SOCIAL SUPPORT, PERSONAL CONTROL, AND PSYCHOLOGICAL FUNCTIONING AMONG INDIVIDUALS WITH HEART FAILURE

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Heart failure, a serious and prevalent chronic disease, places a large psychosocial burden on patients and their families. The research presented in this dissertation focuses on the complex relationship between *social support* and *personal control* and two key psychological outcomes—*depression* and *anxiety*—in individuals coping with heart failure. Theoretically defensible models are developed, drawing on an integrative stress coping framework, and appropriate inferential statistical procedures are implemented to identify the importance of the proposed relationships in a sample of 242 adult men and women being treated for cardiomyopathy.

Two structural models are evaluated. Model 1 examines the mediating influence of personal control between social support and depression, while Model 2 examines the mediating influence of control between social support and anxiety. Statistically significant estimates indicate that social support plays a key role in reducing psychological distress—depressive symptoms and anxiety. The mechanism whereby social support effectively reduces psychological distress is entirely through patients' perceived control.

An additional objective of the study is exploration of gender differences in the two models. A series of empirical analyses, using path analysis and regression-based mediation models, indicate that among heart failure patients there are indeed statistically significant gender differences in the relationships between social support, control, and psychological functioning. These differences were contingent on the measurement of control included in the model.

Evidence suggests that the role of social support is greater for women than for men in reducing psychological distress, as indicated by either depressive symptoms or anxiety.

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PREFACE

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1. INTRODUCTION

Heart failure (HF) is a complex chronic progressive disease that results from any functional or structural disorder of the heart that weakens its pump performance (Cowie & Zaphiriou, 2002; Grady et al., 2000). Close to 5 million Americans suffer from HF, with a startling estimated 550,000 new cases diagnosed each year (Artinian, 2003). Prevalence of HF increases with age, with approximately 5% of people age 60-69 and roughly 10% of people aged 70 or older being said to have the condition (Givertz, Colluci, & Braumwald, 2001). The incidence and prevalence of HF is expected to increase over the coming years, (Artinian, 2003) which will no doubt have an impact on the current 5% rate of HF-related hospitalizations (MacMahon & Lip, 2002). In part, this increase is attributed to an aging population and greater survival rates of individuals with heart disease (Artinian, 2003). Therapeutic advances in the treatment of acute cardiac events and heart disease have resulted in an increase number of individuals surviving heart attacks, which ultimately impacts the incidence of HF (Artinian, 2003; MacMahon & Lip, 2002).

Despite recent advances in the pharmacological management of HF, this disease is characterized by frequent hospitalizations and high mortality rates (Grady et al., 2000). The 1-year mortality rate associated with HF still remains close to 40% (MacMahon & Lip, 2002). Based on the 44-year follow-up of the Framingham Heart Study, approximately 80% of men and 70% of women diagnosed with congestive heart failure who are under the age of 65 die within 8 years (American Heart Association, 2004). HF is the most costly cardiovascular disease in the US (Grady et al., 2000) with indirect and direct costs estimated at $25.8 billion for 2004 (American Heart Association, 2004). While the financial costs of HF are significant, the

associated burden and psychosocial impact on patients and their families are considerable as well.

Results from qualitative studies provide health care professionals with insight into the psychosocial burdens encountered by individuals with heart failure. The results of these studies suggest that individuals with HF often experience impairment in psychological functioning such as a disturbance in mood, anxiety, insecurity, powerlessness, worthlessness, a sense of disruption and incoherence, feelings of being a burden to others, and feeling imprisoned by the illness (Hawthorne & Hixan, 1994; Mahoney 2001; Martensson, Karlsson, & Fridlund, 1997; Martensson, Karlsson, & Fridlund, 1998). The focus of this study is specifically on depression and anxiety as these impairments in psychological functioning have been shown to have serious consequences for patients' future health outcomes. For example, depression is associated with increased morbidity associated with coronary disease (Jiang, Krishnan, & O'Conner, 2002) and mortality post myocardial infarction (Bush et al., 2001; Frasure-Smith, Lesperance, & Talajic, 1993; Writing Committee for the ENRICHD Investigators, 2003).

1.1. BACKGROUND

Across studies the rate of major depression among individuals with heart disease is approximately 20 percent, with rates of minor depression often higher than 25 percent, and depressive symptoms reported in up to 65 percent of patients (Frasure-Smith et al., 1993; Ladwig et al., 1992; Carney, Freedland, Sheline, & Weiss, 1997). The reported rates of depression vary considerably among individuals diagnosed with heart failure, based on the characteristics of the sample studied and the method of measuring and defining depression, with incidence ranging from 11% to 85% (Freedland et al., 2003; Thomas, Friedmann, Khatta, Cook, & Lann, 2003;

Turvey, Schultz, Arndt, Wallace, & Herzog, 2002; Zuccalà, Cocchi, & Carbonin, 1995). A more thorough examination of the high variability in reported rates of depression among individuals with HF and the difficulties associated with recognizing and differentiating depression among individuals with medical illness will be presented in Chapter Two.

There is a lack of studies examining the prevalence of anxiety and its related symptoms among individuals with HF, which is surprising given the probability that anxiety related to the physical symptoms (specifically dyspnea) and poor prognosis of HF may be a significant problem for patients (Artinian, 2003; MacMahon & Lip, 2002). One study that examined quality of life among individuals with advanced HF found anxiety levels as measured by the Multiple Affective Adjective Checklist to be consistent with mild anxiety (Walden et al., 1994). A second study that examined the psychological profile of individuals with HF found that anxiety levels were higher among individuals with New York Heart Association (NYHA) functional class III HF than individuals with class I or II HF (Majani et al., 1999). It is important to note that individuals with class IV HF were excluded from participating in this study.

Although psychological functioning generally improves for most cardiac patients, impairment persists for a significant minority, and for these individuals it can be a serious impediment to present and future well-being. Evidence indicates that psychological impairment is related to higher mortality post-MI, increased morbidity, a reduction in life quality, and increased impairment in physical functioning (Denollet, Sys, & Brutsaert, 1995; Frasure-Smith, Lespérance, & Talajic, 1995; Ladwig, Kieser, Konig, Breithardt, & Borggrefe, 1991). Even more troubling is the evidence that consistently demonstrates the presence of gender differences in psychological functioning, with women reporting higher levels of depression and anxiety (Holahan, Moos, Holahan, & Brennan, 1995; Maeland & Havik, 1987; Schleifer et al., 1989).

It has been proposed that personal resources, such as social support and coping style, facilitate adaptation to threatening events including chronic illness and thus impact psychological outcomes (Holahan & Moos, 1990). Further, evidence suggests that there may be gender differences and variations in the effect of personal resources and coping on psychological outcomes among the medically ill (Holahan et al., 1995). To date, our knowledge of the predisposing factors in the development of depression and anxiety among the medically ill is incomplete and based on multifaceted complex theoretical models. A clear need exists for further empirical work based on a parsimonious model that can explain the variation in vulnerability to psychological impairment among the medically ill.

1.2. PURPOSE

The overall purpose of the proposed study is to develop and test two structural models derived from Holahon, Moos, and Bonin's (1999) integrative stress and coping theoretical framework in a sample of individuals coping with a chronic illness. The primary objective is to examine the mediating influence of personal control between social support and depression (Model 1) and anxiety (Model 2) in a sample of individuals diagnosed with congestive heart failure. A secondary objective is to explore gender differences in the two proposed models.

1.3. SPECIFIC AIMS AND RESEARCH HYPOTHESES

Specific Aim #1: To propose and test a theoretical mediating model of the relationship between social support, personal control, and depression among individuals with congestive heart failure.

The explicit hypotheses for Model 1 to be examined in this empirical work are as follows:

H1.1 Social support has an inverse direct effect on depressive symptomatology in individuals diagnosed with congestive heart failure.

H1.2 Social support has a positive effect on personal control through a direct path.

H1.3 Personal control has an inverse direct effect on depressive symptomatology in individuals diagnosed with congestive heart failure.

H1.4 Social support has an inverse effect on depression through an indirect path - specifically, social support has a positive effect on personal control, which in turn reduces depressive symptoms in individuals diagnosed with congestive heart failure.

Specific Aim #2: To propose and test a theoretical mediating model of the relationship between social support, personal control, and anxiety among individuals with congestive heart failure. Similarly, the hypotheses for the second model are outlined below. Again, these four hypotheses will be tested against the null hypothesis of no effect.

H2.1 Social support has an inverse direct effect on anxiety in individuals diagnosed with congestive heart failure.

H2.2 Social support has a positive effect on personal control through a direct path.

H2.3 Personal control has an inverse direct effect on anxiety in individuals diagnosed with congestive heart failure.

H2.4 Social support has an indirect inverse effect on depression through personal control, which directly reduces symptoms of anxiety in individuals diagnosed with congestive heart failure.

Specific Aim #3: To explore gender differences in a theoretical mediating model of the relationship between social support, personal control, and depression among individuals with congestive heart failure.

H3.0 The relationships will be different for males and females.

Specific Aim #4: To explore gender differences in a theoretical mediating model of the relationship between social support, personal control, and anxiety among individuals with congestive heart failure.

H4.0 The relationships will be different for males and females.

The ultimate goals of this study are twofold. The first goal is to assist clinicians in understanding the relationships between personal and social resources that are central to psychological functioning among individuals with cardiac disease. Second, the results of this study will serve to inform theoretical work designed to understand gender differences in psychological functioning among the medically ill.

1.4. DEFINITION OF TERMS

1.4.1. Social Support

Theoretical Definition: In principle, it would seem that the concept social support would be easy to define. Thoits (1995), for example, provides one reasonable definition, suggesting that social support can be viewed as a source of coping assistance – a social fund so to speak from which one can draw upon when faced with stressors. However, social support is a complex and multidimensional phenomenon, with a wide variety of theoretical definitions involving both qualitative and quantitative conceptualizations (Dracup, 1994; Sarason & Sarason, 1994). Indeed, from the perspective of researchers, the concept of social support suffers from an obvious lack of specificity (Antonucci & Johnson, 1994).

For this study, the theoretical definition of social support will be limited to perceived levels of functional social support, defined as one's subjective appraisal of availability and

adequacy (Sarason & Sarason, 1994) to the specific function that one's social ties provide, and includes such elements as informational support, social companionship, self-esteem, and tangible support (King, 1997).

Operational Definition: Social support will be measured using the Interpersonal Support Evaluation List (ISEL) developed by Cohen, Mermelstein, Kamarck, and Hoberman (1985). This scale consists of 40 items concerning the perceived availability of potential functional sources of social support. The scale is constructed in such a way that items are counterbalanced for desirability, meaning that half of the items are positive statements about social support and half are negative in nature. The theoretical underpinnings for the scale development closely matches the theoretical framework for this study, in that social support was conceptualized to potentially facilitate one's coping with stressful life events and circumstances. The ISEL has four subscales that are designed to measure distinct functions of social support: tangible support, appraisal support, self-esteem support, and belonging support. Tangible support refers to perceived availability of instrumental aid, such as being able to find a ride if needed. The appraisal subscale measures one's perceived availability of a confidant to talk to about personal problems. Self-esteem support is the perceived availability of a positive comparison of one's self in relation to others. Finally, belonging is the perceived availability of friends or family members with whom one can do things.

1.4.2. Control

Theoretical Definition: Folkman (1984) defines control as "a generalized belief of an individual concerning the extent to which he or she can control outcomes of importance and as a situational appraisal of the possibilities for control in a specific stressful encounter" (p. 839). Inherent in this definition is the notion that control is experienced at a personal and perceived level in that an

individual believes that things are under one's own control whether or not they actually are (Walker, 2001). Control can therefore be defined as the belief or perception that a person has about one's actual or potential capacity and power to determine or influence events or circumstances in one's life (Walker, 2001).

Operational Definition: Three separate scales will be used to operationalize the concept of control. The first measurement will be a seven-item Mastery scale used to measure general feelings of personal control (Pearlin & Schooler, 1978). The purpose of the Mastery scale is to measure a person's perceived control over events in his/her life. The second scale is the Coping with Serious Illness Battery (CSIB) Sense of Control subscale that measures an individual's perceived ability to solve problems and influence others (Stewart, 1983). The third measurement is a four-item scale intended to measure the extent to which an individual believes that he/she has the ability to control the day-to-day symptoms associated with one's illness (P. Bohachick, personal communication, August 3, 2004).

1.4.3. Depressive Symptoms

Theoretical Definition: The concept of depression brings to mind a wide range of definitions ranging from normal temporary mood states associated with feeling sad or down to a serious and debilitating illness called Major Depressive Disorder (MDD). A diagnosis of MDD requires the presentation of specific essential features meeting duration and functional impairment criteria established by the American Psychiatric Association (Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition, 1994). For this study depressive symptoms is defined in terms of the cognitive, behavioral, and affective symptomatology commonly associated with MDD, but not to the extent that the symptoms meet established criteria for MDD (Kaelber, Moul, & Farmer, 1995). Attributes of depressive symptomatology include feeling down, sad or

blue, loss of pleasure, feeling worthless, guilty, and hopeless, decreased energy, loss of sexual interest or pleasure, feeling as if everything is an effort, episodes of crying, and thoughts of suicide.

Operational Definition: To operationalize this construct two measurements will be used – the Symptom Checklist-90-R (SCL-90-R) depression subscale (Derogatis, Rickels, & Rock, 1976) and the Profile of Moods States (POMS) depression subscale (McNair, Lorr, & Droppleman, 1981). The SCL-90-R is designed to assess for the presence of concomitant clinical depressive symptoms. The POMS depression subscale measures the presence of specific affect adjectives such as feelings of sadness and unworthiness.

1.4.4. Anxiety

Theoretical Definition: Anxiety is theoretically defined as an internal and personal state (Breznitz & Goldberger, 1993) in which an individual feels uneasy and/or apprehensive in the face of a perceived or actual threat to one's well-being (Lazarus, 1993). It is important to point out that for the purpose of this study anxiety is conceptualized as a personal perception of experiencing symptoms of anxiety but not to the level indicating an anxiety disorder. In order to avoid confusion between symptoms and disorder, there is reason for labeling this construct "anxiety symptoms". However, in keeping consistent with the current terminology used in the literature, "anxiety" is identified as the construct under study. The symptomatic manifestation of anxiety includes, but is not limited to, feeling nervous, an internal sensation of shakiness and/or trembling, feeling scared for no reason, feeling fearful, experiencing episodes of terror or panic, and experiencing a sense of impending doom.

Operational Definition: To operationalize anxiety information from two measurements will be used – the SCL-90-R anxiety subscale and the POMS anxiety subscale. The SCL-90-R

anxiety subscale measures the presence of common affective (feeling that something bad is going to happen) and somatic (heart pounding) symptoms associated with anxiety (Derogatis et al., 1976). The POMS anxiety subscale measures the extent to which an individual experiences common symptoms related to anxiety such as feeling tense or uneasy (McNair, Lorr, & Droppleman, 1981).

1.5. CONCLUSION

Health care professionals have long understood that heart failure—a serious and prevalent chronic disease—places a large psychosocial burden on patients and their families. The research project proposed here focuses on the structure of this problem, seeking to understand the complex interaction of several key factors: depression, anxiety, the patient's sense of personal control, and social support. The immediate goal of the research is to construct a theoretically defensible model that relates these constructs, and to use modern tools of inferential statistics to identify the importance of the proposed relationships. The ultimate aim is to contribute to a body of work that will lead to appropriate evidence-based practice in the treatment of heart failure patients.

2. LITERATURE REVIEW

The research proposed for this dissertation draws from a wide and disparate literature that spans a number of relevant concepts. The goal of the literature review presented in this chapter is to provide a reasonably concise guide to previous research that most closely relates to the proposed research. The chapter begins with a discussion of the literature that informs the general theoretical framework. After a brief overview of the model used in the dissertation, the chapter provides a discussion of the following key areas: depression among the medically ill; social support, especially focusing on the role of social support among those with heart disease and its relationships with depression and anxiety; personal control; and, finally, the role of gender as it relates to the concerns of the proposed research.

2.1. THEORETICAL FRAMEWORK

The conceptual framework guiding the proposed investigation is an *integrative stress and coping model* that focuses on the role of personal and social resources in dealing with acute and chronic life crises (Holahan & Moos, 1999; Holahan, Moos, & Bonin, 1999). The integrative stress and coping model was developed primarily to facilitate the integration and understanding of research relevant to predicting adjustment and coping responses under conditions of stress (Holahan et al., 1999) and draws heavily on the work of Lazaras and Folkman (1984). A social-ecological perspective is central to the development of this framework in that three social-ecological principles generally guide the work: positive adaptation, the influence of social context in

shaping individual personality and behavior, and interdependence and dynamic interrelationships among variables (Holahan et al., 1999).

The basic framework, depicted in Figure 1, focuses on five domains relevant to the stress and coping process: environmental system, personal system, life crises/transitions, cognitive appraisal/coping responses, and health/well-being. The environmental system comprises ongoing stressors (such as chronic illness) as well as social coping resources. Social coping resources include, but are not limited to, one's unique interpersonal networks, including the social support one receives directly from family and others (Holahan et al., 1999). Although Holahan and colleagues (1999) do not specifically provide theoretical definitions to the concepts in their model, they do draw heavily on the work of Lazarus and Folkman. As such, resources are broadly conceptualized as what a person draws on in order to facilitate coping (Lazarus & Folkman, 1984) and include both personal (personality strengths) and social (social environment, social support) resources (Holahan et al., 1999). For the present study, the focus is restricted to *social support* as a primary indicator of one's social coping resources.

The second domain is the personal system, which primarily comprises an individual's sociodemographic and personal coping resources. Coping resources include such factors as self-esteem and self-confidence. The environment and personal systems are believed to be relatively stable and have a direct influence on individual life crises and transitions, which comprises the third domain. The life crises/transition domain comprises event-related factors and changes in one's life circumstances that are potentially viewed as stressful.

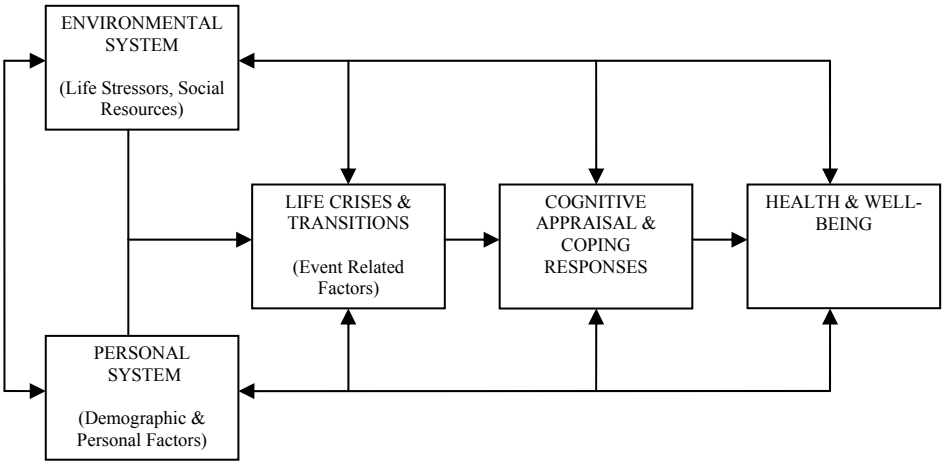

Figure 1 Integrative Stress Coping Conceptual Framework (Holohan, Moos, & Bonin, 1999, p. 41)

The fourth domain, cognitive appraisal and coping responses, comprise the coping strategies that an individual utilizes. Holahan and colleagues (1999) do not provide specific definitions for the components associated with the cognitive appraisal and coping responses domain, but again rely on Lazarus and Folkman (1984) to provide an understanding of the concepts in this domain. Coping, thus, is conceptualized as a stabilizing factor that promotes adaptation or protection during stressful times, and consists of cognitive and behavioral endeavors.

Inherent to an understanding of the protective nature of coping is a consideration of the strategies that one engages during times of stress. According to Holahan, Moos, and Schaefer (1996), coping responses include both approach coping strategies and avoidance coping strategies. Avoidance coping strategies, including denial and withdrawal, are by and large associated with maladaptive behavioral outcomes and psychological distress (Holahan et al., 1999; Rohde, Lewinsohn, Tilson, & Seeley, 1990). Approach coping strategies involve strategies to confront the stressor, such as problem solving and seeking information, and are generally viewed as adaptive in nature (Holahan et al., 1999; Sherbourne, Hays, & Wells, 1995).

The final domain encompasses health and well-being. The direct and indirect influences of each domain on the others are captured by the paths depicted in the model. In addition, the bi-directional arrows in the model point to the interdependence among domains and specify the dynamic reciprocal feedback that can occur at any point in the stress/coping process (Holahan et al., 1999).

2.2. OVERVIEW OF MODEL

The model for the proposed research is presented in some detail in Chapter 3. It is nonetheless helpful to provide a preliminary overview of the concepts as a means of informing the literature review that follows.

In rough terms one can think of the proposed research as providing careful empirical evaluation of a subset of the relationships depicted in the broader theory of Holohan and colleagues (1999), as outlined in Figure 1. In particular, the proposed research (a) focuses on social support as a key element of the Environmental System, (b) studies personal control as playing a key role in Cognitive Appraisal and Coping Responses, and (c) evaluates depression and anxiety as key outcomes for Health and Well-Being. It is worth noting that while the theoretical framework specifies the relationship between the domains as being reciprocal, the present study focuses on the relationships between the variables in a non-reciprocal manner. The rationale motivating this approach is the cross-sectional nature of the data. Figure 2 illustrates these relationships.

Because the proposed research focuses on a medically ill population, it is important that the literature reviewed include studies that deal with the theoretical and practical difficulties of studying this population. Also, the proposed investigation focuses on the role of gender (see

Figure 3), so the literature review also touches on the broad theoretical and empirical literature relevant to this issue.

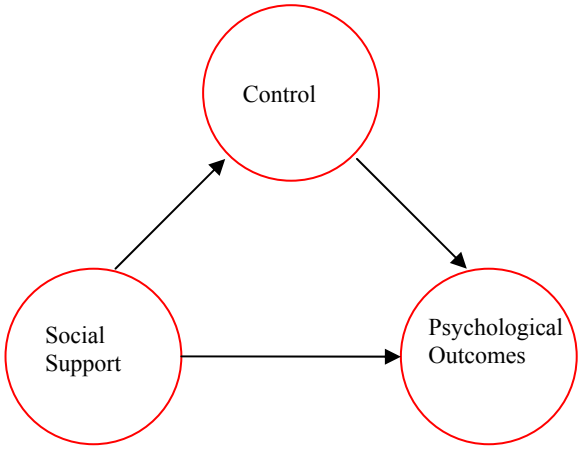

Figure 2 Proposed Conceptual Model

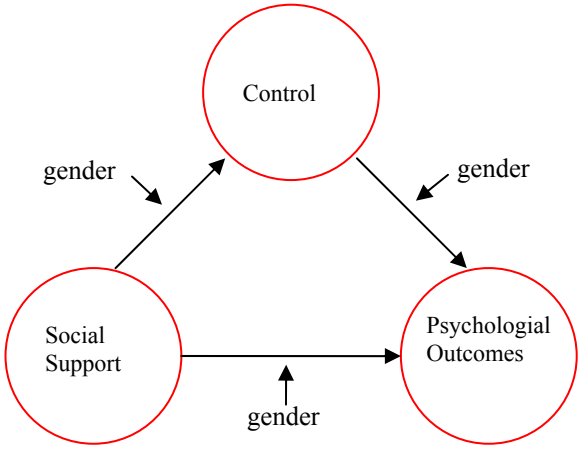

Figure 3 Proposed Conceptual Model with Gender Influences

2.3. DEPRESSION AMONG THE MEDICALLY ILL

Depression is an under-recognized medical condition that is often experienced as a comorbid problem for people who are diagnosed with chronic medical illnesses. Current lifetime rates of

depression among the general population ranges from 6% to 25% (Kessler, 2004), while higher rates of depression are often found in individuals with chronic medical conditions. The estimated rates for depression vary between the different types of medical illnesses, with reported rates ranging between 5 and over 50% (Sutor et al., 1998). A potential explanation for the higher rates of depression among patients with medical conditions is that the very presence of the illness serves to act as stress-related catalyst for depression (Slimmer, Lyness & Caine, 2001).

Further complicating the problem of under-recognition is evidence demonstrating that the presence of depression among the medically ill negatively impacts health related outcomes, including increased medical utilization and costs, increased levels of symptom burden, higher functional impairment, increased rates of mortality and morbidity, decreased quality of life, and diminished patient compliance with treatment regimens (Carney, Freedland, Eisen, Rich & Jaffe, 1995; Ciechanowski, Katon, & Russo, 2000; Denollet et al., 1995; Forrester, Lipsey, Teitelbaum, DePaulo, & Andrezejewski, 1992; Frasure-Smith et al., 1993; Greigo, 1993; Ladwig, Kieser, Konig, Breithardt, & Borggrefe, 1991; Ladwig, Roll, Breithardt, Budde, & Borggrefe, 1994; Shively, Fox, Brass-Mynderse, 1996). Despite this troubling evidence, depression remains poorly understood and often under-treated among the medically ill, which is especially disturbing given that depression is highly treatable among individuals with medical conditions (Hirshfeld, 1998; Sheikh et al., 2004)

Central problems in the understanding of depression among the medically ill are the lack of precise definitions and the difficulties in accurately diagnosing depression in the research setting. The problems with diagnosing depression in combination with a medical disorder are clear. Of primary concern are the confounding or overlapping symptoms of depression and the medical disorder of interest. For example, somatic symptoms, such as fatigue, sleep disturbances,

and appetite changes, are commonly associated with various medical disorders and as such may not necessarily be a sign of depression, thus making it difficult to tease out the relative importance of somatic symptoms among the medically ill to symptom criteria for depression (Creed, 1997).

