

**University of Alberta**

Exploring social factors affecting health in cardiac rehabilitation patients

by

Shawn N. Fraser



A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment  
of the requirements for the degree of Doctor of Philosophy

Faculty of Physical Education and Recreation

Edmonton, Alberta  
Spring 2006



Library and  
Archives Canada

Bibliothèque et  
Archives Canada

Published Heritage  
Branch

Direction du  
Patrimoine de l'édition

395 Wellington Street  
Ottawa ON K1A 0N4  
Canada

395, rue Wellington  
Ottawa ON K1A 0N4  
Canada

*Your file* *Votre référence*

*ISBN: 0-494-13974-9*

*Our file* *Notre référence*

*ISBN: 0-494-13974-9*

#### NOTICE:

The author has granted a non-exclusive license allowing Library and Archives Canada to reproduce, publish, archive, preserve, conserve, communicate to the public by telecommunication or on the Internet, loan, distribute and sell theses worldwide, for commercial or non-commercial purposes, in microform, paper, electronic and/or any other formats.

The author retains copyright ownership and moral rights in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

#### AVIS:

L'auteur a accordé une licence non exclusive permettant à la Bibliothèque et Archives Canada de reproduire, publier, archiver, sauvegarder, conserver, transmettre au public par télécommunication ou par l'Internet, prêter, distribuer et vendre des thèses partout dans le monde, à des fins commerciales ou autres, sur support microforme, papier, électronique et/ou autres formats.

L'auteur conserve la propriété du droit d'auteur et des droits moraux qui protègent cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

---

In compliance with the Canadian Privacy Act some supporting forms may have been removed from this thesis.

Conformément à la loi canadienne sur la protection de la vie privée, quelques formulaires secondaires ont été enlevés de cette thèse.

While these forms may be included in the document page count, their removal does not represent any loss of content from the thesis.

Bien que ces formulaires aient inclus dans la pagination, il n'y aura aucun contenu manquant.

  
**Canada**

## Abstract

Two background studies and two dissertation studies that served as the basis of this dissertation project examined the role of social support in cardiac rehabilitation (CR). The first study examined the role of higher-level social factors (age, income) on social support and exercise tolerance of patients before and after CR. Results showed that select sociodemographic factors were related to social support, and both social support and select sociodemographic factors were related to exercise tolerance. Results highlighted a need to more carefully examine the relationship between sociodemographic factors and social support. The second study examined the impact of functional and exercise specific social support and stress on self-efficacy for overcoming barriers to CR participation. Results showed that functional social support was related to more exercise specific support and less stress. Higher stress and less exercise specific support was related to less confidence to overcome barriers to exercise in CR. Study 1 of the dissertation examined the sociodemographic determinants of structural and functional support. It was hypothesized that sociodemographic factors would be related to the nature of one's social network and that one's social network would influence the available functional support. Results showed that sociodemographic factors were related to social network size and diversity and were differently related to aspects of functional social support. Study 2 of the dissertation examined how social support and stress might impact on cardiovascular reactivity (CVR) among CR patients. Results showed that heart rate (HR), blood pressure, and mean arterial pressure all increased from resting to anticipation of exercise. Social support was related to less HR-reactivity in women and more in men. Anxiety was positively related to HR-reactivity and HR-reactivity was negatively related to self-

efficacy and self-rated health in women. In men, self-efficacy and self-rated health were positively related to HR-reactivity. Systolic blood pressure (SBP) reactivity had the lowest relationships with social support variables. Men and women with more self-efficacy had lower SBP-reactivity, and anxiety was positively related to SBP-reactivity in women. Results showed that social support was related to less stress and more self-efficacy suggesting a potential path from support to CVR responses.

## Acknowledgements

This dissertation was partially funded by a Doctoral Dissertation fellowship from the Social Sciences and Humanities Research Council of Canada. I am also grateful to the staff and the participants at the Northern Alberta Cardiac Rehabilitation Program who generously gave their time for the sake of this dissertation.

Personally, I am indebted to my parents, family, and friends and of course to Terra Murray. You are all responsible for keeping me grounded and for recognizing my efforts. Special recognition goes to my supervisor Dr. Wendy Rodgers, and my former supervisor Dr. Kevin Spink.

## Table of Contents

Section		Page	
1. Introduction	General introduction	1	
	Social support and health	6	
	Social support/networks and mortality	7	
	Social support/networks and morbidity	9	
	Explanations of how social support affects health	12	
	Theoretical perspectives on social support	13	
	Direction of relationship	17	
	Support interventions	17	
	A conceptual framework for studying social factors and health	19	
	Mechanisms	27	
	Cardiovascular reactivity	28	
	References	37	
	2. Study 1	Study 1 - Sociodemographic correlates of social networks and social support among people attending cardiac rehabilitation	63
		Method	70
		Participants	70
Measures		70	
Procedures		75	
Analyses		76	
Results		77	
Data screening		77	
Regression assumptions		78	
Descriptive results		82	
Regressions analyses predicting social networks		83	
Regression analyses predicting functional social support		84	
Discussion		86	
Social network characteristics		86	
Social support functions		87	
References	94		
3. Study 2	Study 2 – Social support and cardiovascular reactivity to anticipation of a treadmill test	108	
	Medication effects on CVR	112	
	Calcium channel blockers	113	
	Beta-blockers	114	
	ACE-inhibitors	118	
	Alpha-blockers	121	
	Medication comparison studies	123	
	Medication combination studies	127	
	Miscellaneous medications	130	
	Method	133	
Participants	134		

	Measures	134
	Procedures	142
	Analyses	144
	Results	145
	Data screening	145
	Analysis assumptions	147
	Sample characteristics	150
	Cardiovascular responses	150
	Correlates of cardiovascular reactivity	151
	Psychological correlates	153
	Discussion	154
	Psychosocial correlates of CVR	155
	Relationships among psychosocial variables	159
	Limitations	160
	References	164
4.	Discussion	185
	References	193
Appendices		
A	The enduring impact of social factors on exercise tolerance in men attending cardiac rehabilitation	195
	The social and clinical context of study	200
	Method	203
	Participants	203
	Measures	203
	Procedures	205
	Analyses	206
	Results	207
	Adjusted regression analyses testing mediation	207
	Summary of results	209
	Discussion	210
	References	216
B	A test of a model of stress and multiple levels of social support influences on efficacy for overcoming barriers to cardiac rehabilitation	228
	Purpose	234
	Method	234
	Participants	234
	Measures	235
	Procedures	236
	Data analysis	237
	Results	237
	Demographic and descriptive	238
	Measurement model	238
	Structural equation model	238
	Discussion	239





## List of Tables

Table		Page
2-1	Demographic and medical information of women and men	102
2-2	Means and standard deviations of study variables among women and men	103
2-3	Means, standard deviations, and correlations among study variables	104
2-4	Regression analysis predicting social networks diversity from demographic characteristics	105
2-5	Regression analysis predicting social network size from demographic characteristics	105
2-6	Regression analysis predicting total social support from demographic characteristics and social networks	106
2-7	Regression analysis predicting appraisal social support from demographic characteristics and social networks	106
2-8	Regression analysis predicting belonging social support from demographic characteristics and social networks	107
2-9	Regression analysis predicting tangible social support from demographic characteristics and social networks	107
3-1	Demographic characteristics of women and men	179
3-2	Means and standard deviations of psychosocial variables for women and men	180
3-3	Means, standard deviations and ranges of mean resting and peak pre-exercise HR, BP, and MAP for men and women	183
3-4	Correlations among psychosocial predictors and cardiovascular changes from resting to anticipation for men and women	184
A-1	Socio-demographic and comorbidities of men	223
A-2	Correlations among study variables	224
A-3	Adjusted regression examining effect of sociodemographic factors on social support	225
A-4	Adjusted regression analyses examining effect of sociodemographic factors on exercise tolerance	226
A-5	Adjusted regression analyses examining mediated effect of social support between sociodemographic factors and exercise tolerance	227
B-1	Demographic characteristics of study sample	249

