Patient Perceptions of Comorbid Depression in Heart Disease

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Patient Perceptions of Comorbid Depression in Heart Disease

by

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Abstract

Depression is a risk factor for the development of heart disease, as well as for poor prognosis among patients with existing disease. However, clinical trials of treatment of depression in patients with heart disease have not resulted in improved cardiac outcomes, and have demonstrated modest effects on depression. The nature of depression in heart patients must be better understood in order to improve treatment and health outcomes. In samples of patients with medical illness as well as in patients with depression, illness perceptions have been useful in predicting both treatment outcomes and self-management behaviors such as coping and adherence. This is the first study to examine patient perceptions of comorbid depression in heart disease. The purpose of the study was to identify baseline correlates of illness perceptions in the context of a depression treatment study. Results from 112 patients with comorbid depression and heart disease, manifested as stable coronary heart disease or heart failure, show that depression is strongly associated with perceived consequences, but that personality factors are more strongly related to several other illness perception dimensions. Depression history variables did not predict illness perceptions, but moderated the effect of personality on timeline perceptions. Relationships among dimensions of illness perceptions were somewhat consistent with findings from other study populations. This study represents an important first step in clarifying how patients with heart disease conceptualize comorbid depression. Future research is needed to determine if these perceptions predict coping with depression, adherence to treatment, or treatment outcomes.
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Heart Disease and Depression

An important link between depression and the heart has been suspected by lay persons for many centuries. It is common for individuals in intense psychological pain to speak of experiencing a “broken heart” or “heartache.” However, it has only been in recent decades that this purported link has been empirically investigated. Over the past 20 years, a robust literature has emerged showing depression to be not only a relatively common problem in patients with various kinds of cardiovascular disease, but an important prognostic indicator as well. Estimates of the prevalence of depression in heart patients vary according to the method of assessment used and the particular patient population under investigation. However, point prevalences have ranged from 14-47% among patients with CAD (Lett et al., 2004). The diagnostic criteria for DSM-IV major depressive disorder are met by an estimated 20% of patients after an acute myocardial infarction (MI), with a similar percentage showing symptoms of mild depression (Frasure-Smith et al., 1993; Hance, Carney, Freedland, & Skala, 1996). This percentage decreases somewhat over time in the months following the MI as patients’ coronary condition stabilizes. Patients hospitalized for heart failure (HF) are affected by depression at comparable rates, with one study reporting 20% major depression and 16% minor depression (Freedland et al., 2003). These rates correspond to a 3-4-fold increase compared to prevalence estimates of depression in the general population (Rumsfeld & Ho, 2005). When self-report depression questionnaires such as the Beck Depression Inventory (BDI) are used, over 50% of cardiac patients score in the mild to clinically
depressed range (Freedland et al., 2003). Psychologists and cardiologists alike are now involved in efforts to improve recognition and treatment of depression in patients with heart disease.

It is important for many reasons to treat depression in patients with cardiac disease. Depression in its own right has a tremendous effect on both society and the individual. It is recognized as a leading cause of disability world-wide, and is a major cause of workplace absenteeism, decreased productivity, and increased use of health care resources (Murray & Lopez, 1996). Depression can lead to decreased quality of life, hopelessness, and even suicide (Rumsfeld & Ho, 2005). In heart patients, depression is associated with several additional adverse outcomes.

Depression has consistently been associated with increased morbidity and mortality in patients with various manifestations of cardiac illness (Bush et al., 2005; Carney et al., 1988; Connerney et al., 2001; Everson et al., 1998; Frasure-Smith et al., 1993; Jiang et al., 2001; Whooley et al., 2008). Patients with existing heart disease who are depressed are at increased risk for recurrent cardiovascular events, and have a two- to four-fold increased risk of death after experiencing a cardiac event, with the greatest risk occurring in the first six months after the event (Joynt, Whellan, & O’Conner, 2003). Depressive symptoms, even in the absence of a diagnosable depressive episode, are associated with an increased risk of cardiac events (Anda et al., 1993). Moreover, evidence exists for a “dose-response” relationship, such that more severe depressive symptoms pose a greater risk for future cardiac events (Anda et al, 1993; Pratt et al., 1996). These relationships remain significant after controlling for potential confounding
factors such as age, gender, history and severity of heart disease, and risk factors such as smoking, high blood pressure, body mass index, and socioeconomic status. Minor and major depression increase the risk of rehospitalization and emergency department visits after acute MI, thereby increasing health care costs (Reese et al., 2011). Depressed cardiac patients also demonstrate more detrimental health behaviors than their nondepressed counterparts. Patients with depression are significantly less likely to adhere to lifestyle recommendations such as smoking cessation and exercise after experiencing a cardiac event (Ziegelstein et al., 2000) and are less likely to adhere to their prescription medication regimen (Gehi, Haas, Pipkin, & Whooley, 2005). Depression is associated with decreased likelihood of attendance at, and less improvement in, cardiac rehabilitation (Glazer, Emery, Frid, & Banyasz, 2002; Lane, Carroll, Ring, Beevers, & Lip, 2001), and decreased likelihood of obtaining recommended medical tests (Rumsfeld & Ho, 2005).

For all the reasons stated above, the treatment of depression in heart patients is an important endeavor. Additionally, when one considers the independent contribution of depression to cardiac morbidity and mortality, it seems possible that treatment of depression may be able to reduce at least some of its adverse effect on cardiac outcomes. Relatively few large studies to date have investigated this question. The Sertraline Antidepressant Heart Attack Trial (SADHART; Glassman et al., 2002) was a double-blind, randomized, placebo-controlled trial designed to determine the safety and efficacy of sertraline in depressed patients with a recent acute coronary syndrome (ACS). Sertraline was found to be a safe medication in this population. Although the sample was
not sufficiently large to study the effect of treatment on cardiac outcomes, there was a
trend toward decreased incidence of severe cardiac events in the treatment arm. The
SADHART study investigators recently conducted an analysis with sufficient statistical
power to model cardiac events occurring during the study’s seven year follow-up period
(Glassman, Bigger, & Gaffney, 2009). There was no difference in mortality between
individuals assigned to six months of sertraline treatment versus placebo. The original
SADHART report also examined the efficacy of sertraline therapy in treating depression.
There was no significant difference in Hamilton depression inventory scores (HAM-D;
Hamilton, 1960) between the sertraline and placebo groups. A significant improvement in
depression was observed in the subgroup of patients with severe, recurrent major
depression, although the treatment effect was modest (corresponding to about three points
on the HAM-D).

The Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD;
Berkman et al., 2003) study was a multicenter, randomized, controlled trial funded by the
National Heart, Lung, and Blood Institute (NHLBI), and was the first large trial in a
cardiac sample to test the effect of treating depression primarily with psychotherapy. It
was designed to assess whether treating depression after MI could reduce death and
recurrent nonfatal infarction. Low perceived social support (LPSS) was also a target of
treatment, due to its status as a risk factor for morbidity and mortality (Berkman, Leo-
Summers & Horwitz, 1992; Case, Moss, Case, McDermott, & Eberly, 1992). The 2481
participants were randomized to the treatment group or usual care (UC) within 28 days of
their MI. Treatment consisted of individual cognitive behavior therapy (CBT) augmented
for a minority of participants by group therapy. Participants who were severely depressed at enrollment or who did not show improvement after five weeks of CBT were also treated with sertraline. Individual therapy continued until rigorous criteria for successful treatment were met, or for a maximum of six months. Participants were followed up, and any deaths or recurrent MIs were tracked, for an average of 29 months. Survival analyses showed no significant difference between the treatment group and UC in the number of recurrent MIs or deaths, or in the secondary medical endpoints. Similar to the SADHART trial, ENRICHD analyses showed that treatment of depression was associated with statistically significant but clinically modest improvement of depression.

Although the results of these two trials disappointed many researchers who had hoped to demonstrate that treatment of depression could benefit cardiovascular prognosis, the negative results have inspired much subsequent work. Interesting secondary analyses have emerged out of both data sets, such as the 2008 report by Cowan et al., which found that adherence to CBT homework assignments was the only consistent predictor of improved depression in ENRICHD. Perhaps most important to advancing the field have been the reflections on what could have been done differently in these trials. Sheps, Freedland, Goldman, and McMahon (2003) suggest that, while the rationale for starting treatment as early as possible seemed reasonable (since the highest risk of reinfarction or death occurs within the first six months of the cardiac event), this likely resulted in the inclusion of many transient cases of depression in the analyses – cases which would have resolved quickly even without treatment. Indeed, recovery rates in the UC arm were high (Berkman et al., 2003). Many researchers have also taken a step back after seeing these
results, and concluded that perhaps these studies attempted to do too much, too soon. Results of both studies indicated that not only were the treatments not associated with improvement in measured cardiac outcomes, improvement of depressive symptoms was modest. If the ultimate goal is to improve cardiac endpoints by treating depression, care must be taken to develop maximally effective treatments for depression that occurs in the context of cardiac problems.

The results of SADHART and ENRICHD suggest that there is room for improvement in the methods used to treat depression in cardiac patients. In order to improve treatment, we need to know more about the nature of depression in this population. However, relatively few studies have examined psychological factors which may be important for understanding depression in this population. No previous study has examined cardiac patients’ perceptions of their depression and how these perceptions may be related to depression severity, coping strategies, or treatment adherence, even though these perceptions have been shown to relate to depression treatment in medically well depressed patients.

**The Common-Sense Model of Illness Representations**

**Theoretical model.** The Common Sense Model of Illness Representations (CSM; Leventhal et al., 1980, Leventhal, Leventhal, & Breland, 2011) provides a useful framework for conceptualizing how individuals formulate and organize beliefs about an illness and how these beliefs influence behavior. This theoretical model, which has also been referred to as the “Self Regulation Model,” emerged out of Leventhal’s work on fear communication (Leventhal, 1970). The CSM states that individuals organize
information about illnesses (both medical and psychiatric) into a cognitive representation or model within an underlying processing system. These illness representations, or illness perceptions, are influenced by factors such as one’s culture and prior experience with a particular illness. Leventhal describes the individual as an active information-seeker and problem-solver who, when confronted with a new sensation or experience (e.g., a symptom), seeks to understand the experience and incorporate it into his/her cognitive representation of that illness. He further characterizes this processing system as having two parallel pathways in which separate cognitive (or objective) and emotional representations exist for a given health threat. They are processed in a parallel but relatively independent fashion. Additionally, representations can be either perceptual, as in symptoms which are experienced (e.g., headache pain), or conceptual (e.g., an abstract thought, such as “I must have a migraine”). These two levels may be at odds with one another. For example, a person undergoing chemotherapy for cancer may conceptually believe that the treatment will improve his or her cancer prognosis, while perceptually (somatically), may feel much worse due to the treatment.

The content of illness representations can be organized into a number of dimensions which have been empirically derived (Leventhal et al., 1980). Although other dimensions have recently been added to the theoretical model, five primary dimensions of illness representations have been supported by the literature, and therefore constitute the “basic building blocks” of illness representations (Heijmans and de Ridder, 1998, p. 486). Illness identity is composed of the label an individual assigns to an illness and the symptoms s/he associates with the illness. This is often measured by asking the individual
to check on a symptom inventory all of the symptoms s/he has experienced as part of the illness in question. The timeline dimension represents the individual’s beliefs about the expected course of the illness—whether it will be acute or chronic, for example. The consequences dimension is the extent to which the individual believes that the illness will have significant effects on his/her life. An individual’s beliefs about what caused his/her illness comprise the cause dimension. Causes are often grouped into categories such as biological (e.g., germs or viruses), emotional/psychological (e.g., stress or personality), and environmental (e.g., pollution or chemicals) (Hagger & Orbell, 2003). The fifth major dimension is referred to as cure/control and represents the extent to which the individual believes that his/her illness is amenable to cure or control by either medical treatments or personal efforts. A factor analysis of one of the most common measures of illness representations, showed that the cure/control items load onto two separate factors—one related to personal control/self-efficacy, and the other related to perceived treatment control/outcome expectancies (Moss-Morris et al., 2002). For this reason, the cure/control dimension is often split into two separate dimensions of personal control and treatment control. Illness representation dimensions are assessed either through open-ended interviews (Leventhal & Nerenz, 1985) or by self-report questionnaires such as The Illness Perception Questionnaire (IPQ; Weinman, Petrie, Moss-Morris, & Horne, 1996), and The Revised Illness Perception Questionnaire (IPQ-R; Moss-Morris et al., 2002). The latter are commonly used as they are relatively easy to administer and provide quantitative measures of illness representation dimensions based on Likert scale ratings.