Freedland, Lustman, Carney, and Hong (1992) became interested in this very phenomenon and designed a study to specifically examine the role of nonspecific symptoms that potentially contribute to the underdiagnosis of depression among individuals with coronary artery disease (CAD). Participants completed the Beck Depression Inventory (BDI) to assess for the presence of depressive symptomatology. Major depression was differentially diagnosed using a modified version of the National Institute of Mental Health Diagnostic Interview Schedule (DIS). Among the 114 participants, 31 individuals met criteria for current major depression (MD group), while 83 individuals did not meet criteria (non-MD group). The mean total score on the BDI for the non-MD group fell within the "nondepressed" range ($\bar{x}$ =6.2 ± 4.0) and the "mildly depressed" range ($\bar{x}$ =13.6 ± 5.4) for the MD group (t=6.91, p=.0001). The interesting result is that nine of the 21 BDI symptoms were experienced by at least 20% of participants in both groups and thus were identified as nonspecific symptoms. These "nonspecific" symptoms included inertia, insomnia, fatigue, weight loss, health worries, disinterest in sex, dissatisfaction, self-blame, and irritability. The authors rank ordered all 21 symptoms within groups and found that the most frequently reported symptoms fell within the somatic domain for both groups and were highly correlated. Not surprisingly, fatigue was the most commonly reported symptom for both groups, which ranked first among 100% of the MD group and almost 80% of the non-MD group. Although the authors point out that these results may in fact be a function of the validity of the DIS among patients with CAD, the study also raises questions concerning the validity of

diagnosing depression in this group given that a number of the depression symptoms were confounded with classic symptoms of CAD.

To address the symptomatology confounding issue, Freedland and colleagues (1991) conducted an analysis in a group of patients hospitalized with congestive heart failure (CHF). Among the 60 patients who participated, 10 (17%) met DSM-III-R criteria for major depression using the DIS. In order to better understand the role of confounding symptoms of depression and CHF in making a differential diagnosis of major depression, the authors removed reports of fatigue and insomnia from the symptom profile for each of these ten subjects. Interestingly, even with the application of this more stringent diagnosing strategy, all ten subjects continued to meet DSM-III-R criteria for major depression.

In short, while evidence is quite clear that depression is especially common among individuals with chronic medical conditions, documenting the extent of the depression is clearly complicated by confounding symptomatology. Researchers have made only limited progress in dealing with this issue.

A second, related issue that further clouds the understanding of depression among the medically ill is a lack of consistency in defining depression across research studies, making it difficult to interpret research results concerning the prevalence and consequences of depression among the medically ill. As pointed out in Chapter 1, the reported rates of depression among individuals with HF vary significantly with rates ranging from 11%-85%. This considerable inconsistency can be attributed in part to the sample characteristics (e.g. age, illness severity, inpatient versus outpatient), but also to whether or not depression was defined in terms of symptoms or syndrome, and the method (e.g., self-report versus clinical interview) and measurement utilized to establish the presence of depression.

By far the largest variability in reported rates of depression was among studies that defined depression through the use of self-report scales of depressive symptomatology. Such studies report rates ranging from 24%-85%. The lowest rate of depression via self-report was reported by Havranek, Ware, and Lowes (1999), who found that 24.4% of their sample scored 16 or higher on the Center for Epidemiologic Studies Depression Scale (CES-D). The CES-D is a 20-item self-report scale that reportedly is not influenced by concomitant medical illness. The sample in this study consisted of 45 ambulatory, clinically stable individuals ($\bar{x}$ age = 54) diagnosed with congestive heart failure. These findings are strikingly different from the results of Zucalà and colleagues (1995), who also used the CES-D to assess depressive symptoms, and found that 85% of their sample met criteria for "severe" depressive symptomatology. It is interesting to note that the authors do not specifically discuss what standard they used to define symptoms as "severe." The sample in this study was somewhat older ($\bar{x}$ age = 72), which might account for the vast difference in rates of depressive symptoms between these two studies. It is difficult to draw further conclusions regarding the two samples as Zucalà and colleagues (1995) do not provide detailed information concerning the level of physical impairment and disease severity associated with the participants' HF. It is possible that the study by Zucalà and colleagues involved much more physically ill subjects than those in the Havranek, Ware, and Lowes study.

Vaccarino, Kasl, Abramson, and Krumholz (2001) found that 77.5% of individuals with heart failure reported the presence of clinically relevant depressive symptoms on the Geriatric Depression Scale – Short-Form. Of these individuals, 35% scored in the mild range for depressive symptoms, 33.5% scored in the moderate range, and 9% scored in the severe range. The sample consisted of 391 patients aged 50 or older who met criteria for heart failure

(diagnosis or radiologic signs of HF) at the time of their hospital admission. The high rate of depressive symptomatology in this group is attributed to the fact that the sample was considered to be severally ill.

Two studies that used the Beck Depression Inventory (BDI) reported rates of depression in patients with HF as 35.3% (Jiang, Alexander, Christopher, Kuchibhatla, Gaulden, Cuffe, Blazing, Davenport, Califf, Krishnan, & O'Conner, 2001) and 51% (Freedland et al., 2003) as defined by a score of ten or higher for both studies. The BDI is a well known instrument to assess the severity of self-reported symptoms of depressive symptoms. These two studies also used the National Institute of Mental Health Diagnostic Interview Schedule (DIS) to confirm the presence of major depression. In both studies, a significant proportion of the individuals experienced syndromal depression, with 14% (Jiang et al., 2001) and 20% (Freedland et al., 2003) meeting full criteria for major depression. Likewise, a significant proportion (16% for both studies) met criteria for minor depression. Although both these studies involved hospitalized patients with similar age, ejection fraction, and NYHA class distribution, there were differences in the gender makeup of the sample. In the Freedland study, women comprised 52% of the sample while Jiang's study included 38% women. It is possible that the higher proportion of women in the Freedland study accounted in part for the higher rate of depression in this sample.

Similarly high levels of depression were found by Koenig (1998). In this study of 107 hospitalized patients aged 60 or older with HF, 36.5% and 21.5% met criteria for major and minor depression, respectively. It is interesting to note that Koenig applied a more inclusive set of standards to classify depression than previously discussed studies. Major depression was defined as a score of 16 or higher on the DIS, having at least 3 of the 13 DIS expanded criterion

symptoms for major depression for 2 weeks or longer in the past month, and scoring 11 or higher on the Hamilton Depression Rating Scale.

Among the studies reviewed, Turvey and colleagues (2002) reported the lowest level of depression. They used the Composite International Diagnostic Interview to assess depression in a sample of community living older people with self-reported HF. They found that 11% of the sample met criteria for syndromal depression, but noted that DSM criteria were not used to establish depression.

While the studies cited in the preceding paragraphs are hardly a comprehensive listing of investigations relating depression and medical illness, they serve to illustrate the considerable variability in the estimated prevalence of depression among the medically ill. They also suggest that much of this variability is due to the lack of consistency among researchers in measuring and defining depression among the medically ill, and underscore the importance of using more precise measures of depression or depressive symptomatology in on-going research.

2.4. ANXIETY AMONG THE MEDICALLY ILL

Individuals coping with a chronic illness often experience anxiety, and heightened levels of anxiety have been related to poorer physical and psychological functioning (Januzzi, Stern, Paternak & DeSanctis, 2000; Taylor & Aspinwall, 1993). Descriptive research studies indicate that a significant fraction of patients post-MI have levels of anxiety that are as high as, or even higher than, psychiatric populations. For example, Havik and Maeland (1990) found high levels of anxiety (as defined as 14 or higher on the SED-questionnaire of state-dependent feelings of anxiety) among 20% of the individuals hospitalized for a MI. The level of anxiety increased drastically over the first 1-2 weeks post discharge and then stabilized at six months to the 3-5

year follow-up period. Similarly, Crowe, Runions, Ebbesen, Oldridge, and Streiner (1996) found that among hospitalized post-MI patients 10% had state anxiety levels and 14% had trait anxiety levels that were higher than levels reported for psychiatric patients. Similar trajectories of anxiety level over the first year post-MI were found in the two studies.

Studies have also documented a relationship between anxiety and subsequent morbidity and mortality among individuals with heart disease. Moser and Dracup (1996) found that patients who reported higher levels of anxiety were 4.9 times more likely to experience in-hospital post-MI complications than those who reported lower levels of anxiety. Complications included any of the following: acute ischemia, reinfarction, sustained ventricular tachycardia, or in-hospital death. Anxiety was related to subsequent complications even when controlling for more traditional risk factors.

Sullivan, LaCroix, Baum, Grothaus, and Katon (1997) found that anxiety assessed at the time of cardiac catheterization predicted impairment in role and physical functioning at one year in a sample of patients diagnosed with CAD, even after controlling for disease severity. These findings were confirmed in a follow-up study of patients with CAD (Sullivan, LaCroix, Spertus, & Hecht, 2000). Specifically, anxiety was significantly related to overall functional status at the 6-year follow-up, even when adjusted for age, gender, education, and disease severity. The relationship between anxiety and functional status was strongest in the domains of physical role, pain, social function, mental health, vitality, and general health. This is the first study to document the role of anxiety in patients with heart disease over such an extended period.

A number of studies have also documented a relationship between anxiety and mortality among individuals with heart disease (Carney, Freedland, Sheline, & Weiss, 1997; Denollet & Brutsaert, 1998; Frasure-Smith et al., 1995; Moser & Dracup, 1996; Thomas, Friedmann,

Wimbush, & Schron, 1997). However, Lane, Ring, Beevers, and Lip (2000) found that baseline anxiety (as measured in-hospital 2-15 days post-MI) did not predict fatal and non-fatal coronary events over the course of a year in a sample of 288 patients. The incongruous results of this study were not attributed to level of anxiety (as levels were similar to that reported in previous studies) or power issues related to sample size. The authors speculate that their findings may disagree with previous results due to differences across studies in controlling for potential confounding factors between anxiety and disease severity.

2.5. SOCIAL SUPPORT

2.5.1. Definitional and Theoretical Considerations

There is growing evidence that social support is a significant determinant of individual differences in reactions to stress and recovery from illness (Saraon & Sarason, 1994; Schaefer, Coyne, & Lazarus, 1981; Yates, Skaggs, & Parker, 1994). In principle, it would seem that the concept *social support* would be easy to define, but in practice social support has been defined from a number of theoretical and operational perspectives (Antonucci & Johnson, 1994). For example, Monroe, Imhoff, Wise, and Harris (1983) view social support as an objective number of resources available to a person. In contrast, Leavy (1983) believes that social support is the process by which a person develops, draws on, and preserves their available resources. Kessler, Price, and Wortman (1985) suggest that social support involves "mechanisms by which interpersonal relationships presumably protect people from the deleterious effects of stress" (p. 541). Thoits (1995) defines social support in terms of a social fund, or source of coping assistance from which an individual can draw upon when faced with stressors. Social support can

also be defined from a view that gives emphasis to "feeling loved and cared about by others as the central element in the protective effect of social support" (Sarason & Sarason, 1994, p. 47).

Broadly speaking, one can think of two distinct types of social support: structural support and functional support (Kessler et a., 1985; Sarason & Sarason, 1994). The structural aspects of social support are delineated by such concrete features as one's living arrangements and the number and frequency of one's social interactions. The functional components are a bit more intangible and are related to the specific function that one's social ties provide. Functional components of social support include instrumental aid (such as physical assistance), informational guidance, social interaction, belonging, self-esteem, tangible, emotional, affectionate, and appraisal support (Cohen et al., 1985; King, 1997; Langford, Bowsher, Maloney, & Lillis, 1997; Sherbourne & Stewart, 1991).

Yet another distinction in the characterization of social support is "perceived versus received" (Helgeson, 1993). Perceived support relies on the expressed views of an individual about the availability of support, while received support makes reference to actual quantitative measures of specific types of support. Sarason and Sarason (1994) argue that a primary confusion regarding social support can be attributed to the linkage of support to both objective networks and events as well as one's subjective estimates of availability and adequacy. The relative importance of perceived versus received support to health outcomes has become a key topic of interest in health outcomes research.

From the literature it appears that perceived support is a better predictor of health outcomes than the actual support received (Cohen & Hoberman, 1983; Cohen & Wills, 1985; Wethington & Kessler, 1986). Helgeson (1993) specifically addressed this premise in a study of 64 patients hospitalized for a first coronary event and their spouses. Using the UCLA-Social

Support Inventory, received support was assessed for three support sources (spouse, close family member, and personal physician). Questions paralleling those for received support were developed to measure perceived levels of support. In general, perceived social support was found to have a greater influence on the level of psychological distress, life satisfaction, and overall adjustment to the illness than received support. More specifically, Helgeson found that perceived availability of informational support (during the past three months) predicted reduced psychological distress. Perceived availability of emotional support and spouse disclosure (during the past three months) predicted an increase in life satisfaction, whereas received emotional support predicted decreased life satisfaction.

Complicating matters further is that interpersonal interactions generally thought of as "social support" can be viewed as either positive or negative in nature. As Rook (1984; 1992) notes, there can be problematic facets to social interactions; social ties and relationships can, by acts of omission or commission, cause psychological distress. Rook (1990) denotes social bonds and interactions that are negative in nature as social strain. For some interactions, it might be reasonable to think of positive and negative social interaction as polar extremes on the same continuum, but more generally, as Coyne and Downey (1991) argue, "social support may not be a fundamentally unipolar construct…while the reports of *low support* may sometimes reflect the absence of a supportive relationship, they may more often signify the presence of a negative, conflictive relationship" (p. 412). Coyne and Downey (1991) stress that self-report scales anchored with high and low indicators of perceived support will fail to capture the subtleties of this alternative view of *low* support. Flor, Kerns, and Turk (1987) found that among individuals coping with chronic pain, overly solicitous family members was associated with increased levels

of impairment. In sum, social networks can simultaneously provide support and invoke stress (Fiore, Becker, & Coppel, 1983).

From these various definitions it is apparent that social support is a complex and multidimensional construct, with a wide variety of theoretical underpinnings involving both qualitative and quantitative conceptualizations (Dracup, 1994; Sarason & Sarason, 1994). Even though there are fundamental complexities associated with fully understanding the concept, social support has been shown to be an important coping resource and has a significant influential role on outcomes in psychological functioning and adjustment among individuals with chronic illness (Elizer & Hirsch, 1999; Taylor & Aspinwall, 1993).

The mechanisms by which social support purportedly influence health related outcomes are grounded in two theoretical viewpoints. The first is a direct effects framework in which social support has a direct positive influence on psychological functioning and well-being regardless of the degree of stressors that an individual is experiencing (Aneshensel & Frerichs, 1982; Thoits, 1983; Williams, Ware, & Donald, 1981). The second framework is the stress buffering hypothesis, which argues that social support has a protective influence on an individual's well-being by helping to reduce the negative impact of life stressors (Cohen & Wills, 1985).

2.5.2. Relationship between Social Support and Heart Disease

An important focal point of social support research over the past decade has encompassed the area of recovery and survival from physical illness. Although mortality and morbidity are not a focus of the research presented in this dissertation, it is useful to briefly review this literature as a means of understanding the broader context of the influence of social support on health related

outcomes. In particular, as discussed below, social support and depression are closely linked, as are depression, morbidity, and mortality among individuals with medical illness.

Although few studies have addressed the role of social support on morbidity and mortality in individuals with heart failure, prospective studies have established an association between low levels of social support and risk of morbidity and mortality associated with cardiovascular disease more generally (for reviews see Krantz & McCeney, 2002; Moser, 1994; Reifman, 1995). The majority of the studies have found a significant relationship, with individuals reporting lower levels of social support being at higher risk for morbidity and mortality associated with heart disease. For example, Williams and colleagues (1992) found a significant relationship between marital status and 5-year unadjusted survival (adjusted $x^2 = 4.6$, $p = .032$) in a cohort study of 1368 patients with coronary artery disease. In addition, a significant interaction between marriage and availability of a confidant was observed. Patients who were unmarried and without a confidant had an unadjusted 5-year survival rate of 0.50. In comparison, patients who were either married or reported availability of a confidant had a 5-year survival rate of 0.82.

However, not all studies have arrived at this same conclusion. In contrast to the previous study, Greenwood, Packham, Muir, and Madeley (1995) did not find a significant relationship between social support and mortality in a sample of 1283 patients post-myocardial infarction in a longitudinal study where patients were followed for between 4.7 and 6.3 years. Social support was defined in terms of marital status and social contacts. Social contact was measured by the frequency of formal social contact with friends and relatives through organizations or visits and was dichotomized into “high” or “low” categories. Neither social contact nor marital status

reached significant levels (.05) in terms of their effect on survival over time; however, marital status demonstrated a stronger effect on survival than that of social contact.

It is impossible to fully appreciate the relationship between social support and mortality among individuals with heart disease on the basis of these two studies alone. However, the differing findings from these two studies demonstrate the importance of interpreting results of studies in the context of the overall research design, population studied, and measures used to assess the concept of social support, as well as the broader literature on the subject. Although both studies were comprised of similarly large samples with comparable time frames for follow-up, the study conducted by Williams and colleagues measured the availability of a confidant in the context of theoretically defined functional components of social support, which affords a more robust measure of one's social network. The difference in instruments used to measure the social support construct could, in part, help explain the inconsistency in the findings from these two studies.

Studies have also begun to provide information related to the actual type of support that accounts for the most influence on outcomes related to morbidity and mortality. For example, Woloshin and colleagues (1997) found that perceived tangible support was related to 1-year mortality and physical functioning in a sample of 734 patients with coronary artery disease. Individuals who reported needing "much more help" had 6.5 times greater odds of dying (95% CI = 2.0-21.6) as compared to those individuals who reported no perceived tangible needs (i.e., needs were met). Interesting, this finding held even when controlling for age, baseline physical functioning score, dyspnea, and mental health score. Another study found that lack of social support (defined as the structure of and received emotional support from one's social network) significantly predicted subsequent 6-month survival among 194 patients post-myocardial

infarction (Berkman, Leo-summers, & Horowitz, 1992). This relationship held even after controlling for severity of MI, comorbidities, sociodemographic variables, and other common risk factors such as smoking and hypertension.

Krumoholz and colleagues (1998) found results similar to Berkman and colleagues in their study on the effects of social ties, instrumental support, and emotional support on fatal and nonfatal cardiovascular events among elderly patients hospitalized with heart failure. They found that lack of perceived emotional support was related to both fatal and nonfatal cardiovascular events in the year after hospitalization (odds ratio = 2.4; 95% CI = 1.1-4.9). However, in contrast to the previous mentioned study, patients who reported no social ties at baseline had a marginally higher incidence of events as compared to their counterparts, but this finding was not significant at traditional .05 levels. There was no relationship between instrumental support and 1-year fatal or nonfatal cardiovascular events.

In a landmark multi-site randomized clinical trial – Enhancing Recovery in Coronary Heart Disease (ENRICHD) – investigators examined the relative importance of treating depression and low perceived social support (LPSS) on mortality and reinfarction in a sample of 2481 men and women post-MI (Writing Committee for the ENRICHD Investigators, 2003). Patients were randomized to an intervention group or usual care. The intervention consisted of cognitive behavior therapy, supplemented with a selective serotonin reuptake inhibitor (SSRI) antidepressant when clinically indicated. Usual care consisted of the care provided by each participant's treating physician. Although the intervention resulted in increased perceived social support and decreased depression, the relative effect on mortality and subsequent infarction was not significant.

Though mortality and morbidity are not a focus of the research in the present dissertation, the literature on the relationships between social support and mortality and morbidity are relevant in the broader context. This literature, by and large, finds that social support is an important factor in improving health prospects for individuals with heart disease. These findings motivated a randomized clinical trial that attempted to reduce the perceived lack of social support, and evaluated the impact on subsequent mortality. While this latter study did not establish the effectiveness of the intervention in reducing mortality or morbidity, it does underscore that the relationship between social support and health outcomes is viewed as an important one, deserving of further study.

2.5.3. The Relationship between Social Support and Depression

There is a long history of interest in the relationship between social support and depression among health professionals. Indeed, there is now substantial and convincing evidence documenting the existence of a relationship between social support and depression across research designs and methodologies. The consistency of these finding extend to community (Billings & Moos, 1981; Cohen & Hoberman, 1983; Holahan & Moos, 1991; Russell & Cutrona, 1991), outpatient (Flaherty, Gaviria, Black, Altman, & Mitchell, 1983), and inpatient samples (Brummett, Babyak, Barefoot, Boworth, Clapp-Channing, Siegler, Williams, & Mark, 1998) and include individuals with psychiatric (George, Blazer, Hughes, & Fowler, 1989) and medical illnesses (Holahan et al., 1995). Research has been conducted on a cross-sectional and longitudinal basis, with both types of analyses providing insight. Some key studies are reviewed here.

2.5.3.1. Results from Cross-sectional Studies of Social Support and Depression

Cross-sectional studies aimed at informing health care professionals on the relationship between social support and depression have yielded fairly consistent findings, with lower levels of social support being correlated with higher levels of depression. These results hold over a variety of samples studied, including community dwelling elderly (Palinkas, Wingard, & Barret-Conners, 1990; Prince, Harwood, Blizard, Thomas & Mann, 1997; Russell & Cutrona, 1991), outpatients diagnosed with unipolar depression (Flaherty et al., 1983), and individuals with chronic illness (Symister & Friend, 2003), including heart disease (Brummett et al., 1998; Frasure-Smith et al., 2000; Holahan, Moos, Holahan, & Brennan, 1997; Krishnan, 1998).

Although it would seem that the findings from these studies are fairly robust, caution is warranted when making conclusions about the exact nature of the relationship (i.e. causal inferences) based on correlations alone. As discussed by Alloway and Bebbinton (1987), a key problem in interpreting cross-sectional studies is concluding that a direct relationship is supported between variables when in fact none exists. Results from cross-sectional studies are subject to third-factor explanations and reverse causation, necessitating caution in interpretation of findings (Helgeson & Cohen, 1996). Thus, attention is given to results from longitudinal studies, which can help clarify causal relationships.

2.5.3.2. Results from Longitudinal Studies of Social Support and Depression

There is fairly consistent evidence documenting the significant role of social support on subsequent depression, with lower levels of reported social support predicting higher levels of depression at follow-up. These results extend to community samples of college students (Cohen & Hoberman, 1983; Barnett & Gotlib, 1988), adults (Holahan & Moos, 1991; Monroe, Bromet, Connell, & Steiner, 1986), and the elderly (Russell & Cutrona, 1991). In addition, these results

hold in samples of individuals diagnosed with depression in both outpatient and inpatient settings (George et al., 1989; Sherbourne, Hayes, & Wells, 1995; Swindle, Cronkite, & Moos, 1989) and among individuals with chronic illnesses such as end-stage renal disease (Symister & Friend, 2003).

Only two studies were found that specifically examined the relationship between social support and subsequent depression in individuals with heart disease. The earliest of these studies was conducted by Holahan and colleagues (1995), and included a convenience sample of 325 individuals diagnosed with chronic cardiac illness and 71 individuals diagnosed with acute cardiac illness. The sample was comprised of approximately 75% men with a mean age of 61.8 (SD = 3.16) and 61.1 (SD = 2.93) years for the chronic and acute group, respectively. Ten subscales from the Life Stressors and Social Resource Inventory (LISRES) were used to measure perceived social support within three domains – family, work, and social network. This scale taps into the functional characteristics of social support and includes such items as "Does he or she [spouse] really understand how you feel about things" (p. 155). Depression was defined in terms of symptomatology and was assessed using the Research Diagnostic Criteria (RDC) index. Subjects were followed for a period of one year. Results indicate that, among the two cardiac groups, baseline social support had a significant relationship with depression at one year follow-up ($\gamma = -.46$, $p < .01$). Furthermore, this relationship held in a second model that examined the impact of social support on subsequent depression while controlling for initial depression ($\gamma = -.25$, $p < .05$).

Brummett and colleagues (1998) found similar results in a convenience sample of 506 (68.2% male) individuals diagnosed and hospitalized with coronary artery disease. Mean age for the sample was 63.4 (SD = 11.4). A shortened version of the ISEL (16 questions) was used to

assess subject's perceptions of availability of social support in the four separate functional domains: appraisal support, tangible support, self-esteem support, and sense of belonging. These four subscales served as the manifest variables for the latent construct social support in a structural equation modeling analysis. Depression was measured using four subscales (affective, well-being, interpersonal, and somatic) from the CES-D. Results of this study indicated that baseline social support had a significant direct effect on depression at 1-month in both the "direct model" ($\gamma = -.21$, $p < .05$) and the "indirect model" ($\gamma = -.24$, $p < .05$).

In conclusion, the results from these two studies add credence to the larger body of work on the relationship between social support and subsequent depression. The robustness of these findings extend across studies in which a variety of samples have been considered in the context of various methodological approaches, including the use of different instruments to measure social support and depression.

To date, however, there appear to be no studies that specifically examine the relationship between social support and depression among individuals with heart failure, either from a cross-sectional or longitudinal approach. As pointed out by Reifman (1995), further understanding of social support's influence on adaptation and recovery from illness depends in part on the consistent demonstration of associations between specific components of social support and recovery, and verification of the underlying mechanisms through which social support operates. Although it is reasonable to hypothesize that social support will serve as an equally important factor in the development of depression among individuals with heart failure, the exact nature of the relationship is not known at present. The results of the proposed study will serve to help fill the gap.

2.5.4. Relationship between Social Support and Anxiety

The psychosocial mechanisms by which anxiety influences outcomes in individuals with heart disease are currently undetermined and cannot be inferred from the previously presented studies in section 2.4. At present there appears to be no studies that specifically examine the relationship between social support and anxiety among individuals with a chronic illness such as heart failure. This is especially disconcerting given the growing evidence documenting the significant and pervasive effects of anxiety on health-related outcomes among individuals with heart disease. The results of the proposed study will provide preliminary information concerning the role of social support on anxiety.

2.6. CONTROL

2.6.1. Definitional and Theoretical Considerations

Individuals who are diagnosed with a chronic illness often find themselves making ongoing adjustments to maintain quality of life and well-being. It is important for health care professionals to understand the constructs and conditions that potentially facilitate adjustment to chronic illness. A sense of control is one such promising construct central in the coping process fundamental to positive adaptation to chronic illness (Bowsher & Keep, 1995).

The concept of control is complex and multidimensional with many theoretical underpinnings, definitions, and components. This underlying complexity has led to the development of a number of control-related constructs over the years, including personal control, mastery, self-efficacy, locus of control, and hardiness (Bowsher & Keep; 1995; Pearlin, 1989; Walker, 2001). Although these terms have been used interchangeably and have some overlapping conceptual characteristics and consequences, they have very important distinctions

when it comes to generality, definitions, processes, and antecedents (Bowsher & Keep, 1995). For example, central to the concept of mastery is a sense of gaining control over events in one's life, which is "exemplified by, but not exclusively served by, beliefs about personal control" (Taylor, 1983, p. 1161).