B-2	Means, standard deviations, and correlations among study variables	250
B-3	Fit statistics for the structural equation models	251

## List of Figures

Figure		Page
1-1	Berkman and Glass (2000) framework for examining social influences of health	53
1-2	Relationships examined in Fraser a within the Berkman and Glass (2000) framework	55
1-3	Relationships examined in Fraser b within the Berkman and Glass (2000) framework.	57
1-4	Relationships to be examined in proposed Study 1 within the Berkman and Glass (2000) framework	59
1-5	Relationships examined in Study 2 within the Berkman and Glass (2000) framework.	61
3-1	Cardiovascular changes from baseline to anticipation (pre-exercise)	181
B-1	Hypothesized model relating stress and general and specific social support to efficacy	247
B-2	Model with standardized loadings	252

# 1. INTRODUCTION

## General introduction

The following series of research projects was designed to investigate the influence of social support on health among cardiac rehabilitation patients within the multilevel framework of Berkman and Glass (2000). Figure 1-1 outlines the basic framework of Berkman and Glass. Social factors are thought to affect health through a variety of levels. The highest level, macro-level factors represent social structural conditions that are thought to affect the nature of, or give rise to, the mezzo-level factors called social networks. Thus socioeconomic factors, for example, occurring at the macro-level are thought to influence the nature of one's social support. Specifically, people with more income and education are likely to know more people and have regular contact with a greater variety of people compared to poorer people.

In the middle, mezzo-level factors are thought to provide opportunities for the micro-level factors called psychosocial mechanisms. Thus, a larger and more varied social network would provide opportunities for social engagement, access to resources, and different kinds of social support, such as emotional and informational support.

Last these micro-level factors are thought to impact health through a variety of pathways that are thought to be behavioral, psychological and/or physical. For example, social support is thought to attenuate physiological arousal to stress and so may be protective in terms of developing hypertension (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Also, social support is thought to enhance the maintenance of exercise behavior among clinical (Fraser & Spink, 2002) and nonclinical populations (Duncan, Duncan, & McAuley, 1993).

Thus, the current investigations were intended to explore some of the relationships among macro, mezzo, micro, and pathway-level factors that influence health using social support as a dependent, independent, and mediator variable. To this end, two studies were included as background for the current dissertation (Appendix A and B) and two new studies were included as the dissertation projects.

The two appended studies provide a context for the work done prior to starting the dissertation projects and provided some direction to the two dissertation studies. The first study (Fraser, Rodgers, Murray, & Daub, 2005; Appendix A) sought to explain how social factors such as education, marital status, work status, and presence of support from family and friends (structural support) contributed to recovery in cardiac rehabilitation. The study investigated the pathways outlined in Figure 1-2. The goal was to examine how macro-level sociodemographic factors affected social support, and how these macro and mezzo-level factors affected a pathway factor, exercise tolerance. Exercise tolerance is a prognostic physical pathway factor thought to influence success following a cardiac event. This study involved the examination of archival data from records of patients who have gone through the cardiac rehabilitation program. Social status factors were used to predict social support and exercise tolerance. Macro level sociodemographic factors were shown to influence the mezzo level factor social support. These two factors were then shown to have an enduring impact on a physiological pathway level factor: exercise tolerance.

It was still unclear, however, how sociodemographic factors affected social support at the micro-level. Specifically, it was unclear what functional aspects of social

support were determined by sociodemographic factors at the macro-level and how social network factors affected social support at the micro-level.

A second study (Fraser, Rodgers, Daub, & Black, 2005; Appendix B) was conducted to examine how the micro level factors affected psychological pathway variables represented in Figure 1-3. Specifically the goal was to determine how general functional perceptions of social support (e.g., emotional, informational) influence specific support for exercise in cardiac rehabilitation. This study also examined the role of stress and social support in self-efficacy for overcoming barriers to physical activity among cardiac rehabilitation patients.

Results testing this model of general support and exercise specific support and stress predicting confidence for overcoming barriers to exercise in cardiac rehabilitation showed how higher-level support (general support) led to exercise specific support. The micro-level factor social support was related to the psychological pathways of stress and self-efficacy. Specifically, more support was related to less stress and more self-efficacy. However, it was still unclear how micro-level social support affects health, and what aspects of functional social support are related to psychological and physical pathways, such as anxiety and cardiovascular reactivity in CR patients.

To build on the results of the completed studies two new studies were conducted. The goal of the first study was to examine how the macro-level socioeconomic factors were related to the mezzo-level factor social networks and general social support, a micro-level factor. This relationship is depicted in Figure 1-4. This study sought to answer the question: how do macro and mezzo-level social factors influence the functional aspects of social support? This study follows from some of the information

gleaned in Fraser a, and further explores pathways identified within the framework of Berkman and Glass (2000).

The second study, outlined in Figure 1-5, explored the role of micro-level social support on psychological and physiological pathways. Specifically, stress, social support and self-efficacy were examined as potential contributors to CVR in cardiac patients. This study sought to answer the question: does social support attenuate CVR to a stressor among cardiac patients using the Berkman and Glass (2000) framework? In other words, the micro-level factor social support should influence pathway factors. This study builds on the results of Fraser b and sought to further explicate the relationships from social support to health by examining how social support and stress affected a physical health indicator, reactivity.

Together, these two new studies expanded on the results of Fraser, Rodgers, Murray, et al. (2005) and Fraser, Rodgers, Daub, et al. (2005). These two new studies further examined upstream social factors influencing social support and health as well as downstream social factors affecting health. Thus, these two dissertation studies should add to Fraser, Rodgers, Murray et al. and Fraser, Rodgers, Daub et al. by proving a further examination of the Berkman and Glass (2000) framework.

Before presenting the dissertation studies a review of the extant literature that has examined the relationship between social support and health is presented. This review highlights some of the research indicating the importance of social support in shaping one's health. After reviewing the evidence supporting the role of social support in health, social support is explored as a concept. A number of papers are cited that offer some important conceptual clarity in the study of social support and health. The conceptual

model offered by Berkman and Glass (2000) is described in detail. The two studies that follow were conceived using this framework and research offered in the literature review.



## Social support and health

Social support has been identified as a social or group level factor, as opposed to an individual factor, with great importance to the study of chronic illness and disease. Durkheim's (1897/1951) early studies of suicide helped to launch the early research on the importance of social ties and health (Berkman & Glass, 2000; Brownell & Shumaker, 1984). Durkheim's studies of suicide established a connection between social factors and the individual behavior of suicide. Specifically, Durkheim found suicide rates within particular communities (or districts, or countries) were stable from year to year, and that the social integration of the particular society was responsible for suicide rates. In other words, the individual act of suicide was attributed to a fundamental cause related to characteristics of the community.

Cassel (1976) and Cobb (1976) are generally regarded as the major contributors to the recent study of the influence of social support on health (Berkman & Glass, 2000). Both authors provided reviews of the literature that demonstrated the protective health benefits of social support. For illnesses and health outcomes as varied as arthritis, depression, coronary heart disease, and tuberculosis, low social support was identified as a significant contributor to morbidity and mortality. Cassel argued that the social environment was an important, fundamental cause of disease and, therefore, a better target for intervention than reducing exposure to stressors (see p. 121). Recent commentary by Auerbach and Krimgold (2001) and Link and Phelan (1995) support the view that one's social environment is a critical determinant of health.