Several studies of the CSM dimensions have reported a consistent pattern of inter-
relationships among the dimensions. This was confirmed by a meta-analysis of 45 studies (Hagger & Orbell, 2003). Specifically, high scores on the identity dimension (indicating that the individual experiences many symptoms associated with the illness) are related to greater perceived consequences and a longer perceived timeline. Greater perceived consequences are also associated with belief in a longer timeline. Higher scores on the cure/control dimension (indicating a stronger belief that the illness is controllable) are associated with fewer perceived consequences and a shorter perceived timeline. Finally, higher illness identity scores are related to lower scores on cure/control. This consistency in the inter-relationships of dimensions across samples lends support to the validity of the CSM model as specified by Leventhal.

An important application of the CSM dimensions relates to their purported effects on self-management behaviors such as coping. Leventhal posits that cognitive and emotional representations are primary determinants of coping behavior, defined as the procedures for preventing and controlling health threats (Leventhal, Nerenz, & Steele, 1984). Although Leventhal’s model does not specify exactly how CSM dimensions relate to coping, this has been examined in numerous empirical investigations, and this work will be reviewed shortly. While illness representations are thought to directly influence coping behaviors, a feedback loop exists by which subsequent appraisal of the effectiveness of coping can influence future coping strategies and, often, the very definition or representation of the problem being defined or represented (Leventhal, 1984). Therefore, three stages of information processing influence coping behavior: 1) the definition or representation of the problem; 2) planning and implementation of coping
procedures; and 3) appraisal of the effectiveness of the coping procedure. Processing of illness-related information proceeds to cycle through these three stages, with effectiveness of current coping influencing future definitions of the problem as well as future coping efforts.

It is through this process of influencing coping that illness representations are thought to have an impact on illness outcomes. The CSM is therefore, in essence, a mediational model (Brown et al., 2007; Hagger & Orbell, 2003) in which coping (or health behavior) mediates the relationship between a person’s illness representations and important functional outcomes. A major interest of researchers utilizing the CSM has therefore been the investigation of relationships between CSM dimensions and illness outcomes, and between CSM dimensions and coping.

**Application of the Common-Sense Model to physical illness.** Research on a number of different medical populations has shown that illness representations are related to coping behaviors as well as functional outcomes, including physical or emotional functioning. Hagger and Orbell’s (2003) meta-analysis examined relationships among the major CSM dimensions and various types of coping in a large sample of studies. The studies included in the analysis involved 23 different illnesses, such as diabetes, chronic fatigue syndrome, arthritis, and myocardial infarction. A number of significant relationships were found between CSM dimensions and coping. Higher scores on the cure/control dimension were associated with greater use of problem-focused coping, cognitive reappraisal, and seeking social support. Higher scores on the consequences dimension were related to greater avoidance, denial, and emotional expression, while
higher scores on the timeline dimension were related to greater use of cognitive reappraisal and modestly related to more avoidance and denial. This pattern suggests that belief that an illness has serious consequences is associated with passive coping behaviors, while belief in the controllability of the illness is associated with more active and perhaps more adaptive coping.

Hagger and Orbell (2003) also examined relationships between CSM dimensions and indicators of illness outcomes, and uncovered a number of patterns across illnesses. The cure/control dimension was positively related to psychological well-being, social functioning, and vitality, and was inversely related to distress and objective measures of illness status (e.g., CD4 count for HIV status). Consequences and timeline were both found to be inversely related to physical functioning, psychological well-being, role functioning, social functioning, and vitality. These two dimensions were found to be positively related to psychological distress. In other words, stronger beliefs in the controllability of an illness are linked with positive outcomes such as well-being and decreased distress. Conversely, stronger beliefs in the adverse consequences of an illness are associated with poorer outcomes, such as decreased physical functioning and decreased well-being, with the same pattern holding true for beliefs in a longer illness timeline.

More recent studies have prospectively shown that “negative” perceptions of illness (e.g., belief in a longer timeline, more consequences, and less controllability) are related to negative outcomes, suggesting the predictive utility of the CSM framework. Petrie et al. (1996) found that illness representations assessed during hospitalization for
myocardial infarction (MI) predicted outcomes assessed three and nine months later. Participants who perceived their illness as having less serious consequences were more likely to return to work within three months. Patients who perceived their illness as being more amenable to cure/control were more likely to attend cardiac rehabilitation. Additionally, belief that the illness has serious consequences predicted disability at three and six month follow-ups. In a sample of UK patients undergoing hemodialysis, illness perceptions predicted all-cause mortality over a 16-month follow-up, even after adjusting for known predictors of mortality in renal patients (Chilcot, Wellsted, & Farrington 2011). Galli et al. (2010) found that, in a sample of patients with chronic pain, illness perceptions were strong predictors of disability, depression, and quality of life outcomes measured after three and six months, adjusting for baseline pain and mood. A six-year longitudinal study of patients with osteoarthritis found that changes toward more negative illness perceptions were associated with increases in pain and functional disability, while illness perceptions that became more positive over time were associated with improved outcomes (Kaptein et al., 2010). Dickens et al. (2008) found that illness perceptions predicted new onsets of depression in the first year of recovery from MI. Belief in a longer timeline predicted new onsets of depression, while belief in the controllability of heart disease was associated with a decreased risk of developing depression. Similar prospective relationships have also been found in other illnesses, such as head and neck cancer (Llewellyn, McGurk, & Weinman, 2007) and irritable bowel syndrome (Rutter & Rutter, 2001), among others. Across prospective studies as well as the aforementioned meta-analysis (Hagger & Orbell, 2003), certain dimensions of illness perceptions have
been more consistently predictive of outcomes. Specifically, higher scores on the
dimensions of timeline and consequences are positively associated with poor outcomes,
while higher cure/control scores are positively associated with better outcomes.

One possible explanation for the relationship between negative illness perceptions
and negative outcomes could be that individuals who perceive more negative
consequences experience more negative outcomes because they have objectively worse
disease. Until recently, few studies had included in their models more objective measures
of disease state, making this a potential confound. However, several recent investigations
have found a significant effect for illness perceptions above and beyond the effect for
illness severity (e.g., Chilcot et al., 2011, Galli et al., 2010), suggesting that the
relationship between illness perceptions and health outcomes is not spurious. This
remains an important issue for future research nevertheless.

As the literature continues to build a strong case for the impact of illness
perceptions on coping behaviors and illness outcomes, only a few studies have tested
whether interventions to change maladaptive perceptions can improve outcomes. In a
randomized, controlled trial, Petrie, Cameron, Ellis, Buick, and Weinman (2002) showed
that a brief, in-hospital intervention was able to change the illness representations of MI
patients. Illness representations were measured at baseline and the intervention was then
individualized, based on each patient’s specific beliefs. The intervention attempted to
change illness representations toward being more adaptive, rather than simply being more
optimistic. For instance, participants who believed that their MI resulted in many
negative consequences often believed that they needed to decrease or discontinue many
of their usual activities due to their heart disease. As a part of the intervention, such beliefs were challenged and participants were given instruction with regard to what kinds of activities are safe after MI. At discharge, patients demonstrated less negative beliefs about the consequences, timeline, and controllability of their heart disease relative to their beliefs assessed at hospital admission. At a three month follow-up, patients in the intervention group were found to have returned to work sooner than the control group, and they reported fewer symptoms of angina. Patients in the intervention arm were more likely to report that they planned to attend cardiac rehabilitation, although actual participation did not differ between the groups. Finally, participants in the intervention group reported better understanding of their illness and better preparedness for hospital discharge than those in the control group.

The same research team designed an innovative text message intervention targeted at improving illness perceptions and adherence among patients with asthma (Petrie, Perry, Broadbent, & Weinman, 2011). Patients who were randomized to the intervention arm received via text message statements about asthma that were accurate and encouraged self-efficacy. At 18 weeks, patients in the intervention arm reported changes in illness perceptions that were more accurate (e.g., a chronic timeline) and more adaptive (e.g., increased perceived personal control). Adherence to prescription inhalers also improved.

Keogh et al. (2011) conducted a randomized trial of a two-session motivational interviewing intervention designed to target negative or inaccurate illness perceptions about type 2 diabetes, and to improve self-management of diabetes. Illness perceptions
and hemoglobin A1C (an index of recent blood glucose levels) were measured at baseline and six-month follow-up. Patients who received the intervention showed an increase in their perception that diabetes can be controlled through their own efforts as well as through medical treatments, and demonstrated statistically significant improvements in their A1C.

Key features of the above studies appear to be the identification of illness perceptions that might be problematic for successful self-management of illness, and the delivery of an individualized intervention directly targeted at changing these perceptions. These studies are encouraging as they suggest that maladaptive illness perceptions are amenable to change through intervention, and that these changes are associated with improved functional outcomes and self-management of illness. Cognitive therapy is a particularly promising approach for changing inaccurate or overly negative illness perceptions because it is designed specifically to modify maladaptive thoughts, beliefs, and attitudes (Beck, Rush, Shaw, & Emery, 1979).

**Application of the CSM to depression.** Although the Common Sense Model is purported to be applicable to mental disorders as well as to physical illness (Leventhal, Diefenbach, & Leventhal, 1992), applications of the model to psychological phenomena have lagged behind the physical illness literature. However, this has recently become an active research area, and a growing number of studies are applying the CSM framework to depression, in particular (e.g., Glattacker, Heyduck, & Meffert, 2012; Vollmann et al., 2010). The results of these studies suggest that CSM dimensions are indeed useful for predicting treatment adherence and outcomes in depression and other mental disorders.
An important initial question is whether the same CSM dimensions operate in mental and physical disorders. To investigate this, Fortune, Barrowclough, and Lobban (2004) examined written descriptions of patients’ last depressive episode among a group of currently or previously depressed women. Statements provided in the descriptions were coded with respect to the five dimensions of the CSM. For example, an individual’s statement that “I was so depressed I could not go to work for a month,” was categorized as a “consequence” perception. Inter-rater reliability among the coders was high (0.82). Few participants spontaneously wrote about all five dimensions of illness representations, and most wrote about only two or three. This finding is consistent with previous work done in physical illness (Lau, Bernard, & Hartman, 1989). The dimensions that were most often written about were identity and consequences. The participants also completed the IPQ (Weinman et al., 1996), and analyses revealed the same inter-relationships among scales that have been found in the physical illness literature (described above), lending further evidence to the utility of the CSM dimensions for the study of depression. The authors concluded that patients’ “models of depression are similar in content and structure to the models of physical ill health that have been identified in the Self Regulation Model literature” (Fortune et al., 2004, p.357).