The collection of control constructs has led to some debate and confusion over a precise definition of control. To illustrate this confusion, consider one definition of control: "the responsiveness of an event to human intervention (things are 'under control'" (Walker, 2001, p. 10). This definition of control entails a fairly concrete and simplistic viewpoint in that an event is either controlled or not, but doesn't take into account the more subtle aspects of control, such as illusion of control (Taylor & Brown, 1988) or one's perception of control (Pearlin, 1989). Therefore, it is essential to further develop this definition in order to foster insight into important characteristics of control, such as perceived control, personal control, and perceived personal control.

Perceived control, the extent to which an event is alleged to be under control, implies that the event can be controlled directly through actions on the part of self or others (Walker, 2001). The source of control, internal versus external (either through others or fate), has been found to have significant implications for health related outcomes, with evidence suggesting that an external control orientation tends to be associated with more negative psychosocial outcomes such as depression and anxiety (Hunter & Locke, 1984; Johnson & Sarason, 1978; Shaw, 1999).

The construct *personal control* clarifies that control of the situation or event is self-determined rather than determined by others or chance (Walker, 2001). Finally, *perceived personal control* is the extent to which one believes that he/she has the ability to influence events or situations. It is the element of belief that distinguishes these two constructs of control, though,

as pointed out by Walker (2001), personal control and perceived personal control are often used interchangeably.

The relationship of personal control and adaptation to illness has been the focus of a number of research studies. Findings from these studies indicate that personal control is related to quality of life (Fitzgerald, Tennen, Affleck, & Pransky, 1993; Kempen, Jelicic, & Ormel, 1997), recovery from illness (Partridge & Johnston, 1989), disease severity (Seeman, 1991), self-assessments of health (Pulkkinen, Kokkonen, & Mäkiaho, 1998), and psychological adjustment (Helgeson, 2003).

Researchers have also begun to explore the more subtle aspects of control and found that central to our understanding of personal control is the answer to the question "control over what?" For example, a number of studies have found the perceived control over one's emotions, current symptoms, and the recovery process is more consistently associated with better adjustment to illness than control over disease progression (Affleck, Tennen, Pfeiffer, & Fifield, 1987; Jenkins & Pargament, 1988; Newsom, Knapp, & Schultz, 1996; Thompson, Sobolew-Shubin, Galbraith, Schwankovsky & Cruzen, 1993).

2.6.2. Relationship of Control to the Coping Process

According to Folkman and Moskowitz (2004) coping is a complex and multidimensional process that is sensitive to environmental demands and resources. Coping can be either positive or negative in nature and involves cognitive strategies that engage primary and secondary appraisals (Folkman, 1984). While coping is generally found to be strongly associated with emotional regulation throughout the stress process, the fundamental relationship between the various types of coping are not quite so easily knowable and indeed are not as simple as postulated (Folkman, 1984). For example, certain kinds of escapist coping strategies consistently have been linked to

poor mental health outcomes. In contrast, instrumental problem-focused styles of coping and seeking social support have been found to have positive, negative, as well as no impact on health outcomes (Folkman & Moskowitz, 2004).

In general, though, it is believed that having the capacity and resources to engage in problem-focused coping reduces both the psychological and physiological impact of daily hassles and stressors. One such proposed resource is a sense of personal control (Folkman, 1984). Personal control is believed to have contextual meaning to the coping process in two ways: first, as a generalized belief that an individual has regarding the extent that he/she can control relevant outcomes, and second, as a situational appraisal of the potential for control in a given stressful situation. Control as a generalized belief is a form of primary appraisal, while control in the context of a situational appraisal is viewed as secondary appraisal (Folkman, 1984).

In a related manner, Pearlin (1989) and his colleagues (Pearlin, Menaghan, Lieberman, & Mullan, 1981; Pearlin & Schooler, 1978) posit that exposure to stress, either due to discrete life events (losing a job) or enduring stressors (chronic illness), increases one's risk for adverse outcomes. Personal resources and coping strategies help to attenuate the negative impact of the stress. Resources, by their very nature, are delineated not by what people do, but by "what is available to them in developing their coping repertoires" (Pearlin & Schooler, 1978, p. 5). One such available coping strategy is control oriented coping which involves strategies to eliminate or modify the conditions giving rise to the stressful experience and/or by perceptually controlling the meaning of the experience (Pearlin & Schooler, 1978). Pearlin and his colleagues further argued that a sense of personal control is a component of control-oriented coping strategies. As such, personal control serves as an important medium to subsequent health outcomes in the

presence of or exposure to stressful situations. Pearlin (1989) further suggested that the mechanism by which stress exposure affects outcomes is influenced by the mediational properties of personal control, mastery, and supportive personal relationships.

2.6.3. Relationship between Control and Psychological Outcomes

Personal control has been linked to positive psychological outcomes among individuals with chronic illness. For example, higher reported levels of personal control were correlated with lower levels of depression among individuals with rheumatoid arthritis (Affleck et al., 1987), end-stage renal disease (Christensen, Turner, Smith, Holman, & Gregory, 1991), diabetes (Macrodimitris & Endler, 2001), and cancer (Newsom, Knapp, & Schulz, 1996; Thompson et al., 1993).

The finding that personal control has an impact on psychological functioning has also been found among individuals with heart disease. In an evaluation of 80 patients hospitalized for a cardiac event, Helgeson (1992) established a relationship between perceived control and adjustment to the illness; those with higher perceived control fared better on overall adjustment, defined as absence of depression, anxiety, and hostility. In a follow-up study of 278 patients treated for a cardiac event with percutaneous transluminal coronary angioplasty, Helgeson (1999) found that cognitive adaptation (e.g., self-esteem, optimism, and mastery) was strongly related to positive illness adjustment, which in the study included measures of mental health (e.g., depression and anxiety). Although both of these studies suggest an important relationship between perceived control or mastery constructs and depression and anxiety, in neither of these studies does the author report on these direct relationships specifically.

In studies that have examined this relationship directly, perceived control was found to predict subsequent depression. Research by Moser and Dracup (1995), studying 176 post-MI

patients, evaluated baseline perceptions of control using the Control Attitude Scale. Subjects who had high control, as indicated by a scoring above the median, were found to have significantly lower levels of depression (p-value 0.001) at the 6-month follow-up. Similarly, research by Bohachick, Taylor, Sereika, Reeder, and Anton (2002) found a relationship between baseline sense of control and depression at 6-months. They evaluated a sample of 28 heart transplant patients, using a subscale of the Coping with Serious Illness Battery to measure sense of control. A correlation of -0.52 (significant at the 0.05 level) was found between baseline sense of control and depression measured at the six month follow-up.

Only one study was found that examined the relationship between perceived control and emotional distress among individuals with heart failure (Dracup, Westlake, Erickson, Moser, Caldwell, & Hamilton, 2003). In this study, the Control Attitude Scale (CAS) was used to measure perceived control among 222 heart failure patients. The scale was modified by experts (nurses and physicians) in the area of heart failure, which resulted in a control scale reflecting the specific context of heart failure. Scores ranged from 4-20, with higher scores indicating higher perceived control. Patients were grouped into those with low perceived control (≤ 10, $n = 91$) and higher perceived control (≥ 11, $n = 131$). Patients who scored in the low perceived control group had significantly higher levels of depression as compared to those in the high control group.

To summarize, there is ample literature to suggest a link between personal control and depression in individuals with chronic illness. However, very little of this research has focused on individuals with heart failure. The present study will contribute to this stream of research.

While there is only a limited literature that examines the relationship between personal control and anxiety, three of the studies reviewed in the previous sub-section do provide such analyses. Moser and Dracup (1995) found higher baseline levels of perceived control were

associated with lower levels of anxiety at six months (p value = 0.002) in their sample of 176 hospitalized patients. Dracup and colleagues (2003) found that perceived control was a significant explanatory variable of anxiety in a cross-sectional analysis. Similarly, Bohachick and colleagues (2002) found a negative relationship between baseline sense of control and anxiety.

2.6.4. Control as a Mediating Variable between Social Support and Outcomes

Although there is a literature that identifies personal control as an important factor in health-related outcomes, limited empirical work appears to place this relationship in a broader theoretical context that includes the role of social support. One of the earliest studies to examine the mediational nature of personal control found that self-efficacy did not serve as a significant mediator between social support and depression among individuals with diabetes (Connell, Davis, Gallant, & Sharpe, 1994). It is important to note that the authors used a single item, "I am able to do the things I need to do for my diabetes" (p. 266) rated on a five-point scale to assess subjects' level of self-efficacy. They acknowledged that the use of a single item to capture the latent variable self-efficacy raised considerable questions concerning reliability. As such, conclusions drawn from this study should be viewed with caution.

Manne and Glassman (2000) also found that perceived control did not serve as a mediator between perceived negative spousal social support and psychological distress (anxiety and depression) in a sample of 191 married individuals with cancer. Perceived control was assessed using a five-item scale with questions about control over emotions, relationships, and disease progression. The authors suggest that the findings may be related to a multicollinearity problem between the construct perceived control and coping efficacy (coping efficacy was a significant mediator between negative spousal support and psychological distress in their model). However,

another plausible explanation is that the construct psychological distress was comprised of both depression and anxiety; it is possible that a mediational mechanism of perceived control acts differently for these two outcomes when considered in the context of negative social support.

It is difficult to draw clear and definitive conclusions from these two studies alone, given the measurement issues related to the construct perceived control and the limitations of each study. It is therefore important to consider a related set of studies that look at coping more broadly as a mediating construct between social support and depression. Holahan and colleagues (1995) conducted a one-year study of 396 individuals with cardiac illness. Using a structural equation modeling framework, they demonstrated that baseline social support – a construct based on measures of family support, work support, and social network support – had a direct effect of depressive symptoms one year later. They also found an indirect effect: social support was found to affect coping which in turn was found to affect depressive symptoms. That is, coping was found to be a significant mediating construct.

A second study conducted by Holahan and colleagues (1997) was based on a convenience sample of 183 patients diagnosed with chronic cardiac illness followed for a period of four years. For this study, only four subscales from the LISRES – family support, family stressors, extrafamily support, extrafamily stressors – were used to measure social support. This measurement method resulted in a vastly different cumulative operationalization of social support than that from the previously mentioned study, with social support being defined more in line with one's social context. Approximately 77% of the sample were men and the average age was 61.5 (SD = 3.2) for the group as a whole. Structural equation modeling was used to test the overall model in which social support was hypothesized to have an indirect relationship on depression through coping mechanisms. Results from this study support those previously found

by Holahan and colleagues (1995) and demonstrated that baseline social support predicted a significant indirect relationship between positive social context (time 1) and depressive symptoms (time 2) through percentage approach coping (time 2). It is interesting to note that for this study, a direct relationship between positive social context (time 1) and depressive symptoms (time 2) was not found to be significant.

2.7. GENDER DIFFERENCES

The concern over gender differences stems from a number of observations. First, the prevalence of depression is generally higher for women than men. Among medically well individuals, women are twice as likely as men to develop major depression or dysthymia (Weissman, Bland, Newman, Wells, & Wittchen, 1993). Similarly, being female is a risk factor associated with depression among the medically ill (Parker & Kalucy, 1999). For example, depression generally is higher among women with cardiac disease than men (Forrester et al., 1992; Frasure-Smith et al., 1993). Although limited research has been conducted examining gender differences in depression among individuals with heart failure, there is preliminary evidence to suggest that this pattern may hold for individuals with heart failure as well (Murberg, Bru, Aarsland, & Svebak, 1998).

Similarly, women tend to report higher rates of anxiety when compared to men. This finding extends to the psychiatric clinical population (Foot & Koszycki, 2004; Simonds & Whiffen, 2003) as well as non-clinical populations (Armstrong & Khawaja, 2002; Costa, Terracciano, & McCrae, 2001; Egloff & Schmukle, 2004). There are also important observed gender differences in levels of anxiety among individuals who have suffered a myocardial infarction, with women reporting higher levels of anxiety than men (Garvin, Moser, Riegel,

McKinley, Doering, & An, 2003). At present, no studies were found that specifically examined gender differences in anxiety among individuals with heart failure.

Second, there are important gender differences observed in the recovery process associated with cardiac illness, with women having higher death rates (Wilkinson, Laji, Ranjadayalan, Parsons, & Timmis, 1994) and generally poorer medical outcomes (Conn, Taylor, & Abele, 1991). In addition, the negative impact of depressive symptoms on functional status appears to be stronger for women than for men after coronary artery bypass surgery (Mallki, et al., 2005). Gender differences have also been documented related to the epidemiology, etiology, diagnosis, prognosis, and treatment of heart failure (Jessup & Piña, 2004; Petrie, et al., 1999). For example, while women with advanced heart failure tend to have better overall rates of survival than men (Ho, Anderson, Kannel, Grossman, & Levy, 1993; Adams, et al., 1999), women have significantly more symptoms associated with heart failure (Jessup & Piña, 2004), less improvement in physical health status, and perceived lower levels of quality of life (Chin & Goldman, 1998) when compared to men with heart failure.

Since evidence suggests that gender differences in rates of depression and anxiety are associated with both physiological (Thase & Howland, 1995; Young & Korszun, 2002) and psychosocial determinants (Brems, 1995; Hammen, 2003a, 2003b), it may be that there is some continuity between these determinants and the poorer outcomes observed for women with heart failure. It seems entirely plausible that the role of depression and anxiety in adaptation to chronic illness, such as heart failure, might be quite different for men than for women. Similarly, within the heart failure population, relationships between depression, anxiety, and other potentially relevant factors – illness severity, functional status, mortality, and indeed social support and personal control – might be quite different for women than for men. Tamres, Janicki, and

Helgeson (2002), in their meta-analysis of gender differences in coping behavior, find that when individuals face personal health as a stressor, the effect of active coping behaviors is stronger for women than for men in reducing psychological distress. As a second example, in Neff and Karney's (2005) study of husband-wife pairs, the level of positive support women provided was positively correlated with the severity of their husbands' problems, while the level of support men provided was *not* associated with the severity of their wives' problems. While neither of these studies relates directly to the exploratory analysis undertaken in this study, they provide insight into possible gender differences in the role of social support. At present though, few studies have examined the coping and adaptation process of patients with heart failure in terms of gender differences. A review of the literature suggests that a clearer comprehension on the role of gender, or for that matter, a more complete understanding of the many other subtle issues at work in psychosocial adaptation to heart failure, will require further studies using large enough samples to undertake convincing multivariate analysis.

2.8. CONCLUSION

The points raised in this literature review relevant to the proposed research are easily summarized: First, a vast literature documents a large problem of depression among the medically ill. Second, the literature provides consistent and strong evidence to indicate that social support has a direct relationship on depression even when controlling for relevant potential confounding factors. This finding extends to cross-sectional and longitudinal studies that control for baseline depression. Third, however, there appears to be a paucity of studies documenting the psychosocial mechanisms through which social support affects anxiety among individuals with chronic illness. In particular, while related research on social support and coping more globally

indicate the value in studying these key constructs in the context of anxiety among the medically ill, there appears to be no studies that focus specifically on this topic in individuals with heart failure. Finally, while a large body of evidence points to substantial gender differences in many of the constructs and outcomes related to depression among the medically ill, there is only a small literature that studies these issues carefully, and no work that directly studies gender differences in the role of social support and control as they relate to depression and anxiety among patients with heart disease.

3. METHODS

3.1. RESEARCH DESIGN

3.1.1. Secondary Analysis

The design of the proposed secondary analysis is a cross-sectional survey using data collected from the parent study. The primary aim of this study is to examine two mediating models of the relationship between social support and psychological outcomes among individuals with chronic heart failure. The first model examines the role of social support and personal control on depression, while the second considers the role of these same variables on anxiety. A secondary objective is to explore gender differences in the two proposed models.

The hypotheses for the first model are: (H1.1) Social support has an inverse direct effect on depressive symptomatology in individuals diagnosed with chronic heart failure. (H1.2) Social support has a positive effect on personal control through a direct path. (H1.3) Personal control has an inverse direct effect on depressive symptomatology in individuals diagnosed with chronic heart failure. (H1.4) Social support has an inverse effect on depression through an indirect path; specifically, social support has a positive effect on personal control, which in turn reduces depressive symptoms in individuals diagnosed with chronic heart failure.

Similarly, the hypotheses for the second model are outlined as follows: (H2.1) Social support has an inverse direct effect on anxiety in individuals diagnosed with chronic heart failure. (H2.2) Social support has a positive effect on personal control through a direct path. (H2.3) Personal control has an inverse direct effect on anxiety in individuals diagnosed with

chronic heart failure. (H2.4) Social support has an indirect inverse effect on depression through personal control, which directly reduces symptoms of anxiety in individuals diagnosed with chronic heart failure.

Finally, the last hypotheses examined addresses gender differences in each of the two models. (H3.0 and H4.0) The relationships will be different for males and females.

3.1.2. Description of the Parent Study

The design of the parent study, "Medication Adherence and Quality of Life in Chronic Illness" (National Institute of Health, National Institute of Nursing Grant #5 KO1 NR00083), was a panel survey focusing on a sample of patients diagnosed with congestive heart failure (CHF) receiving treatment with ACE inhibitors. The primary aim of this study was to test a multicomponent conceptual model of health related quality of life and medication adherence. The proposed hypotheses were: (1) psychosocial factors (social support and personal control) have a direct positive effect on medication adherence; (2) social support and personal control have an inverse effect on health related quality of life (psychological status, physical functioning, and symptomatology) in individuals who are chronically ill with CHF; and (3) psychological status, physical functioning, and symptomatology have a direct effect on medication adherence. The principal investigator examined the influential effects of psychosocial factors on quality of life and adherence through structural equation modeling (Bohachick, Burke, Sereika, Murali, & Dunbar-Jacob, 2002).

3.2. SETTING AND SAMPLE

3.2.1. Setting

The setting for the parent study was an outpatient Heart Failure Clinic associated with a University of Pittsburgh Health Center Clinic in Southwestern Pennsylvania. The convenience

sample included 243 men and women who met criteria to participate in the study. Individuals met inclusion criteria if they were: (a) 21 years of age or older; (b) diagnosed with congestive heart failure; (c) receiving treatment with an ACE inhibitor; and (d) were able to read and write English. Individuals were excluded from the study if they: (a) presented with a selected unstable medical conditions (unstable angina, recent myocardial infarction or cardiac surgery, and/or a noncardiac life-threatening disease); (b) had a major psychiatric disorder; and/or (c) were obviously confused. Potential study participants were not excluded on the basis of race, ethnicity, gender, or HIV status. Every effort was made during recruitment to enroll individuals in the parent study such that the demographic characteristics of the sample reflected the patient population of the urban area in which the study was being conducted.

3.2.2. Sample

For this analysis the sample consisted of 242 men and women between 21 and 82 years old being treated primarily for either idiopathic (55.8%) or ischemic (39.7%) cardiomyopathy. Descriptive statistics for the entire sample and by gender are presented in Table 1 and Table 2.

The sample is predominantly male (68.6%), married (74.8%), white, Non-Hispanic (91.7%), with a high-school education (48.3%). The majority of the participants reported that they were unemployed due to their disability (51.2%). Participants reported on average 7.13 total number of medications and 2.69 concurrent diagnoses, the diagnoses reported most frequently being coronary artery disease (29.8%), diabetes mellitus (25.8%), dysrhythmias (35.1%), gastrointestinal disorders (23.0%), hyperlipidemia (21.4%), hypertension (27.8), and previous myocardial infarction (25.4%).

Table 1 Demographic Characteristics of Sample

Variable	Total Sample (n=242)	Male (n=166)	Female (n=76)	Test For Gender Difference
Age – Mean	55.33	56.10	53.64	$T = 1.49$
(SD)	(11.87)	(10.84)	(13.79)	
Marital Status				
Currently Married	181	131 (78.9%)	50 (65.8%)	$\chi^2 = 6.55$
Never Married	22	13 (7.8%)	9 (11.8%)	
Separated	3	2 (1.2%)	1 (1.3%)	
Divorced	22	13 (7.8%)	9 (11.8%)	
Widowed	13	6 (3.6%)	7 (9.2%)	
Other	1	1 (0.6%)	0 (0.0%)	
Main Racial/Ethnic Group				
White (Not of Hispanic Origin)	222	161 (97.0%)	61 (80.3%)	$\chi^2 = 29.22$**
Black (Not of Hispanic Origin)	15	1 (0.6%)	14 (18.4%)	
Asian or Pacific Islander	3	2 (1.2%)	1 (1.3%)	
Other	2	2 (1.2%)	0 (0.0%)	
Highest Level of Education				
Grade School	20	12 (7.2%)	8 (10.5%)	$\chi^2 = 9.90$*
High School Diploma	117	72 (43.4%)	45 (59.2%)	
Vocational School	11	10 (6.0%)	1 (1.3%)	
Some College	38	27 (16.3%)	11 (14.5%)	
Associate Degree	12	9 (5.4%)	3 (3.9%)	
Bachelor Degree	26	21 (12.7%)	5 (6.6%)	
Graduate Degree	11	9 (5.4%)	2 (2.6%)	
Postgraduate Degree	7	6 (3.6%)	1 (1.3%)	
Current Occupation				
Employed – Full time	50	42 (25.3%)	8 (10.5%)	$\chi^2 = 36.47$**
Employed – Part time	8	5 (3.0%)	3 (3.9%0	
Unemployed – Due to lay off or employment factors	1	0 (0.0%)	1 (1.3%)	
Unemployed – Due to disability	124	86 (51.8%)	38 (50.0%)	
Retired – Not due to ill health	46	33 (19.9%)	13 (17.1%)	
Full Time Homemaker	13	0 (0.0%)	13 (17.1%)	

*Significant at the 0.05 level. **Significant at the 0.01 level.

The results of a preliminary analysis focusing on a comparison of social support, control and psychosocial variables (depressive symptoms and anxiety) by gender are presented in Chapter 4.

Table 2 Medical and Psychological Characteristics of Sample

Variable	Total Sample (n=242)	Male (n=166)	Female (n=76)	Test For Gender Difference
Type of Cardiomyopathy				
Idiopathic	135	82 (49.4%)	53 (69.7%)	$\chi^2 = 27.57$**
Toxic	2	2 (1.2%)	0 (0.0%)	
Familial	3	2 (1.2%)	1 (1.3%)	
Ischemic	96	80 (48.2)	16 (21.1%)	
Other	6	0 (0.0%)	6 (7.9%)	
Total Number of Concurrent Diagnosis	2.69 (1.64)	2.60 (1.56)	2.87 (1.79)	$t = -1.17$
Total Number of Medications	7.13 (2.97)	6.80 (2.81)	7.84 (3.18)	$t = -2.56$*
Ejection Fraction	0.29 (0.11)	0.29 (0.12)	0.30 (0.10)	$t = -0.32$
Depression				
1. POMS Depression Subscale	3.50 (3.91)	3.07 (3.57)	4.43 (4.45)	$t = 2.56$*
2. SCL-90-R Depression Subscale	0.87 (0.73)	0.78 (0.63)	1.12 (0.89)	$t = 3.40$**
Anxiety				
1. POMS Anxiety Subscale	4.27 (3.79)	1.96 (3.58)	4.96 (4.14)	$t = 1.92$
2. SCL-90-R Anxiety Subscale	0.54 (0.62)	0.44 (0.49)	0.76 (0.80)	$t = 3.92$**

*Significant at the 0.05 level. **Significant at the 0.01 level.

Information related to the New York Heart Association (NYHA) Functional Classification was either not available from the medical chart or not adequately documented such that one could discriminate a subject's class accurately. For example, a subject's NYHA class might be charted as "2-3" instead of exclusively class 2 or class 3. There was, however, sufficient information on physical activity levels from self-report questionnaires to arrive at an alternate classification of physical functioning among this sample. Information related to functional status was collected using the Functional Status Questionnaire (Jette et al., 1986). This 6-item self-report was designed to assess the level of difficulty in performing activities among ambulatory care patients. Items reflect ability to engage in intermediate activities of daily living and include the following: (1) "walking several blocks"; (2) "walking one block or climbing one flight of stairs"; (3) "doing work around the house such as cleaning, light yard work, home maintenance"; (4) "doing errands, such as grocery shopping"; (5) "driving a car or using public transportation"; and (6) engaging in vigorous activities such as running, lifting heavy objects or participating in strenuous sports" (Jette et al, 1986, p. 144). Subjects were asked to rate each item

in terms of the level of difficulty over the last four weeks. Items were rated on a four-point scale with the following anchors: (1) usually did with no difficulty; (2) some difficulty; (3) much difficulty; or (4) usually did not do because of health. Information from these questions was used to classify subjects into the following categories of functional impairment: mild, moderate, severe, and extremely severe. The majority of subjects fell within the moderate (36.3%) and severe (51.3%) categories, which can be thought of as a proxy to NYHA Class II and Class III (Heart Failure Society of America, 1999). Subjects who reported mild levels of functional impairment accounted for 7.9% of the total sample. Only 4.5% of subjects were classified as having extremely severe levels of impairment.

The principal investigator (PI) or the project nurse recruited all subjects following the IRB approved procedures as follows. The Medical Director of the Heart Failure Clinic referred patients to the study and provided medical backup for the study. Clinic clinicians met with potential subjects in order to explain the study and secure permission to give names of those interested in participating to the PI and/or project nurse. The clinic secretarial staff provided the PI and/or the project nurse with the names of potential participants along with their next scheduled appointment time.

The project nurse reviewed the medical record of each of these identified individuals in order to determine eligibility status based on study criteria. The project nurse then met with those patients who met record review eligibility criteria to explain the study and obtain informed consent. Informed consent included a description of the nature and purpose of the study, potential risks and benefits associated with study participation, and information related to subject rights (including confidentiality and the right to withdraw from the study at any time with no risk of consequences to heath care). During the informed consent process, subjects were given an

opportunity to ask questions related to the study. In addition, subjects were given contact information for the PI in the event that questions arose at a later time. Subjects who agreed to participate signed and dated three copies of the informed consent. In addition, the PI and/or project nurse along with an impartial witness from the clinic staff signed and dated the consents. Study participants received a copy of the informed consent and the additional copies were included in the research record and the clinic medical record. A recruitment log was kept of all potentially eligible participants documenting the reason(s) for exclusion from the study.

