013). Additionally, these findings align with past research among patients with breast cancer that showed BSB was related to poor psychological functioning (Glinder & Compas, 1999), and to higher distress, including anxiety and depression (Bennett, Compas, Beckjord, & Glinder, 2005). However, these results differ from studies that found BSB to be positively associated with good adjustment among women with breast cancer (Timko & Janoff-Bulman, 1985), and fewer symptoms of depression and less impairment in health-related quality of life among patients with chronic obstructive pulmonary disease (Plaufcan, Wamboldt, and Holm, 2012).

After calculating a comparison between BSB scores from the 6-item CSBA subscale to the one-item measure used in previous literature, the results indicated that BSB levels in the present sample ($M = 2.31$) were higher than past studies involving patients with cancer that ranged from 1.40-1.90 (Bennett, Compas, Beckjord, & Glinder, 2005; Glinder & Compas, 1999; Malcarne, Compas, Epping-Jordan, & Howell, 1995). However, the present levels of BSB were similar to those reported in other studies with patients with CVD, ranging from 2.25-2.58 (Bennett, Howarter, & Clark, 2013; Harry, Bennett, Clark, Howarter, & Eways, 2015). Therefore, blaming one's past behavior seems more prevalent in patients with CVD compared to cancer. Given the well-established behavioral risk factors associated with CVD onset, these mean differences are not surprising.

Hypothesis #3 was not supported, as BSB at the beginning of CR was not significantly related to any of the health outcomes longitudinally, after controlling for baseline levels of the health outcomes. Although cited literature has shown longitudinal relationships between BSB and mental health outcomes among patients with cancer (Malcarne et al., 1995) and CVD (Bennett, Howarter, & Clark, 2013), these were not found in the present sample of patients with CVD. It is possible that barriers (e.g., low SES, transportation, insurance) associated with this specific population of underrepresented patients at a safety-net hospital may affect participation in CR, health outcomes, as well as how perceived blame impacts health outcomes at the completion of CR.

Hypotheses for Characterological Self-Blame and Outcomes

Hypothesis #4 was partially supported, in that baseline CSB was positively related to depressive symptoms and negatively related to mental HRQoL cross-sectionally. CSB was not significantly related to the other health outcomes cross-sectionally. Therefore, attributing

your cardiac event to your character was related to poor mental health outcomes, in the form of more depressive symptoms and worse mental HRQoL at the beginning of CR. Janoff-Bulman (1979) hypothesized that characterological self-blame is maladaptive for adjustment because blame is attributed to one's personality that is presumed to be non-modifiable. Our findings align with past research showing relationships between CSB and poor adjustment (Timko & Janoff-Bulman, 1985), poor psychological functioning (Glinder & Compas, 1999), and higher distress (Bennett, Compas, Beckjord, & Glinder, 2005) among patients with breast cancer. Also, the relationship between CSB and depressive symptoms has been previously found in patients with chronic obstructive pulmonary disease (Plaufcan, Wamboldt, & Holm, 2012).

After calculating a comparison between CSB scores from the five-item CSBA subscale to the one-item measure used in previous literature, the results indicated that CSB levels in the present sample (M = 1.39) were similar to past studies involving patients with cancer that ranged from 1.30-1.56 (Bennett, Compas, Beckjord, & Glinder, 2005; Glinder & Compas, 1999; Malcarne, Compas, Epping-Jordan, & Howell, 1995). However, present levels of CSB were slightly lower than those reported in other studies among patients with CVD, ranging from 1.72-1.74 (Bennett, Howarter, & Clark, 2013; Harry, Bennett, Clark, Howarter, & Eways, 2015). It is noteworthy, then, that these patients with CVD seemed to blame their character to similar degrees as patients with various forms of cancer.

Hypothesis #5 was partially supported; CSB at the beginning of CR was negatively related to physical HRQoL at Time 2. CSB was not related to the other health outcomes longitudinally. Thus, attributing your cardiac event to your character or personality was related to worse physical HRQoL at the completion of a CR program. Research has shown

that CSB has been related to worse physical health outcomes, including cardiac symptom experience (Harry, Bennett, Clark, Howarter, & Eways, 2015). In congruence, our findings suggest that CSB at the beginning of CR is associated with reported lower levels of physical HRQoL.