In one of the first papers to report on the application of the CSM to depression, Brown et al. (2001) argue for the utility of the CSM in studying how patients manage their depression and respond to treatment. The authors assert that depression is similar to any other chronic illness for which self-management is important, and that illness representations may be useful for understanding coping, help-seeking, and treatment
adherence in depression. The authors examined illness representations in a sample of depressed primary care patients and found that representations were related to past treatment experience, coping strategies, and medication adherence. Specifically, patients who had previously received treatment for depression perceived their disorder as having a longer timeline, and as having greater consequences. Individuals who were currently taking an antidepressant medication viewed their depression as having a longer timeline than those who were not on medication. Although these results shed some light on the ways in which an individual’s depression history may influence his or her cognitive representation of current depression, they seem somewhat counterintuitive. To the extent that treatment is efficacious, it should shorten the duration of depression and help to minimize its adverse consequences, and yet individuals who had undergone treatment perceived a longer timeline and more consequences. This is likely due to the fact that individuals with more severe, protracted depressive episodes are more likely to get treatment than patients with shorter, milder episodes. Similarly, individuals who are taking antidepressant medication are more likely to have more severe depression than those who are not taking medication. This question could be addressed by testing in a multiple regression framework whether the current severity of depression is a better predictor of illness representations than is past treatment for depression.

Research also suggests that illness perceptions may influence behaviors such as coping and medication adherence in patients with depression. Brown et al. (2001) report that, after adjusting for the severity of depression, illness perceptions were related to coping strategies. Individuals who endorsed more adverse consequences of their
depression tended to use active coping, religious coping, and self-blame. Patients who perceived their depression as less controllable tended to use religious coping. Also, participants who cited interpersonal factors as a cause of depression were less likely to reliably take their antidepressants. The results of this study are somewhat inconsistent with previously reported findings on the relationships between CSM dimensions and coping in physical illness, which has found higher consequence scores to be associated with less active coping and more avoidance. The authors note that their sample was quite small, and that the study was intended as pilot work. A pattern more consistent with the findings in physical illness emerged in a study by Kelly, Sereika, Battista, and Brown (2007). Depressed patients who perceived more severe consequences of their depression engaged in more maladaptive coping behaviors, such as rumination and engagement in dangerous activities. More adaptive coping strategies such as positive reframing and problem solving were endorsed by individuals who perceived greater control over their depression. This pattern of results mirrors findings regarding illness representations and coping in physical illness which have shown that higher scores on the consequences dimension tend to be related to more maladaptive coping, while higher ratings of cure/control are associated with more adaptive or active coping behaviors.

A German study recently reported on the utility of illness perceptions in predicting depression treatment outcomes. Illness perceptions were assessed in a sample of patients with depressive disorders, two weeks before they entered a 4-6 week inpatient psychosomatic rehabilitation center (Glattacker et al., 2012). After accounting for known predictors of depression, timeline and treatment control perceptions predicted depression
severity and quality of life six months following treatment. However, the authors did not investigate the mechanisms (e.g., adherence or coping) through which illness perceptions influence depression outcomes.

Some research on patients’ beliefs about their depression has not specifically employed the CSM model as a framework, but has used similar constructs. For example, questionnaires that assess patients’ beliefs about medications would overlap with treatment control perceptions. Russell and Kazantzis (2008) found that depressed patients who reported more concerns about medication use were less likely to adhere to their antidepressants. Increased concerns about medications may correspond to a lower score on the CSM treatment control dimension. A corresponding decrease in adherence to antidepressant medication would be consistent with the pattern that weaker beliefs in cure/control are related to less adaptive coping behaviors (e.g., decreased adherence). Other studies have also demonstrated that illness beliefs and beliefs about medications are associated with adherence to antidepressant medications (Brown et al., 2005, and Burra et al., 2007) as well as with reluctance to seek treatment (Edwards, Tinning, Brown, Boradman, & Weinman, 2007).

Many investigations have focused exclusively on the cause dimension, often referred to as “illness attribution.” A study by Lynch, Kendrick, Moore, Johnston, and Smith (2006) found that patients with depression who believed their depression was caused by genetic factors were less likely to adhere to their antidepressant medication regimen. Perhaps a strong belief that depression is caused by genetic factors leads to an erroneous conclusion that there is nothing one can do to alleviate this seemingly
inevitable condition. Such pessimistic beliefs may lead to decreased motivation to adhere to treatment regimens. Addis, Truax, and Jacobson (1995) have reported some intriguing findings which suggest that particular kinds of attributions may be related to differential performance across different types of therapy. Patients who attributed their depression to existential causes (e.g., “I’m stuck where I am in life, nothing ever changes”) had better therapy outcomes when assigned to cognitive therapy but worse outcomes when assigned to behavioral therapy (Addis & Jacobson 1996). A different study found that cognitive therapy was less effective in patients who made biological attributions for their depression (Leykin, DeRubeis, Shelton, & Amsterdam, 2007). Schweizer et al. (2009) examined how depression attributions related to treatment preference in a naturalistic setting. Individuals with depression were informed about the treatment options available to them at a mood disorders clinic (cognitive-behavioral therapy, interpersonal therapy, or antidepressant medication [ADM]), and then chose their preferred treatment. Participants who made intraindividual (e.g., characterological) attributions were more likely to choose CBT, while those who made biological attributions were more likely to choose ADM.

The CSM states that illness perceptions affect illness outcomes through behaviors such as coping or adherence. A weakness of the literature on illness perceptions in depression is the relative lack of longitudinal studies, which limits the conclusions that can be drawn about how illness perceptions relate to coping, adherence, and depression outcomes. Studies that have looked at illness perceptions and depression in a prospective fashion have failed to report on hypothesized mechanisms of action. An ongoing study by
Brown and colleagues will examine coping behavior as a mediator of the relationship between illness representations and psychosocial functioning in depressed primary care patients over a one year period. Results from baseline data have been published and are consistent with the possibility that individuals who perceive a longer timeline are more likely to use strategies such as self-blame and behavioral disengagement, which leads to lower psychosocial functioning (Brown et al., 2007). Although these are cross-sectional findings, they raise the possibility that some of the effects of illness representations are mediated by coping strategies. If this finding is confirmed in the longitudinal analysis, it would be consistent with findings from the physical illness literature.

In summary, recent investigations applying the CSM to depression have found that illness representation dimensions are linked to self-management behaviors such as coping and adherence, as well as to depression outcomes (Brown et al., 2001; Kelly et al., 2007; Brown et al., 2007; Glattacker et al., 2012). Related constructs, such as beliefs about antidepressant medications (which correspond to the CSM dimension of cure/control) predict adherence to prescribed medication regimens (Russell & Kazantzis, 2008). Beliefs in certain causes of depression may be related to antidepressant medication adherence (Lynch et al., 2006), likelihood of successful outcomes across different treatments (Addis & Jacobson, 1996; Leykin, et al., 2007), and treatment preferences (Schweizer et al., 2009). Although the study of illness representations in depression is still developing, it appears that these representations may be important for understanding the process of recovery from depression. The proven utility of the CSM in predicting important health behaviors and outcomes in physical illnesses also suggests the potential
of this approach for characterizing comorbid depression in heart disease. The study of illness representations in depressed cardiac patients may eventually inform treatments in this population and help researchers develop more effective treatments—a necessary step if the goal is to improve cardiovascular outcomes through alleviation of depression.

**Personality, Heart Disease, Depression, and Illness Representations**

Personality traits such as the five domains originally described by Costa and McCrae in 1992 (Extraversion, Agreeableness, Conscientiousness, Emotional Stability/Neuroticism, and Intellect/Openness) are related to coronary artery disease (e.g., Smith et al., 2008), depression (e.g., Trull & Sher, 1994), and perceptions of health and illness (e.g., Goodwin & Engstrom, 2002). Moreover, personality disorders have been shown to relate to these variables as well. The following is a brief review of the personality literature relevant to this dissertation.

Personality traits such as neuroticism and hostility have been shown to be risk factors for the development of coronary heart disease. For instance, Smith et al. (2008) examined the relationship between spouse ratings of negative affectivity or neuroticism facets, dimensions of the Interpersonal Circumplex (IPC), and coronary artery calcification (CAC) in a sample of healthy older adults. The results showed that spouse ratings of anger, anxiety, and depression were significantly related to CAC. Higher scores on dominance and lower scores on affiliation were also related to CAC. There is also evidence that personality pathology, particularly borderline personality disorder, is a risk factor for cardiovascular disease (e.g., Moran et al., 2007).

Personality variables are related to mood states, including depression, in complex
ways. Although a thorough review of this literature is outside the scope of this dissertation, this section summarizes the key findings. Individuals with major depressive disorder have been shown to score high on neuroticism, low on extraversion, high on openness, and low on conscientiousness (Trull & Sher, 1994). Trait extraversion is robustly correlated with positive affect (Watson & Clark, 1997). Personality pathology is also related to depression in several important ways. First, there is a high rate of co-occurrence between major depressive disorder and personality disorders (e.g., Yen, McDevitt-Murphy, & Shea, 2006). Comorbid personality disorders are associated with increased distress, decreased functioning, a more difficult course, and poorer prognosis (Skodol et al., 2005) among patients with major depressive disorder. Additionally, the severity of personality pathology predicts recurrence of major depressive disorder (Craighead, Sheets, Craighead, & Madsen, 2011). Finally, there is evidence for differential treatment effects in patients with major depressive disorder and comorbid personality disorder. Fournier et al. (2008) found that depressed individuals with comorbid personality disorder responded less favorably to cognitive therapy than to treatment with antidepressant medication, while individuals without personality disorder showed a more positive response to cognitive therapy.

Leventhal’s Common Sense Model states that personality factors are likely to be important determinants of illness representations and coping (Diefenbach & Leventhal, 1996). Previous research suggests a strong relationship between personality and perceptions of health, although such perceptions have not typically been conceptualized within a CSM framework. Much of this work has utilized measures of global
expectancies about health, such as optimism that one will remain healthy (Rasmussen et al., 2006). Goodwin and Engstrom (2002) investigated the relationships between “big five” personality traits and perceptions of health in a large community sample. Participants were asked to rate their perceived health on a 5-point Likert scale ranging from very poor to excellent. Among individuals with self-reported medical problems, perceptions of good health were associated with high scores on Agreeableness, Openness, Extroversion, Conscientiousness, and low scores on Neuroticism. Powers and Oltmanns (in press) also found that more negative perceptions of health were related to neuroticism, and additionally to three personality disorders (schizoid, antisocial, and borderline personality disorder).

One of the few studies to examine the relationship between personality variables and CSM-specified illness perceptions was conducted among a sample of patients who had recently had a myocardial infarction (Williams, O’Connor, Grubb, & O’Carroll, 2011). Perceptions of heart disease were measured using the Brief Illness Perception Questionnaire (BIPQ; Broadbent, Petrie, Main, & Weinman, 2006), and were examined with respect to Type D personality, defined as the tendency toward negative affectivity and social inhibition. Patients with Type D personality differed from patients without Type D on all 7 dimensions of illness perceptions. Specifically, patients with Type D perceived more adverse consequences, a longer timeline, less personal and treatment control, lower illness coherence, higher emotional representation, and greater symptomatology.

Goetzmann et al. (2005) conducted a cross-sectional examination of the “big five”
personality factors (NEO-FFI; Costa & McCrae, 1992) and their relationships to illness representations among lung transplant recipients. They found that individuals who perceived having more control over their illness tended to have lower scores on neuroticism and higher scores on openness to experiences.

Taken together, these findings suggest that personality variables influence patient perceptions of health and illness. Personality is therefore important to take into account in the context of the current study.

An additional consideration concerns the measurement of personality during a depressive episode. Some researchers have warned that self-reports of personality assessed during a depressive episode may be influenced by the depressive state (Hirschfeld et al., 1983; Griens, Jonker, Spinhoven, & Blom, 2002), although others have argued that this is not a serious concern (Bagby et al., 1998; Morey et al., 2010). Griens et al. (2002) found that individuals with a depressive disorder reported levels of neuroticism and extraversion which fluctuated with changes in depression severity over 12 weeks. The authors argue that their findings provide evidence that these personality factors reflect states, and not traits, during a depressive episode. Although the change is statistically significant, its clinical significance is questionable. Over the course of 12 weeks, extraversion and neuroticism scores on the NEO-FFI (Costa & McCrae, 1992a) changed by only 1.6 and 3 points, respectively. Bagby et al. (1998) argue that the reports of knowledgeable informants are less likely to be influenced by the patients’ depressive mood state. They found that depressed patients’ ratings of their own personality on the NEO PI-R (Costa & McCrae, 1992b) converged with ratings made by knowledgeable
informants, suggesting that depressed mood may not unduly influence self-reported personality traits. There is no consensus regarding the degree to which depressed mood contaminates self-reported personality, so the potential effects of mood state should be evaluated in future studies of the relationships among personality factors and illness representations. Repeated administrations of self-report personality inventories will make it possible to determine whether ratings vary with depressed mood state, and informant ratings are believed to be less contaminated by the patient’s mood state. Finally, participants should be instructed that when making ratings of personality, long-term, stable characteristics should be considered over current emotional state (Griens et al., 2002).