The following sections provide a brief review of the current literature examining the influence of social support and social networks on health. Before presenting this

research, it is important to note the considerable controversy surrounding the conceptual definition of social support and social networks. O'Reilly (1988) should be consulted for an introduction to the controversy, which cannot be resolved here. Instead a rudimentary explanation of social support and networks will be briefly introduced.

Social networks, or network ties, typically refers to the number of relationships one has with other people. For example, ties to friends, family and children or a confidant (Glass, Mendes de Leon, Seeman, & Berkman, 1997) or involvement in community organizations or church membership (e.g., Berkman & Syme, 1979). The mere presence of these relationships is also known as structural support (Cohen, Kaplan, & Manuck, 1994). The concept of social support also includes the resources (e.g., information, emotional support) provided from the relationships one has with other people (Cohen & Wills, 1985), or functional support (Cohen et al.). The exact nature of the relationships between structural and functional social support and health is unclear (Thoits, 1995). The following studies, and most of the recent literature, use some variation of these general descriptions to operationalize social support or social networks.

#### *Social support/networks and mortality*

Prospective longitudinal epidemiological studies provide compelling evidence for an independent negative relationship between social support and all-cause mortality (Knox & Uvnäs-Moberg, 1998; Stansfeld, 1999). One of the first of several studies was the Alameda County study (Berkman & Syme, 1979). The authors found that individuals low in social ties (married, contacts with friend and family, church membership, group memberships) were 2.3 (for men) to 2.8 (for women) times more likely to die from all

causes over a nine year period. This result was independent of individual health behaviors such as smoking, physical activity, and alcohol consumption.

The Evans County, Georgia study (Schoenbach, Kaplan, Fredman, & Kleinbaum, 1986) used a measure of social network modified from Berkman & Syme's (1979) study. Participants were followed from 1967-1980. The results showed that older people reporting fewer social ties at baseline had a higher risk for mortality, and white males with low social index scores at baseline had 1.5 times the risk of death, after controlling for prior cardiovascular disease (CVD) risks.

Another prospective study conducted in Tecumseh, Michigan (House, Robbins, & Metzner, 1982) over a 9-12 year period found similar results. Specifically, mortality rates were higher among men who were not married and higher among those who did not engage in a variety of social activities. These results controlled for other factors, such as smoking, coronary heart disease and disability. Among women marital status was not important, and only low church attendance was related to mortality.

Blazer (1982) reports 30-month mortality rates from a study in Durham County, North Carolina. Blazer included in the measurement of social support roles and attachments, perceived social support, and social interaction. After controlling for 10 socioeconomic, health status, and behavior confounds, the relative risks of mortality were estimated. Blazer found impaired social roles and attachments, impaired perceived support, and low frequency of social interactions was associated with relative mortality risks of 2.0, 3.4, and 1.9, respectively.

Vogt, Mullooly, Ernst, Pope, and Hollis (1992) found social networks were strong predictors of 15-year mortality hazard. Kawachi et al. (1996) reported higher risks of

CVD mortality among those who were socially isolated. Studies from Sweden (Orth-Gomer & Johnson, 1987; Rosengren, Orth-Gomer, Wedel, & Wilhelmsen, 1993) and Finland (Kaplan et al., 1994) add to this body of evidence suggesting that a lack of social support is a consistent and powerful predictor of mortality. Finally, Lund et al. (2002) found that living alone versus with someone was a significant predictor of 8-year mortality. Berkman and Glass (2000) conclude from the extant literature that those who are “socially isolated or disconnected to others have between two to five times the risk of dying from all causes compared to those who maintain strong ties to friends, family, and community” (p. 160).

#### *Social support/networks and morbidity*

A number of studies have investigated the influence of social support on the morbidity or onset of disease. The relationship between social support and CVD is probably the most studied of these relationships (Stansfeld, 1999). For example, Vogt et al. (1992) found network scope (number of network domains with one or more ties) was related to onset of ischemic heart disease. Kawachi et al. (1996) found that social isolation was related to stroke but not heart disease. A review by Hemingway and Marmot (1999) concluded that social support is a strong etiological factor in the development of CHD. Specifically, they reported that 5 of 8 prospective cohort studies found social support was a predictor of CHD. More recently, Sundquist, Lindstrom, Malmstrom, Johannson, and Sundquist (2003) found that low social participation was prospectively related to higher rates of CHD after controlling for smoking and demographic factors such as age, sex and education.

Uchino et al. (1996) reviewed the literature and concluded that the association between social support and positive cardiovascular function is consistent. However, cardiovascular function was usually indicated by resting blood pressure or acute cardiovascular reactivity to stress. Although these mechanisms seem plausible, it is currently difficult to firmly conclude that a lack of social support, per se, causes CVD (Berkman, Glass, Brissette, & Seeman, 2000) and more evidence is required (Orth-Gomer, 1994). However, Uchino et al. (1996) and Knox and Unväs-Moberg (1998) offer a number of plausible mechanisms that may help clarify the issue. Like Uchino et al., Knox and Unväs-Moberg conclude that low social support leads to cardiovascular morbidity. Further, the authors outline a number of behavioral and neuroendocrine pathways that may help explain the generally negative influence of low social support on health.

One way that social support is thought to affect health is by attenuating or buffering cardiovascular responses. For example, Uchino and Garvey (1997) examined the effect of a speech stressor on systolic (SBP) and diastolic blood pressure (DBP) and heart rate. Perceived availability of social support was found to moderate the effects of the stressor. That is, participants who were told that the researcher would be available in another room if needed (support availability condition) had lower SBP and DBP responses to the stressor than the control group. Kamarck, Manuck, and Jennings (1990) experimentally examined cardiovascular reactivity (CVR) to a mental arithmetic and concept formation task in college-aged women. Results revealed that SBP and heart rate responses were attenuated with the presence of a close female friend for both tasks compared to participants who completed the tasks alone. Gerin, Pieper, Levy, and

Pickering (1992) examined CVR to a threatening condition. Participants engaged in a debate on a controversial topic (e.g., abortion) with the presence or absence of an actively supportive confederate while being “challenged and attacked by two opponents” (p. 326). Gerin et al. found that participants who were supported had smaller CVR responses than those who were unsupported. In a field setting, Unden, Orth-Gomer, and Elofsson (1991) examined HR, SBP and DBP over a 24-hour period. The authors found that those reporting lower social support at work exhibited higher mean heart rate and blood pressures.

Recently, Hilmert, Kulik, & Christenfeld (2002) have challenged the notion that social support always attenuates CVR. The authors studied the effects of social support on CVR in evaluative and nonevaluative settings. The results showed that social support in the evaluative setting reduced CVR, while social support tended to increase CVR in the nonevaluative setting. The authors conclude that social support might be necessary when a situation is evaluative and therefore anxiety provoking, but that in a nonevaluative situation, social support may serve to increase motivation to perform the task. This study raises at least two important considerations for future research. First, the appraisal of the situation as evaluative or nonevaluative may be important in determining CVR and the role of social support. Second, this study involved the actual receipt of social support. This might partially explain the differences between this study and other studies where support was perceived as available (e.g., Kamarck et al., 1991).