Past studies have demonstrated a relationship between baseline CSB and mental health outcomes at subsequent time points. For example, Glinder and Compas (1999) found that CSB was related to poor adjustment six and 12 months post-cancer diagnosis, and Bennett, Compas, Beckjord, and Glinder (2005) found that CSB was associated with distress at four, seven, and 12 months post-cancer diagnosis. In contrast, these associations were not seen in the present study. Rather, our findings were similar to those reported by Bennett, Howarter, and Clark (2013) that found that CSB was not predictive of distress among CR patients at the beginning or end of the program. Again, patient characteristics and barriers may have impacted the ability to detect these relationships in our population. Additionally, the time between the start of CR and the end (approximately 3-6 months) may not have been enough time to detect the effects of CSB on health outcomes.

Mediation Hypotheses Behavioral Self-Blame and Outcomes

Overall, Hypothesis #6 was not supported because control did not mediate any of the associations with health outcomes. However, results did support longitudinal effects of control appraisals on mental HRQoL. In addition, only in models controlling for CR sessions was BSB significantly negatively related to control appraisals.

Janoff-Bulman (1979) posited that enhancements in control appraisals are predicted to be the mechanism that links behavioral self-blame attributions to improved adjustment. Consistent with this prediction, Bennett, Howarter, and Clark (2013) found that behavioral

attributions specific to diet and exercise were positively related to perceived control over recurrence, but they did not find perceived control to be related to psychological distress. Additionally, our findings are congruent with previous research that did not find a relationship between BSB and perceptions of control over cancer reoccurrence, and control beliefs did not mediate the relationship between BSB and adjustment among cancer patients (Malcarne, Compas, Epping-Jordan, & Howell, 1995). Additionally, the present findings are similar to those that found perceptions of control did not mediate relationships between self-blame and both types of psychological distress (anxiety and depression symptoms) among patients with breast cancer (Bennett, Compas, Beckjord, & Glinder, 2005).

The present study did not find control appraisals over cardiac symptoms to link behavioral attributions to health outcomes. The CSBA scale asks specifically if you attribute your cardiac event to aspects of behavior, but this blame may not relate to feelings of control of cardiac symptoms. Previous studies have used different, often one to two item measures of control appraisals. In contrast, the present study used an eight-item measure of control appraisals that assesses perceived control over cardiac-related symptoms and behavior related to a heart condition. Previous literature (Moser et al., 2009) using the CAS-R showed that average levels of perceived cardiac-related control in the current sample ($M = 20.9$) were lower than all three CVD disease groups in past research, including coronary heart disease ($M = 30.3$), heart failure ($M = 28.4$), and myocardial infarction ($M = 29.1$).

Thus, the current sample may have felt less perceived control than previous samples that consisted mostly of European American patients not recruited from a safety-net hospital (Moser et al., 2009). It may be that control specifically related to cardiac symptoms may not be associated with behavioral attributions, and therefore it would not serve as a mediator of

the relationship between BSB and mental health outcomes. Additionally, our patient population may feel less control over not only their cardiac health, but also less perceived control in general given their sociodemographic profiles and institutional barriers. Interestingly, only in models controlling for CR sessions was there a negative relationship between BSB and control appraisals. This relationship runs counter to theory-based predictions, and it is difficult to explain. It is possible that this is a spurious relationship, so additional research is needed.

Cardiac control appraisals at baseline were significantly related to mental HRQoL at the end of CR, demonstrating that higher perceptions of control over cardiac-related symptoms were related to better mental HRQoL. Past research has shown the positive impact of perceived control on health outcomes among various chronic illness populations, as well as among CVD patients (Dracup et al., 2003; Evangelista, Moser, Dracup, Doering, & Kobashigawa, 2004). Among those with CVD populations, past studies have shown that higher perceived control was related to lower levels of anxiety and depression (Dracup et al., 2003; Evangelista, Moser, Dracup, Doering, & Kobashigawa, 2004). We found similar relationships in the present sample relative to mental HRQoL. It may be that feelings of control over specific cardiac symptoms were related to better psychological well-being. These relationships were not found for the other health outcomes, however. In the case of the current sample of patients with CVD at a safety-net hospital, other variables or contextual factors besides control over cardiac symptoms may explain more of the variance in health outcomes.