**Research Goals**

Recent work on depression in non-cardiac samples suggests that individuals’ beliefs about their depression are related to treatment outcomes (Addis & Jacobson, 1996; Leykin et al., 2007; Glattacker et al., 2012) as well as to coping strategies (Brown et al., 2001, Kelly et al., 2007), adherence to pharmacological treatment (Brown et al., 2001; Russell & Kazantzis, 2008), and treatment preferences (Schweizer et al., 2009). Leventhal’s Common Sense Model of Illness Representations is the prevailing framework for understanding these beliefs. The CSM states that individuals organize their beliefs about an illness within a common-sense cognitive representation containing a number of dimensions, including information about the illness’s cause, consequences, timeline, perceived controllability, and associated symptoms (Leventhal et al., 1980). These beliefs are proposed to be major determinants of coping behaviors, which in turn
influence health and functional outcomes (Leventhal et al., 1984). Across a broad literature, the most relevant illness perceptions appear to be consequences, cure/control, and timeline. Higher ratings on the consequences and timeline dimensions consistently relate to worse functional outcomes (e.g., physical functioning and emotional distress), and often predict maladaptive coping. Conversely, the cure/control dimension appears to be positively related to better outcomes (such as well-being and social functioning) and to more adaptive or active coping procedures.

Given that illness representations are related to treatment, it is also important to understand factors that relate to, or perhaps influence, CSM dimensions. Leventhal’s Common Sense Model states that illness representations are determined by stable characteristics such as personality traits, as well as past experiences with the illness in question. However, very few studies have examined this. An additional problem with many previous investigations of illness representations is the failure to account for the potential confounding effect of illness severity. It is important to understand whether illness representations are merely a reflection of the patient’s mood state or illness severity. In other words, illness representations are expected to relate to these factors, but not so strongly as to suggest they are measuring the same thing.

The purpose of this dissertation study is to examine cognitive representations of depression (referred to as “illness perceptions”) in persons with depression and heart disease. This is the first study to examine patients’ common-sense perceptions of depression in a group of individuals with comorbid heart disease and depression. It is designed to determine relationships among illness perception dimensions, depression
severity, depression history, and personality traits in patients with comorbid depression and heart disease, defined as either stable coronary heart disease or chronic heart failure. As a primary aim, this study tested whether timeline, consequence, personal control, and treatment control perceptions are influenced by more than just the severity of depression. It was hypothesized that factors such as a patient’s previous experience with depression as well as personality traits are uniquely related to CSM dimensions, after taking into account depressive severity. A secondary aim of the study is to determine whether similar patterns of relationships exist among the CSM dimensions in a depressed cardiac sample, compared to the pattern which has been reported in medically-ill nondepressed patients, as well as in depressed, medically-well populations. In future analyses, the relationship between illness perceptions and depression treatment outcomes will be investigated.

Assessments were obtained at the baseline evaluation and the first intervention session of a depression treatment study. The Illness Perception Questionnaire-Revised (IPQ-R; Moss-Morris, 2002) was used to measure illness representation dimensions. Analyses were performed at the subscale level, with subscales representing the illness perception dimensions. The timeline, consequences, personal control, and treatment control subscales were selected for analysis because these dimensions have shown the most consistent pattern of relationships with coping and health outcomes in past research.

**Primary Aims**

**Hypothesis 1.** Individuals who view their depression as having many adverse consequences are more severely depressed, have had more previous episodes of depression, and are higher on neuroticism than individuals who report few adverse
consequences. 1a) Depression severity is positively associated with adverse consequences. 1b) Number of previous depressive episodes is positively related to consequences independently of the severity of the current depressive episode. 1c) Neuroticism is positively correlated with adverse consequences independently of the severity of the current depressive episode.

These hypotheses are consistent with the cognitive model of depression, which states that more severely depressed persons tend to hold more negative beliefs about the future (Beck et al., 1979). It is also consistent with the Haggar and Orbell (2003) meta-analytic findings that, across a large number of studies, people who perceived greater consequences of their illness reported higher emotional distress. Additionally, it is plausible that individuals who have had more experience with (e.g., more episodes of) depression have a higher probability of experiencing negative consequences in the past, and therefore would perceive their depression as resulting in more negative consequences. Finally, individuals who are high on trait neuroticism experience more negative affectivity and may therefore be more likely to experience (and report) negative consequences of their illness.

**Hypothesis 2.** Individuals who expect a longer (more chronic) timeline are more severely depressed, have had longer past depressive episodes, and are higher on trait neuroticism. 2a) More severe depression is associated with a longer perceived timeline. 2b) The experience of having longer previous depressive episodes is associated with a longer perceived timeline for depression, independent of severity of current depression.
2c) Neuroticism is positively correlated with timeline, independently of depressive severity.

This hypothesis is again consistent with what is known about the cognitive aspects of depression. Individuals who are more severely depressed tend to be more pessimistic about the future, and are therefore expected to perceive a longer timeline for their depression. In addition, a recent study by Freedland et al. (Freedland, Carney, Steinmeyer, & Reese, 2010) found that ACS patients who were more severely depressed following their cardiac event did indeed experience longer depressive episodes. This hypothesis is also consistent with Hagger and Orbell’s (2003) meta-analysis which found that a longer expected timeline was positively associated with distress across many studies of various physical illnesses. It is also plausible that individuals who report having had past episodes which were more protracted expect a longer timeline for their depression. Finally, individuals who score higher on neuroticism tend to perceive poorer health status (Goodwin & Engstrom, 2002), and are therefore predicted to expect a longer timeline.

**Hypothesis 3.** Individuals who perceive more **personal control** over their depression are less depressed, have lower levels of neuroticism, and higher levels of conscientiousness than those who score low on personal control. 3a) Severity of depression is inversely related to perceived personal control. 3b) Neuroticism is inversely related to personal control, independent of depressive severity. 3c) Conscientiousness is positively associated with personal control, independent of depressive severity.
This hypothesis is consistent with meta-analytic findings in the illness representation literature (Haggar & Orbell, 2003). Across numerous patient samples, the cure/control scale (which has since been split into separate personal and treatment control scales) was negatively associated with psychological distress. Goetzmann et al. (2005) showed that lung transplant patients who perceived higher cure/control for their illness scored lower on neuroticism.

Hypothesis 4. Participants who perceive their depression as being more amenable to treatment control are less depressed, are more likely to have received previous treatment for depression, and report lower levels of neuroticism. 4a) Depression severity is inversely related to treatment control. 4b) Neuroticism is inversely related to treatment control, independent of depressive severity. 4c) Previous treatment of depression is associated with greater perceived treatment control, independent of depressive severity.

This hypothesis is consistent with meta-analytic findings in the illness representation literature (Haggar & Orbell, 2003). Across numerous patient samples, the cure/control scale was negatively associated with psychological distress. Additionally, Goetzmann et al. (2005) showed that lung transplant patients who perceived higher cure/control for their illness scored lower on neuroticism. Finally, it is expected that individuals who perceive higher treatment control are more likely to have sought treatment for depression in the past. Although Brown et al. (2001) found that individuals who had previously received treatment for depression perceived more pessimistic illness representations (longer timeline and perceived consequences), depressive severity was
not included in their analyses. Therefore, it is expected that when depression severity is accounted for, previous treatment should relate to reports of greater treatment control.

The aforementioned predictions state that individuals who are more severely depressed hold more pessimistic beliefs about their illness. These hypotheses could also be consistent with the possibility that more severe depression simply has worse consequences, a longer timeline, and is less amenable to control, suggesting that patients’ perceptions are merely a reflection of the severity of their illness. If this study shows that personality traits and past experience with depression are significantly related to illness perceptions independent of depressive severity, this would suggest that illness perceptions reflect more than just the severity of one’s depression.

**Secondary Aims**

Correlations among CSM dimensions were investigated to determine if the pattern of interrelationships among illness representation domains is consistent with the pattern found in nondepressed medically ill samples and medically-well depressed samples. It was hypothesized that scales of the IPQ show interrelationships that are consistent with both the physical illness literature (Hagger & Orbell 2003) and investigations of the IPQ in depressed samples (Fortune et al., 2004). The following a priori hypotheses were tested: Worse perceived consequences are associated with a longer expected timeline (Hypothesis 5); greater perceived treatment control is associated with fewer perceived adverse consequences (Hypothesis 6); greater perceived treatment control is associated with a shorter expected illness timeline (Hypothesis 7); greater perceived personal control is associated with fewer perceived adverse consequences (Hypothesis 8); and greater
perceived personal control is associated with a shorter expected illness timeline (Hypothesis 9).

**Method**

The hypotheses outlined above were tested in the context of two larger studies conducted by Robert M. Carney, Ph.D. (Study A), and Kenneth E. Freedland, Ph.D. (Study B). Only those aspects of the parent studies which are relevant to the current analyses are described here.

**Participants**

A total of 112 participants (80 from Study A and 32 from Study B) were included in the current study. All participants who completed at least the first intervention session in Study A or B were included in the current analyses.

Study A included participants with major depressive disorder and stable CHD, all of whom received treatment for depression. Participants met the DSM-IV criteria for a current major depressive episode and scored 17 or higher on the 17-item Hamilton Rating Scale for Depression (HAM-D-17; Hamilton, 1960) approximately 3 months after hospitalization for an acute coronary syndrome (ACS). ACS was defined as the occurrence of acute MI or unstable angina, evidenced by (1) symptoms of acute myocardial ischemia in conjunction with (2) significant ST segment elevations or depressions on electrocardiography (ECG) and/or (3) an elevation of serum troponin I greater than 0.5 ng/ml. Patients were excluded from Study A for the following reasons: 1) significant cognitive impairment as evidenced by a score equal to or greater than 20 on the Short Blessed Test (SBT; Katzman et al., 1983); 2) severe Axis 1 comorbidities such
as schizophrenia, bipolar disorder, active substance abuse, or alcoholism; 3) current use of antidepressants; 4) severe medical conditions such as recurrent MI within 3 months of the index event, severe congestive heart failure, malignancy, or serious physical limitations; 5) exemption by the patient’s cardiologist or primary care physician; 6) medical contraindications to the use of sertraline; 7) participation in a competing research protocol; or 8) refusal to participate.

Study B included patients with depression and chronic heart failure (HF), who were randomly assigned to a cognitive-behavioral intervention or to a usual care (UC) control condition which did not provide any depression intervention. HF was evidenced by 1) clinically diagnosed heart failure for at least three months prior to screening; 2) any prior hospitalization with HF listed as a diagnosis; and 3) classification of New York Heart Association (NYHA) class II or III. Participants met the following criteria for depression: 1) DSM-IV criteria for a current major depressive episode, and 2) a score of 14 or higher on the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). Patients were excluded for the following reasons: 1) exemption by the patient’s physician; 2) dementia or significant cognitive impairment; 3) major mobility-limiting physical disability; 4) poor one-year prognosis due to a condition other than heart failure; 5) logistical barriers to participation in laboratory visits; 6) less than 30 years of age; 7) alcoholism or substance abuse, bipolar disorder, schizophrenia, or other psychotic disorder; 8) high risk of suicide; 9) current nonstudy psychotherapy for depression or other psychiatric problems; 10) initiation of antidepressant therapy in the past 8 weeks; and 11) hospitalization for HF or ACS in the past month. In Study A, the decision was
made to administer the current study’s primary measures at the first intervention session, in order to minimize participant burden at the baseline visit (when a large battery of assessments was already planned). To keep the procedures consistent, these measures were also administered at the first intervention session in Study B. Therefore, the current study includes only those Study B participants who randomized to receive CBT.