Rather than onset of disease, Berkman and Glass (2000) suggest that social support may have a greater influence on prognosis. For example, Berkman, Leo-Summers, & Horowitz (1992) found patients hospitalized for an acute myocardial

infarction were 2.9 times more likely to die within only 6 months if they reported a lack of emotional support. Vogt et al. (1992) found social support was a strong predictor of mortality among those who had survived an incidence of ischemic heart disease, cancer or stroke. Williams et al. (1992) reported better survival among CVD patients who were married. A review by Hemingway and Marmot (1999) showed that 9 of 10 prospective studies found social support was a significant prognostic factor in those with coronary heart disease. Additionally, some evidence suggests that social support can enhance recovery from cancer (Kravdal, 2001; Spiegel, Bloom, Kraemer, & Gottheil, 1989) as well as adjustment or coping with cancer (Helgeson, Cohen, Schultz, & Yasko, 2000). Social support also may help recovery from illness in general (e.g., Cohen, Doyle, Skoner, Rabin, Gwaltney, 1997; Statistics Canada, 1999).

Thus, it currently appears that social support is a consistent predictor of mortality from all-causes. The fact that social support is protective against all-causes offers some support for Link and Phelan (1995) who identify social factors, including social support and social networks, as potential fundamental causes of disease. Further, social support may be more useful in helping individuals recover from illness rather than preventing the onset of illness. However, the findings regarding onset of disease are somewhat conflicting, requiring more research.

#### *Explanations of how social support affects health*

The extant literature clearly implicates social support as an important factor influencing health. In fact, Statistics Canada (1999) has identified a lack of social support as a risk factor for health problems and death. What remains unclear, however, is how social support affects health (e.g., DiMatteo, 2004). That is, what are the mechanisms and

what other factors are responsible for the influence of social support on health outcomes? Few studies have examined this question, and the answers provided could lead to appropriate and improved interventions (Cassel, 1976; Yen & Syme, 1999).

Unfortunately, a review of the social support literature shows a consistent and pervasive call for conceptual and methodological clarification of the notion of social support (e.g., Berkman & Glass, 2000; Brownell & Shumaker, 1984; O'Reilly, 1988). Interestingly however, this lack of conceptual clarity has not interfered with the strong relationships observed in the literature, perhaps adding strength to the findings. That is, the results are continually replicated across settings using a variety of different methodological approaches. However, the drawback is the potential to impair the understanding of exactly *how* social support influences health, a question that remains unresolved (Yen & Syme, 1999).

#### *Theoretical perspectives on social support*

Although the research on social support and health is hampered by a lack of clear gold standard measures, some advances have been made towards clarifying the concept. One clear distinction offered is between perceived and received (or enacted) social support (see Dunkel-Schetter & Bennet, 1990; Sarason, Sarason, Brock, & Pierce, 1996).

The distinction between received and perceived support has been made to differentiate between support that is actually received and support that is perceived to be available (Dunkel-Schetter & Bennet, 1990). Empirical research has supported this conceptual distinction. Cohen and Hoberman (1983) reported a moderate relationship ( $r^2 = .21$ ) between perceived and received social support, while Sarason, Shearin, Pierce, &



Sarason (1987) reported a small relationship ( $r^2 = .06$ ). Newcomb (1990) used structural equation modeling to show that received and perceived support were two distinct factors.

The question is whether it is actually necessary to receive support if it is perceived to be available (Rodriguez & Cohen, 1998). Rodriguez and Cohen suggest that received support is often not related to better emotional adjustment to stressful experiences. Further, some reports suggest that received support may be associated with increased stress and physical symptoms (e.g., Cohen & Hoberman, 1983). However, this may be due to the fact that support was sought when needed (Cohen and Hoberman; Sarason, Sarason, & Pierce, 1990). Hence, the recipient recalls receiving support during particularly stressful times.

A number of explanations for the discrepancy between perceived and received support have been posited. For example, a support recipient's report of enacted support that does not seem to match with reports of others suggests that the perception of support may be more accurate, and important. Sarason, Sarason, and Gurung (1997) and Lakey and Drew (1997) suggest that the way in which an individual perceives social support operates like an enduring personality characteristic. Evidence for this comes from a study by Pierce, Sarason, & Sarason (1992). The authors gave undergraduate students standardized, identical notes written by the students' mothers prior to a stressful task, giving a speech. The researchers found that those reporting lower perceived support interpreted their mother's notes as less supportive than those with high support perceptions.

Dunkell-Schetter and Bennett (1990) acknowledge that individual differences may influence the discrepancy, but they also suggest three other explanations. It may be

that the recipient has simply overestimated the availability of support. When that support is enacted or drawn upon, it may be less responsive than expected due to a situation which is stressful for the providers. Second, it could be that the support dissipates over time. The providers simply get exhausted or tired of providing support. Finally, the recipient could be overwhelmed by support in the case where it is easy for the provider to help, for example. Although, these issues remain to be resolved (Dunkel-Schetter & Bennett, 1990; Rodriguez & Cohen, 1998), perceptions of support appear to be most relevant to health.

Structural and functional characteristics of support have also been mentioned as important distinctions in the social psychological literature (see Cohen & Syme, 1985; Cohen, 1988). Structural characteristics refer to the existence and nature of social ties. Social network approaches would be subsumed under the category of structural support. Functional support refers to the actual psychological or material resources available. These also have been described as types, dimensions or provisions of support (e.g., Cutrona & Russell, 1987). Further, these dimensions or provisions are usually considered as perceived support. In other words, the perception that a particular provision is available is thought to be more important than whether the support is actually available (Cohen, Mermelstein, Kamarck, & Hoberman, 1983). For example, in a recent review, DiMatteo (2004) concluded that functional social support was more related to patient adherence to medical regimens.

Several approaches have been taken in the study of functional characteristics of social support. Rodriguez and Cohen (1998) outline three functional dimensions of social support: instrumental, informational, and emotional. Cohen et al. (1983) outline the four

dimensions: belonging, self-esteem, tangible, and appraisal support. Cobb (1976), as mentioned previously, outlined social support in terms of the perception of feeling esteemed and valued, loved and cared for, and feeling like one belongs to a network.

These provisions are thought to be essential to avoid feeling isolated or lonely, although certain provisions may be more important depending on the circumstances. Further, these provisions may have different sources. That is, different relationships may provide different provisions, yet the same person may provide several of these provisions (Weiss, 1974).

Other considerations for the study of social support are the satisfaction with available support, the source of support, and the specificity of support. Sarason, Levine, Basham, & Sarason (1983) argued that in addition to perceived available support, the satisfaction with one's support is important. For example, Malcolm and Janisse (1991) showed that satisfaction with support was related to lower systolic blood pressure in men.

The source of the support may be important as well. For example, Dakof and Taylor (1990) examined the sources of support among cancer patients. The authors found that physicians and other cancer patients were more valued for their informational support, while emotional support was provided most by intimate others. This study also touches on the specificity of support. The patients tended to value information from other patients and the physician, suggesting that the support was probably specific to the cancer experience, as opposed to the general emotional support that was likely provided by the intimate other.

Oka, King, and Young (1995) also examined source and specificity of support among exercisers aged 50 to 65 years. The authors found that the exercisers preferred less

support from exercise staff at the beginning of the program. Support from family and friends predicted long-term adherence to the exercise program. Further, specific support around exercise was associated with better adherence. These results further underscore the importance of source and specificity of support.