3.3. MEASUREMENTS

3.3.1. Social Support

Subscales from the Interpersonal Support Evaluation List (ISEL) were utilized to provide the manifest variables for the construct social support. The ISEL is a 40 item self-report designed to measure the perceived availability of potential social support resources in four separate domains: appraisal support, tangible support, self-esteem support, and sense of belonging (Cohen et al., 1985). Item selection was guided by a stress/coping theoretical framework with the goal of covering the domains of social support resources important to facilitating coping with stressful life events. To counterbalance for desirability, 20 items are worded in a positive manner and 20 items are negative statements. Respondents are asked to rate each item in terms of whether the statement is *definitely false*, *probably false*, *probably true*, or *definitely true*, with response scores ranging from 0-3. An overall functional support score is derived by simply adding up the numbers corresponding to each of the responses. The overall functional support score ranges

from 0-120, with higher scores indicating higher levels of social support. Subscale scores are calculated in a similar manner and range from 0-30, with higher scores indicating higher support.

The ISEL is widely used in research relating to social support and is a valid and reliable instrument. In studies of the general population, internal consistency reliability (alpha coefficient) ranges from 0.88 to 0.90 for the total ISEL and 0.70-0.82 for appraisal, 0.62-0.73 for self-esteem, 0.73-0.78 for belonging, and 0.73-0.81 for tangible support (Cohen, et al., 1985). The estimated reliability coefficients for this study are 0.88 for appraisal, 0.71 for tangible, 0.80 for self-esteem, 0.85 for belonging, and 0.94 for the total scale.

Test-retest reliability information is reported for two-day, six-week, and six-month intervals for the general population. Test-retest scores range from 0.74-0.87 for the total scale, 0.49-0.87 for tangible, 0.54-0.74 for self-esteem, 0.67-0.82 for belonging, and 0.60-0.87 for the appraisal subscale in a general population. Cohen and colleagues (1985) demonstrate the convergent construct validity of the ISEL through correlation studies with other social support measures such as the Inventory of Socially Supportive Behaviors (0.46), Moos Family Environment Scale (0.30), and the Partner Adjustment Scale (0.31).

Cohen and colleagues (1985) report on the means and standard deviations resulting from three collection time-points with the Oregon Smoking sample. Means and standard deviations ranged from 32.9 to 34.4 and 4.96 to 5.98 respectively. In comparison, the estimated mean and standard deviation for this sample was 89.62 and 17.27 for the total scale.

3.3.2. Control

Three scales were used to provide observed measurements for the construct personal control: the Mastery scale, the Coping with Serious Illness Battery (CSIB) Sense of Control subscale, and the Control of Symptoms scale.

The Mastery scale is a 7-item self-report designed to measure a global sense of personal control (Pearlin & Schooler, 1978). A total mastery score is derived by first reverse scoring two items and then summing the item responses. Total scores range between 7 and 35, with higher scores indicating higher perceived personal control. The mastery scale was originally constructed for a study aimed at understanding the social origins of personal stress (Pearlin & Schooler, 1978). In this study, a sample of 2300 individuals, aged 18-65, were systematically interviewed regarding potential life strains, coping strategies commonly employed, and emotional stresses experienced. Seven questions related to mastery were posed to individuals participating in the study. Individual items are rated on a 5-point scale that assesses the extent to which people agree or disagree that things that happen in their lives are under their control.

Principal component analysis with varimax rotation revealed item loadings ranging from 0.47-0.76. The authors do not specifically report on reliability or validity in this study. However, in a later study conducted by Skaff, Pearlin, and Mullan (1996) the alpha reliability was estimated at 0.75 in a sample of 456 spouses and children caring for a family member with Alzheimer's disease. Other studies have reported alpha levels between 0.73 and 0.77 (Armstrong-Stassen & Cameron, 2003; Bengtsson-Tops, 2004). The estimated reliability coefficient for this study was 0.82.

The CSIB Sense of Control subscale is a 7-item self-report where 6 items are rated on a 5 point Likert scale and 1 item is rated on a 6 point scale (Stewart, 1983). Scores range from 6-30, with higher scores indicating higher perceived personal control. The goal of this scale is to provide an assessment of an individual's ability to solve problems and influence others.

Stewart (1983) reported scale internal-consistency reliability in two sub-samples of 0.74 (94 patients with cancer) and 0.72 (156 post-MI patients). The estimated reliability coefficient for

this study was 0.72. Normative information was also described for the cancer and post-MI patients. The reported mean and standard deviation (reported in parenthesis) were 22.15 (3.81) for the cancer group and 23.45 (3.85) for the post-MI sample. In comparison, the mean and standard deviation for this sample were 22.94 and 3.64 respectively. The author also reported investigations of the scale validity via factor analysis. Principal components analysis revealed two factors with eigenvalues greater than 1 with item-scale correlations ranging from 0.36 to 0.59 (cancer sample) and 0.25 to 0.65 (post-MI sample).

The Control of Symptoms scale consist of four items rated on a 10 point scale, with *very uncertain* anchored at 1 and *very certain* anchored at 10. The goal of the four items is to understand the extent to which patients believe that they have the ability to control the day-to-day symptoms associated with their medical illness. The psychometric properties have not been established for this measurement; however, the estimated reliability coefficient for this study was 0.85. To establish convergent validity information, correlations between the Control of Symptoms scale and the two other scales used in this study were conducted. Correlations between the Control of Symptoms scale and the Mastery and CSIB were 0.40 and 0.69 respectively, indicating a moderate relationship between the three methods of evaluating control.

3.3.3. Depression

The Symptom Check List-90-R (SCL-90-R) is a multi-dimensional scale designed to assess psychopathology in both psychiatric and medical patients. The measurement includes a 13-item depression subscale that captures a range of co-occurring clinical depressive symptoms. These items cover symptoms such as feelings of hopelessness, loss of energy, lack of motivation, and thoughts of suicide. Respondents rate each item in terms of the amount of discomfort that the symptom has caused in the past month. Rating is based on a 5-point Likert scale ranging from 0

(*not at all*) to 4 (*extremely*). In this study, scores were calculated by adding together the non-zero scores from the individuals items and then dividing the summed score by 13. Scores range from 0 to 4, with higher scores indicating higher distress from symptoms. Cut-off scores to represent a clinical case are not available. The reported mean was 1.87 (SD=0.84) in a sample of 209 symptomatic volunteers with reactive depression and chronic anxiety states (Derogatis, Rickels, & Rock, 1976). In comparison, the mean for our sample was 0.87 (SD=0.73). In a sample of 565 subjects the internal consistency reliability coefficient for the depression subscale was estimated at 0.90. The estimated reliability coefficient for this study was 0.91. Convergent validity was evaluated by correlating the SCL-90-R scores with results from two scoring versions of the Minnesota Multiphasic Personality Inventory (MMPI) – the MMPI content scale and the MMPI cluster scale. The correlations were 0.75 and 0.68 for the content scale and cluster scale respectively (Derogatis, Rickels, & Rock, 1976).

The Profile of Mood States (POMS) scale is designed to assess transient mood states. The original POMS Scale consists of 65 items that measure the presence of specific affect adjectives such as sad, angry, and shaky (McNair, Lorr, & Droppleman, 1981). The scale consists of seven subscales, including a 15 item depression subscale. Respondents are asked to rate the extent to which they have experienced the affect adjective over the last week on a 5-point Likert scale from 0 (*not at all*) to 4 (*extremely*). McNair and colleagues (1981) estimated the internal-consistency reliability coefficient of the depression subscale at 0.95 in two samples (male and female) of psychiatric outpatients. The POMS depression subscale correlated with the Hopkins Symptom Distress Scale depression score at 0.86, which supports the concurrent validity of the POMS.

A shortened version of the POMS (37 items) is also available with 8 items relating to depression (Shacham, 1983). Correlation coefficients between the short and original scale range from 0.95 to 0.98, which suggests that the shortened version can be used without significantly compromising information and consistency. Internal consistency reliability coefficients for the original and shortened version of the depression subscale are 0.913 and 0.907 respectively (Shacham, 1983). The mean (standard deviation not reported) for the shortened version of the depression subscale was 0.91 in a sample of 83 cancer patients (Shacham, 1983). Direct comparisons between previously reported reliability and normative information and the presented study are not warranted as the present study uses a slightly different version of the scale. For this study, the depression subscale has a total five items. The estimated reliability coefficient for this study was 0.89.

3.3.4. Anxiety

The SCL-90-R anxiety subscale assesses behaviors associated with high manifest anxiety including such indicators as restlessness, nervousness, and the presence of panic attacks. The anxiety subscale has 10 items rated on a 5-point Likert scale. Total subscale scores range from 0 to 4 with higher scores indicating higher degrees of discomfort. According to Derogatis and colleagues (1976) the reported mean for 209 "symptomatic volunteers" was 1.49 (SD=0.78). The estimated mean for our sample is 0.54 (SD=0.62). The internal consistency reliability coefficient was estimated at 0.85 in a sample of 565 individuals (Derogatis, Rickels, & Rock, 1976). The estimated reliability coefficient for this study was 0.89. Convergent validity was established with comparison to a study based on the MMPI with a correlation found at 0.57.

A second anxiety subscale is taken from the POMS Scale described above. The original POMS anxiety subscale consisted of 9 items, while the shortened version includes 6 items

(Sacham, 1983). Items are rated on a 5-point scale ranging from “not at all” to “extremely”. Internal consistency for the original 65-item scale is estimated at 0.92 in a sample of male psychiatric outpatients and 0.90 in a sample of female psychiatric outpatients (McNair et al., 1981). In Shacham’s (1983) analysis, internal consistency reliability for the original 9-item subscale was 0.74, while in the 6-item short form the estimated coefficient was 0.80. The mean score (6-item short form) for a sample of 83 cancer patients was 1.44, calculated by summing the total items score and dividing by six. Comparisons to the present study are to be viewed with caution as the scale used in this study consisted of only five items with an estimated alpha reliability coefficient of 0.87.

3.3.5. Medical and demographic information

Medical information and demographic data were collected from the subject’s medical record and includes the following: type of cardiomyopathy, concurrent diagnoses, ejection fraction, number of medications, age, gender, ethnicity, marital status, and socioeconomic factors.

3.4. DATA COLLECTION PROCEDURES

After informed consent was obtained, the project nurse met with participants in order to review procedures related to study participation. During this time, subjects were given the Medication Event Monitoring System (MEMS) cap along with extensive instructions on how to use the cap over the duration of the study. Subjects were instructed to place the MEMS cap on the vial containing their ACE inhibitor medication. Each subject also received a packet of baseline self-reports with instructions to complete the questionnaires at their leisure over the next day. The estimated time to complete the respondent questionnaires was sixty minutes. Subjects were given a pre-addressed stamped envelope to use for returning the completed questionnaires. The project

nurse contacted subjects within three days after this clinic visit to follow-up on any additional questions and/or concerns related to study participation. MEMS data were collected over a three month period. At the end of the three months, subjects returned the MEMS cap either at a follow-up clinic appointment or through the mail. Final measures were taken at the three month follow-up clinic visits.

3.5. PROTECTION OF HUMAN RIGHTS

An application for Internal Review Board (IRB) approval under exempt status was submitted to the University of Pittsburgh Institutional Review Board prior to conducting the proposed secondary analysis. IRB approval was received on October 8, 2004 under exempt status. See Appendix A for a copy of the IRB approval letter. All data were de-identified (free of all identifying information) as required to meet criteria for IRB exempt status.

3.6. DATA SCREENING PROCEDURES

Data screening procedures were conducted using SPSS 12.0 [SPSS Inc, Chicago, Ill.]. The first step in the data screening process was to inspect the univariate statistics for accuracy of input. Frequency reports were generated for all variables, which demonstrated that all variables fell within expected ranges. Means and standard deviations were reasonable for all measurements. Bivariate correlations were within the expected direction of previous empirical evidence.

3.6.1. Missing Data

Every effort was made in the data collection phase to avoid missing data. However, one subject failed to provide responses on any of the control measurements (i.e., Mastery scale, CSIB Sense

of Control subscale, and Control of Symptoms scale) and was therefore not included in the analyses. The participant dropped from the analyses was a 63 year old man who reported that he was married and unemployed due to his disability. All other subjects provided complete information on all items for every variable, leaving a sample size of 242 for the analyses.

3.6.2. Univariate and Multivariate Outliers

A visual screening of the histograms and boxplots revealed evidence to suggest that univariate outliers existed. Therefore, a more formal statistical analysis was conducted in which standardized z-scores were generated for all variables. Standardized scores higher than 3.29 ($p < .001$) were considered as potential outliers (Tabachnick & Fidell, 1996). For the total group, potential univariate outliers were identified for both of the anxiety measurements. The POMS Anxiety subscale had one outlier (z-score = 3.62) and the SCL-90-R Anxiety subscale had five outliers (z-scores = 3.32, 3.48, 3.64, 3.80, and 3.97). All outliers were in the upper range of the measurement indicating that these subjects had higher levels of anxiety. This same screening method was conducted for men and women separately as the planned analyses also involved an examination of gender differences in the path analysis. For the group of women, two outliers were identified (POMS Anxiety z-score = 3.62 and SCL-90-R Anxiety z-score = 3.97). No outliers were found for the group of men.

The presence of multivariate outliers was evaluated statistically using Mahalanobis distance for both grouped (by gender) and ungrouped data. Mahalanobis distance at $p < .001$ (χ^2 df = to number of variables) was used to identify multivariate outliers (Tabachnick & Fidell, 1996, p. 67). A total of seven subjects were identified as potential multivariate outliers in the total group, with Mahalanobis distance scores ranging between 33.66 and 49.01. For the grouped

data, only one woman (Mahalanobis = 33.98) and five men (Mahalanobis ranging from 33.50 to 49.99) were identified as a potential multivariate outliers.

In order to make an informed decision regarding remedial strategies to reduce the influence of outliers, the identified potential outliers were examined for population fit. In examining each outlier on a case by case basis, it was determined that the identified outliers were indeed a part of the target population and that the information provided from these subjects represented appropriate variability in the measurements. Therefore, these subjects were retained in the sample for the analyses.

3.6.3. Screening for Normality, Linearity, and Multicollinearity

An assessment of univariate normality was completed by examining frequency histograms generated in PRELIS 8.54 [Jöreskog & Sörbom, Scientific Software International, Chicago, Ill.]. These histograms were suggestive of deviations from normality for the depressive symptoms and anxiety measurements. Skewness and kurtosis levels are summarized in Table 3 in order to provide insight into the validity of the study results under normal theory estimation (Hoyle and Panter, 1995).

Threshold levels discussed by Curran, West and Finch (1996) were used for identifying nonnormal univariate distributions. Skewness values ranging from 2.00 to 3.00 and kurtosis values between 7.00 and 21.00 indicate moderately nonnormal distributions. Skewness levels above 3.00 and kurtosis levels above 21.00 are indicative of extremely nonnormal distributions. Accordingly, all univariate distributions demonstrated minimal skewing and kurtosis.

Linearity was assessed through the use of bivariate scatterplots among pairs of measured variables. Scatterplots were roughly oval shaped indicating that the underlying assumption of linearity was tenable.

Table 3 Univariate Skewness and Kurtosis

Variable	Skewness	Kurtosis
1. POMS Anxiety	1.02	0.65
2. POMS Depression	1.19	0.73
3. SCL-90-R Anxiety	1.85	3.46
4. SCL-90-R Depression	1.04	0.46
5. Mastery	-0.26	0.05
6. Personal Control	-0.61	0.72
7. Control of Symptoms	-0.14	-0.93
8. ISEL Appraisal	-0.66	-0.25
9. ISEL Tangible	-1.04	0.41
10. ISEL Self-Esteem	-0.39	0.16
11. ISEL Belonging	-0.74	0.14
12. ISEL Total	-0.68	0.01

Collinearity (multicollinearity and singularity) diagnostics was conducted in order to assure matrix inversion capability or nonsingularity. Bivariate correlation output was examined for correlations equal to or exceeding 0.80, as a correlation of this magnitude is suggestive of collinearity (Maruyama, 1998). The correlation between SCL-90-R Depression and POMS Depression ($r = 0.82$) demonstrates potential collinearity. Further diagnostic information related to collinearity provided in the SEM output was examined after conducting the analysis. For this diagnostic procedure the covariance matrix determinant was examined. In general, an extremely small determinant indicates problems with multicollinearity or singularity. Typically SEM programs will terminate an analysis or will provide warning messages when the covariance matrix is singular.

3.7. DESCRIPTIVE STATISTICS

Descriptive statistics (measures of central tendency, variability, and relationship) were generated using SPSS 12.0 [SPSS Inc, Chicago, Ill.]. Although the median is generally the central tendency measurement of choice for ordinal date, the mean was used as the measure of central tendency for social support, control, depression, and anxiety. These measures are considered to be highly ordinal in nature and as such the mean will provide meaningful information. Independent sample t-tests were conducted to compare means between women and men for the manifest variables. Pearson correlation coefficients were generated to describe the degree of relationship between the manifest variables.

3.8. INFERENTIAL STATISTICS

3.8.1. Specific Aims 1 and 2

Structural Equation Modeling (SEM) was used to test the hypotheses associated with specific aims 1 and 2. SEM is a multivariate statistical methodology that allows for a confirmatory, or hypothesis-testing approach for analyzing theoretically linked relationships between constructs relative to a certain phenomenon (Byrne, 1998). Generally, the theory describes the underlying causal processes and effects between the concepts or constructs within the theory, which then serves to guide the research hypotheses under investigation. For this study, a SEM approach was used to estimate the direct and indirect effects of social support and control on depression (Model 1). Similarly, a second SEM analysis was conducted to estimate the structural effects of social support and control on anxiety (Model 2). SEM analyses were based on the covariance matrix using the maximum likelihood procedure in LISREL 8.54 program [Jöreskog & Sörbom, Scientific Software International, Chicago, Ill.].

3.8.2. Specific Aims 3 and 4

A primary goal of this study is to understand gender differences in two mediating models examining the relationship between social support and psychological outcomes among individuals with chronic heart failure. To achieve this objective, a multi-sample modeling approach was conducted separately for each of the two models. Multi-sample modeling is uniquely suited for testing the hypotheses proposed by this study "because of its ability to test a theoretical model for its applicability to different groups simultaneously" (Marsh & Grayson, 1995, pg. 201). Path analyses were used to test for gender differences as outlined in specific aims 3 and 4 and illustrated in Figure 4. In principle, one could conduct the hypothesis tests within the context of the structural equation modeling procedures described above. However, the sample size for women was relatively small for such analysis.

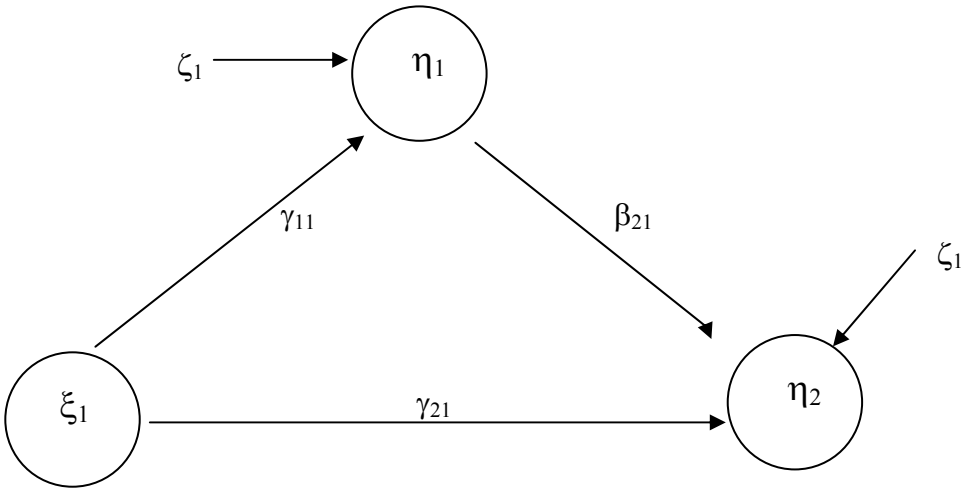

Figure 4 Path Analysis Model in LISREL Notation

Path analysis is similar to SEM in that models specify causal direct and indirect relationships between a number of variables that can be tested for model fit (Hatcher, 1994). However, path analysis differs from SEM in that the relationships between constructs are tested

using only manifest or observed variables (as opposed to latent variables in SEM). As such, an important assumption in path analysis is that independent variables are measured without error. Given that most of the measurements of constructs in the social sciences are typically measured with error, this assumption is frequently violated. Other assumptions associated with path analysis are similar to those for SEM. For this particular study, path analysis is the method of choice as the samples when grouped for males and females are too small to conduct a full SEM analysis (Hatcher, 1994).

For the path analyses, social support was operationalized using the total score of the Interpersonal Support Evaluation List (Cohen, Mermelstein, Kamarck, & Hoberman, 1985). Depressive symptoms are measured using the Symptom Checklist 90-R (SCL-90-R) depression subscale (Derogatis, Rickels, & Rock, 1976), which in this sample is the more reliable of the two available instruments. Finally, separate analyses were conducted with each of the three available control scales: The mastery scale (Pearlin & Schooler, 1978), the Coping with Serious Illness Battery (CSIB) Sense of Control subscale (Stewart, 1983), and the Control of Symptom scale that was constructed specifically for the parent study.

Multi-group path analyses, as outlined by Byrne (1998) and Tabachnick and Fidell (1996) were conducted in order to examine gender differences. This set of analyses was conducted using LISREL 8.54 [Jöreskog & Sörbom, Scientific Software International, Chicago, Ill.]. The first critical step in this process was to develop good-fitting path models for males and females separately. This part of the analysis was conducted through separate LISREL runs for each group. The models were then tested simultaneously in one run without constraining or forcing any parameters across models to be equal. The results of this analysis – the unconstrained multiple group model – served as the baseline for comparison of more restricted models. The

next steps involved testing a sequence of models with more stringent constraints imposed upon chosen parameters. The more restricted models were then tested against the baseline unconstrained multiple group model and/or least restrictive models using a chi-square difference test.

A supplementary method for evaluating gender differences involved estimating a mediator model as described by Barron and Kenny (1986) along with gender interactions. This set of analyses was conducted using SPSS 12.0 [SPSS Inc, Chicago, Ill]. In particular, parameters of the following regression equations were estimated:

(1) $x_{i2} = \beta_0 + \beta_1 x_{i1} + \beta_2 z_i + \beta_3 x_{i1} z_i + \varepsilon_i$
(2) $y_i = \beta_0 + \beta_1 x_{i1} + \beta_2 z_i + \beta_3 x_{i1} z_i + \varepsilon_i$
(3) $y_i = \beta_0 + \beta_1 x_{i1} + \beta_2 x_{i2} + \beta_3 z_i + \beta_4 x_{i1} z_i + \beta_5 x_{i2} z_i + \varepsilon_i$

where y is the dependant variable, depressive symptoms or anxiety; x_1 is the independent variable, social support; x_2 is the mediator, mastery; and z is a gender indicator equal to 0 if female and 1 if male.

3.9. STRUCTURAL EQUATION MODELING

3.9.1. Model Specification

A full latent variable (LV) model (sometimes referred to as a complete model) consists of both a measurement model and a structural model (Byrne, 1998). As such, generally two separate steps are taken when specifying a full LV model. First, the structural model is specified according to the theoretical paradigm under study, which *a priori* connects the structural links between the latent variables in the model. Latent variables, also called unobserved or unmeasured variables (Byrne, 1998), are delineated as either exogenous or endogenous in nature. Exogenous variables are analogous to independent variables and thus, are not directly influenced by other latent

variables in the model. As such, the model does not explain changes in the values of exogenous variables (Byrne, 1998). When more than one exogenous variable exist in the model, they typically share a nondirectional relationship with each other (MacCallum, 1995).

Endogenous variables are dependent variables, and are directionally influenced by other variables in the model (Byrne, 1998). A major advantage of structural analysis is that the relationship between variables can be decomposed into the direct, indirect, and total structural effects for the model under study. The direct effect (DE) of an independent variable on a dependent variable is depicted by the structural coefficient linking the two variables (Mueller, 1996) and is equivalent to the type of relationship evaluated through multiple regression or ANOVA (Hoyle, 1995). An indirect effect (IE) is the influence of an independent variable on a dependent variable through one or more mediating variables (Hoyle, 1995). In this sense, the mediating variable acts as both an independent and dependent variable. Computationally, the IE is the product of the structural coefficients associated with all the linking paths in an identified structural chain (Mueller, 1996). The total effect (TE) of an independent variable on a dependent variable is simply the sum of the DE and IE (Muellller, 1996).

The structural relationship between the latent exogenous and endogenous variables can be characterized through a schematic presentation, as well as through a series of regression or structural equations (Byrne, 1998). For this study, the SEM analytic model in LISREL notation is schematically represented in Figure 5. Refer to Table 4 for a description of the Greek symbols and notation used in the full LV models.