**Procedure**

**Study A recruitment.** Participants for Study A were recruited from three St. Louis area hospitals affiliated with Washington University School of Medicine and BJC Healthcare. With the attending physician’s permission, patients admitted to the hospital for ACS within the past 3 months were contacted about the study. Upon telephone contact, patients were screened for depression by completing the Patient Health Questionnaire (PHQ-9; Spitzer, Kroenke, & Williams, 1999). Patients who screened positive for depression on the PHQ-9 were invited to visit the Behavioral Medicine Center at Washington University School of Medicine, at which time study eligibility was determined by administration of the BDI-II, the DISH depression interview (Freedland et al., 2002), and HAM-D-17, as well as through a detailed medical history and physical examination.

**Study B recruitment.** Study B recruited heart failure patients. Inpatients were identified via systematic surveillance of admission lists at two area hospitals and were contacted as described above for Study A. Outpatients were recruited from clinical echocardiography services, heart failure clinics, general cardiology clinics, and cardiology faculty group practices that are part of the BJC health care network. Study
brochures were also placed at these sites, although few patients were enrolled through this method. Eligibility was determined by telephone administration of the PHQ-9, as well as a brief psychiatric history interview and screening form to assess for other exclusion criteria. Patients who appeared eligible after the screening telephone call were invited to attend a baseline evaluation appointment at the Behavioral Medicine Center.

Measures

**Demographic Variables.** Participants were asked to provide information about their age, gender, race, ethnicity, education, income, and marital status.

**Depression and Psychiatric History.**

**Beck Depression Inventory (BDI-II).** The BDI-II (Beck, Steer, & Brown, 1996) was used to assess self-reported depression symptoms. This 21-item measure is one of the most widely used measures of depression severity, and has also been used extensively in research on comorbid depression and heart disease. Participants were asked to rate the severity of each symptom over the past two weeks, on a scale of 0-3. The total scale score therefore ranges from 0-63. The coefficient alpha for the current study was 0.82.

**Depression Interview and Structured Hamilton (DISH).** The DISH (Freedland, Skala, et al., 2002) was used to determine whether participants met the DSM-IV diagnostic criteria for major depression. The instrument also yields the following variables which were used in the current study: number of previous major depressive episodes, length of the longest previous episode, and history of treatment for depression. For the current study analyses, the field indicating length of the longest episode was left blank for patients who had never had a previous episode. These patients were therefore
not included in analyses of this variable. The DISH was recently recommended by an NHLBI expert panel for the assessment and treatment of depression in patients with heart disease (Davidson et al., 2006).

**Beck Anxiety Inventory (BAI).** The BAI (Beck & Steer, 1993) was used to assess severity of self-reported anxiety symptoms. Participants were asked to rate the severity of 21 symptoms over the past week, on a scale of 0-3. The total scale score therefore ranges from 0-63.

**Health Status and Medical History.** Detailed information about participants’ medical history and current medical status were obtained from both hospital records and from patient self-report. These data included information specific to cardiovascular health (cardiovascular history, cardiological test data, history of revascularization, and cardiac risk factors) and information about any other major medical illnesses (past or current), as well as current medication use.

**Personality traits.** The short form of the International Personality Item Pool (Mini-IPIP) was used to assess the Big Five factors of personality: Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Intellect/Imagination (sometimes referred to as Openness) (Donnellan, Oswald, Baird, & Lucas, 2006). This 20-item measure uses four items to assess each big five trait. It was designed as a short form of the 50-item International Personality Item Pool—Five-Factor Model measure (IPIP-FFM; Goldberg, 1999), which is available in the public domain. Because the participants were both medically ill and depressed, the short form was used in order to minimize participant burden. The Mini-IPIP includes statements describing personality characteristics, such as
“I am the life of the party,” and “I do not have a good imagination.” Participants were asked to indicate the accuracy of each statement on a 5-point Likert scale ranging from 1 (very inaccurate) to 5 (very accurate). Scores were then summed for each factor. Internal consistency reliabilities for each of the five scales have been shown to be well above .60 in a series of five studies conducted by the scale developers. The largest study, which included a sample of over 2500 adults, reported the following coefficient alphas: extraversion = .77; agreeableness = .70; conscientiousness = .69; neuroticism = .68; and intellect/imagination = .65. In the same study, convergent correlations were high between the Mini-IPIP scales and Goldberg’s IPIP-FFM scales ($r = .93$ for extraversion, .89 for agreeableness, .90 for conscientiousness, .92 for neuroticism, and .85 for intellect/imagination). The coefficient alphas for the current study are provided in Table 1. Alpha values for the Mini-IPIP ranged between .63 and .67, with the exception of neuroticism (.49).

**Illness Representations.** The Revised Illness Perception Questionnaire (IPQ-R; Moss-Morris et al., 2002) was used to assess participants’ cognitive representations of their depression. The IPQ-R and the original version of the instrument, the IPQ (Weinman, et al., 1996), were designed to measure the illness representation domains described in Leventhal’s common-sense model of illness representations (Leventhal et al., 1984). Specifically, the IPQ was composed of 5 subscales, corresponding to Leventhal’s domains of illness representations: identity, consequences, cure/control, cause, and timeline. Since its development, the IPQ has been widely used as a measure of illness representations in many chronic diseases, including heart disease (Petrie et al.,
It has also been shown to relate to a number of constructs as proposed by the CSM, including coping (Heijmans & deRidder, 1998), mood (Grace et al., 2005; Hermele, Olivo, Namerow, & Oz, 2007), and adherence (Cooper, Lloyd, Weinman, & Jackson, 1999; Weinman, Petrie, Sharpe, & Walker, 2000).

Revision of the IPQ was undertaken due to concerns about some of the subscales’ psychometric properties (Moss-Morris et al., 2002). In particular, the cure/control and timeline scales lacked internal consistency. When cure/control was reanalyzed using factor analysis, it was discovered that items loaded onto two separate factors—one related to personal control/self-efficacy, and the other related to perceived treatment control/outcome expectancies. Since these two factors were only weakly correlated, two separate subscales were created. Concerns about the internal consistency of the timeline scale were remedied by increasing the number of items on that scale, and by adding an additional subscale assessing cyclical timeline beliefs. The authors also added a subscale to assess emotional representations of illness, in order to more completely reflect the parallel processing of both cognitive and emotional representations described in the CSM. Finally, the authors added another subscale to assess “the extent to which a patient’s illness representations provided a coherent understanding of the illness” (Moss-Morris et al., 2002, p. 2). Including both the new subscales and the original five domains of the IPQ, the IPQ-R contains the following 9 subscales: identity, consequences, cause, personal control, treatment control, timeline (acute/chronic), timeline cyclical, emotional representations, and illness coherence.
Sections of the IPQ-R which were administered in the current study included all scales except the identity and cause scales. The list of symptoms which comprises the IPQ-R identity scale includes experiences typical of physical illness, such as sore throat, nausea, breathlessness, and stiff joints. These symptoms are not necessarily applicable to depression. Similarly, the cause scale includes items that are unlikely to be endorsed as causes of a depressive disorder, such as “a germ or virus,” “diet or eating habits,” and “pollution in the environment.” In order to include either of these scales, lengthy modifications would have been necessary to make the scales more applicable to depression. For these reasons and in order to decrease participant burden, the identity and the cause subscales were excluded from the current study. Participants were administered the free-response causal item, which asks participants to rank the three most important factors they believe to have caused their illness (depression). The IPQ-R was also modified to read “your depression” instead of “your illness,” as has been done in other studies using the IPQ-R in depression (Fortune et al., 2004; Glattacker et al., 2012; Vollmann et al., 2010). It is also recommended in the scoring materials for the IPQ-R that the word “illness” be replaced with the name of the specific illness under study (Moss-Morris et al., 2002).

The IPQ-R asks participants to read various statements about their illness and rate their agreement with each statement on a Likert scale with the following responses: strongly disagree, disagree, neither agree nor disagree, agree, and strongly agree. Each rating has a corresponding numerical value. IPQ-R scale scores are then obtained by summing the values for the items on each scale. Higher scores on the timeline,
consequences, timeline cyclical, and emotional representation scales have a negative valence, indicating beliefs that the illness will have a long timeline, more negative consequences, a more cyclical nature, and a greater emotional impact. High scores on the personal control, treatment control, and coherence scales have a positive valence, representing beliefs that the illness is amenable to control by personal efforts or treatment, and a greater personal understanding of the condition.

Psychometric data on the IPQ-R are reported in the original paper (Moss-Morris et al., 2002). The seven subscales used in the current study demonstrated good internal consistency on a sample of 711 patients from 8 different illness groups, with Cronbach alphas ranging from .79 to .89 (Moss-Morris et al., 2002). In addition, the 38 items corresponding to these seven scales were entered into a principal components analysis, which produced seven factors corresponding to the theoretically derived scales. As described above, inter-relationships among the various scales have also indicated logical relationships. For example, high scores on the control subscale correlate negatively with more pessimistic illness representations in other domains, such as the belief in a long timeline or in serious consequences (Moss-Morris, 2002). The IPQ-R has shown reasonably good test-retest reliability, with correlations ranging from .46 to .88 over a three-week period in renal dialysis patients. The six-month stability in the same sample showed correlations all greater than .5 (Moss-Morris, 2002). In order to demonstrate that the IPQ-R measures more than simply affective states, the scale developers report small to moderate correlations between IPQ-R dimensions and the PANAS (Watson, Clark, & Tellegen, 1988). The largest correlation reported was .54, which was found between
emotional representations and negative affect.

The IPQ-R has also been shown to have acceptable psychometric properties when used to examine illness perceptions of depression (Glattacker et al., 2012). Internal consistency of the timeline, consequences, and cure/control scales ranged from .56 to .76, and two-week test-retest reliability was .68 or above for each scale. In addition, the pattern of correlations among subscales in depressed samples resembles the pattern seen in studies of IPQ dimensions in physically ill patient populations (Fortune et al., 2004). Table 1 displays the coefficient alphas for the current study, which vary between .71 and .82, with the exception of the timeline cyclical scale (.57).

Two studies by Brown et al. lend evidence to the validity of the IPQ as a measure of depressed patients’ cognitive models of depression. Brown et al. (2001) found that after accounting for severity of depression, illness cognitions in depressed primary care patients were associated with current and past treatment-seeking behavior, medication adherence, and coping strategies. Kelly et al., (2007) also found that illness representation dimensions were related to coping in a depressed sample, and the results paralleled findings regarding illness representations and coping in physical illness. Finally, Brown et al. (2007) found evidence that maladaptive coping strategies played a mediational role in explaining the relationship between illness representations and psychosocial well-being. The latter finding is consistent with Leventhal’s (1984) full Common-Sense Model, which states that illness representations influence coping strategies and health behaviors, which in turn influence health outcomes.

**Timeline for assessments.** The BDI-II and the DISH were administered at
baseline. The IPQ-R and the Mini-IPIP were administered to participants in both studies at the first intervention session. All other data used in the current study (such as demographic information and medical history) were collected at baseline.

**Statistical Analysis**

**A Priori Hypotheses.**

**Hypothesis 1.** 1a) Depression severity is positively associated with adverse consequences. 1b) Number of previous depressive episodes is positively related to consequences independent of the severity of the current depressive episode. 1c) Neuroticism is positively correlated with adverse consequences independently of the severity of the current depressive episode. To test these hypotheses, a hierarchical linear regression analysis was conducted with severity of depression (measured by the BDI-II) entered in the first step, number of previous episodes of depression entered in the second step, and Mini-IPIP neuroticism score entered in the third step. It was hypothesized that all predictors would be significant in the complete model, suggesting that personality and experience have independent explanatory power above and beyond the severity of depression.