#### *Direction of relationship*

In interpreting the literature indicating the influence of social support on health, it is apparent that poor health results from low support rather than ill health leading to a lack of support. While the latter is certainly plausible to some extent, the evidence to date from large-scale longitudinal studies supports the temporal association from social support to health. For example, Sundquist et al. (2004) examined a random sample of 6861 adults who were followed over a ten-year period. Low social participation at baseline was predictive of the development of CHD within the following 10-year period after controlling for demographic factors and smoking. Similarly, Berkman and Syme (1979) found those with more support were more likely to be alive 9 years later. Again, the results of these and other prospective studies (e.g., House et al., 1982; Kawachi et al., 1996; Schoenbach, et al., 1986; Vogt et al., 1992) suggest the relationship between social support and health is primarily due to the effect of support on health rather than health leading to poor support.

#### *Support Interventions*

The relationship between social support and health appears to be well established. Given the relationship between social support and health the question of how to intervene on low social support becomes a relevant question. Gottlieb (2000) has outlined two general categories of support interventions. These involve intervening on an existing

support network, or natural ties, and, second, introducing new network ties. Interventions with existing networks involve improving support transactions by giving support providers information on how to help the support recipient. For example, providers might be instructed on how to help a recipient adhere to a medical regimen. Interventions to introduce new ties may involve garnering support from doctors and nurses, for example. In the case that existing network members may not share a similar experience support groups may be a form of intervention. Glass (2000) has outlined professionally led, family led, and mutual support groups as three kinds of support interventions.

A number of studies have examined the influence of support interventions with mixed results (Glass, 2000). According to Glass, the reason for mixed results stems from poor theoretically grounded interventions along with methodological shortcomings, such as small sample sizes. Another concern raised by Glass is that support interventions may go unreported. For example, educational interventions often “contain significant (and sometimes hidden) support components” (p. 283). For example, Blumenthal et al. (1997) found that a stress management program was more effective than usual care or an exercise intervention in reducing episodes of daily ischemia among heart patients in cardiac rehabilitation. The stress management intervention was conducted in a group setting with 8 patients. The program involved education, skill instruction and therapeutic techniques. The format of the program sounds very much like Gottlieb’s (1998) definition of a support group: “6 to 10 people who share a similar life stressor, transition, affliction, or noxious habit, and who receive expert information and training, and exchange mutual aid for a predetermined period of time...to foster improved coping and adjustment” (p. 635). Nevertheless, support intervention strategies have been outlined

(e.g., Cutrona & Cole, 2000; Gottlieb, 2000; Helgeson & Cohen, 1996), and support appears to be amenable to change and thus a target for intervention.

Linden, Stossel, and Maurice (1996) reviewed the effect of psychosocial interventions on the physical and psychological outcomes of coronary artery disease patients. The results of the meta-analysis showed that those with psychosocial interventions experienced greater reductions in stress, cholesterol, systolic blood pressure and mortality. A look at Linden et al.'s Table 1 shows that 11 of the 23 controlled trials used some form of group intervention, and two other trials included spouses as part of the therapy. Thus, one might conclude that support interventions were an integral component of many of the psychosocial interventions.

*A conceptual framework for studying social factors and health*

Berkman and Glass (2000) have outlined a “conceptual model of how social networks impact health” (p. 144). One goal for the development of this model, or framework, was to help consolidate the large and varied research on the topic. For example, Berkman and Glass note that in discussing social support “many terms are used loosely and interchangeably” (p. 137). Thus Berkman and Glass have tried to clarify terminology such as social networks and social support and they have tried to organize the extant literature on social support and health into a meaningful framework. Specifically, Berkman and Glass (2000) draw on research from epidemiology, sociology, psychology and physiology to construct a conceptual framework to “examine how social relations and networks influence a broad array of health outcomes” (p. 142). The framework highlights the efforts of past research on social support and health and offers future directions for research that follows from these past advances. This framework will

also serve as a framework for examining the influence of social support on health for this dissertation. This conceptual model depicted in Figure 1-1 is a framework for examining the role of social support and networks on health outcomes developed from the extant literature.

The model is a multilevel representation of factors influencing health. A multilevel approach to understanding how social support affects health has a number of advantages. This particular framework acknowledges multiple pathways and processes that lead to health rather than focusing on a single mechanism (cf. Link & Phelan, 1995). Thus, in understanding how social factors affect health, a multilevel approach allows for detailed explication at different levels. In other words, this approach allows for the integration of models at different levels (Marmot, 2000). For example, sociological models of health, psychological models of health behaviors, and physiological models of health outcomes could be linked to understand how social factors influence psychological determinants of behaviors thought to affect health outcomes (e.g., blood pressure, fitness). Thus, in understanding how social support affects health, the multilevel approach allows for the determination of psychological pathways from social support to health as well as an understanding of what gives rise to social support. This understanding could lead to targeted interventions at appropriate levels (e.g., family level vs. community level).

Berkman and Glass (2000) describe higher level, or upstream, factors which include social structural conditions and social networks. These macro-level and mezzo-level factors, respectively, are composed of what are traditionally sociological constructs. For example, macro-level factors include culture, socioeconomic status, politics and

social change. Mezzo-level factors include social network structure and characteristics of network ties.

Lower level factors are called downstream factors. These include psychosocial mechanisms at the micro-level, and *pathways*, furthest downstream. Psychosocial mechanisms include social support, social influence, social engagement, person-to-person contacts, and access to resources and material goods. The pathways identified include health behavior, and psychological and physiological pathways. These are thought to be the most proximal *pathways* to health. In other words, these pathways are thought to lead more directly to health outcomes, and hence are more downstream.

The Berkman and Glass (2000) framework implies a causal path from the upstream factors to the downstream factors such that the macro-level factors condition or shape the nature of mezzo-level network factors. The mezzo-level factors, in turn, provide opportunities for the psychosocial mechanisms, which then impact health through the specific pathways that can be behavioral, psychological or physiological. This model offers a specific framework for examining directional and ordered relations among variables.

Most of the work examining social support and health has occurred downstream at the micro and pathway levels (see House, Umberson, & Landis, 1988). Berkman et al. (2000) argue that the study of macro-social factors “is almost completely absent in studies of social network influences on health” (p. 846). Link and Phelan (1995) agree that, as distal causes of disease, social factors, in general, “have received far less attention” (p. 80). This may help explain why Berkman and Glass are somewhat vague in explicating these macro-level factors in the conceptual model. Again, the authors outline



cultural factors, socioeconomic factors, politics, and social change as macro-level components.

Macro-level factors are thought to influence the mezzo-level factors. However, it is important to note that the macro-level factors are also inextricably linked. That is, culture and politics clearly affect each other, and these are both affected by, and affect social change, along with socioeconomic factors. For example, the recent urbanization of North America is surely linked to changes in the cultural norms of society as we move away from an agrarian society. Other cultural factors like sexism and racism clearly affect, or are affected by, politics in the form of laws and public policy directed towards women (e.g., Afghanistan) and minorities (e.g., Nazi Germany, racial profiling), for example. The internment of Japanese Canadians after the bombing of Pearl Harbor may be an example of cultural racism and social change due to war affecting public policy.

Socioeconomic factors have probably received the most attention in examining social factors on health at the macro-level, however. Some of the socioeconomic factors identified by Berkman and Glass (2000) are inequality, discrimination (again related to sexism and racism), conflict, and poverty. Health Canada (1999) has acknowledged the importance of several socioeconomic factors on health. Specifically acknowledged is the relationship between low-income and early death and more illness. The results in the U.S. are no different: “poor and poorly educated people still die at higher rates than those with higher incomes and better educations” (Pappas, Queen, Hadden, & Fisher, 1993, p.103; also see Auerback & Krimgold, 2001 for an extensive review).