Table 4 Greek Symbols and Notation for SEM (Mueller, 1996)

Symbol	Meaning
ξ (ksi)	Latent exogenous variable Model 1: ξ_1 = Social Support Model 2: ξ_1 = Social Support
η (eta)	Latent endogenous variables Model 1: η_1 = Control; η_2 = Depressive Symptoms Model 2: η_1 = Control; η_2 = Anxiety
β (beta)	Structural effect from an endogenous variable to another endogenous variable
B (Beta)	Matrix containing structural effects from endogenous variables to other endogenous variables
γ (gamma)	Structural effect from an exogenous variable to an endogenous variable
Γ (Gamma)	Matrix containing the structural effects from exogenous to endogenous variables
δ (delta)	Measurement error associated with an observed exogenous variable
ε (epsilon)	Measurement error associated with an observed endogenous variable
ζ (zeta)	Error term associated with an endogenous variable
Θ_δ (Theta Delta)	Variance/covariance matrix of observed exogenous measurement errors
Θ_ε (Theta Epsilon)	Variance/covariance matrix of observed endogenous measurement errors
Λ_x (Lambda X)	Matrix containing the structural effects linking the observed and latent exogenous variables
Λ_y (Lambda Y)	Matrix containing the structural effects linking the observed and latent endogenous variables
Σ (Sigma)	Unrestricted variance/covariance matrix of observed variables
$\Sigma(\theta)$	Model-implied observed variables variance/covariance matrix
Φ (Phi)	Exogenous variables variance/covariance matrix
Ψ (Psi)	Variance/covariance matrix of endogenous variable's error terms

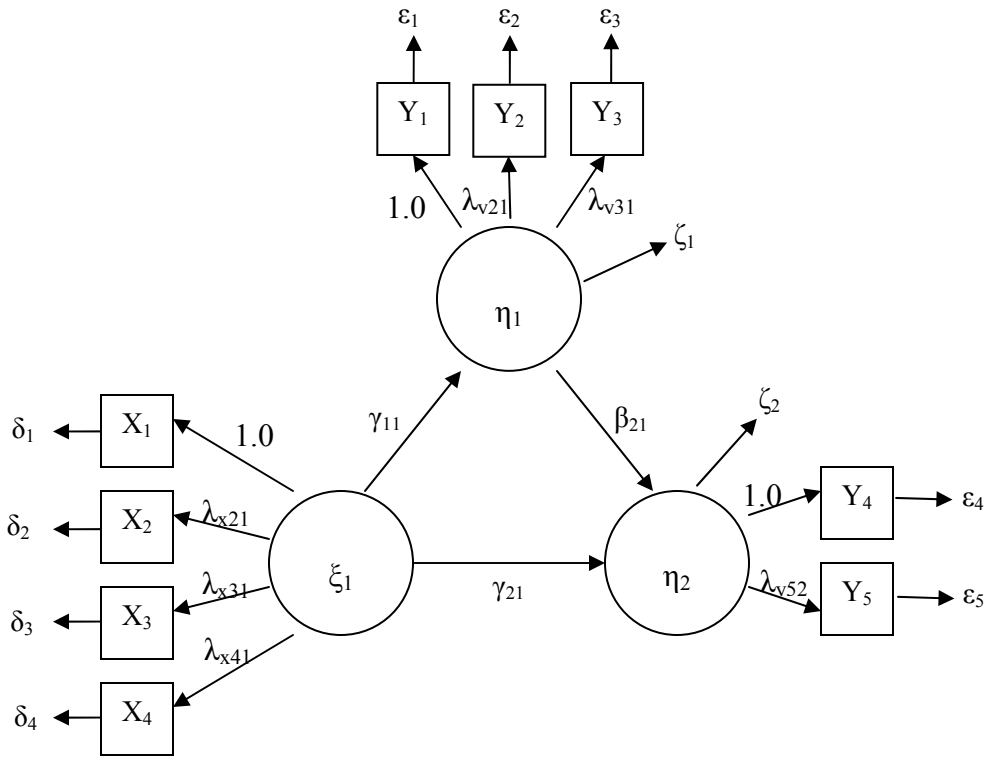

Figure 5 Structural Equation Model in LISREL Notation

Figure 6 structurally relates the latent exogenous variable, social support (ξ_1 = SocSup), to the latent endogenous variables, control (η_1 = Cont) and depressive symptoms (η_2 = DepSy). Looking closely at Figure 6, one can see that *social support* is hypothesized to have a direct structural effect on *depressive symptoms* as indicated by the path γ_{21}. In addition, the model specifies that *social support* has an indirect structural effect on *depressive symptoms* through a single mediating variable *control*, as depicted by paths γ_{11} and β_{21}. The product of these two paths ($\gamma_{11}\beta_{21}$) gives the indirect effect for *social support* on *depressive symptoms*. The sum of the DE and the IE gives the total effect of *social support* on *depressive symptoms.*

One advantage of SEM is that the mediating variable, in this study *control*, acts not only as a dependent variable for social support, but also as an independent variable for depressive symptoms. Thus, the direct effect of *social support* on *control* and likewise the direct effect of *control* on *depressive symptoms* is determined separately, which is represented by the direct paths γ_{11} and β_{21}.

Similarly, Figure 7 depicts the structural relation between the latent exogenous variable, social support (ξ_1 = SocSup), to the two latent endogenous variables (η_r), control (η_1 = Cont) and symptoms of anxiety (η_2 = Anx). For this model the effects can be decomposed into the direct, indirect, and total effects as previously discussed for Model 1.

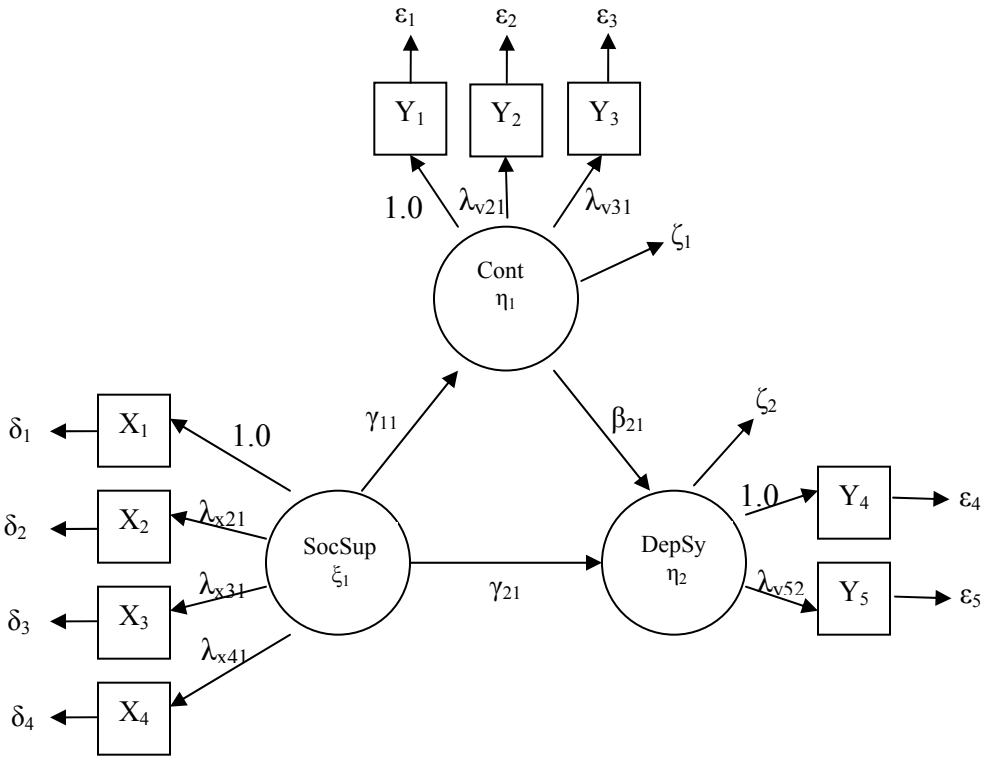

Figure 6 Depressive Symptoms Structural Model in LISREL Notation

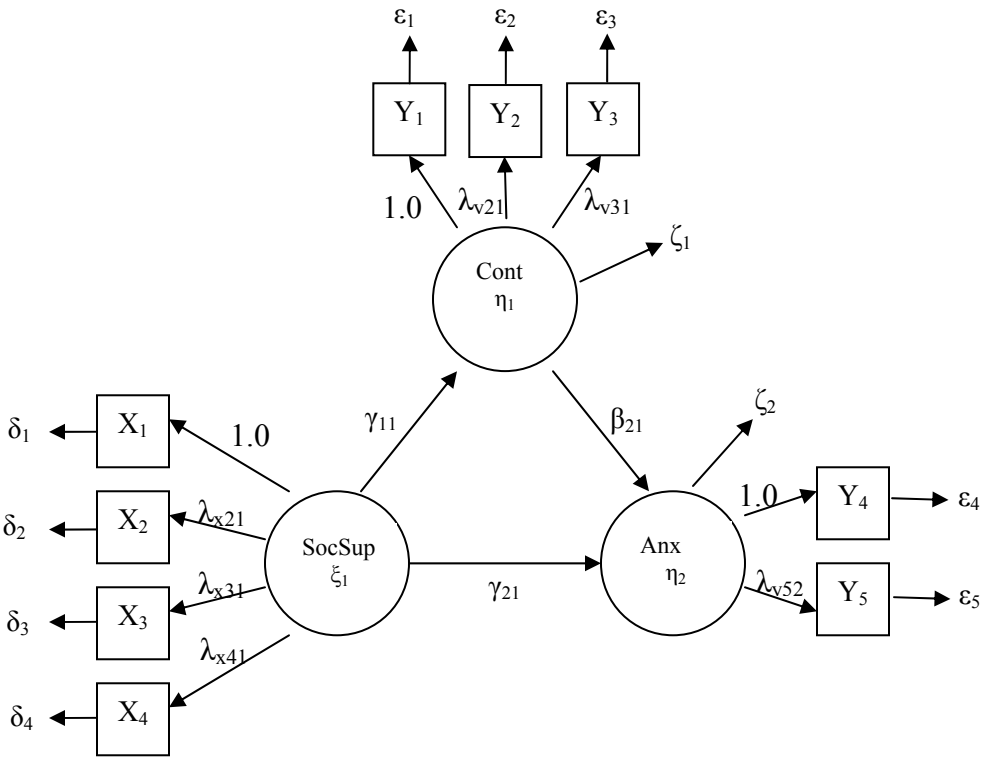

Figure 7 Anxiety Structural Equation Model in LISREL Notation

In addition to demonstrating each model's structural relations pictorially, the structural relations can be represented by a series of regression (or structural) equations. Each model can be represented by the following structural equation:

$$\eta = \mathrm{B}\eta + \Gamma\xi + \varsigma$$

where η (eta) represents the vector of latent endogenous variables, B (Beta) represents the matrix containing the structural effects relating the endogenous variables, Γ (Gamma) represents the matrix containing the coefficients relating the exogenous variable to the endogenous variables, ξ (ksi) represents the latent exogenous variable vector, and ζ (zeta) represents the vector of residual error terms associated with an endogenous variable (Byrne, 1998).

Following Byrne (1998), one can further expand each model's structural equation into the following matrix equation:

$$\begin{matrix} \eta \\ \begin{bmatrix} \eta_1 \\ \eta_2 \end{bmatrix} \end{matrix} = \begin{matrix} \mathrm{B} \\ \begin{bmatrix} 0 & 0 \\ \beta_{21} & 0 \end{bmatrix} \end{matrix} \begin{matrix} \eta \\ \begin{bmatrix} \eta_1 \\ \eta_2 \end{bmatrix} \end{matrix} + \begin{matrix} \Gamma \\ \begin{bmatrix} \gamma_{11} \\ \gamma_{21} \end{bmatrix} \end{matrix} \begin{matrix} \xi \\ [\xi_1] \end{matrix} + \begin{matrix} \varsigma \\ \begin{bmatrix} \varsigma_1 \\ \varsigma_2 \end{bmatrix} \end{matrix}$$

From this equation it is clear that the first of the endogenous variables (η_1, control) does not depend on the second endogenous variable (η_2, depressive symptoms or anxiety), while the second endogenous variable does depend on the first.

3.9.2. Measurement Model

The next step was to identify the measurement model, which involves operationally defining all the latent exogenous and endogenous variables included in the full LV model. Given that latent variables by definition are constructs that are not capable of being directly observed, the measurement model identifies the observed (also referred to as measured or manifest) variables which serve as indicators or approximate measures for the underlying constructs in the model (Holye, 1995). Generally speaking, it is desirable to have a number of distinct indicators for each latent variable, as the LV is defined by the commonalities shared by the multiple indicators. Essentially, one can view the LV's as equivalent to the common factors derived in factor analysis.

The major advantage of using multiple indicators versus a single indicator approach to operationally define hypothetical constructs is that the resulting LVs are free from measurement error (Holye, 1995). This part of the proposed analysis involved fitting and testing a measurement model for the latent variables in Models 1 and 2 to determine whether the proposed indicators do in fact reflect the meaning of the constructs under study. Scales and/or items from

instruments that are purported to measure study constructs were evaluated for reliability, validity, and factor structure. Based on this analysis the appropriate measures for the latent variables (social support, personal control, depression, and anxiety) were selected for each model.

3.9.3. Parameter Estimation Procedures

LISREL 8.54 [Jöreskog & Sörbom, Scientific Software International, Chicago, Ill.] was used to test the measurement model, the structural model, and the overall goodness of fit for both Model 1 and Model 2 proposed in this study. LISREL is a computer software program that allows the researcher to estimate unknown parameters in a set of linear structural equations using Maximum Likelihood (ML). PRELIS was used to prepare the covariance matrix to be read by LISREL.

3.9.4. Data Model Fit

No single fit criterion is available in which one can definitively conclude whether or not the data fit the proposed model. Rather, several methods of gauging data-model fit have been proposed. These methods include the following: (1) χ^2 statistic or alternately the χ^2/df ratio; (2) Goodness-of-Fit (GFI) and Adjusted Goodness-of-Fit Index (AGFI); (3) Normed, Nonnormed Fit Index (NFI and NNFI) and Comparative Fit Index (CFI); and (4) the Root-Mean-Square Residual (RMR). For the purposes of this analysis, each of these methods was used to evaluate data-model fit.

Chi-Square and χ^2/df ratio – Theoretically, the χ^2 will be nonsignificant when the unrestricted population variance/covariance matrix is equal to the model-implied variance/covariance matrix. However, there are several shortcomings associated with using the χ^2 statistic as a single indicator of data-model fit. Specifically, the χ^2 statistic depends on satisfying a number of underlying assumptions – including multivariate normality, validity of the proposed null hypothesis, and having an adequately large sample size – which in reality are

rarely completely achieved (Mueller, 1996). Further, the χ^2 statistic is sensitive to sample size, with larger samples producing larger χ^2 test statistics. Given these concerns, the χ^2 test was considered in comparison to other fit indices when evaluating model fit for the proposed study. In addition, as recommended, χ^2/df ratio was calculated in order to compare the magnitude of the χ^2 statistic with the underlying sampling distribution (Mueller, 1996). Although there is not an absolute cut-off value for evaluating goodness-of-fit based on this ratio, generally values less than 2.00 are desirable. However, Mueller (1996) suggests that values as high as 5.00 may well reflect good data-model fit.

Goodness-of-Fit (GFI) and Adjusted Goodness-of-Fit Index (AGFI) – The GFI, an absolute fit index, is a ratio derived from the sum of the squared weighted variances from the model covariance matrix divided by the squared weighted variances from the sample covariance matrix, yielding an index comparable to the R^2 in multiple regression (Tabachnick & Fidell, 1996). The AGFI, a parsimony index, penalizes model complexity by adjusting for the degrees of freedom relative to the number of estimated parameters in the model (Schumacker & Lomax, 1996). These two indices are especially useful when comparing the fit of two separate models with the same data, and as such are applicable to the proposed study of gender differences. GFI and AGFI values fall between 0 and 1, with values of .90 or above defining overall good data-model fit (Mueller, 1996).

Normed, Nonnormed Fit Index (NFI and NNFI) and Comparative Fit Index (CFI) – The NFI is a comparative fit index that evaluates the estimated model by comparing said model's χ^2 to the χ^2 from an independence model (Tabachnick & Fidell, 1996). In essence this index rescales χ^2 to range between 0 (no fit) to 1, indicating perfect data-model fit (Schumacker & Lomax, 1996), with values greater than .90 indicative of good data-model fit. The NNFI

penalizes for model complexity by adjusting the degrees of freedom downward, which can result in values outside the 0-1 range (Tabachnick & Fidell, 1996). Both the NFI and NNFI are sensitive to sample size and are considered to be marginal fit indices at best in studies with small samples such as the proposed study. Therefore, the CFI was utilized as it is less sensitive to small sample sizes. The CFI is a measure that utilizes the noncentral χ^2 distribution with noncentrality parameters and ranges from 0-1, with the standard .90 cutoff indicating good data-model fit (Tabachnick & Fidell, 1996).

Standardized Root-Mean-Square Residual (SRMR) – The SRMR is the average difference between the sample variances/covariances and the estimated population variances/covariances. SRMR values range between 0 and 1, with values equal to or less than .05 indicating good data-model fit (Tabachnick & Fidell, 1996).

3.10. ISSUES RELATED TO MODEL SPECIFICATION

3.10.1. Underlying Assumptions

The underlying statistical assumptions that are necessary for structural equation modeling are summarized by Mueller (1996) and are briefly outlined here: (1) latent exogenous and endogenous variables have a mean of 0 [$E(\xi) = E(\eta) = 0$]; (2) a linear structural relation exists between the exogenous and the endogenous latent variables; (3) the ζ equation error terms have a mean of 0 with constant variance across observations, are independent (uncorrelated with other observations), and are not correlated with the exogenous latent variables; (4) the (I–B) matrix is nonsingular, i.e., is invertible; (5) both exogenous and endogenous observed variables have a mean of 0 [$E(X) = E(Y) = 0$]; (6) a linear relationship exists between the indicator variables

(exogenous and endogenous) and the associated latent constructs (exogenous and endogenous); and (7) the measurement error terms in δ and ε have a mean of 0, constant variance across observations, are independent (i.e. are uncorrelated across observations), are uncorrelated with the exogenous and endogenous latent variables, and are uncorrelated with each other.

3.10.2. Statistical Identification

Statistical identification is a complex issue in model specification and is often a difficult condition to establish for a given structural model. The basic underlying principle of identification is whether or not there exists a unique set of parameters consistent with the data under study (Byrne, 1998). Identification specifically involves the relationship between the free parameters to be estimated and the observed variances and covariances from which the free parameters are to be estimated. Based on this relationship, one can classify a model as being just-identified, underidentified, or overidentified. In a just-identified model, the number of free estimable parameters is equal to the number of variances and covariances (i.e., the number of "data points" using Byrne's terminology). An underidentified model is one in which the number of data points is less than the number of estimable parameters, and thus the model contains insufficient information to obtain a unique estimated parameter solution. In contrast, an overidentified model is one in which the number of data points is more than the number of parameters to be estimated, resulting in degrees of freedom greater than zero.

The goal in conducting a structural equation analysis is to specify a model that is overidentified. The rationale behind this is that an overidentified model will lead to an infinite number of possible estimable parameter solutions for which the researcher can test against the null hypothesis. In comparison, a just-identified model, having zero degrees of freedom, will

yield only one unique solution for parameter estimates, which essentially fails to enhance scientific knowledge as the resulting solution can never truly be rejected (Holye, 1995).

There are two necessary, but not sufficient, conditions for identification of model parameters (Holye, 1995). First, every LV in the model must have an established scale. For this study, each latent variable was mapped onto an associated observed indicator variable, referred to as the "reference" variable (Byrne, 1998). For both models under study, the reference variable was selected based on reliability estimates, as it is suggested that the measure with the highest reliability serve as the reference variable (Byrne, 1998). The scaling procedure for the LV was established by constraining the reference variable factor loading parameter (λ) for each set of loadings to a non-zero number, which is typically 1.0 (Bryne, 1998).

The second condition refers to the relationship between the number of data points and unknown parameters in the model, such that the number of unknown parameters cannot exceed the number of sample variance/covariance terms (Holye, 1995). The number of data points for each model was verified using the following formula, $p(p+1)/2$, where p is equal to the number of variables (Byrne, 1998). For this study, the number of observed variables for Model 1, and similarly for Model 2, equals nine. Using the above formula ($9(9+1)/2 = 45$), there are 45 data points of information for each proposed model. The number of unknown parameters for each model is 20 – specifically, there are six first order regression coefficients (λ_{x21}, λ_{x31}, λ_{x41}, λ_{y21}, λ_{y31}, λ_{y52}), three second order regression coefficients (γ_{11}, γ_{21}, β_{21}), nine measurement error variances (δ_1, δ_2, δ_3, δ_4, ε_1, ε_2, ε_3, ε_4, ε_5), and two residual error terms (ζ_1, ζ_2). As such, there is evidence suggesting that each proposed model is overidentified with 25 degrees of freedom, as calculated by the number of data points minus the number of unknown parameters (Byrne, 1998).

3.10.3. Principle of Discomfirmability

A critical issue in model specification and evaluation is the degree of disconfirmability – or the degree to which it is possible for the "model to be inconsistent with observed data" (MacCallum, 1995, pg 29). The disconfirmability of a model is directly related to its degrees of freedom, such that the number of effective parameters must be less than the number of measured variables variances/covariances resulting in positive degrees of freedom. Models that are logically specified with a high number of parameters relative to the degrees of freedom are likely to demonstrate good data model fit and are thus more difficult to disconfirm. In contrast, models that have a relatively low number of parameters relative to the degrees of freedom tend to be highly disconfirmable. Under conditions of high disconfirmability, a researcher has more confidence in concluding that a model is a true representation of the data when good data-model fit is found. Although there is no hard and fast rule for determining the degree of disconfirmability, the proposed models in this study have 18 degrees of freedom with a total of 36 parameters that provides a reasonable level of disconfirmability. To further understand the disconfirmability of the proposed models, the root mean square error of approximation (RMSEA) was used as a measure of data model fit, which essentially is a data-model fit measure that takes into account the lack of fit relative to the existing degrees of freedom (MacCallum, 1995).

3.10.4. Respecification and Model Modification

Model respecification or modification is one of the most controversial issues related to structural equation modeling – in part because SEM is primarily a confirmatory analysis and post hoc model respecification is essentially exploratory in nature resulting in an increase risk for capitalizing on chance variations in the data (Kelloway, 1998). Two empirical tests are available

to aid in model respecification – the Lagrange Multiplier (LM) and Wald test. The LM answers the question as to whether adding parameters would improve model fit, while the Wald indicates which parameters would improve model fit if deleted (Tabachnick & Fidell, 1996). For the proposed study, strategies to improve model fit were considered if the modifications are considered to be theoretically sound, as suggested by Kelloway (1998). In the event that post hoc modifications are conducted to improve either the measurement or structural model, such modifications will be clearly identified with an appropriate discussion of the rationale for the modifications.

3.10.5. Problems in Estimation

An important assumption underlying the majority of SEM estimation procedures is that the sample covariance matrix must be positive definite or nonsingular, meaning that the determinant of the matrix is non-zero (Schumacker & Lomax, 1996). If the covariance matrix is not positive definite (or singular) than the inverse of the covariance matrix is nonexistent, which results in an inability to generate valid statistics related to the covariance matrix. Typically a non-positive definite covariance matrix is a problem associated with either pairwise deletion of missing data or a linear dependency among variables. For this analysis, procedures were incorporated to deal with missing data appropriately. In addition, collinearity diagnoses were conducted for the purpose of identifying linear dependency among variables. Variables that are found to contribute to collinearity were removed from the SEM analysis.

Estimation procedures in SEM involve an iterative process that typically ends when necessary statistical criteria have been attained. Nonconvergence (or early termination of the iterative process) occurs under conditions of a poorly specified model or faulty start values. SEM output generally warns of nonconvergence problems (Hoyle, 1995). For this study, strategies to

deal with nonconvergence problems will include modifying start values and/or improving model specification.

3.11. SAMPLE SIZE CONSIDERATIONS

Structural equation modeling is considered a large sample statistical technique, with the estimation procedures and model fit tests based on the assumption of large samples (Kelloway, 1998). “Large” is somewhat of an ambiguous term. However, Bentler and Chou (1987) suggested that power is most likely adequate when there is a ratio of ten subjects per estimated parameter regardless of distributional assumptions. Under normal and elliptical theory, ratios of 5:1 (sample size to number of free parameters) may be acceptable when latent variables have many indicators and related factor loadings are large. SEM models proposed in this study have no more than 20 parameters to be estimated with a total sample of 243.

Power considerations for path analysis are similar to those for SEM. The two path models for this study have 5 parameters to be estimated for the male and female groups. Given that this sample is comprised of 167 men and 76 women, the sample size for each group is sufficient to conduct multi-group path analysis.

4. RESULTS

4.1. DESCRIPTIVE RESULTS FOR MANIFEST VARIABLES

Independent sample t-tests, using SPSS 12.0 [SPSS Inc, Chicago, Ill.], were conducted in order to compare means between women and men for the observed variables. The results of a preliminary analysis examining gender differences among the variables of interest are reported in Table 5. Results indicate that women and men differed in levels of depressive symptoms and anxiety at statistically significant levels. In general, women reported higher levels of depressive symptoms and anxiety as compared to men in this sample. Women also reported lower levels of mastery than men. No gender differences were found for the social support variables, sense of personal control, and control of symptoms. Means, standard deviations, and zero-order Pearson correlations for the observed variables are presented in Table 6 for the entire sample and in Table 7 for males and females separately.

4.2. RESEARCH HYPOTHESES

4.2.1. Measurement Model for Social Support, Control, and Depressive Symptoms

The measurement model examined for this hypothesis includes three latent variables: social support, control, and depressive symptoms. The constructs social support and control are measured by three manifest variables, while the construct depressive symptoms is measured by only two manifest variables. The resulting estimated parameters for the measurement model are

presented in Figure 8. Turning to the parameter estimates as a start for understanding data-model fit, the results show the signs of the parameter estimates are all consistent with expectations and previous empirical evidence. The majority of the standardized residuals are well below 2.00, which is an acceptable cutoff level for evaluating the "largeness" of standard errors. However, six standard residuals are above 2.00 (see Table 8), indicating some model misspecification among these manifest variables. Given the number of residuals over 2.00 it is reasonable to consider model modifications to improve model-fit. However, given that the factor analysis procedure is confirmatory in nature, and the modifications are not considered to be theoretically sound, model modification to capture the relationship between these paths was not conducted.

Table 5 Gender Differences for Manifest Variables

Variable	Men (N=166) M (SD)	Women (N=76) M (SD)	t^a
Social Support			
1. ISEL Appraisal Subscale	22.13 (6.15)	23.51 (5.87)	1.64
2. ISEL Tangible Subscale	23.41 (3.88)	23.26 (3.90)	-0.27
3. ISEL Self-Esteem Subscale	20.67 (4.56)	20.67 (5.24)	0.04
4. ISEL Belonging Subscale	22.86 (5.19)	23.39 (5.66)	0.73
5. ISEL Total Score	89.07 (16.89)	90.84 (18.14)	0.74
Control			
1. Mastery Scale	3.69 (0.77)	3.46 (0.86)	-2.05*
2. CSIB Sense of Control Subscale	23.13 (3.55)	22.54 (3.83)	-1.18
3. Control of Symptoms Scale	6.00 (2.40)	5.41 (2.59)	-1.71
Depression			
3. POMS Depression Subscale	3.07 (3.57)	4.43 (4.45)	2.56*
4. SCL-90-R Depression Subscale	0.78 (0.63)	1.12 (0.89)	3.40**
Anxiety			
3. POMS Anxiety Subscale	3.96 (3.58)	4.96 (4.14)	1.92
4. SCL-90-R Anxiety Subscale	0.44 (0.49)	0.76 (0.80)	3.92**

[a] Mean differences tested via two-tailed independent sample t test at the 0.05 level with equal variances assumed. * $p < 0.05$. ** $p < 0.01$

Table 6 Means, Standard Deviations, and Zero-Order Correlations Among the Manifest Variables

Variable	Mean (SD)	1	2	3	4	5	6	7	8	9	10	11	12
1. POMS Anxiety	4.27 (3.79)	—											
2. POMS Depression	3.50 (3.91)	.73*	—										
3. SCL-90-R Anxiety	0.54 (0.62)	.74*	.64*	—									
4. SCL-90-R Depression	0.89 (0.73)	.71*	.82*	.80*	—								
5. Mastery Scale	3.61 (0.80)	-.47*	-.51*	-.53*	-.62*	—							
6. CSIB Sense of Control Subscale	22.94 (3.64)	-.44*	-.55*	-.46*	-.57*	.69*	—						
7. Control of Symptoms Scale	5.81 (2.47)	-.31*	-.36*	-.36*	-.44*	.39*	.42*	—					
8. ISEL Appraisal	22.57 (6.09)	-.20*	-.28*	-.23*	-.32*	.48*	.39*	.26*	—				
9. ISEL Tangible	23.36 (3.88)	-.28*	-.35*	-.28*	-.40*	.48*	.41*	.35*	.57*	—			
10. ISEL Self-Esteem	20.67 (4.78)	-.39*	-.51*	-.38*	-.54*	.61*	.56*	.36*	.59*	.53*	—		
11. ISEL Belonging	23.02 (5.33)	-.28*	-.44*	-.28*	-.44*	.55*	.49*	.34*	.77*	.68*	.71*	—	
12. ISEL Total	89.62 (17.27)	-.32*	-.46*	-.34*	-.47*	.61*	.53*	.37*	.80*	.78*	.82*	.93*	—

* Significant at the 0.01 level (2-tailed).