Overall, patients' averages on the health outcomes reflected a significant improvement in physical HRQoL, better heart-healthy diet scores, and better physical

functioning (see Table 4). These highlight the importance of CR programs on improving physical health outcomes and align with previous research denoting the positive effects of CR participation (Barzi et al., 2003; Lisspers et al, 2005). Contrary to literature on the positive effects of psychosocial intervention on physical health outcomes, the present study showed a significant increase in depressive symptoms from Time 1 to the end of CR. Thus, the limited psychosocial intervention included in the CR program at the research site hospital may not be enough to ameliorate symptoms of depression in this patient population.

Mediation Hypotheses Characterological Self-Blame and Outcomes

Overall, Hypothesis #7 was not supported because control did not mediate any of the associations. However, results did again support longitudinal effects of control appraisals on mental HRQoL, and CSB on physical HRQoL. Similar to BSB, blaming your cardiac event on your character or personality did not translate to feelings of control over cardiac-related symptoms. Consistent with the prediction that CSB attributions are related to less control, Bennett, Howarter, and Clark (2013) found that CSB was negatively associated with general control over one's cardiac health and control over recovery. In contrast, our findings mirrored studies that reported no mediation by control appraisals (Bennett, Compas, Beckjord, & Glinder, 2005; Malcarne, Compas, Epping-Jordan, & Howell, 1995).

Limitations

Although the proposed study aimed to fill two gaps in the literature by further validating the CSBA scale and assessing whether it predicted physical and mental health outcomes among underserved patients with CVD, there were a few limitations. First, the sample size for this study was small, despite extending the study's recruitment period due to unanticipated difficulties recruiting eligible participants. It is hypothesized that reduced

patient flow, and thus lower numbers of eligible patients referred to CR, may have affected the recruitment efforts. Thus, the small convenience sample may not generalize to the entire CVD population. However, those who did not complete the study did not significantly differ from the present sample on demographic characteristics. The sample size in this project did not reach the anticipated level to ensure at least 80% power to detect relationships between variables at both time points. For the cross-sectional analyses, the sample size at time 1 was close to the number required to detect 80% power ($N = 92$). For analyses examining health outcomes at Time 2, our sample size did not meet the required number of participants to detect significant relationships. In fact, our sample size at time 2 indicated approximately 50% power to detect significant relationships. Thus, relationships with health outcomes at Time 2, including the mediation analyses, may have shown significant results in a larger sample size.

Additionally, there is a low percentage of CVD patients who are referred to CR (Brown et al., 2009), and an even lower percentage that actually participate (Beswick et al., 2005; Clark et al., 2013; Suaya et al., 2007). Moreover, these results may not generalize to patients who are ineligible for CR or to patients who are eligible but decided not to participate. The present sample consisted predominantly of patients with low socioeconomic status and thus may not generalize to all CVD patients. This study also relied on self-report data and thus will need to be interpreted with some caution due to shared method variance, social desirability, and other response biases. Although the present study assessed longitudinal health outcomes, three to six months is a short-time period and may not reflect patients' actual long-term health status.

Also, attrition for follow-up self-report data at the end of CR occurred for 47% of participants. Of those who did not complete the study, 57% dropped out of CR early, 26% were still enrolled in CR at the time the study completed, and 15% were lost due to medical data archive. According to discussions with CR staff, a majority of the participants was unable to complete CR due to mental, physical, or institutional barriers. Given the low reported income in our sample (as evidenced by the high rates of receiving the hospital's income-based discount), it is possible that these sociodemographic factors were related to their adherence to CR, and thus the outcomes assessed as part of this project.

Lastly, reliability coefficients could not be calculated for the four health outcomes because they were extracted from patients' medical files. However, all of these measures have been shown to be valid and reliable among samples of CVD patients in previous studies.