These macro-level factors are thought to influence the mezzo-level factors, such as social networks. The broad category of social networks, as described by Berkman and

Glass (2000), include an individual's social network (or structural social support) and the characteristics of an individual's network ties. Social network characteristics are basically structural and include size, range, density, homogeneity and reachability of the network. The characteristics of network ties are closely related to social networks, describing frequency of face-to-face contact, nonvisual contact and organizational participation, as well as reciprocity, duration, and intimacy. Again, these two broad components of the mezzo-level are inextricably linked, and are influenced by macro-level factors. However, Berkman and Glass (2000) provide no specific hypothesized relationships between the macro and mezzo-levels, although the conceptual framework does allow for some hypothesized links. For example, it is conceivable that cultural characteristics will influence the nature of one's social networks. Specifically, a racist culture, or a political climate that disenfranchises a person might limit the number of contacts that person has available. Also, the characteristics of that network would likely differ from the majority group. Turner and Marino (1994) provide evidence for the link between a macro-level factor and a mezzo-level factor. Specifically, the authors found that people with higher education and incomes had more contact with their own network members. In addition, Statistics Canada (1999) reports that people with the lowest incomes have the lowest social support (micro-level). This and other evidence (Cattell, 2001; Krause & Borawski-Clark, 1995) may support a mediated relationship between socioeconomic status (SES), social network contact and social support.

The micro-level factors that have been studied are primarily psychosocial mechanisms. That is, social support, social influence, social engagement, and person-to-person contact. Also included are access to resources and material goods. Again, one can

see that the components at this level are interrelated. For example, social support consists of instrumental, informational, and emotional support (Cohen & Hoberman, 1983).

Instrumental support refers to access to tangible resources. Clearly, access to resources is influenced by, or may influence, our perceptions of instrumental support (see Kessler, Price, & Wortman, 1985 for a discussion of perceived versus actual support).

Access to resources presents an interesting component of the micro-level factors. In fact, access to resources does not appear to be a psychosocial mechanism, as implied by the framework. Rather, access to resources refers to the actual availability of tangible resources, such as money and transportation. However, the presumption seems to be that access to resources resides at the same level as the other micro-level factors. This is similar to the distinction between actual and perceived support.

At this level the conceptual model can be used to explain how access to resources may be affected by upstream factors. For example, it was shown that SES might influence the composition of one's social network. If one's social network is small it may be limited in providing access to transportation, or housing, or even economic opportunities, for example (Cattell, 2001). Further, Berkman and Glass (2000) suggest that the degree to which one's networks overlap (e.g., some of my friends from work might be some of my friends at school and hence the school and work networks overlap) may influence access to various opportunities. For example, consider a homogeneous or tight knit culture within in a broader cultural context of inequality. If this homogenous culture happens to be on the low end of growing inequality the result is low social cohesion (i.e., connectedness between groups; see Kawachi & Berkman, 2000).

Conceivably, such a situation could result in fewer overlapping networks, and hence less access to resources.

However, there may be a more direct influence of SES on health. Syme (2001) argues that SES is the most “significant risk factor for health and well-being” (p. 12). In Canada, for example, far more Aboriginal families have problems securing housing and affording food, and live in low-income situations. Further, they tend to suffer from three to five times the rates of diabetes, cancer, hypertension, arthritis, and heart problems (Health Canada, 1999, p. 17). It is debatable whether this is entirely a direct influence of cultural factors like racism, or racist public policies, or if these outcomes are influenced by access (see Krieger, 2000 for a review). Williams (2001) argues that SES accounts for most of the racial difference in health in the US. However, when compared to Whites of similar SES, Blacks are still worse off for heart disease incidence and multiple indicators of health. Williams suggests that these differences are not biologically based. In other words, although these minority groups are clearly worse off in terms of SES, there also exist other societal influences that negatively impact on health. Other evidence exists to suggest that discrimination can directly affect health as well (Krieger & Sidney, 1996).

Finally, there are the (proximal) pathways to health. These include behavioral, psychological, and physiological mechanisms. Behavioral pathways involve health behaviors, like smoking, diet and exercise. The psychological pathways involve depression and well-being, as well as self-esteem and coping. Physiological pathways are probably the most direct pathways to health involving immune system function, cardiovascular reactivity (CVR) and hypothalamic-pituitary-adrenal axis responses (Knox & Unväs-Moberg, 1998; Uchino et al., 1996).

The proximal pathways to health represent a particular challenge to the study of social influences on health. For example, the health consequences of physical inactivity, smoking, and a poor diet are well known, yet it is unclear if social support affects health through these behaviors, or if there is a more general effect of social support on health. Nevertheless, it is clear that social factors are an important influence of individual health behaviors. For example, among adolescents, smoking is best predicted by the smoking of one's peers (Landrine, Richardson, Klonoff, & Flay, 1994). Social support is also linked to higher rates of adherence to exercise (Duncan & McAuley, 1993; Duncan & Stoolmiller, 1993; McAuley, Courneya, Rudolph, & Lox, 1994) compliance with medical regimens (DiMatteo, 2004; Meichenbaum & Turk, 1987; Taylor & Aspinwall, 1993) and cardiac rehabilitation in particular (Dracup, 1994). Yet a recent review by Uchino et al. (1996) shows that in studies examining the influence of social support on health, the health behaviors, per se, are not the *major* pathways impacting on health. Knox and Unväs-Moberg (1998) stress the importance of the influence of social support on health behaviors. However, like Uchino et al., the authors stress the importance of direct physiologic pathways, rather than behavioral. Specifically, Knox and Unväs-Moberg argue that social isolation affects pituitary-adrenal cortical factors and sympathetic-adrenomedullary influences on heart rate (HR) and blood pressure (BP), which increases the risk of CVD. These responses are referred to as cardiovascular reactivity (CVR).

Thus, there is empirical support for the micro-level and pathway-level factors in the conceptual model. There also exists some evidence for the upstream factors, where support for the influence of macro-level factors on mezzo-level factors was introduced.

The primary contribution of the model, however, is to systematically organize the extant literature and to provide a framework for future research.

### *Mechanisms*

The framework described by Berkman and Glass (2000) offers several hypothesized mechanisms that can be explored. A number of approaches to the conceptual study of the influences on social support and health were reviewed above. The evidence seems to indicate that social support does have a consistent positive effect on health. However, it is necessary to identify the mechanisms of this phenomenon. In other words, how does social support affect health (Cohen, 1988)? Without a precise conceptual definition of social support this may be difficult. However, two basic theoretical approaches to this question have been undertaken.

The two approaches from a psychosocial perspective are typically referred to as the direct or main-effects model, and the stress-buffering model (Cohen, 1988). The main effect model argues that social support has a direct effect on health, which may be mediated by behavior or physiological processes. Evidence from studies like that of Berkman & Syme (1979) indicate that the main-effects model is likely associated with structural forms of support (Cohen et al., 1994) such as contact with friends and family. Thus, the main effect model suggests that mezzo-level social networks affect health through some mediating mechanism such as behavior. Evidence for this model comes from the early epidemiological studies mentioned above where social influences predicted mortality while controlling for a variety of other health factors like smoking and exercise (Berkman & Syme). A recent review (Uchino et al., 1996), however, suggests that behavior may not be the only pathway through which social support

influences physiological function (p. 522). So, although social support may positively influence health behaviors, the effect on health may be through a more proximal mechanism. That is, social support may directly affect health by impacting one of the physiological mechanisms mentioned above.