**Hypothesis 2.** More severe depression is associated with a longer perceived timeline. 2b) The experience of having longer previous depressive episodes is associated with a longer perceived timeline for depression, independent of severity of current depression. 2c) Neuroticism is positively correlated with timeline, independently of depressive severity. To test these hypotheses, a hierarchical linear regression analysis was conducted with BDI entered in the first step, length (in weeks) of the longest previous
depressive episode entered in the second step, and Mini-IPIP neuroticism entered in the final step. It was predicted that all variables would be significant in the complete model, indicating that personality and experience help to explain expected timeline, after accounting for depression severity.

**Hypothesis 3.** 3a) Severity of depression is inversely related to perceived personal control. 3b) Neuroticism is inversely related to personal control, independent of depressive severity. 3c) Conscientiousness is positively associated with personal control, independent of depressive severity. To test these hypotheses, a hierarchical regression analysis was conducted with BDI entered as the first predictor, followed by the Mini-IPIP neuroticism and conscientiousness scores. It was hypothesized that all predictors would be significant in the complete model, suggesting that neuroticism and conscientiousness help to explain perceived personal control, after adjusting for the effect of depression severity.

**Hypothesis 4.** 4a) Depression severity is inversely related to treatment control. 4b) Neuroticism is inversely related to treatment control, independent of depressive severity. 4c) Previous treatment of depression is associated with greater perceived treatment control, independent of depressive severity. To test these hypotheses, a hierarchical regression analysis was conducted with BDI entered as the first predictor, followed by Mini-IPIP Neuroticism score. In the last step of the model, a binary indicator of the presence or absence of past treatment for depression was entered. It was expected that all predictors would be significant in the complete model, indicating that
neuroticism and past experience with treatment helps to explain perceived treatment control, after adjusting for the effect of depression severity.

**Hypotheses 5-9.** 5) Worse perceived consequences are associated with a longer expected timeline, 6) greater perceived treatment control is associated with fewer perceived adverse consequences, 7) greater perceived treatment control is associated with a shorter expected illness timeline, 8) greater perceived personal control is associated with fewer perceived adverse consequences, and 9) greater perceived personal control is associated with a shorter expected illness timeline. To test these hypotheses, bivariate correlations were calculated between scores on each relevant pair of IPQ-R scales.

**Exploratory analyses.**

Relationships among Big Five personality traits, depression severity, and all seven illness representation dimensions were explored in correlational analyses. Depression was expected to relate modestly, if at all, with illness perception dimensions. In other words, depression scores were expected to relate to illness perception scores, but not so strongly as to suggest they are measuring the same construct.

**Tests of statistical assumptions.** For the primary analyses (hypotheses 1-4), data were examined for violations of statistical assumptions. Scatter plots were examined for evidence of linearity in the relationships between predictors and outcomes. Scatter plots were also used to examine outliers. Dependent variables (IPQ-R consequences, timeline, personal control, and treatment control) were tested for skewness and kurtosis. Distributions of regression residuals were compared to the normal distribution. To test for homoscedasticity of errors, plots of predicted values versus residuals were examined.
The assumption of independence of errors was examined using the Durbin-Watson statistic. Finally, tests for multicollinearity of predictors were conducted. No evidence was found for skewness or kurtosis in the distributions of the four primary dependent variables (IPQ-R consequences, timeline, personal control, and treatment control). Scatter plots of each predictor versus dependent variable were examined for outliers. One major outlier was found on the duration of longest episode variable (included in Hypothesis 2). This value was removed and the regression analysis was re-run, with no change in results. The results reported here therefore include this observation. Scatter plots were then examined with respect to the linearity of the relationships between predictors and outcomes. No evidence was found for nonlinear relationships. For each regression analysis, a histogram of residuals was compared to a normal distribution curve and normal probability plots were examined. All residual distributions were approximately normal, consistent with multivariate linear regression assumptions. To test for homoscedasticity of the errors, plots of predicted values versus residuals were examined. Scatter plots revealed no evidence for heteroscedasticity. The assumption of independence of errors was examined for each analysis using the Durbin-Watson statistic. The computed statistic did not indicate a violation of this assumption. Finally, tests for multicollinearity of predictors were conducted. Tolerance and VIF statistics were computed and did not indicate any significant multicollinearity issues.

Results

Descriptive Statistics

**Demographic variables.** A total of 112 patients were enrolled in the study,
including 80 patients (71%) from Study A, and 32 (29%) from Study B. Table 2 displays demographic and medical characteristics of the sample. The complete sample includes 45 women (40%) and 67 men (60%). The participants ranged in age from 30 to 81, with a mean (SD) age of 59 (10.3) years. The sample was 79% white (N=88) and 20% African-American (N=23). Thirty-one percent of the participants (N=28%) reported having a high school education or less, while 72% (N=81) had more than a high school education.

Depression, personality, and illness perceptions. Table 3 describes the psychological variables. Baseline BDI scores ranged from 16 to 51, with a mean score of 30 (8). As part of the DISH, patients were asked whether they had ever experienced a depressive episode prior to the current one, and if they ever received treatment for depression. Those who reported having a previous episode were asked how many episodes they had, when they occurred, and how many weeks the longest episode had lasted. Eighty-one percent of patients (N=91) reported having a previous depressive episode, and over two thirds of the sample (71%; N=80) stated that they had previously received treatment for depression. Among the 91 patients who reported having had a previous episode of depression, the average number of reported episodes was approximately 3 (2.6), while the modal number of episodes was 1 (32%). The mean reported duration of the longest depressive episode was 82 weeks (123; range: 2-884). The average age at first episode was 32 years (16), while age at last episode was 48 (14). The average number of days between baseline assessment and initial CBT session was 11.4 (7).

Primary Analyses
Hypothesis 1. The results of the regression analysis are presented in Table 4. 1a) The BDI accounted for a significant amount (15%) of the variance in IPQ-R consequences in the first step of the model \((p < .001)\). 1b) The model was not significantly improved by adding neuroticism in step two of the model. 1c) The model was also not improved by adding number of depressive episodes in step three. All two-way interactions were tested, and none were found to be significant.

Hypothesis 2. Table 5 displays the results of the regression model predicting IPQ-R timeline. 2a) The BDI accounted for 6% of the variance in timeline in the first step of the model \((p < .05)\). 2b) Neuroticism accounted for an additional 10% of the variance in timeline when it was added in the second step of the model \((p < .01)\). With neuroticism in the model, BDI was no longer significantly related to timeline \((p = .40)\). 2c) The addition of the duration of longest depressive episode variable did not account for a significant additional proportion of variance in the third step of the model. The only significant regression coefficient in the full model was neuroticism \((p < .01)\). All two-way interactions were tested. A significant interaction was found between neuroticism and duration of longest depressive episode \((p < .05)\). To examine the nature of the interaction, the duration variable was divided into three groups: short duration (less than or equal to 6 months), medium duration (greater than 6 months but less than two years), and long duration (greater than or equal to two years), and the relationship between neuroticism and IPQ-R timeline was plotted for each group. As can be seen in Figure 1, the relationship between neuroticism and timeline decreases as a function of increasing depressive episode duration \((r = .59\) for short, \(r = .41\) for medium, and \(r = .12\) for long).
Hypothesis 3. Results of the personal control regression analysis can be found in Table 6. 3a) BDI did not account for a significant portion of variance in step 1 of the model. 3b) Neuroticism did not significantly relate to personal control. 3c) Conscientiousness did not significantly relate to personal control. The full model accounted for only 4% of the variance in personal control. All two-way interactions were tested, and none were found to be significant.

Hypothesis 4. Results of the regression analysis for treatment control can be found in Table 7. 4a) The BDI did not account for a significant proportion of the variance in IPQ-R treatment control in the first step of the model. 4b) The addition of neuroticism accounted for a significant proportion of variance (6%) in step two ($p < .01$). 4c) Past treatment of depression in step three did not account for a significant portion of the variance in the outcome. In the full regression model, neuroticism remained the only variable significantly (negatively) related to treatment control ($p < .01$). Two-way interactions were tested, and none were found to be significant.

Secondary Analyses

Hypotheses 5-9. The bivariate correlations between scales of the IPQ-R can be found in Table 8. As predicted, higher scores on the consequences scale were significantly associated with higher scores on the timeline scale ($r = .263, p = .005$). Treatment control was not correlated with the consequences scale ($p = .919$), but higher scores on treatment control were associated with lower scores on the timeline scale ($r = -.41, p < .001$). The personal control scale was not related to the IPQ-R consequences scale ($p = .66$), but was significantly negatively associated with the IPQ-R timeline scale.
$r = -.47, p < .001$).

**Exploratory analyses**

Pearson correlations were calculated to explore relationships between illness perceptions and mood, depression history, and personality.

**Relationships between IPQ-R scales and mood.** Table 9 displays results for bivariate correlations between IPQ-R scales and mood. In Pearson correlations, BDI was significantly positively associated with three of the IPQ-R scales: consequences ($r = .38, p < .001$), timeline ($r = .24, p = .01$), and emotional representation ($r = .53, p < .001$). Anxiety (measured by the BAI) was significantly related to the emotional representation scale ($r = .37, p < .001$). To determine if this relationship could be accounted for by neuroticism, a multiple regression analysis was conducted. In the model, both BAI and neuroticism were significantly related to emotional representation (both $p < .01$).

**Relationships among IPQ-R scales.** Bivariate correlations among IPQ-R scales can be found in Table 8. In addition to the hypothesized relationships described above, significant bivariate relationships were also found between IPQ-R personal control and the treatment control ($r = .57, p < .001$), between emotional representations and consequences ($r = .58, p < .001$), and between emotional representations and timeline ($r = .32, p = .001$).

**Relationships between IPQ-R scales and personality.** Bivariate correlations between IPQ-R scales and Personality can be found in Table 10. Among Mini-IPIP scales, neuroticism was correlated with five of the seven IPQ-R scales. In Pearson correlations, neuroticism was significantly related to consequences ($r = .30, p = .001$),
timeline ($r = .39, p < .001$), personal control ($r = -.19, p = .040$), treatment control ($r = -.22, p = .020$), and emotional representations ($r = .49, p < .001$). Conscientiousness was related to consequences ($r = .28, p < .01$) and emotional representation ($r = .26, p < .01$). Openness was significantly associated with illness coherence ($r = .33, p < .001$). Agreeableness was related to personal control ($r = .26, p < .01$), and extraversion was related to timeline ($r = -.25, p < .01$). Regression analyses from Hypothesis 2 (IPQ-R timeline) and Hypothesis 3 (IPQ-R Personal Control) were re-run post hoc with the addition of extraversion and agreeableness, respectively. The regression analysis predicting timeline therefore included the following predictors: BDI, neuroticism, duration of longest depressive episode, and extraversion. In the full model, the original results remained unchanged; extraversion was not significantly related to timeline and neuroticism was the only significant predictor of timeline. The post hoc regression analysis predicting personal control included BDI, neuroticism conscientiousness, and agreeableness. In the original analysis, no predictors were significant in the full model. In the post hoc analysis, agreeableness was significantly positively related to personal control in the full model ($p < .01$).