Clinical and Theoretical Implications

Despite these limitations, this study is the first of which we are aware to examine how the CBSA relates to physical and mental health outcomes, thereby filling a large gap in the literature. In CVD, where emphasis is place on behavioral risk factors and lifestyle modification changes, self-blame may be an unintended consequence. The present study showed that blaming one's behavior for a cardiac event at the beginning of CR was related to more depressive symptoms and worse mental HRQoL concurrently, but not related to outcomes at the end of CR. Thus, CR staff should identify patients' levels of BSB at the start of CR in order to design and implement interventions in hopes of affecting positive health outcomes, specifically related to psychological functioning. Psychosocial interventions that are part of CR programs may want to add assessment of cardiac self-blame attributions to

their curricula to 1) protect against immediate negative effects on mental health outcomes and psychological adjustment, and 2) protect against negative effects on perceived control.

Blaming your cardiac event on your character or personality at the beginning of CR was related to more depressive symptoms and worse mental HRQoL at baseline, but not at the end of CR. In addition, CSB was related to worse physical HRQoL at the end of CR. Thus, CR staff should also identify patients' levels of CSB at the beginning of CR in order to implement strategies in hopes of ameliorating the negative effects on psychological distress and physical HRQoL.

The CSBA scale may be added to the battery of health outcome measures used in many CR programs, as an indicator at the beginning of CR of who to target for intervention based on self-blame levels. In addition, exploring patients' attributions about the causes of their CVD may lead to a better understanding of adaptive and maladaptive responses to illness and treatment. Additionally, the CSBA scale may be used in future research examining self-blame attributions to examine its relationship among other psychosocial outcomes in patients with CVD.

Future Directions

This was the first study to assess relationships between the CSBA scale and physical and mental health outcomes among CVD patients, therefore future research is needed to provide further evidence of these relationships with other health outcomes to strengthen the predictive validity of the CSBA scale. Research should be conducted in larger samples of patients with CVD in other safety-net hospitals, as well as other institutions that serve cardiac populations. In addition, future studies should evaluate other potential variables in addition

to cardiac control appraisals that may link cardiac self-blame attributions to physical and mental health outcomes.

APPENDIX A.

MEASURES

A-1. Cardiac Self-Blame Attributions Scale, Times 1 & 2

Cardiac Self-Blame Attributions Scale

Some individuals blame themselves for their cardiac events, whereas some individuals do not. Please read these questions and circle the answer that best reflects how you feel.

		Not at all	A little	Somewhat	A lot	Completely
1.	How much do you blame yourself for past behaviors that may have caused your cardiac event?	0	1	2	3	4
2.	To what extent do you accept fault for behaviors that may have caused your cardiac event?	0	1	2	3	4
3.	How much do you think your past behaviors contributed to your cardiac event?	0	1	2	3	4
4.	To what extent do you believe that a change in your behavior could have prevented your cardiac event?	0	1	2	3	4
5.	To what extent do you feel accountable when thinking about past behaviors that may have caused your cardiac event?	0	1	2	3	4
6.	When discussing possible causes of your cardiac event with important people in your life, to what extent have you blamed your past behavior?	0	1	2	3	4
7.	How much do you blame the type of person you are for your cardiac event?	0	1	2	3	4
8.	To what extent do you believe that a change in the type of person you are could have prevented your cardiac event?	0	1	2	3	4
9.	How much do you blame your personality for your cardiac event?	0	1	2	3	4
10.	How much do you blame yourself for being the type of person who has bad things, like a cardiac event, happen to them?	0	1	2	3	4
11.	When discussing possible causes of your cardiac event with important people in your life, to what extent have you blamed your personality?	0	1	2	3	4

A-2. Control Attitudes Scale-Revised, Times 1 & 2

The Control Attitudes Scale-Revised						
		Totally Disagree	**Disagree**	**Neither agree nor disagree**	**Agree**	**Totally Agree**
1.	If I do all the right things, I can successfully manage my heart condition.	1	2	3	4	5
2.	I can do a lot of things myself to cope with my heart condition.	1	2	3	4	5
3.	When I manage my personal life well, my heart condition does not bother me as much.	1	2	3	4	5
4.	I have considerable ability to control my symptoms.	1	2	3	4	5
5.	No matter what I do, or how hard I try, I just can't seem to get relief from my symptoms.	1	2	3	4	5
6.	I am coping effectively with my heart condition.	1	2	3	4	5
7.	Regarding my heart problems, I feel lots of control.	1	2	3	4	5
8.	Regarding my heart problems, I feel helpless.	1	2	3	4	5

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