The second approach is the stress-buffer hypothesis and was essential to Cobb's (1976) argument. He thought social support moderated the relationship between stressors and health. The basic idea is that people who perceive high levels of support are better equipped to deal with stressors that they may encounter (Cohen et al., 1994). Cohen (1988) outlines two ways in which the buffering may occur. First, the recipient's perception of his or her ability to cope with the stressor is enhanced at the onset of the stressor with the help of support. Second, after the onset of the stressor and a response, support could reduce the reaction to the stress. Related to this concept is the idea of stress-matching or optimal-matching. Cutrona and Russell (1990) argue that the particular type of social support should match the type of stressor to be effective. For example, Cutrona and Russell's notion of optimal matching suggests that emotional support would be more appropriate in situations where events are uncontrollable. This is because a person cannot actively cope with an uncontrollable situation by problem solving, for example, so emotional support might aid in coping with the emotional consequences of the event. However, when an event is thought to be controllable, it may be more useful to provide informational support that could help the recipient actively cope. Thus, within the Berkman and Glass (2000) framework, social support at the micro-level might moderate the influence of stress on health.

*Cardiovascular reactivity*

At the pathways level, researchers have begun further exploration of physiological and psychological pathways on health including the relationship between social support and cardiovascular reactivity. Kamarck and Lovallo (2003) note that there is currently “little consensus on the meaning and measurement of CVR” (p. 9). Nevertheless, cardiovascular reactivity has been characterized by “exaggerated heart rate and blood pressure responses when encountering behavioral stimuli experienced as engaging, challenging, or aversive” (Rozanski, Blumenthal, Kaplan, 1999; p. 2207). This is consistent with Kamarck and Lovallo, who argue that the challenging situation must be motivationally meaningful such that there is a personal consequence. Further, the situation should require some kind of adaptive response that is either physical or cognitive. Schwartz et al. (2003) describe CVR as “BP and HR elevations occurring during presentation of a discrete stressor” (p. 22). Trieber et al. (2003) define CVR more generally “as the magnitude or pattern of an individual’s hemodynamic responses to behavioral stressors” (p. 46). On assessing reactivity, Manuck, Kasprovicz, Monroe, Larkin, and Kaplan (1989) note the method typically includes assessing “the difference between physiologic states recorded during such ‘stimulus’ periods and comparative observations made under ‘baseline’ conditions” (p. 366). Hence, CVR is typically calculated as the mathematical difference between baseline and stimulus. Baseline and stimulus may be measured as the average baseline and the average stimulus, thus enhancing reliability (Manuck et al.). However, baseline has also been compared to maximum or peak task/stimulus related changes. If these parameters are ‘reactive’ (i.e., dynamic) then it might be that peak changes are arguably more meaningful than average changes, however.



Cardiovascular reactivity is the general term for the multiple responses of the cardiovascular system to psychological stress, also referred to as mental-stress responses, hemodynamic responses, stress responsivity, acute psychophysiologic reactivity and simply cardiovascular reactions. These responses include the results of catecholamine and corticosteroid release: increased HR, cardiac output, blood pressure (Herd, 1991; Krantz, Kop, Santiago, & Gottdiener, 1996). Also included is increased platelet activity and sodium retention (Herd). However, most researchers seem to focus on specific cardiovascular responses such as BP responses or HR and BP responses (Kamarck, Jennings, Pogue-Geile, & Manuck, 1994; Krantz & Manuck, 1984; cf. Christenfeld & Gerin, 2000; Schwartz et al., 2003). Yet increases in BP may be the result of increased cardiac output (CO), increased total peripheral resistance (TPR) or a combination of the two (Rüddel, Langewitz, Schächinger, & Schmeider, 1988), for example. Thus, Turner (1994) suggests that individuals may have a vascular response (e.g., TPR) or a cardiac response (e.g., CO; cf. Kamarck et al.; Kamarck & Lovallo, 2003; Kelsey, 1993; Rüddel, Langewitz, Schächinger, Schmieder, & Schulte, 1988) to a stressor. Thus, two people may display similar BP responses to a stressor yet one may be experiencing a rise in TPR while another may experience a rise in CO. So, while HR and BP reactivity have been most commonly studied and powerfully linked to prognosis, it may be useful to understand the underlying pattern of hemodynamic responding including other factors such as stroke volume (SV), pre-ejection period (PEP), and mean arterial pressure (MAP).

However, HR and BP are typically assessed for at least two reasons. First, these are usually easily obtained from standard noninvasive equipment such as a HR monitor and a sphygmomanometer. Second, HR and BP measures have been linked to prognosis,

morbidity and mortality most frequently. In contrast SV and TPR require specialized equipment such as impedance cardiography (e.g., Kamarck et al., 1994) and more particularly are prognostically less clear than BP.

The *reactivity hypothesis* generally suggests that exaggerated responses will lead to a greater risk of developing hypertension and possibly heart disease (Christenfeld & Gerin, 2000; Kamarck & Lavallo, 2003; cf. Treiber et al., 2003). A key aspect of CVR is a response to psychologically relevant stimuli that is fairly consistent (Turner, 1994). It is this tendency to respond to potentially aversive stimuli in an exaggerated manner that places the person at risk. Thus, a reactive individual may be exposed to relatively high heart rate and blood pressures, for example, over extended periods, and/or with relatively high frequency. Cohen and Manuck (1995) noted that evidence is now convincing that CVR is a stable individual difference variable.

Several studies have demonstrated the effects of CVR per se on long-term health. For example, Lynch, Everson, Kaplan, Salonen, and Salonen (1998) found that CVR in anticipation of a maximal exercise test was predictive of four-year progression of atherosclerosis. Similarly, Kamarck et al. (1997) found that an exaggerated response to a mental stressor was related to atherosclerosis in a sample of Finnish men followed over a four-year period. Barnett, Spence, Manuck, and Jennings (1997) found that a heightened response to mental stress was predictive of 2-year change in atherosclerosis. Everson et al. (2001) found that an exaggerated response to an anticipated exercise tolerance test in middle-aged men was associated with an increased risk of stroke over an 11-year follow-up period controlling for a variety of risk factors including, for example, antihypertensive medications, VO<sub>2</sub>max, smoking, BMI, diabetes, and alcohol consumption. Waldstein et

al. (2004) found systolic and diastolic blood pressure reactivity were related to a higher number of silent cerebrovascular infarctions and cerebrovascular disease as indicated by magnetic resonance imaging. Kop, Gottdiener, Patterson, & Krantz (2000) found that increased SBP reactivity was significantly and independently related to left ventricular (LV) mass, a risk factor for cardiac morbidity and mortality. A recent review by Treiber et al. (2003) concludes that CVR can lead to the development of increased LV mass and high BP, as well as clinical events in those with hypertension and heart disease.

Among cardiac patients, CVR appears to be an important contributor to prognosis. For example, Blumenthal et al. (1995) examined the effects of mental stress on coronary artery disease (CAD) patients who had previously shown evidence of myocardial ischemia induced through exercise. The authors found that mental stress induced ischemia was related to greater acute CV responses to stress. Further, these patients were also more likely to experience mental-stress induced ischemia outside of the lab setting. Thus, CVR is thought to be related to risk of myocardial ischemia. This is echoed in a review by Rozanski et al. (1999) where they point out that mental stress leads to substantial BP increases that are on par with exercise elevated increases in BP. However, these BP increases occur more rapidly than with exercise, and at a lower heart rate. Kral et al. (1997) similarly found that myocardial ischemia induced by mental stress was associated with exercise induced ischemia among at-risk asymptomatic individuals. Manuck, Olson, Hjemdahl, and Renhqvist (1992) found an exaggerated CV response to a mental stressor among patients with a recurrent infarction compared to patients who did not experience a recurrent event. Further, mental stress is thought to trigger not only myocardial ischemia, but also myocardial infarctions or heart attacks (Krantz et al.,

1996). These results appear to show that mental stress responses can be significant prognostic indicators for cardiac patients as Krantz, Sheps, Carney, and Natelson (2000) have argued. However, these studies occur exclusively within the pathway level of the Berkman and Glass (2000) framework examining only psychological (i.e., mental stress) precursors to CVR, ischemia and infarctions.