Table 7 Zero-Order Correlations for Manifest Variables by Gender

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. POMS Anxiety	—											
2. SCL-90-R Anxiety	M: .76* F: .74*	—										
3. POMS Depression	M: .69* F: .78*	M: .59* F: .69*	—									
4. SCL-90-R Depression	M: .68* F: .75*	M: .73* F: .86*	M: .80* F: .84*	—								
5. Mastery Scale	M: -.39* F: .-.58*	M: -.43* F: -.66*	M: -.44* F: -.60*	M: -.55* F: -.70*	—							
6. CSIB Sense of Control Subscale	M: -.45* F: -.41*	M: -.45* F: -.50*	M: -.56* F: -.52*	M: -.59* F: -.55*	M: .67* F: -72*	—						
7. Control of Symptoms Scale	M: -.34* F: -.24*	M: -.41* F: -.28*	M: -.40* F: -.27*	M: -.50* F: -.34*	M: .41* F: .34*	M: .40* F: .45*	—					
8. ISEL Appraisal	M: -.09* F: -.45*	M: -.19* F: -.39*	M: -.19* F: -.51*	M: -.20* F: -.49*	M: .45* F: .61*	M: .26* F: .64*	M: .27* F: .28*	—				
9. ISEL Tangible	M: -.21* F: -.41*	M: -.29* F: -.32*	M: -.32* F: -.41*	M: -.42* F: -.40*	M: .47* F: .51*	M: .35* F: .51*	M: .36* F: .32*	M: .53* F: .67*	—			
10. ISEL Self-Esteem	M: -.35* F: -.46*	M: -.36* F: -.53*	M: -.52* F: -.53*	M: -.52* F: -.59*	M: .55* F: .71*	M: .50* F: .70*	M: .37* F: .34*	M: .56* F: .67*	M: .53* F: .55*	—		
11. ISEL Belonging	M: -.21* F: -.43*	M: -.23* F: -.57*	M. -.39* F: -.57*	M: -.42* F: -.53*	M: .53* F: .61*	M:.42* F: .63*	M: .34* F: .36*	M: .73* F: .85*	M: .69* F: .66*	M: .74* F: .66*	—	
12. ISEL Total	M: -.24* F: -.50*	M: -.30* F: -59*	M: -.40* F: -.59*	M: -.47* F: -.58*	M: 58* F: .71*	M: .45* F: .71*	M: .39* F: .37*	M: .86* F: .93*	M: .78* F: .80*	M: .82* F: .83*	M: .93* F: .92*	—

* Significant at the 0.01 level (2-tailed).

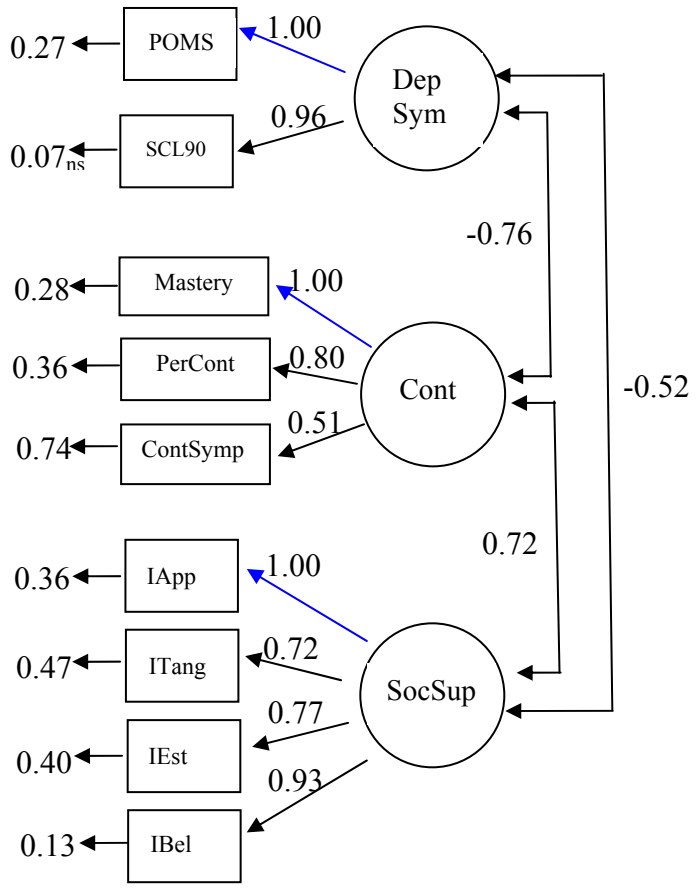

Figure 8 Confirmatory Factor Analysis for Social Support, Control, and Depressive Symptoms Measurement Model.

Observed variables are as follows. IAPP=ISEL Appraisal Subscale; ITANG=ISEL Tangible Subscale; IEST=ISEL Self-Esteem Subscale; IBEL=ISEL Belonging Subscale; Mastery=Mastery Scale; PerCont=Coping with Serious Illness Battery (CSIB) Sense of Control Subscale; ContSymp=Control of Symptoms Scale; POMS=Profile of Moods States (POMS) Depression Subscale; and SCL90=Symptom Checklist 90-R (SCL-90-R) Depression Subscale. Latent variables are as follows. SocSup=social support; Cont=control; and DepSym=depressive symptoms. All paths are significant at $p < 0.05$ except for paths marked ns.

Table 8 Standardized Residuals for Social Support, Control, and Depressive Symptoms Measurement Model

Manifest Variables	Standardized Residual
ISEEST and POMS-D	-4.52
ISEEST and SCL90R-D	-4.55
ISEAPP and SCL90R-D	2.90
ISEEST and Mastery	4.37
ISEEST and PerCont	3.41
ISEBEL and ISEAPP	3.59

Fit indices for the measurement model are presented in Table 9. The fit statistics for this model suggest an adequate fit to the data. The χ^2 with 24 degrees of freedom is statistically significant ($\chi^2 = 68.91$, p =0.00) which is indicative of poor data-model fit. However, this is to be somewhat expected given a sample size of 242. The χ^2/df ratio is above 2, which is somewhat larger than desired for adequate data-model fit. With the exception of the Adjusted Goodness of Fit Index (AGFI = 0.88) all fit indices are above 0.90, indicating good data-model fit. The Standardized Root-Mean-Square Residual (RMSR) also provides evidence for good data-model fit with a value of 0.05. In contrast, the Root-Mean-Square Error of Approximation (RMSEA) is above the desired 0.05 level, and marginally above the more liberal cut-off of 0.08, indicating questionable data-model fit. Taken together, the evidence indicates an adequate data-model fit for the measurement model.

Table 9 Model 1 Measurement Model Fit Statistics

Statistic	Measurement Model
χ^2	68.91 (df=24)*
χ^2/df	2.87
Goodness of Fit Index	0.94
Adjusted Goodness of Fit Index	0.88
Normed Fit Index	0.97
Non-Normed Fit Index	0.97
Comparative Fit Index	0.98
Standardized Root-Mean-Square Residual	0.05
Root-Mean-Square Error of Approximation	0.08
90% CI for RMSEA	[0.06; 0.11]

*Significant at the 0.01 level.

4.2.2. Structural Model for Testing Mediation Effect of Control on Depressive Symptoms

The first specific aim for this study was to propose and test a theoretical mediating model of the relationship between social support, personal control, and depression among individuals with congestive heart failure. The original model (Model 1) proposed and tested in this study is a

partially mediating model identical to the theoretical model presented in Figure 5 of the previous chapter. The research hypotheses tested in this analysis are as follows:

> H1.1 Social support has an inverse direct effect on depressive symptomatology in individuals diagnosed with congestive heart failure.
> H1.2 Social support has a positive effect on personal control through a direct path.
> H1.3 Personal control has an inverse direct effect on depressive symptomatology in individuals diagnosed with congestive heart failure.
> H1.4 Social support has an inverse effect on depression through an indirect path - specifically, social support has a positive effect on personal control, which in turn reduces depressive symptoms in individuals diagnosed with congestive heart failure.

The estimated parameters of the model are given in Figure 9. As a starting point, it is worth noting that all indicators load on the respective latent variable as expected. Before presenting key empirical findings it is also worth discussing the latent constructs themselves. Social support and control are scaled such that higher scores are associated with perceived higher levels of each of these constructs. In contrast, the construct, depressive symptoms, is scaled so that higher values indicate more depressive symptoms. With this in mind, the estimated path coefficients between the latent variables load in the expected direction, except for the direct path between social support and depressive symptoms.

The path coefficient from social support to depressive symptoms ($\beta = 0.05$) was positive and not statistically significant. Therefore, H1.1 purporting a direct effect of social support on depressive symptoms was not supported. In contrast, hypotheses H1.2, H1.3, and H1.4 were supported in that social support has a direct positive effect on control, control has an inverse direct effect on depressive symptoms, and social support has an indirect effect on depressive symptoms through control. The total structural effects of social support on depressive symptoms, the product of the indirect effects plus the direct effect ($[-0.79 \times 0.72] + 0.05$), is -0.52. This suggests that 52% of the variance in depressive symptoms is explained by the total effects of social support, and that these effects serve to reduce symptoms of depression.

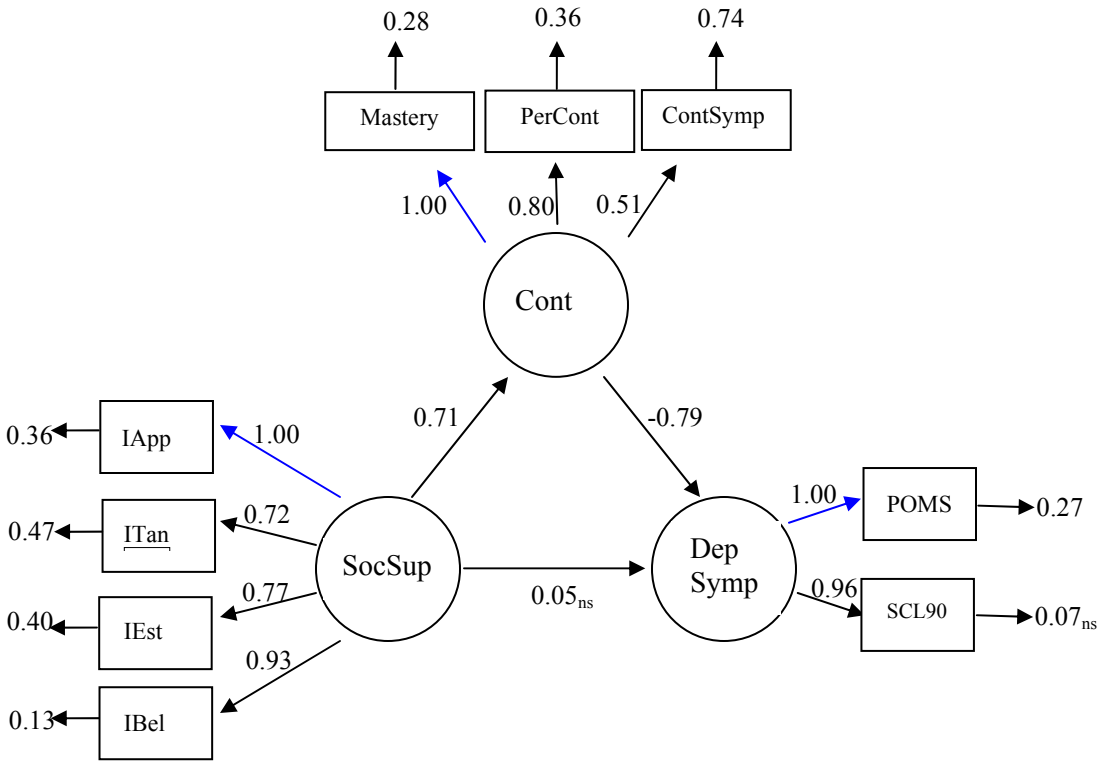

Figure 9 Model 1: Hypothesized Partially Mediating Model

All paths are significant at $p<0.05$, except the two designated as *ns*.

The fit statistics for this model suggest an adequate fit to the data (see Table 10). As with the measurement model, the χ^2 with 24 degrees of freedom is statistically significant ($\chi^2 = 68.91$, p =0.00) and the $^{\chi 2}$/df ratio is above 2. All fit indices are above 0.90, except for the Adjusted CFI. The Standardized RMSR is 0.05 and RMSEA is above 0.08. Standardized residuals are identical to that reported for the measurement model. Overall, the results indicate an adequate data-model fit for the original model.

Table 10 Model 1 and 1A Fit Statistics

	Model	
Statistic	**Model 1**	**Model 1A**
X^2	68.91 (df=24)*	69.63 (df=25)*
X^2/df	2.87	2.79
Goodness of Fit Index	0.94	0.94
Adjusted Goodness of Fit Index	0.89	0.90
Normed Fit Index	0.97	0.97
Non-Normed Fit Index	0.97	0.97
Comparative Fit Index	0.98	0.98
Standardized Root-Mean-Square Residual	0.05	0.05
Root-Mean-Square Error of Approximation	0.09	0.09
90% CI for RMSEA	[0.06; 0.11]	[0.06; 0.11]

*Significant at the 0.01 level.

A second analysis was conducted in which a fully mediating model (Model 1A) was tested in order to see if this improved data-model fit. This analysis was motivated by the outcome showing that social support did not have a direct effect on depressive symptoms in Model 1. In this analysis the path between social support and depressive symptoms was constrained to zero (see Figure 10). Fit indices for the Model 1A are likewise reported in Table 10. The results of this model show a similar fit to the data as compared to Model 1. The standardized RMSR and the RMSEA are identical. Standardized residuals above 2.00 involve the same pairs of manifest variables and are similar in magnitude as those in Model 1 (see Table 11). However, the fit indices are marginally better for Model 1A. For example, all the fit indices now reach or are above the 0.90 cut-off indicating good model-fit. Given that the two models are nested, a chi-squared difference test was conducted to compare the two models more formally. The results, $\chi_1^2 = 0.72$, $p<0.05$ indicate that there is no difference in the fit between the two models. This result suggests that the direct path between social support and depressive symptoms did not make a significant contribution to the model and thus H1.1 was not supported.

For this model, the total structural effects of social support on depressive symptoms is simply the product of the indirect effects, -0.76 ×0.71 = -0.54. This suggests that 54% of the variance in depressive symptoms is explained by the total effects of social support.

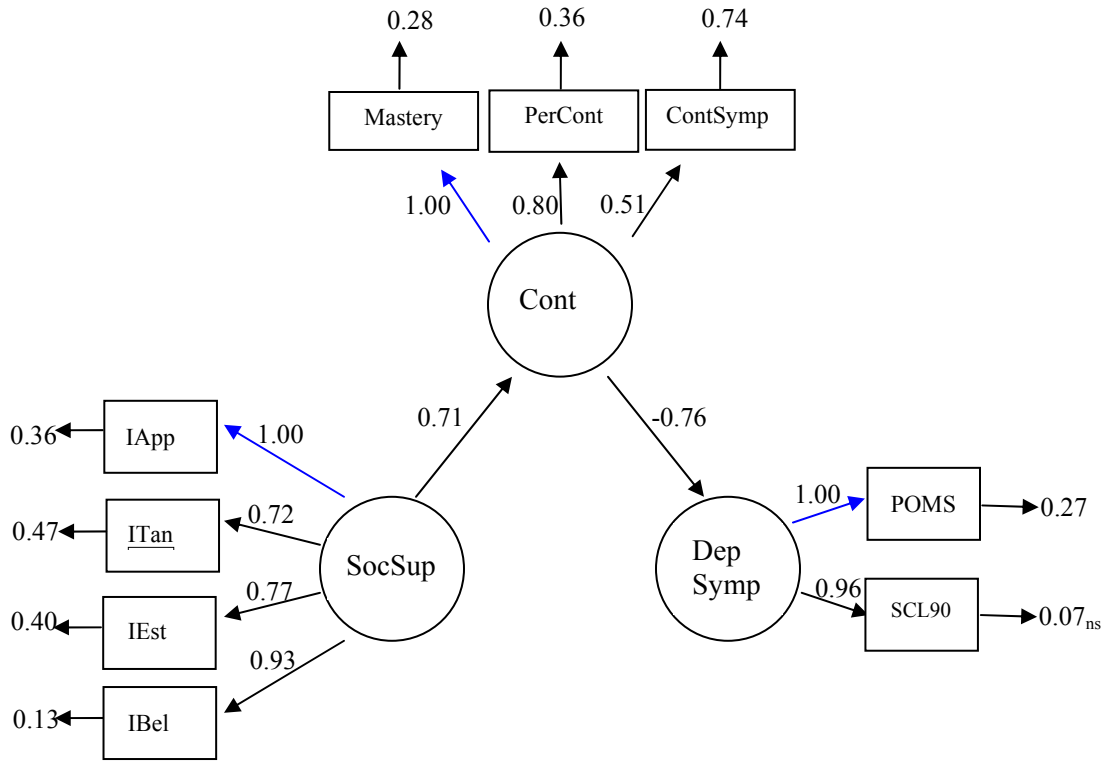

Figure 10 Model IA: Fully Mediating Model

All paths are significant at $p < 0.05$, except the one designated *ns*.

Table 11 Model 1A Standardized Residuals

Manifest Variables	Standardized Residual
ISEEST and POMS-D	-3.87
ISEEST and SCL90R-D	-3.59
ISEAPP and SCL90R-D	2.65
ISEEST and Mastery	4.38
ISEEST and PerCont	3.41
ISEBEL and ISEAPP	3.64

4.2.3. Measurement Model for Testing Mediation Effect of Control on Anxiety

The second set of analyses, which are related to specific aim 2, follows the same analytic structure as those for specific aim 1. For these analyses the outcome latent variable is anxiety rather than depressive symptoms. The beginning point of these analyses is the measurement model presented in Figure 11. All parameter estimates are consistent with expectations regarding the signs of the loadings. Five standard residuals are above 2.00 (see Table 12), indicating some model misspecification among these manifest variables. As with the previous measurement model, model modification was not conducted.

Fit indices for the Model 2 measurement model are presented in Table 13. The fit statistics for this model suggest good data-model fit. While the χ^2 with 24 degrees of freedom is statistically significant (χ^2 = 47.67, p =0.00), the $^{\chi 2}$/df ratio is less than 2, which takes into consideration χ^2 in relation to the sample size. All fit indices are above 0.90, indicating good data-model fit. Both the standardized RMSR and the RMSEA provide evidence for good data-model fit.

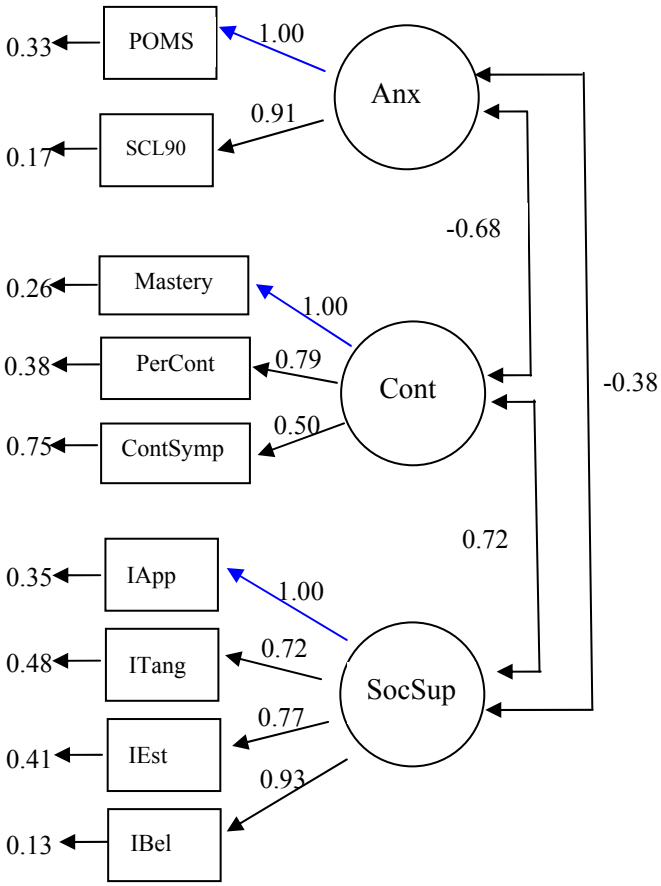

Figure 11 Confirmatory Factor Analysis for Social Support, Control, and Anxiety Measurement Model

Observed variables are as follows. IAPP=ISEL Appraisal Subscale; ITANG=ISEL Tangible Subscale; IEST=ISEL Self-Esteem Subscale; IBEL=ISEL Belonging Subscale; Mastery=Mastery Scale; PerCont=Coping with Serious Illness Battery (CSIB) Sense of Control Subscale; ContSymp=Control of Symptoms Scale; POMS=Profile of Moods States (POMS) Anxiety Subscale; and SCL90=Symptom Checklist 90-R (SCL-90-R) Anxiety Subscale. Latent variables are as follows. SocSup=social support; Cont=control; and DepSym=depressive symptoms. All paths are significant at $p < 0.05$.

Table 12 Standardized Residuals for Social Support, Control, and Anxiety Measurement Model

Manifest Variables	Standardized Residual
ISEEST and POMS-D	-3.57
ISEEST and SCL90R-D	-3.26
ISEEST and Mastery	4.30
ISEEST and PerCont	3.59
ISEBEL and ISEAPP	3.19

Table 13 Model 2 Measurement Model Fit Statistics

Statistic	Measurement Model
χ^2	47.67 (df=24)*
χ^2/df	1.99
Goodness of Fit Index	0.96
Adjusted Goodness of Fit Index	0.92
Normed Fit Index	0.98
Non-Normed Fit Index	0.98
Comparative Fit Index	0.99
Standardized Root-Mean-Square Residual	0.05
Root-Mean-Square Error of Approximation	0.06
90% CI for RMSEA	[0.04, 0.09]

*Significant at the 0.01 level.

4.2.4. Structural Model for Testing Mediation Effect of Control on Anxiety

The second specific aim was to propose and test a theoretical mediating model of the relationship between social support, personal control, and anxiety among individuals with congestive heart failure. Model 2 is identical to the theoretical model presented in Figure 7 (previous chapter), and represents a partially mediating model. The research hypotheses tested in this analysis are as follows:

> H2.1 Social support has an inverse direct effect on anxiety in individuals diagnosed with congestive heart failure.
> H2.2 Social support has a positive effect on personal control through a direct path.
> H2.3 Personal control has an inverse direct effect on anxiety in individuals diagnosed with congestive heart failure.
> H2.4 Social support has an indirect inverse effect on depression through personal control, which directly reduces symptoms of anxiety in individuals diagnosed with congestive heart failure.

Estimated model parameters are presented in Figure 12. As with the model examining depressive symptoms, social support and control are scaled such that higher scores are associated with perceived higher levels of each of these constructs and anxiety is scaled so that higher values indicate more anxiety related symptoms.

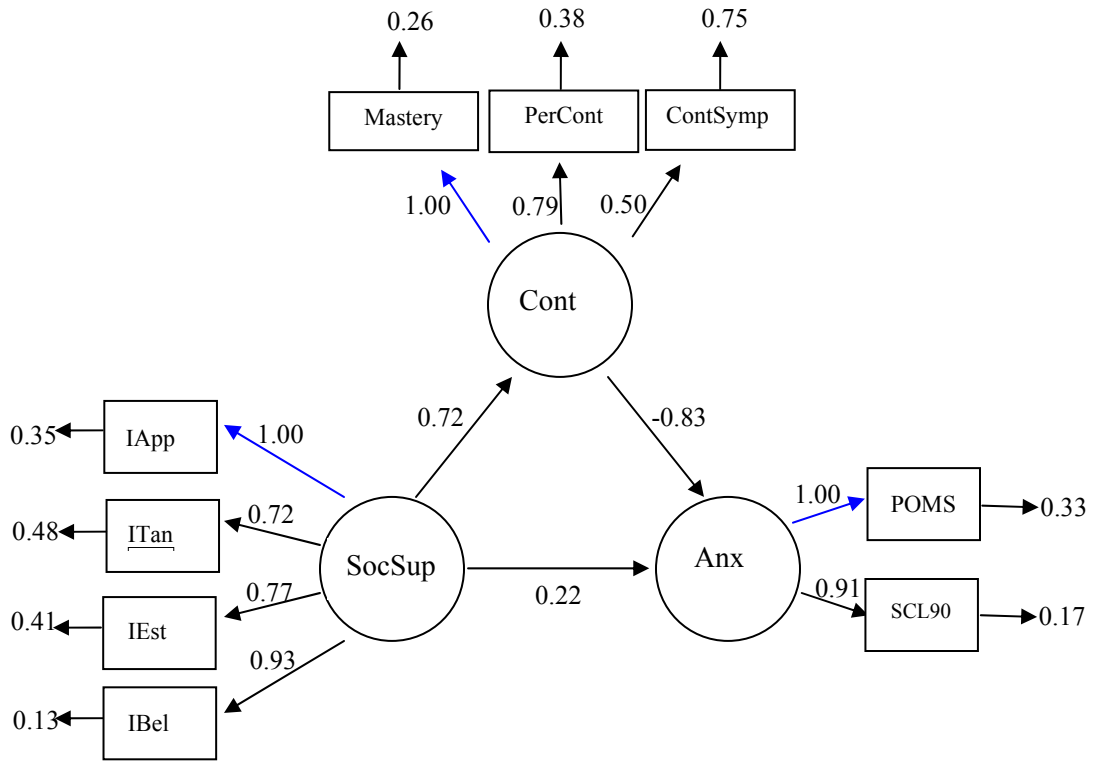

Figure 12 Model 2: Hypothesized Partially Mediating Model

All paths significant at $p < 0.05$.