**Relationships between IPQ-R scales and depression history variables.** The following depression history variables were examined with respect to their correlations with IPQ-R scales: history of depressive episode, history of past treatment for depression, number of previous episodes and duration of the longest episode. Duration of the longest depressive episode was significantly negatively correlated with personal control ($r = -.22, p < .05$). No other indices of depression history were related to illness perceptions.
Discussion

This study is the first to examine illness perceptions in patients with comorbid depression and heart disease. The primary aim of the study was to examine baseline relationships between patients’ perceptions of depression and depression severity, personality, and depression history. A priori predictions were made regarding correlates of consequences, timeline, personal control, and treatment control. It was predicted that depression severity, neuroticism, and number of depressive episodes would be significantly associated with consequences in multiple regression. Consequences were strongly related to depression, but were not further explained by neuroticism or depressive episodes. Exploratory analyses examined bivariate relationships between consequences and personality, mood, and depression history. Notably, although it was not a significant predictor in the multivariate model, neuroticism was significantly correlated with consequences at the bivariate level. Since depression and neuroticism were significantly correlated with each other, when both variables were in the regression model, depression was revealed as a stronger predictor of consequences. Conscientiousness and anxiety were also positively related to consequences at the bivariate level. Taken together, these results suggest that cardiac patients’ perceptions of the consequences of their depression are strongly influenced by negative affect, and are more strongly related to state (e.g., depression) than trait factors. Additionally, having had more depressive episodes does not lead patients to expect more serious consequences of their depression.

Although depression, neuroticism, and duration of the longest depressive episode
were all hypothesized predictors of timeline perceptions, neuroticism was the only significant predictor in the multivariate model. Indeed, depression was significant until neuroticism was added to the model, indicating that the latter demonstrates stronger explanatory power. Exploratory analyses also suggest that individuals who are higher on extraversion perceive a shorter timeline for their depression. To determine whether extraversion, or positive affect, could account for these findings, a post hoc analysis was conducted including the original predictors and extraversion. The original results remained unchanged, with neuroticism remaining the only significant predictor of timeline. Interestingly, a significant interaction was found between duration of longest depressive episode and neuroticism, demonstrating that the relationship between neuroticism and timeline was stronger for individuals with shorter previous episodes of depression, compared to individuals with longer previous episodes. It may be that, for individuals who have less experience with depression (e.g., shorter episodes), perceived length of current depression is influenced strongly by personality tendencies. However, in individuals with longer past episodes, neuroticism is not as influential in determining perceived timeline.

Contrary to the a priori prediction, none of the variables were significant in the multiple regression model for personal control. Neuroticism was significantly inversely related to personal control at the bivariate level. Exploratory analyses also revealed that patients who were more agreeable endorsed more perceived personal control. This again suggests an influence of personality factors on patients’ perceptions. Post hoc regression analyses revealed that when agreeableness is added as a predictor to the original set of
variables, it accounted for 5.6% of the variance in personal control ($p < .05$), and was the only significant predictor in the model. It was not predicted that agreeableness would be related to personal control, and an explanation for this finding was not readily available in the literature. There is some evidence that agreeableness is related to more active coping (e.g., Lawson, Bundy, Belcher, & Harvey, 2010), and personal control is also related to active coping. Additionally, individuals who are high on agreeableness are likely to foster more social support from others, and such support may lead one to feel a greater sense of self-efficacy to manage depression.

Regression analyses predicting treatment control showed that while neuroticism was significantly inversely related to treatment control, depression and number of depressive episodes showed no relationship. Additionally, neuroticism was the only variable that was significantly correlated with treatment control in exploratory bivariate correlations. It is notable that depression severity showed no relationship (in multivariate or bivariate analyses) to patients’ perception of the controllability of their depression. Rather, control perceptions appear to be more strongly related to personality variables.

A priori predictions about the correlations among the IPQ-R scales were supported by the data in three out of five cases. As expected, greater perceived consequences were related to a longer perceived timeline, and both perceived treatment control and personal control were associated with the perception of a shorter illness timeline. However, there was no significant relationship between perceived treatment control and consequences, or between personal control and consequences, even though these are relationships that have been found in many previous studies across different
patient samples (e.g., Hagger & Orbell, 2003). Although not predicted a priori, treatment control and personal control were strongly correlated, which makes conceptual sense. The emotional representations scale was significantly correlated with consequences and timeline in post hoc analyses. The two scales that were most strongly related were consequences and emotional representation, and each of these scales showed significant positive correlations with the same four variables: depression, anxiety, neuroticism, and conscientiousness. This raises the question of whether there is actually too much overlap in the constructs measured by these two scales. Indeed, previous factor analytic studies in other populations have demonstrated that the consequences and emotional representations items consistently loaded onto one factor (Wittkowski et al., 2008).

A main goal of this study was to determine whether cardiac patients’ perceptions of depression reflect more than just the severity of their depressive disorder. Personality and/or depression history were therefore expected to relate to illness perceptions independently of depression severity, and correlations between BDI and illness perceptions were expected to be modest. The regression findings for the timeline, personal control, and treatment control perceptions support the above hypothesis. Only the consequences and emotional representation scales related particularly strongly to the BDI in exploratory analyses (both p < .001). In the regression analysis of consequences, other hypothesized predictors did not relate to consequences independently of depression. Therefore, it cannot be ruled out that, among cardiac patients with depression, perceptions of consequences represent little more than patients’ current depressive severity, or are influenced by the depressive state. Indeed, it is reasonable to expect that
patients would base their perceptions of the consequences of their depression on the severity of symptoms they are currently experiencing. However, in order for this scale to be helpful for understanding depression outcomes in heart patients, it should demonstrate predictive utility above and beyond that of depression severity. It remains to be seen if the consequences scale will be useful in this capacity; however, this will be examined in future work.

The study did not confirm that the consequences scale related to other variables as expected based on theory and previous research. Previously well-replicated associations between consequences and other IPQ-R scales were also not found. These results raise some concern about the application of the consequences scale to patients with comorbid depression and heart disease. It is possible that the scale is approached differently by this patient population. A confirmatory factor analysis could be conducted in the future to determine if the 7 subscale model used by the IPQ-R fits well with data from the current sample. This may be possible once a larger sample of data is accrued, as data collection is ongoing. It may also be interesting to collect IPQ-R data from a depressed, otherwise healthy sample and use confirmatory factor analytic techniques to compare the best fitting subscale models between these two samples. This would help clarify if there is something different about the dimensions that cardiac patients consider when conceptualizing their depression, compared to medically healthy patients with depression.

Contrary to a priori predictions, the depression history variables were not associated with illness perceptions, even though similar variables have been related to perceptions of depression in other research (Brown et al., 2001). The most notable
finding for depression history emerged as an interaction between neuroticism and duration of longest past episode in the IPQ-R timeline analysis. It could be that past experience with depression influences perceptions of current depression in complex ways, such as through interactions with personality characteristics, and that more complex analyses are needed to uncover the effects of depression history on perceptions of depression. Alternatively, patients may not have been able to precisely recall the answers to questions about their history of depression in the current study. For example, patients were asked how many depressive episodes they had as well as the duration of the longest episode. Many patients reported a history of depression spanning years or even decades, and may therefore have had difficulty with accurate recall of these details. In other words, the current study’s assessment of past experience with depression may represent an unreliable measure. A more precise measure could be obtained through longitudinal follow-up of patients, or by verifying facts about patients’ past depression through chart review. However, a longitudinal study of this kind would need to be conducted over a very long period of time and would be extremely costly. Chart review would present its own logistical problems, as this data may not be readily available in medical records for many patients. Although the CSM posits that patients’ experience with an illness influences their conceptualization of that illness, it is also possible that depression history does not play a major role in the conceptualization of comorbid depression among patients with heart disease. For instance, if patients truly have poor recall for the details of their past depressive episodes (particularly those that occurred in the remote past), their memories may not have an impact on their perceptions about their
current episode.

In many cases, IPQ-R scales showed stronger relationships with personality variables than with current depressive state. Previous work has not looked extensively at the relationship between personality and illness perceptions, but several studies have found a correlation between personality and subjective perceptions of overall health. The Common Sense Model specifies that individuals’ illness perceptions are influenced by several factors, including personality, so the current study’s findings fit within the CSM framework. Additionally, this study replicated findings by Goetzmann et al. (2005) that perceptions of control are negatively correlated with neuroticism. However, the fact that perceptions related strongly to indices of personality traits, which are thought to be more stable than mood, raises the question of whether illness perceptions in this population are less dynamic than the CSM would specify. The CSM states that patients’ illness perceptions change with experience and with the acquisition of new information. Previous work has also found that patients’ beliefs about an illness are amenable to change through intervention. This is particularly important because perceptions of control may be promising as a focus for treatment, as they have been associated with adaptive coping, and have predicted depression treatment outcomes (Glattacker et al., 2012). It is possible that patients with comorbid depression and heart disease may differ from previously studied populations in that they have more stable perceptions which are based more strongly on personality factors. An alternative explanation is that patients form beliefs about depression based upon their own personality tendencies in the absence of objective information or efforts to change such beliefs. Preliminary evidence for this
notion emerged in the significant interactive relationship found between neuroticism and duration of longest episode on IPQ-R timeline. For individuals who had shorter depressive episodes (and therefore had less information about depression), the effect of neuroticism on timeline was large \( (r = .59) \). However, for patients with longer episodes, the effect of neuroticism was relatively small \( (r = .12) \). Future work is needed to clarify the potentially complex interactive effects between personality, depression history, and perceptions of depression.

Several of the study’s findings did not fit with predictions based on the Common Sense Model of Illness Perceptions. Specifically, perceptions of depression did not reliably relate to past experience with depression. The consequences scale did not show predicted relationships with other variables and may have been influenced by depression state. Finally, perceptions of depression’s consequences did not relate to perceptions of control, as has been established in many previous studies. Illness perceptions for depression in patients with heart disease may behave somewhat differently than illness perceptions in other illnesses or in medically healthy individuals with depression. There could be several possible explanations for this. The current examination of illness perceptions was conducted in the context of a treatment trial, which differs from the context in which previous research has been conducted. Additionally, there may be complex ways in which patients’ experience of heart disease influences their perceptions of depression. This could be examined in several ways. First, patients’ perceptions of both their heart disease and their depression could be measured, and relationships between these two sets of perceptions could be investigated. Additionally, it would be
useful to compare perceptions of depression in matched samples of medically healthy patients and patients with cardiac disease. This is the first study to look at patient’s perceptions of comorbid depression in a sample that has significant medical illness, and future work should help clarify how this comorbidity may affect patient perceptions.

The study has several limitations. First, for the sake of project feasibility, relationships were only examined among variables collected at baseline and the first intervention session. However, longitudinal data for this study are currently being collected and will be available in the future. Second, in order to minimize participant burden, measures of coping were not collected. Given results from other research, it will be important to examine relationships among illness perceptions and coping strategies in this population. Additionally, the Big Five personality factors were assessed, but the facets that make up these traits were not. Data on the relationship of personality facets to illness perceptions may help explain what is driving the observed associations. Lastly, coefficient alpha values observed in this sample indicated that the Mini-IPIP may not have been reliable. In particular, the alpha for the neuroticism scale was quite low (.49), suggesting that larger effects might have been observed with a more reliable measure. If possible, future work should make use of a personality measure with more items per trait.

Data collection has continued since analysis of the data reported here, allowing several of the above questions to be examined in future work. Additionally, hypotheses pertaining to the process and outcome of treatment represent future research questions that will be addressed using data collected as part of this study. Various measures of the treatment process are being collected, including patients’ adherence to homework and
number of intervention sessions attended. Initially, data will be examined to determine if illness perceptions relate to depression treatment outcomes. IPQ-R scores before and after undergoing CBT for depression will be examined to determine if patients’ perceptions of their depression change over time, and whether any changes correspond to changes in depression severity. Future work will also examine how illness perceptions relate to markers of treatment adherence such as adherence to CBT homework and attendance of therapy sessions. If illness perceptions do impact treatment outcomes via mediating factors such as adherence, interventions akin to those used in previous research may be able to modify important illness beliefs. This line of work will contribute to our knowledge of how individual difference characteristics relate to treatment in patients with comorbid depression and heart disease, and may be used to develop more refined or individually-tailored treatments.