Path loadings are consistent with theoretical expectations except the direct path from social support to anxiety, which is positive and statistically significant. This finding indicates that H2.1 can be rejected. The remaining hypotheses (H2.2, H2.3, and H2.4) are supported. The total structural effects of social support on anxiety are [-0.83 ×0.72] + 0.22 = -0.38, indicating that 38% of the variance in anxiety is explained by the total effects of social support, and that these effects serve to reduce anxiety.

The fit statistics are presented in Table 14. With exception to the χ^2 result, all of the other fit indices suggest a good data-model fit. Standardized residuals are identical to that reported for the measurement model. A visual examination of the standardized residuals Q-plot showed that the x's hovered around the diagonal, indicating that the underlying assumption of normality was

met. In addition, bivariate scatterplots of the social support and anxiety measures are all roughly oval-shaped, thus a linear relationship between social support and anxiety is supported.

A second analysis was conducted in which seven identified outliers were removed from the analysis. The rationale for conducting this additional analysis was to provide a test of sensitivity to the finding that social support had a direct, but positive effect on anxiety, which was in contrast to the hypothesized direction of the relationship. The results are consistent with those reported for Model 2, such that all structural coefficients load in the same direction and with similar magnitude. Likewise, the fit statistics are similar and provide evidence for good data-model fit. The results of this analysis provides further support for the conclusion to reject H2.1.

Table 14 Model 2 Fit Statistics

Statistic	Measurement Model
χ^2	47.67 (df=24)*
χ^2/df	1.99
Goodness of Fit Index	0.96
Adjusted Goodness of Fit Index	0.92
Normed Fit Index	0.98
Non-Normed Fit Index	0.98
Comparative Fit Index	0.99
Standardized Root-Mean-Square Residual	0.05
Root-Mean-Square Error of Approximation	0.06
90% CI for RMSEA	[0.04, 0.09]

*Significant at the 0.01 level.

4.2.5. Gender Differences in the Models Involving Depressive Symptoms

The third aim of this study is to explore potential gender differences in a theoretical mediating model of the relationship between social support, control, and depressive symptoms among individuals with congestive heart failure. Specifically, the goal is to examine H3.0 – the relationships will be different for males and females.

4.2.5.1. Tests for Invariance across Gender for Social Support, Mastery, and Depressive Symptoms

Figure 13 presents baseline path analyses separately for men and women for social support, mastery, and depressive symptoms. For both men and women the estimated parameters are consistent with theoretical expectations. For clarity, the following notation is used to specify paths. Two gamma paths, GA(1,1) and GA(2,1) refer, respectively, to the direct effects of social support on the mediating control variable and the outcome variable. The beta path, BE(2,1) specifies the direct path between the mediating control variable and the outcome variable. The GA(1,1) path from social support to mastery is positive and statistically significant for both genders, the GA(2,1) path from social support to depressive symptoms is negative and statistically significant for men only, and the BE(2,1) path from mastery to depressive symptoms is negative and statistically significant for men and women.

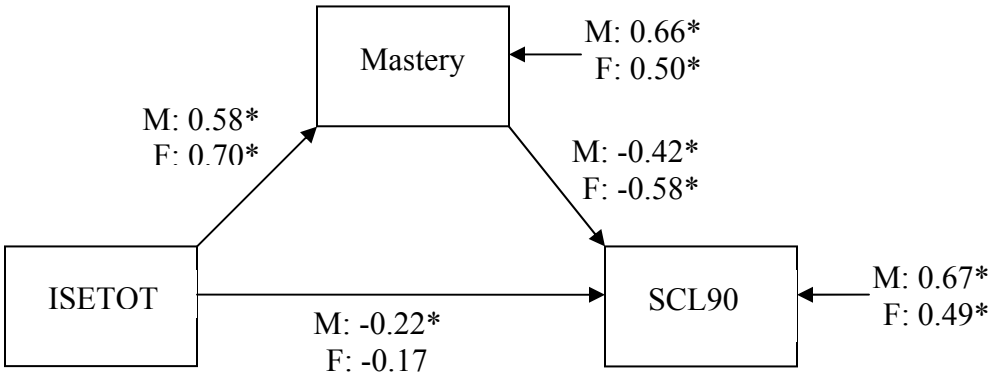

Figure 13 Path Analyses for Social Support, Mastery, and Depressive Symptoms for Men (Above) and Women (Below)

Manifest variables are as follows. ISETOT = ISEL Total Score; Mastery = Mastery Scale; and SCL90 = Symptom Checklist 90-R (SCL-90-R) Depression Subscale.
* Significant at the 0.05 level.

The results of the tests of gender differences, using simultaneous path analysis, are presented in Table 15. Tests of gender differences in parameters are conducted on all possible combinations of the paths. Consistent with model fitting, the $\Delta\chi^2$ between the various models provides evidence concerning the hypothesized equality constraints, with a significant $\Delta\chi^2$ indicates non-invariance (paths are not the same across genders). For this analysis, the hypothesis that all paths are the same (i.e., all paths are invariant) is rejected. Similarly, each evaluation involving the path BE(2,1) indicates a trend toward gender differences, with p-values of 0.06 or lower. None of the invariance tests involving the gamma parameters alone (paths GA(1,1) and GA(2,1)) are significant.

Table 15 Summary of LISREL Tests for Invariance across Gender for Social Support, Mastery, and Depressive Symptoms

Model	χ^2 (p-value)	df	CFI	Model Comparison	$\Delta\chi^2$	Δdf
(1) Baseline multigroup model	0.00 (1.00)	0	___	___	___	___
(2) All paths Invariant	9.24 (0.03)	3	0.98	2 vs. 1	9.24*	3
(3) Path GA(1,1) Invariant	1.95 (0.16)	1	0.99	3 vs. 1	1.95	1
(4) Path GA(2,1) Invariant	0.00 (0.97)	1	1.00	4 vs. 1	0.00	1
(5) Path BE(2,1) Invariant	3.64 (0.06)	1	0.99	5 vs. 1	3.64	1
(6) Paths GA(1,1) GA(2,1) invariant	1.95 (0.38)	2	1.00	6 vs. 1	1.95	2
(7) Paths GA(1,1) BE(2,1) invariant	5.54 (0.06)	2	0.98	7 vs. 1	5.54	2
(8) Paths GA(2,1) BE(2,1) invariant	6.77 (0.03)	2	0.98	8 vs. 1	6.77*	2

* Significant at the 0.05 level.

A supplementary means of evaluating gender differences entailed estimating a mediator model (Barron & Kenny, 1986) with gender interactions using regression. Estimates of the coefficients are reported in Table 16. Estimates are again consistent with theoretical

expectations: In equation (1) the independent variable, social support, is found to have a positive effect on the mediator, mastery; in equation (2) social support is found to have a negative effect on the dependent variable, depressive symptoms; and in equation (3) mastery is found to have a negative effect on depressive symptoms. A fully mediating model is supported as social support is found to have statistically significant effects on the outcome variables in equations (1) and (2), but not in equation (3).

Turning attention to estimated gender effects, equation (3) parallels the results of the path analysis previously reported. Specifically, the estimated regression coefficient -0.603 indicates a negative effect of mastery on depressive symptoms for women, while the statistically significant positive interaction term mastery × gender of 0.258 indicates that for men the effect of mastery on depressive symptoms is smaller in absolute value (i.e., the effect for men is -0.603 + 0.258 = -0.345).

Table 16 Regression Results: Mediation Model of Social Support, Mastery, Depressive Symptoms by Gender

	Equation (1) Outcome Variable is Mastery	**Equation (2) Outcome Variable is Depressive Symptoms**	**Equation (3) Outcome Variable is Depressive Symptoms**
Predictor Variables			
Social Support	0.033**	-0.028**	-0.008
	(0.004)	(0.004)	(0.005)
Mastery	-	-	-0.603**
			(0.105)
Gender (1=male)	0.890*	-1.387**	-1.191**
	(0.450)	(0.443)	(0.411)
Social Support × Gender	-0.007	0.011*	0.000
	(0.005)	(0.005)	(0.006)
Mastery × Gender	-	-	0.258*
			(0.125)
R^2	0.41	0.31	0.45

Standard errors are given in parentheses below the estimated regression coefficients.
*Significant at the 0.05 level. **Significant at the 0.01 level.

The explorations in both the path analysis and the regression analysis indicate gender differences in the relationships between mastery and depressive symptoms. Both sets of results indicate that social support is important in improving mastery for men and women alike. Mastery in turn is associated with reduced depressive symptoms, but the strength of this relationship is significantly stronger for women than men.

4.2.5.2. Tests for Invariance across Gender for Social Support, Personal Control, and Depressive Symptoms

Figure 14 presents baseline path analyses, separately for men and women, for social support, personal control, and depressive symptoms. The estimated parameters are consistent with theoretical expectations for both men and women and are statistically significant at the 0.05 level.

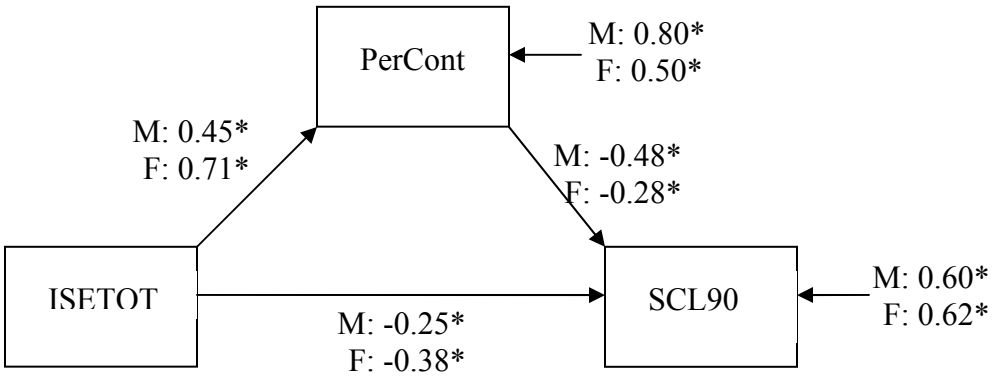

Figure 14 Path Analyses for Social Support, Personal Control, and Depressive Symptoms for Men (Above) and Women (Below)

Manifest variables are as follows. ISETOT = ISEL Total Score; PerCont = Coping with Serious Illness Battery (CSIB) Sense of Control Subscale; and SCL90 = Symptom Checklist 90 (SCL-90-R) Depression Subscale.
* Significant at the 0.05 level.

The results of the simultaneous path analysis for gender differences are presented in Table 17. For this analysis, the hypothesis that all paths are invariant (i.e., the same) is rejected. Models involving the path between social support and personal control (GA(1,1)) are statistically significant for gender differences. None of the invariance tests involving exclusively BE(2,1) and/or GA(2,1) are significant.

Estimates of the regression coefficients for social support, personal control, and depressive symptoms are reported in Table 18. The signs of the estimated coefficients are consistent with theoretical expectations. The key gender difference found in the path analysis is supported by the regression analysis. In particular, the effect of social support × gender on personal control estimated in equation (1) is negative and statistically significant, indicating that the role of social support is smaller for men than women in improving personal control. The estimated regression coefficient 0.150 indicates a positive effect of social support on personal control, while the statistically significant interaction term social × gender of -0.055 indicates that for men the effect of social support on personal control is smaller in absolute value (i.e., the effect for men is -0.055 + 0.150 = 0.095). The interactions social support × gender and personal control × gender were not statistically significant in equation (3).

The explorations in both the path analysis and the regression analysis indicate that gender differences exist in the relationship between social support and personal control. Both sets of results indicate that the separate roles of social support and personal control on depressive symptoms are equally important for men and women.

Table 17 Summary of LISREL Tests for Invariance Across Gender for Social Support, Personal Control, and Depressive Symptoms

Model	χ^2 (p-value)	Df	CFI	Model Comparison	$\Delta\chi^2$	Δdf
(1) Baseline multigroup model	0.00 (1.00)	0	___	___	___	___
(2) All paths invariant	7.66 (0.05)	3	0.98	2 vs. 1	7.66*	3
(3) Path GA(1,1) Invariant	5.81 (0.02)	1	0.98	3 vs. 1	5.81*	1
(4) Path GA(2,1) Invariant	1.87 (0.17)	1	0.99	4 vs. 1	1.87	1
(5) Path BE(2,1) Invariant	0.34 (0.55)	1	1.00	5 vs. 1	0.34	1
(6) Paths GA(1,1) GA(2,1) invariant	7.37 (0.03)	2	0.97	6 vs. 1	7.37*	2
(7) Paths GA(1,1) BE(2,1) invariant	6.14 (0.05)	2	0.98	7 vs. 1	6.14*	2
(8) Paths GA(2,1) BE(2,1) invariant	2.08 (0.35)	2	0.99	8 vs. 1	2.08	2

* Significant at the 0.05 level.

Table 18 Regression Results: Mediation Model of Social Support, Personal Control, Depressive Symptoms by Gender

	Equation (1) Outcome Variable is Personal Control	**Equation (2) Outcome Variable is Depressive Symptoms**	**Equation (3) Outcome Variable is Depressive Symptoms**
Predictor Variables			
Social Support	0.150** (0.019)	-0.028** (0.004)	-0.019** (0.005)
Personal Control	-	-	-0.065** (0.024)
Gender (1=male)	5.790** (2.203)	-1.387** (0.443)	-0.731 (0.505)
Social Support × Gender	-0.055* (0.024)	0.011* (0.005)	0.009 (0.006)
Personal Control × Gender	-	-	-0.019 (0.028)
R^2	0.31	0.31	0.42

Standard errors are given in parentheses below the estimated regression coefficients.
*Significant at the 0.05 level. **Significant at the 0.01 level.

4.2.5.3. Tests for Invariance across Gender for Social Support, Symptom Control, and Depressive Symptoms

Figure 15 presents baseline path analyses separately for men and women for social support, control of symptoms, and depressive symptoms. The estimated parameters are consistent with theoretical expectations for both men and women. All paths are statistically significant at the 0.05 level except the BE(2,1) for women (i.e. the path between symptom control and depressive symptoms).

The results of the simultaneous path analysis for gender differences are presented in Table 19. For this analysis, the hypothesis that all paths are the same (Model 2) is supported. Even so, models involving the path GA(2,1) (i.e., the path between social support and depressive symptoms) are statistically significant for gender differences. None of the invariance tests involving solely GA(1,1) and/or BE(2,1), which corresponds to the paths between social support and symptom control and the path between social support and depressive symptoms respectively, were statistically significant.

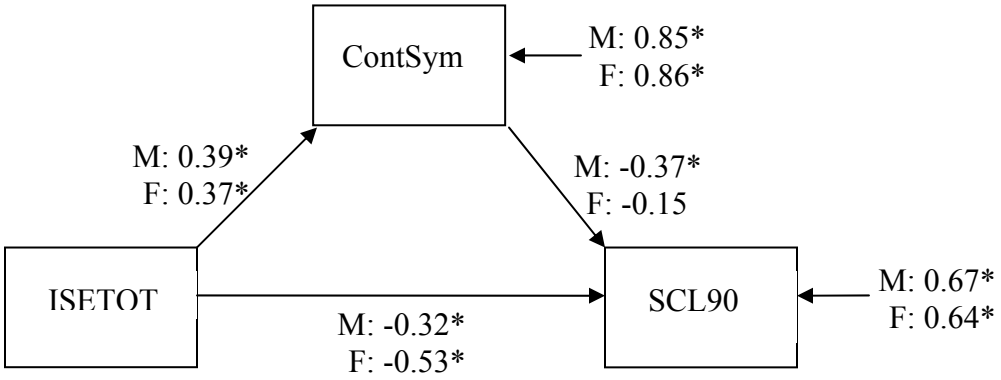

Figure 15 Path Analyses for Social Support, Symptom Control, and Depressive Symptoms for Men (Above) and Women (Below)

Manifest variables are as follows. ISETOT = ISEL Total Score; ContSym = Control of Symptoms Scale; and SCL90 = Symptom Checklist 90-R (SCL-90-R) Depression Subscale.
* Significant at the 0.05 level.

Table 19 Summary of LISREL Tests for Invariance across Gender for Social Support, Symptom Control, and Depressive Symptoms

Model	χ^2 (p-value)	df	CFI	Model Comparison	$\Delta\chi^2$	Δdf
(1) Baseline multigroup model	0.00 (1.00)	0	___	___	___	___
(2) All paths Invariant	6.11 (0.11)	3	0.98	2 vs. 1	6.11	3
(3) Path GA(1,1) Invariant	0.02 (0.90)	1	1.00	3 vs. 1	0.02	1
(4) Path GA(2,1) Invariant	6.00 (0.01)	1	0.96	4 vs. 1	6.00**	1
(5) Path BE(2,1) Invariant	1.36 (0.24)	1	0.99	5 vs. 1	1.36	1
(6) Paths GA(1,1) GA(2,1) invariant	6.05 (0.05)	2	0.97	6 vs. 1	6.05*	2
(7) Paths GA(1,1) BE(2,1) invariant	1.38 (0.50)	2	1.00	7 vs. 1	1.38	2
(8) Paths GA(2,1) BE(2,1) invariant	6.06 (0.05)	2	0.97	8 vs. 1	6.06*	2

* Significant at the 0.05 level. **Significant at the 0.01 level.

Estimates of the regression coefficients for social support, symptom control, and depressive symptoms are reported in Table 20. The signs of the estimated coefficients are consistent with theoretical expectations. Moreover, in equation (3) the interaction term social support×gender is positive and statistically significant. For women the direct effect of social support on depressive symptoms is -0.026 while the effect for men, -0.026 + 0.014 = -0.012, is smaller in absolute value.

The explorations in both the path analysis and the regression analysis indicate that gender differences exist in the direct relationship between social support and depressive symptoms. Both sets of results indicate that social support has a larger direct role in reducing depressive symptoms for women than for men.

Table 20 Regression Results: Mediation Model of Social Support, Symptom Control, and Depressive Symptoms by Gender

	Equation (1) Outcome Variable is Symptom Control	Equation (2) Outcome Variable is Depressive Symptoms	Equation (3) Outcome Variable is Depressive Symptoms
Predictor Variables			
Social Support	0.053**	-0.028**	-0.026**
	(0.015)	(0.004)	(0.004)
Symptom Control	-	-	-0.051
			(0.028)
Gender (1=male)	0.467	-1.387**	-1.313*
	(1.654)	(0.443)	(0.424)
Social Support ×	-0.002	0.011*	0.014**
Gender	(0.018)	(0.005)	(0.005)
Symptom Control ×	-	-	-0.045
Gender			(0.035)
R^2	0.16	0.31	0.38

Standard errors are given in parentheses below the estimated regression coefficients.
*Significant at the 0.05 level. **Significant at the 0.01 level.

4.2.5.4. Tests for Invariance across Gender for Social Support, Mastery, and Anxiety

The final aim of this study is to explore gender differences in a theoretical mediating model of the relationship between social support, control, and anxiety among individuals with congestive heart failure. Specifically, the goal is to examine H4.0 specifying that the relationships will be different for males and females. This hypothesis was examined following the same path analysis and regression methodology reported in the previous sections. Figure 16 presents baseline path analyses, separately for men and women, for social support, mastery, and anxiety. The estimated parameters are consistent with theoretical expectations for both men and women. All paths are statistically significant at the 0.05 level for men. For women, GA(2,1), the path between social support and anxiety is the only path that is not significant.

The results of the simultaneous path analysis for gender differences are presented in Table 21. The hypothesis that all paths are invariant is rejected at the standard 0.05 level. As was found in the depressive symptoms path models reported above, each analysis involving the path BE(2,1) (i.e., the path between mastery and anxiety) is statistically significant, indicating that this relationship differs for men and women. None of the invariance tests involving exclusively the paths GA(1,1) and/or GA(2,1)) are significant. These paths refer to the relationship between social support and mastery and social support and anxiety, respectively.

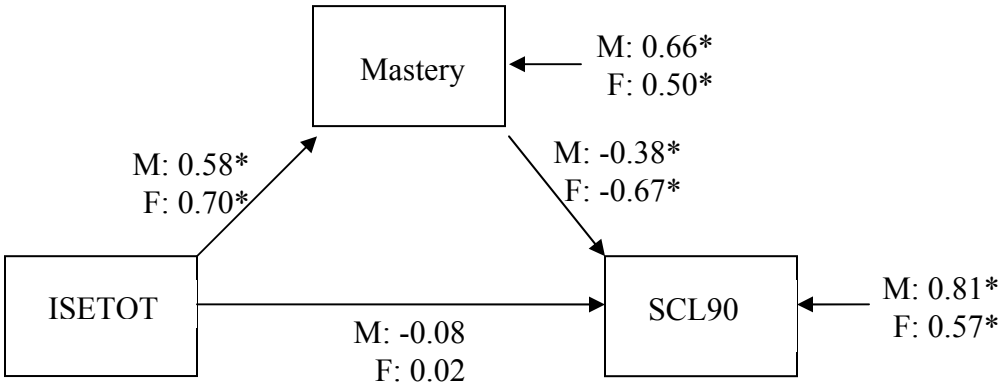

Figure 16 Path Analyses for Social Support, Mastery, and Anxiety for Men (Above) and Women (Below)

Manifest variables are as follows. ISETOT = ISEL Total Score; Mastery = Mastery Scale; and SCL90 = Symptom Checklist 90-R (SCL-90-R) Anxiety Subscale.
*Significant at the 0.05 level.

Estimates of the regression coefficients are reported in Table 22. Estimates are again consistent with theoretical expectations: In equation (1) the independent variable, social support, is found to have a positive effect on the mediator, mastery; in equation (2) social support is found to have a negative effect on the dependent variable, anxiety; and in equation (3) mastery is found to have a negative effect on anxiety. The results of this analysis are consistent with a fully

mediating model in which social support is found to have statistically significant effects on the outcome variables in equations (1) and (2), but not in equation (3).

Table 21 Summary of LISREL Tests for Invariance across Gender for Social Support, Mastery, and Anxiety

Model	χ^2 (p-value)	Df	CFI	Model Comparison	$\Delta\chi^2$	Δdf
(1) Baseline multigroup model	0.00 (1.00)	0	___	___	___	___
(2) All paths Invariant	14.85 (0.00)	3	0.94	2 vs. 1	14.85**	3
(3) Path GA(1,1) Invariant	1.95 (0.16)	1	0.99	3 vs. 1	1.95	1
(4) Path GA(2,1) Invariant	0.32 (0.57)	1	1.00	4 vs. 1	0.32	1
(5) Path BE(2,1) Invariant	8.44 (0.00)	1	0.96	5 vs. 1	8.44**	1
(6) Paths GA(1,1) GA(2,1) invariant	2.24 (0.32)	2	0.99	6 vs. 1	2.24	2
(7) Paths GA(1,1) BE(2,2) invariant	10.26 (0.00)	2	0.95	7 vs. 1	10.26**	2
(8) Paths GA(2,1) BE(2,1) invariant	11.98 (0.00)	2	0.94	8 vs. 1	11.98**	2

**Significant at the 0.01 level.

The estimated gender effects as reported in equation (3) are consistent with the results of the path analysis previously reported. The estimated regression coefficient -0.624 for mastery in equation (3) indicates a negative effect of mastery on anxiety for women at a statistically significant level. The statistically significant positive interaction term mastery×gender of 0.383 indicates that for men the effect of mastery on anxiety is smaller in absolute value (i.e., the effect for men is -0.624 + 0.383 = -0.241).

The results of both the path analysis and the regression analysis indicate gender differences in the relationships between mastery and anxiety. The results indicate that social support plays a role in improving mastery for men and women alike. Mastery in turn is

associated with reduced levels of anxiety, but the strength of this relationship is significantly stronger for women than men.

Table 22 Regression Results: Mediation Model of Social Support, Mastery, and Anxiety by Gender

	Equation (1) Outcome Variable is Mastery	Equation (2) Outcome Variable is Anxiety	Equation (3) Outcome Variable is Anxiety
Predictor Variables			
Social Support	0.033**	-0.020**	-0.001
	(0.004)	(0.004)	(0.004)
Mastery	-	-	-0.624**
			(0.095)
Gender (1=male)	0.890	-1.345**	-1.295**
	(0.450)	(0.403)	(0.374)
Social Support ×	-0.007	0.011*	0.003
Gender	(0.005)	(0.004)	(0.005)
Mastery ×Gender	-	-	0.383**
			(0.114)
R^2	0.41	0.20	0.36

Standard errors are given in parentheses below the estimated regression coefficients.
*Significant at the 0.05 level. **Significant at the 0.01 level.

4.2.5.5. Tests for Invariance across Gender for Social Support, Personal Control, and Anxiety

Figure 17 presents baseline path analyses, separately for men and women, for social support, personal control, and anxiety. The estimated parameters are consistent with theoretical expectations for both men and women. The path GA(2,1) representing the relationship between social support and anxiety is not statistically significant for either men or women. All other paths are statistically significant at the 0.05 level for both men and women.

The results of the simultaneous path analysis for gender differences are presented in Table 23. For this analysis, the hypothesis that all paths are the same (Model 2) is rejected. Models involving GA(1,1), the path between social support and personal control, are statistically significant for invariance across genders. None of the invariance tests involving exclusively

BE(2,1) and/or GA(2,1) are significant, which represents the paths from personal control to anxiety and social support to anxiety respectively, indicating that for men and women these paths are equal.