Finally, illness perceptions may also be related to coping and treatment outcomes in more complex ways than have been studied thus far. The impact of one illness perception dimension on coping might be moderated by higher or lower scores on a different dimension. For instance, patients with high scores on illness consequences but also high scores on personal control may cope differently than patients with high consequences but low personal control perceptions. However, no previous studies have examined interactions between illness perceptions. This should be a goal of future work.

In summary, this study represents an important first step in elucidating how patients with cardiac disease conceptualize comorbid depression, and the role that illness perceptions may play in depression treatment among patients with heart disease. The
results show that most dimensions of illness perceptions (with the exception of consequences) are not influenced by depression, and in most cases relate to one another in ways that are consistent with previous research findings. This suggests that assessment of illness perceptions can increase our understanding of depression in heart patients, and that illness perceptions represent individual differences which may inform treatment. Finally, illness perceptions showed strong relationships with personality variables, which has not been consistently studied and suggests an area where much future work is needed. Data currently being collected for this study will be used in the future to investigate important questions about the role of illness perceptions in the treatment of depression among patients with heart disease.
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Table 1  
\textit{Coefficient Alpha}  

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Table 2

Demographic and Medical Characteristics

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Note. MI = myocardial infarction; PTCA = percutaneous transluminal coronary angioplasty; CABG = coronary artery bypass grafting; CVA = cerebrovascular accident.
Table 3

*Psychological Variables*

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<td></td>
<td><em>M (SD)</em></td>
</tr>
<tr>
<td><strong>Total sample</strong></td>
<td><em>(N = 112)</em></td>
</tr>
<tr>
<td>BDI-II at baseline</td>
<td>30.4 (8)</td>
</tr>
<tr>
<td>BAI at baseline</td>
<td>15.5 (9.5)</td>
</tr>
<tr>
<td>IPQ-R</td>
<td></td>
</tr>
<tr>
<td>Consequences</td>
<td>22.7 (3.7)</td>
</tr>
<tr>
<td>Emotional Representation</td>
<td>21.4 (3.6)</td>
</tr>
<tr>
<td>Illness Coherence</td>
<td>14.3 (4.1)</td>
</tr>
<tr>
<td>Personal Control</td>
<td>22.0 (3.6)</td>
</tr>
<tr>
<td>Timeline</td>
<td>18.9 (4.0)</td>
</tr>
<tr>
<td>Treatment Control</td>
<td>18.1 (2.6)</td>
</tr>
<tr>
<td>Treatment Cyclical</td>
<td>13.55 (2.6)</td>
</tr>
<tr>
<td>Mini-IPIP</td>
<td></td>
</tr>
<tr>
<td>Agreeableness</td>
<td>14.4 (3.1)</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>11.9 (2.2)</td>
</tr>
<tr>
<td>Extroversion</td>
<td>10.2 (3.6)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>14.8 (3.0)</td>
</tr>
<tr>
<td>Openness</td>
<td>13.2 (3.3)</td>
</tr>
<tr>
<td>DISH Depression History</td>
<td></td>
</tr>
<tr>
<td>History of depressive episode</td>
<td>91 (81)</td>
</tr>
<tr>
<td><em>(n)</em></td>
<td></td>
</tr>
<tr>
<td>History of treatment of depression</td>
<td>80 (71)</td>
</tr>
<tr>
<td><em>(n)</em></td>
<td></td>
</tr>
<tr>
<td>Number of previous episodes</td>
<td>2.7 (2.7)</td>
</tr>
<tr>
<td>Duration of longest depressive episode (weeks)</td>
<td>81 (123)</td>
</tr>
<tr>
<td><em>(n)</em></td>
<td></td>
</tr>
<tr>
<td>Length of depression history (years)</td>
<td>15.7 (16)</td>
</tr>
</tbody>
</table>

*Note.* BDI-II = Beck Depression Inventory; BAI = Beck Anxiety Inventory; IPQ-R = Illness Perception Questionnaire, Revised; Mini-IPIP = International Personality Item Pool, short form; DISH = Depression Interview and Structured Hamilton.
Table 4
*Predictors of IPQ-R Consequences*

<table>
<thead>
<tr>
<th>Predictor variables</th>
<th>Consequences</th>
<th>( \Delta R^2 )</th>
<th>( \beta )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td></td>
<td>.15***</td>
<td>.38***</td>
</tr>
<tr>
<td>BDI-II</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td>.02</td>
<td>.31**</td>
</tr>
<tr>
<td>BDI-II</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuroticism</td>
<td></td>
<td>.17</td>
<td></td>
</tr>
<tr>
<td>Step 3</td>
<td></td>
<td>.02</td>
<td>.31**</td>
</tr>
<tr>
<td>BDI-II</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuroticism</td>
<td></td>
<td>.16</td>
<td></td>
</tr>
<tr>
<td>Number of previous episodes</td>
<td></td>
<td>-.15</td>
<td></td>
</tr>
<tr>
<td>Model ( R^2 )</td>
<td></td>
<td>.19</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* *p < .05, **p < .01, ***p < .001.*
Table 5
*Predictors of IPQ-R Timeline*

<table>
<thead>
<tr>
<th>Predictor variables</th>
<th>Timeline</th>
<th>$\Delta R^2$</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
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<td>.06*</td>
<td>.24*</td>
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<td>BDI-II</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td>.10**</td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td></td>
<td></td>
<td>.09</td>
</tr>
<tr>
<td>Neuroticism</td>
<td></td>
<td></td>
<td>.35**</td>
</tr>
<tr>
<td>Step 3</td>
<td></td>
<td>.01</td>
<td>.08</td>
</tr>
<tr>
<td>BDI-II</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuroticism</td>
<td></td>
<td></td>
<td>.34**</td>
</tr>
<tr>
<td>Duration of longest episode</td>
<td></td>
<td></td>
<td>.11</td>
</tr>
<tr>
<td>Model $R^2$</td>
<td></td>
<td>.17</td>
<td></td>
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</tbody>
</table>

*Note. *p < .05, **p < .01, ***p < .001.*
Table 6

*Predictors of IPQ-R Personal Control*

<table>
<thead>
<tr>
<th>Predictor variables</th>
<th>Personal Control</th>
<th>$\Delta R^2$</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
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<td>Step 1</td>
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<tr>
<td>Step 2</td>
<td></td>
<td>.033</td>
<td>.02</td>
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<td>BDI-II</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuroticism</td>
<td></td>
<td>-.20</td>
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</tr>
<tr>
<td>Step 3</td>
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<td>.015</td>
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<tr>
<td>BDI-II</td>
<td></td>
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<td>Neuroticism</td>
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<td>-.21</td>
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</tr>
<tr>
<td>Conscientiousness</td>
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<td>.03</td>
<td></td>
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<tr>
<td>Model $R^2$</td>
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*Note.* *p < .05, **p < .01, ***p < .001.*
Table 7

Predictors of IPQ-R Treatment Control

<table>
<thead>
<tr>
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<tr>
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<td>( \Delta R^2 )</td>
</tr>
<tr>
<td>Step 1</td>
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</tr>
<tr>
<td>BDI-II</td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td>.06**</td>
</tr>
<tr>
<td>BDI-II</td>
<td></td>
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<tr>
<td>Neuroticism</td>
<td></td>
</tr>
<tr>
<td>Step 3</td>
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<tr>
<td>BDI-II</td>
<td></td>
</tr>
<tr>
<td>Neuroticism</td>
<td></td>
</tr>
<tr>
<td>Past treatment for depression</td>
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<tr>
<td>Model ( R^2 )</td>
<td>.07</td>
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Note. *p < .05, **p < .01, ***p < .001.
Table 8
*Bivariate Correlations Between IPQ-R Scales*

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<th>4</th>
<th>5</th>
<th>6</th>
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</thead>
<tbody>
<tr>
<td>1. Consequences</td>
<td></td>
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<td>2. Timeline</td>
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<td></td>
<td></td>
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<td>3. Personal Control</td>
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<td>-.41***</td>
<td>.57***</td>
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<td>5. Emotional Representation</td>
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<td>.32**</td>
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<td>.01</td>
<td></td>
<td></td>
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<td>6. Illness Coherence</td>
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<td>.13</td>
<td>-.07</td>
<td>-.14</td>
<td>-.12</td>
<td></td>
</tr>
<tr>
<td>7. Timeline Cyclical</td>
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<td>-.12</td>
<td>-.07</td>
<td>-.08</td>
<td>-.01</td>
<td>-.13</td>
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</tbody>
</table>

*Note. *p < .05, **p < .01, ***p < .001.*
Table 9

*Correlations Between IPQ-R Scales and Mood*

<table>
<thead>
<tr>
<th></th>
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<th>BAI</th>
</tr>
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<tbody>
<tr>
<td>Consequences</td>
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<tr>
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<td>.11</td>
</tr>
<tr>
<td>Personal Control</td>
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<td>-.03</td>
</tr>
<tr>
<td>Treatment Control</td>
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<td>.15</td>
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<tr>
<td>Emotional Representations</td>
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<td>.37***</td>
</tr>
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<td>Illness Coherence</td>
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<td>-.05</td>
</tr>
<tr>
<td>Timeline Cyclical</td>
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<td>.08</td>
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</table>

*Note.* *p* < .05, **p* < .01, ***p* < .001.
Table 10
*Correlations Between IPQ-R Scales and Personality*

<table>
<thead>
<tr>
<th></th>
<th>Mini-IPIP</th>
<th></th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>C</td>
<td>A</td>
<td>E</td>
<td>O</td>
</tr>
<tr>
<td>Consequences</td>
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<td>-.22*</td>
<td>.04</td>
<td>-.04</td>
<td>.04</td>
</tr>
<tr>
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<td>.39***</td>
<td>-.15</td>
<td>-.09</td>
<td>-.25**</td>
<td>-.13</td>
</tr>
<tr>
<td>Personal Control</td>
<td>-.20*</td>
<td>.23*</td>
<td>.26**</td>
<td>.02</td>
<td>.08</td>
</tr>
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<td>.19*</td>
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<td>.02</td>
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<td>-.12</td>
<td>-.13</td>
<td>-.19*</td>
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<td>.03</td>
<td>.12</td>
<td>.15</td>
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<td>Timeline Cyclic</td>
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<td>.05</td>
<td>-.09</td>
<td>.16</td>
<td>-.05</td>
</tr>
</tbody>
</table>

*Note. N = Neuroticism; C = Conscientiousness; A = Agreeableness; E = Extroversion; O = Openness
*p < .05, **p < .01, ***p < .001.*
Table 11  
*Correlations Between IPQ-R Scales and Depression History Variables*  

<table>
<thead>
<tr>
<th></th>
<th>DISH Depression History</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td># of Previous Episodes</td>
<td>Duration of longest episode</td>
<td>Previous treatment of depression</td>
</tr>
<tr>
<td>Consequences</td>
<td>-.15</td>
<td>-.05</td>
<td>.05</td>
</tr>
<tr>
<td>Timeline</td>
<td>-.04</td>
<td>.17</td>
<td>.11</td>
</tr>
<tr>
<td>Personal Control</td>
<td>-.01</td>
<td>-.22*</td>
<td>.04</td>
</tr>
<tr>
<td>Treatment Control</td>
<td>-.11</td>
<td>-.11</td>
<td>-.08</td>
</tr>
<tr>
<td>Emotional Representations</td>
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<td>-.02</td>
<td>-.04</td>
</tr>
<tr>
<td>Illness Coherence</td>
<td>.10</td>
<td>.10</td>
<td>.05</td>
</tr>
<tr>
<td>Timeline Cyclical</td>
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<td>.05</td>
<td>-.05</td>
</tr>
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</table>

*Note. *p* < .05, **p** < .01, ***p** < .001.*
Figure 1. Scatter plot of significant interaction in Hypothesis 2. Duration_Group = Duration of longest depressive episode; Group 1 = short duration (≤ 6 months), group 2 = medium duration (> 6 months but < 2 years), group 3 = long duration (≥ 2 years).