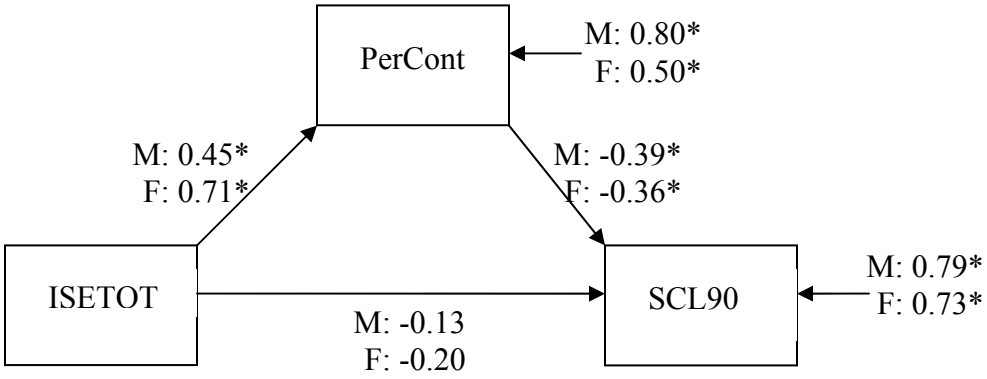

Figure 17 Path Analyses for Social Support, Personal Control, and Anxiety for Men (Above) and Women (Below)

Manifest variables are as follows. ISETOT = ISEL Total Score; PerCont = Coping with Serious Illness Battery (CSIB) Sense of Control Subscale; and SCL90 = Symptom Checklist 90-R (SCL-90-R) Anxiety Subscale.
*Significant at 0.05 level.

Estimates of the regression coefficients for social support, personal control, and anxiety are presented in Table 24. The signs of the estimated coefficients are consistent with theoretical expectations. The results of the regression analyses support the primary finding of the path analysis indicating a gender difference in the path between social support and personal control. As seen in equation (1), the effect of social support×gender on personal control is negative and statistically significant, indicating that the role of social support is smaller for men than women in improving personal control. The estimated regression coefficient 0.150 indicates a positive effect of social support on personal control, while the statistically significant interaction term social×gender for men is -0.055. This finding indicates that for men the effect of social support

on personal control is smaller in absolute value (i.e., the effect for men is -0.055 + 0.150 = 0.095). Neither the interactions social support×gender nor personal control×gender were statistically significant in equation (3).

The results from this exploratory set of analyses indicate that gender differences exist in the relationship between social support and personal control. However, the unique effect of social support and personal control on the outcome anxiety are equally important for men and women.

Table 23 Summary of LISREL Tests for Invariance across Gender for Social Support, Personal Control, and Anxiety

Model	χ^2 (p-value)	Df	CFI	Model Comparison	$\Delta\chi^2$	Δdf
(1) Baseline multigroup model	0.00 (1.00)	0	___	___	___	___
(2) All paths Invariant	9.24 (0.03)	3	0.96	2 vs. 1	9.24*	3
(3) Path GA (1,1) Invariant	5.81 (0.02)	1	0.97	3 vs. 1	5.81*	1
(4) Path GA (2,1) Invariant	0.57 (0.45)	1	1.00	4 vs. 1	0.57	1
(5) Path BE (2,1) Invariant	0.44 (0.51)	1	1.00	5 vs. 1	0.44	1
(6) Paths GA(1,1) GA(2,1) invariant	6.28 (0.04)	2	0.97	6 vs. 1	6.28*	2
(7) Paths GA(1,1) BE(2,1) invariant	6.23 (0.04)	2	0.97	7 vs. 1	6.23*	2
(8) Paths GA(2,1) BE(2,1) invariant	3.07 (0.21)	2	0.99	8 vs. 1	3.07	2

*Significant at the 0.05 level.

Table 24 Regression Results: Mediation Model of Social Support, Personal Control, and Anxiety by Gender

	Equation (1) Outcome Variable is Personal Control	Equation (2) Outcome Variable is Anxiety	Equation (3) Outcome Variable is Anxiety
Predictor Variables			
Social Support	0.150**	-0.020**	-0.009
	(0.019)	(0.004)	(0.005)
Personal Control	-	-	-0.075**
			(0.023)
Gender (1=male)	5.790**	-1.345**	-1.218*
	(2.203)	(0.403)	(0.473)
Social Support ×	-0.055*	0.011*	0.005
Gender	(0.024)	(0.004)	(0.005)
Personal Control ×	-	-	-0.021
Gender			(0.026)
R^2	0.31	0.20	0.29

Standard errors are given in parentheses below the estimated regression coefficients.
*Significant at the 0.05 level. **Significant at the 0.01 level.

4.2.5.6. Tests for Invariance across Gender for Social Support, Symptom Control, and Anxiety

Figure 18 reports baseline path analyses for men and women separately for social support, control of symptoms, and anxiety. The estimated parameters are consistent with theoretical expectations for both men and women. Each path is statistically significant at the 0.05 level, except for the BE(2,1) path for women. The path BE(2,1) represents the relationship between social support and anxiety.

The results of the simultaneous path analysis for gender differences are presented in Table 25. For this analysis, the hypothesis that all paths are invariant (Model 2) is supported. However, the path GA(2,1) (i.e., the path between social support and anxiety) is statistically significant for gender differences, but only when examined on its own. None of the invariance

tests involving GA(1,1) and/or BE(2,1) were statistically significant. GA(1,1) corresponds to the path between social support and symptom control and BE(2,1) represents the path between social support and anxiety.

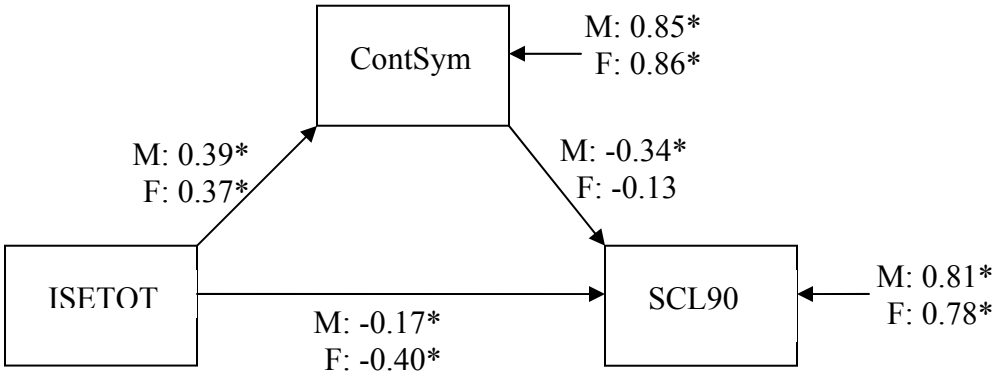

Figure 18 Path Analyses for Social Support, Symptom Control, and Anxiety for Men (Above) and Women (Below)

Manifest variables are as follows. ISETOT = ISEL Total Score; ContSym = Control of Symptoms Scale; and SCL90 = Symptom Checklist 90-R (SCL-90-R) Anxiety Subscale.
*Significant at 0.05 level.

Table 26 reports the regression coefficient estimates for social support, symptom control, and anxiety. The signs of the estimated coefficients are consistent with theoretical expectations. In equation (3), the interaction term social support×gender is positive and statistically significant. For women the estimated direct effect of social support on anxiety is -0.018 while for men, the estimate is -0.018 + 0.013 = -0.05, which is substantially smaller in absolute value.

The explorations in both the path analysis and the regression analysis indicate that gender differences exist in the direct relationship between social support and anxiety. In both cases, social support is found to have a greater effect in reducing anxiety for women than for men.

Table 25 Summary of LISREL Tests for Invariance across Gender for Social Support, Symptom Control, and Anxiety

Model	χ^2 (p-value)	df	CFI	Model Comparison	$\Delta\chi^2$	Δdf
(1) Baseline multigroup model	0.00 (1.00)	0	___	___	___	___
(2) All paths Invariant	5.51 (0.14)	3	0.98	2 vs. 1	5.51	3
(3) Path GA (1,1) Invariant	0.02 (0.90)	1	1.00	3 vs. 1	0.02	1
(4) Path GA (2,1) Invariant	5.44 (0.02)	1	0.95	4 vs.1	5.44*	1
(5) Path BE (2,1) Invariant	0.58 (0.44)	1	0.99	5 vs. 1	0.58	1
(6) Paths GA(1,1) GA(2,1) invariant	5.49 (0.06)	2	0.96	6 vs. 1	5.49	2
(7) Paths GA(1,1) BE(2,1) invariant	0.60 (0.74)	2	1.00	7 vs. 1	0.60	2
(8) Paths GA(2,1) BE(2,1) invariant	5.46 (0.07)	2	0.96	8 vs. 1	5.46	2

*Significant at the 0.05 level.

Table 26 Regression Results: Mediation Model of Social Support, Symptom Control, and Depressive Symptoms by Gender

	Equation (1) Outcome Variable is Symptom Control	**Equation (2) Outcome Variable is Anxiety**	**Equation (3) Outcome Variable is Anxiety**
Predictor Variables			
Social Support	0.053** (0.015)	-0.020** (0.004)	-0.018** (0.004)
Symptom Control	-	-	-0.041 (0.026)
Gender (1=male)	0.467 (1.654)	-1.345** (0.403)	-1.295** (0.392)
Social Support × Gender	-0.002 (0.018)	0.011* (0.004)	0.013** (0.005)
Symptom Control × Gender	-	-	-0.028 (0.032)
R^2	0.16	0.20	0.25

Standard errors are given in parentheses below the estimated regression coefficients.
*Significant at the 0.05 level. **Significant at the 0.01 level.

4.2.5.7. Observations About the Explorations for Gender Differences

The preceding sections, 4.2.5.1 through 4.2.5.6, present six statistical exercises exploring gender differences in the relationships between social support, control (as measured by three different scales), and key outcomes—depressive symptoms or anxiety. Each of the six exercises uses both path analysis and regression. It is important to recognize that the sample size for women is quite small, so that all results must be viewed with caution. Given this caveat, though, a remarkably consistent pattern emerges.

First, estimated coefficients in each of the estimated models are consistent with general theoretical expectations. Second, statistically significant differences between results for men and women emerge in each of the explorations. Moreover, in each of the six cases the path analysis and regression approaches yield the same interpretation. Third, the patterns of gender differences are precisely the same for each of the outcome variables, depressive symptoms and anxiety. Fourth, and perhaps most importantly, in each case the finding is that the role of social support is greater for women than for men in reducing psychological distress, as indicated by either depressive symptoms or anxiety. The way in which social support influences the outcome varies, however, depending on which manifest control scale is used in implementing the analysis. Table 27 summarizes these results for the path analyses (which, in turn, are entirely consistent with the regression findings).

Table 27 Summary of Key Gender Differences in Path Analyses

Outcome is Depressive Symptoms			
		Control Scale	
	Mastery	Personal Control	Symptom Control
BE(2,1), the direct effect of control in reducing depressive symptoms	greater for women		
GA(2,1), the direct effect of social support in improving control		greater for women	
GA(1,1), the direct effect of social support in reducing depressive symptoms			greater for women*

Outcome is Anxiety			
		Control Scale	
	Mastery	Personal Control	Symptom Control
BE(2,1), the direct effect of control in reducing anxiety	greater for women		
GA(2,1), the direct effect of social support in improving control		greater for women	
GA(1,1), the direct effect of social support in reducing anxiety			greater for women*

*For these models, all-path invariance was not rejected at the 0.05 level, but GA(1,1) invariance is rejected.

5. DISCUSSION

Heart failure, a serious and prevalent chronic disease, places a large psychosocial burden on patients and their families. The research presented in this dissertation attempts to increase understanding about the complex relationship between social support and control and two key psychological outcomes in individuals coping with heart failure: depressive symptoms and anxiety. A theoretically defensible model is constructed that relates these constructs, and modern tools of inferential statistics are used to identify the importance of the proposed relationships. This chapter discusses the key findings, limitations of this work, and implications for future research.

The first major contribution of the study was developing and testing two structural models derived from Holahon, Moos, and Bonin's (1999) integrative stress and coping theoretical framework. The objective was to test for a mediating influence of control between social support and depression (Model 1) and anxiety (Model 2) in a sample of individuals diagnosed with heart failure. The second contribution is an empirical exploration of gender differences in the estimated relationships.

5.1. Results from the Structural Equations Models

The key findings from the structural equations models are easily summarized: Among heart failure patients, social support plays a key role in reducing psychological distress—depressive

symptoms and anxiety. The mechanism whereby social support effectively reduces psychological distress is entirely through patients' perceived control.

The first piece of evidence in support of this summary follows directly from Model 1, which uses depressive symptoms as the psychological functioning outcome. In this structural equation model, the path from social support to control is, as expected, positive and highly significant; social support is found to improve patients' sense of control. In turn, the path from control to depressive symptoms is negative and highly significant; personal control is found to reduce depressive symptoms. The direct path from social support to depressive symptoms, however, is estimated to be very close to zero. Thus, the beneficial effects of social support on depressive symptoms operate entirely through control.

The second piece of evidence in support of the summary follows from Model 2, which uses anxiety as the indicator of psychological functioning. Here again, the path from social support to control is estimated to be positive, while the path from control to anxiety is negative; social support improves control, which in turn reduces levels of psychological distress in heart failure patients. Although the *total* effect of social support on anxiety, including both indirect and direct effects, is negative, as expected, it is remarkable to note that in this model the direct path from social support to anxiety is found to be *positive* and statistically significant. Social support—when understood in the context of a direct relationship—may actually act to increase anxiety among individuals with heart failure. This particular finding is potentially important in that it demonstrates that the direct effect of social support on anxiety is quite different when compared to the direct effect on depressive symptoms. This finding suggests that clinical interventions designed to incorporate social support in reducing psychological distress may need to be different when the targeted outcome is anxiety versus depressive symptoms. In sum, the

findings suggest that while social support does serve to reduce anxiety, the beneficial role for social support operates entirely through the mediating factor, perceived control.

These findings provide empirical support for the integrative stress and coping framework as proposed by Holahan, Moos, and Bonin (1999) and suggest that a resources coping framework is applicable to understanding the context of psychological adjustment, specifically as it relates to depressive symptoms and anxiety, among individuals with a chronic disorder such as heart failure. Although this study did not attempt to examine all the components of the theoretical framework, simplifying the structural models to focus exclusively on the relationship between social support and control for separate models of depressive symptoms and anxiety allows us to tease out the contextual complexities associated with the adaptation process and to better understand the idiosyncratic nature of the relationships identified within this framework. Furthermore, by focusing on three aspects of control (mastery, personal control, and control of symptoms) as a form of coping resources, these results serve to clarify the mediational role of control in the adaptation process by building on the work of Holahan and colleagues (1991; 1995; 1997) in which coping is conceptualized more broadly in terms of cognitive and behavioral strategies. In addition to providing support to our theoretical understanding of adaptation, these results have potentially important implications for clinical practice. Because each path tested in the two structural models identifies a dynamic process that is considered amenable to change, the results provide a starting point for clinicians to design and test interventions aimed at enhancing adaptation to chronic illness.

In general terms, the key result of the study—that social support is important in reducing depressive symptoms and anxiety among heart patients—contributes to a large and growing literature that emphasizes the importance of social support for healthy psychological functioning

among the medically ill (Taylor & Aspinwall, 1993; Yoel & Hirsh, 1999). The second, more surprising finding is that the positive role of social support on depressive symptoms and anxiety operates entirely through control. This result is inconsistent with previous research establishing a direct positive link between social support and depressive symptoms among individuals with medical illnesses (Brummett et al., 1998; Frasure-Smith et al., 2000; Krishnan, 1998; Symister & Friend, 2003). However, much of this research tests this relationship within the context of a direct association and not within the context of a third mediating variable. Nonetheless, this finding is consistent with Holahan, Moos, Holahan, and Brennan (1997), who found that the beneficial effects of social support on depressive symptoms operated entirely through the mediator, approach coping in a sample of individuals diagnosed with cardiac illness. The result provides support, moreover, for Walker's (2002) contention that it is

> necessary to take account of individual desire for control and the controllability of the situation. Placing social support in the context of a theory of control enables researchers to understand how different types of support are likely to impact on health outcomes. Failure to do so will continue to lead to conflicting and confusing research findings. (p. 6)

The finding that social support has the direct effect of *increasing* anxiety should not be considered entirely unexpected. Scholars have noted that "social support" is a broad multidimensional concept with both positive and negative attributes. Rook (1984; 1992) points to problematic facets of social interactions; social ties and relationships, by acts of omission or commission, can create psychological distress. Coyne and Downey (1991) make a related point—that it may not be appropriate to conceptualize social support as a unipolar construct. Social networks can simultaneously provide support and invoke stress (Fiore, Becker, & Coppel, 1983). Flor, Kerns, and Turk (1987) found that among individuals coping with chronic pain,

overly solicitous family members were associated with increased levels of impairment. Furthermore, social transactions that are thought to be supportive may lead to decreased feelings of self-efficacy and threats to one's self-esteem (Holahan, Moos, & Bonin, 1999). Holahan and colleagues (1999) put forward that "excessive support in the context of chronic illness may be perceived as suggesting that the recipient is incompetent to manage for him- or herself" (p. 51). It is interesting to note that "excessive support" does not imply that the support is positive or negative in nature, rather simply in excess. Although this premise was not tested directly in this analysis, it is quite possible that the individuals in this study, when given higher amounts of social support felt a sense of incompetency and/or inadequacy in coping with their heart failure. This relationship emerged even in the context of social support being positive in nature as conceptualized by Cohen, Mermelstein, Kamarck, and Hoberman (1985) – higher levels on the ISEL are indicative of higher levels of social support. While one should always be cautious in making strict causal arguments based on empirical findings, the interpretation of the paths estimated in Model 2 is that, absent the improvement in control, higher levels of social support lead to increased anxiety. Future research studies are needed to confirm and clarify this finding.

5.2. Explorations of Gender Differences

The second broad objective in the research reported in this dissertation is to provide evidence concerning gender differences in psychological functioning among the medically ill. An extensive series of empirical analyses, using path analysis and mediation models based on linear regression, lead to the following conclusions: among heart failure patients there are indeed statistically significant gender differences in the relationship between social support, control, and psychological functioning, with consistent evidence suggesting that the role of social support, in

the context of an integrative stress coping model, is greater for women than for men in reducing psychological distress, as indicated by either depressive symptoms or anxiety.

Empirical evidence that leads to this conclusion can be constructed using results from either the path analyses or the regression analyses (as the two methods produced results that are consistent with one another). In these analyses, indicators for "control" are three independent manifest variables: mastery, personal control, and control over symptoms. Empirical evidence demonstrates that for both depressive symptoms and anxiety the following statistically significant relationships hold: (1) the effect of mastery in reducing psychological distress is greater for women than for men, (2) the effect of social support in increasing personal control is greater for women than for men, and (3) the direct effect of social support in reducing psychological distress is greater for women than for men when "symptom control" is the proposed mediator.

Given the substantial literature on gender differences in the levels of depression among patients with cardiac disease (Forrester, Lipsey, Teitelbaum, DePaulo, & Andrezejewski, 1992; Frasure-Smith, Lesperance, & Talijic, 1993), and given evidence that gender differences in rates of depression and anxiety are associated with psychosocial determinants (Hammen, 1999; Radloff, 1985), one would expect to find gender differences in the role of social support and control. Little is known, however, about the nature of these gender differences, especially in the context of individuals with heart failure. The present research represents an initial attempt at such an exploration.

The first finding, that the effect of mastery in reducing psychological distress is greater for women than men, appears consistent with Tamres, Janicki, and Helgeson's (2002) observation that when individuals face personal health problems as a stressor, the effect of active

coping behaviors has a stronger effect for women than for men in reducing psychological distress. This is relevant to finding (1) because in the theoretical framework guiding this study control is conceptualized as a component of active coping strategies (Pearlin & Scholler, 1978).

The second finding indicates that having a sense of personal control—the perceived ability to solve problems—also matters differently for men and women. For men and women alike, an increased sense of personal control is found to reduce depressive symptoms and anxiety. However, women and men differ in the extent to which social support affects personal control; the role of social support in improving personal control is greater for women than for men.

The third finding suggests that there are no gender differences in the mediating role of symptom control—daily pain, fatigue, shortness of breath, and difficulties sleeping—on depressive symptoms and anxiety. This dimension of control matters equally for men and women, so in a model that has symptom control as the lone mediating variable, gender differences in the effect of social support on psychological outcomes are found to operate solely through the direct path.

The possibility that separate facets of control might operate differently in health-related contexts is underscored by Folkman (1984). It is interesting that among the variables measuring control, gender differences were observed for the effect of mastery on both depressive symptoms and anxiety, while no such differences were observed for personal control and symptom control. As indicated by finding (1), mastery—a person's perceived control over events and one's life—appears more important than other dimensions of control in reducing psychological distress for women than men. This finding is not surprising given that heart failure appears to manifest differently for men and women (Jessup & Piña, 2004; Petrie, et al., 1999). For example, even in

light of the fact that women with advanced heart failure tend to have better survival rates than men (Ho, Anderson, Kannel, Grossman, & Levy, 1993; Adams, et al., 1999), women report more symptoms associated with heart failure (Jessup & Piña, 2004), less improvement in physical health status, and perceived lower levels of quality of life (Chin & Goldman, 1998) when compared to men with heart failure. In addition, the negative impact of depressive symptoms on functional status appears to be stronger for women than for men with heart disease (Mallki, et al., 2005). In light of these findings, it becomes imperative that women find effective strategies that enhance adaptation to heart failure, and that these strategies may differ for men and women when mastery is employed.

The broader finding, concerning the gender differences in effects of social support on personal control and psychological outcomes, is potentially important in light of recent work that highlights differences in the way women and men provide support to one another. For example, Neff and Karney (2005) find that in a study of husband-wife pairs, the level of positive support women provided was positively correlated with the severity of their husbands' problems, while the level of support men provided was *not* associated with the severity of their wives' problems.

It is worth noting that a possible explanation in the observed gender differences may result from potentially confounding variables that were not included in the path analyses. For example, gender differences were present for a number of demographic and medical variables, including racial/ethnic group, education, current occupation, type of cardiomyopathy, and number of medications. It is possible that gender differences in these variables partially explain the gender differences observed in this study. Future research aimed at understanding the demographic and medically related variables in the context of an integrative stress coping model are warranted to better explain gender differences in adaptation to heart failure.

5.3. LIMITATIONS

This secondary analysis is subject to several limitations. The first limitation concerns the cross-sectional nature of the data, limiting the ability to make causal inferences. While the hypotheses underlying model development may be causal in nature, structural models do not provide definitive proof for causal inferences (Kelloway, 1998). A closely associated consideration is the interpretation of the nature of the relationships specified in these models. For example, in this study social support and control were hypothesized to affect depressive symptoms and anxiety. A rather intriguing idea is the potential bidirectional relationship between these variables. For example, it is quite possible that depressive symptoms and anxiety may ultimately affect social support and control, but the directionality of the relationship cannot be verified within the context of this study. Well designed longitudinal studies are needed to make causal inferences about the exact nature of these relationships.

Second, the population studied was a sample of convenience and is limited to patients with heart failure who are followed up as outpatients in a heart failure clinic. Whether these results extend to patients who are suffering from more severe heart failure or other conditions of medical illness is uncertain. Related to this limitation is a concern that the sample is predominantly white men and women, limiting the generalizability of the findings to individuals of other races and ethnicity. In addition, the small ratio of women to men in the sample size prevented the use of full structural equation modeling to test for gender differences. As such, the findings from the exploratory gender difference analyses should be viewed with caution and serve as a starting point for future studies.

The final limitation concerns the use of a secondary analysis. The overall research design and research strategies (instrument selection, recruitment, and data collection) are not carried out within the context of the proposed theoretical framework, specific aims, and hypotheses. As such, there is a forcing of the research methodology to fit the overall context and aims of the study. There is also limited insight into the nuances of the study when conducting a secondary analysis.

5.4. FUTURE RESEARCH

The first key long-run goal of this study is to make a contribution, however modest, to the growing body of research that informs theoretical work designed to understand gender differences in psychological functioning among the medically ill. The research presented here provides support for existing theories that emphasize important roles of social support and control in improving psychological functioning among the chronically ill. The empirical explorations make a new contribution by highlighting potentially important gender differences in the dynamic relationship between social support, control and psychological distress. Further research is needed to further explore and confirm both of these findings.

Ideally, future research in this domain would be conducted in a longitudinal context, which would increase the researchers' ability to establish causal relationships. In pursuing this research it would be preferable to collect measures of depression and anxiety using refined diagnostic tools administered by clinicians, along with self-reports measuring symptomatology. Given the preliminary work reported here, future work should be designed to explicitly test for

gender invariance; it may well be that among heart failure patients there are many fundamentally important gender differences.

The second long-run objective of any applied research on psychosocial outcomes among chronic patients is to assist clinicians in developing effective strategies to improve the lives of patients and their families. The present study of social support and control among heart failure patients raises the possibility that strategies designed to improve social support can be useful in reducing heart failure patients' depressive symptoms and anxiety. The study indicates that to be effective such strategies would operate primarily (or even exclusively) through improving patients' sense of control. Finally, the study suggests that intervention programs might in some cases be fruitfully tailored to reflect the patient's gender. For example, a program that improves symptom control—the patient's ability to control daily pain, fatigue, shortness of breath, and lack of sleep—would likely be equally effective for men and women in reducing depressive symptoms and anxiety. A program that provides the patient with improved mastery—perceived control over one's life—might, instead, prove particularly effective for women in reducing such psychological distress.

Of course, extensive clinical work, guided by empirically informed theory, would be required to develop and test such programs. The present research is a small, but hopefully useful, step forward in the important effort to identify personal and social resources that are central to psychological functioning among individuals with heart failure.

6. APPENDIX

University of Pittsburgh
Institutional Review Board

Exempt and Expedited Reviews
Christopher M. Ryan, Ph.D., Vice Chair

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TO: Melissa Taylor

FROM: Christopher M. Ryan, Ph.D., Vice Chair

DATE: October 8, 2004

PROTOCOL: Social Support, Personal Control, and Psychological Functioning Among Individuals with Heart Failure

IRB Number: 0409111

The above-referenced protocol has been reviewed by the University of Pittsburgh Institutional Review Board. Based on the information provided in the IRB protocol, this project meets all the necessary criteria for an exemption, and is hereby designated as "exempt" under section 45 CFR 46.101(b)(4).

The regulations of the University of Pittsburgh IRB require that exempt protocols be re-reviewed every three years. If you wish to continue the research after that time, a new application must be submitted.

- If any modifications are made to this project, please submit an 'exempt modification' form to the IRB.
- Please advise the IRB when your project has been completed so that it may be officially terminated in the IRB database.
- This research study may be audited by the University of Pittsburgh Research Conduct and Compliance Office.

Approval Date: October 7, 2004
Renewal Date: October 7, 2007

CR:ky

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