Social support is thought to dampen CV responses to stressful situations. Lepore (1998) calls the pathway from social support to CVR to health the “social support-reactivity hypothesis” (p. 257). The hypothesis suggests that social support acts as a buffer to stressors that are responsible for excessive responses of the cardiovascular system. In other words, social support at the micro-level is thought to directly dampen the generally arousing effects of stress on the individual at the pathways level. Conversely, the lack of social ties, or social isolation, is a stressor itself, and a risk for mortality and morbidity (Kiecolt-Glaser, McGuire, Robles, Glaser, 2002). Thus, the mezzo-level factor social networks might directly affect the pathway level factor stress, where poor social networks lead to higher stress. However, the mechanism of social support reducing chronic exposure to a stressor and consequently attenuating the effects on the cardiovascular system is likely very similar. For example, Cohen (1988) has suggested that social support might dampen the effects of a stressor by influencing the initial appraisal of the stressor. Thus, knowing support is available may lessen the perceived impact of the stressor one is faced with. Second, social support may intervene with an ongoing stressor by providing resources to reduce the perceived impact of the stressor or as a distraction. If the stressor was a health problem, social support may also aid by providing recourses to encourage the appropriate health behaviors (e.g., exercise and diet

change). However, in considering the Berkman and Glass framework there is an apparent need to investigate how mezzo-level social network support might lead to the micro-level functional social support and how these two levels might influence the impact of stress and CVR on health.

Despite the efforts to examine the role of CVR in the development and progression of heart disease, and the influence of social support on CVR, little research has been conducted examining the role of social support on CVR in cardiac patients. Craig, Lynch and Quartner (2000) further note that the influence of CVR on prognosis is strongest among patients with coronary artery disease (CAD; e.g., Blumenthal et al., 1995; Kamarck et al., 1997; Lynch et al., 1998) and it is therefore surprising that the relationship between social support and CVR has not been studied in cardiac patients. That is, several studies have found that CVR to a mental stressor may be an important predictor of future recurrent cardiovascular events in patients with CAD. For example, Manuck et al. (1992) found that patients with a recurrent event exhibited higher SBP and DBP responses to a mental stress task 39 to 64 months previously. One reason for the poorer prognosis among more reactive patients may be that mental stressors can induce silent myocardial ischemia both in the laboratory and during daily activities. Gottdiener et al. (1994) found that CAD patients who experienced ischemia during a laboratory mental stressor also experienced ischemia during normal sedentary daily activities. Blumenthal et al. (1995) examined mental stress-induced ischemia in patients with CAD who had previously experienced exercise-induced ischemia. The authors found that patients who experienced mental stress-induced ischemia in the laboratory were more likely to experience ischemia during daily living and from daily mental stress than those

who did not experience the mental stress-induced ischemia. This is particularly important given the recent conclusions of Strike and Steptoe (2003), who argue “stress-induced ischemia is not simply a reflection of disease severity, but an indicator of a particular susceptibility to psychological factors” (p. 700). Thus, the importance of understanding the influence of psychosocial factors on CVR in heart patients seems critical. Further, there is a need to expand these studies beyond the pathway level to higher levels of influence. In other words, it is important to understand the factors leading to, or perhaps attenuating, the “susceptibility to psychosocial factors.”

Craig et al. (2000) examined the role of social support in reducing CVR among cardiac patients with a recent MI or coronary artery bypass graft (CABG). The authors found that patients with higher perceptions of available social support had a reduced cardiovascular response to a stressful situation. Further, Craig et al. found that the CVR differences were observed in patients taking medications designed to attenuate this effect. While these results are promising, much remains unknown. For example, it is unclear how social support affects CVR changes as a result of cardiac rehabilitation. Exercise based CR is thought to improve autonomic function among CR patients resulting in improved heart rate recovery (Nishime, Cole, Blackstone, Pashkow, & Lauer, 2000) and increased heart rate variability (Malfatto et al., 1998). Whereas recent research suggests a moderate bout of exercise reduces psychological stress responses (Brownley et al., 2003), Herd (1991) suggests that physical conditioning, per se, may not improve CVR to psychological stress. Social support, however, might act more generally by attenuating CVR responses to psychological stress.

Thus the purpose of the present investigation was to explore the influence of social support on recovery in cardiac rehabilitation within larger Berkman and Glass (2000) framework of social influences on health. Therefore, both the determinants and the effects of social support were explored including the upstream effects of social influences. Fraser, Rodgers, Murray et al. (2005) examined sociodemographic factors and social support influencing exercise tolerance and Study 1 examined the higher level determinants of different forms of functional social support. Fraser, Rodgers, Daub et al. (2005) examined functional social support and exercise specific support effects on CR patient confidence about overcoming barriers to exercise in CR. Study 2 examined the role of social support and stress in cardiovascular responses to anticipation of an exercise tolerance test.

## References

- Auerbach, J.A., & Krimgold, B.K. (Eds.).(2001). *Income, socioeconomic status, and health: Exploring the relationships*. Washington, DC: National Policy Association.
- Barnett, P.A., Spence, J.D., Manuck, S.B., & Jennings, J.R. (1997). Psychological stress and the progression of carotid artery disease. *Journal of Hypertension, 15*, 49-55.
- Berkman, L.F., & Glass, T. (2000). Social integration, social networks, social support, and health. In L.F.Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 137-173). New York: Oxford University Press.
- Berkman, L.F., Glass, T., Brissette, I., & Seeman, T.E. (2000). From social integration to health: Durkheim in the new millenium. *Social Science & Medicine, 51*, 843-857.
- Berkman, L.F., Leo-Summers, L., & Horwitz, R.I. (1992). Emotional support and survival after myocardial infarction: A prospective, populations-based study of the elderly. *Annals of Internal Medicine, 117*, 1003-1009.
- Berkman, L.F., & Syme, S.L. (1979). Social networks, host resistance, and mortality: a nine year follow-up study of Alameda County residents. *American Journal of Epidemiology, 109*, 186-203.
- Blazer, D.G. (1982). Social support and mortality in an elderly community population. *American Journal of Epidemiology, 115*, 684-694.
- Blumenthal, J.A., Jiang, W., Babyak, M.A., Krantz, D.S., Frid, D.J., Coleman, R.E., et al. (1997). Stress management and exercise training in cardiac patients with myocardial ischemia: Effects on prognosis and evaluation of mechanisms. *Archives of Internal Medicine, 157*, 2213-2224.



- Blumenthal, J.A., Jiang, W., Waugh, R.A., Frid, D.J., Morris, J.J., Coleman, E., et al. (1995). Mental stress-induced ischemia in the laboratory and ambulatory ischemia during daily life. *Circulation*, *92*, 2102-2108.
- Brownell, A.B., & Schumaker, S.A. (1984). Social support: An introduction to a complex phenomenon. *Journal of Social Issues*, *40*, 1-9.
- Brownley, K.A., Hinderliter, A.L., West, S.G., Girdler, S.S., Sherwood, A., & Light, K.C. (2003). Sympathoadrenergic mechanisms in reduced hemodynamic stress responses after exercise. *Medicine & Science in Sports & Exercise*, *35*, 978-986.
- Cassel, J. (1976). The contribution of the social environment to host resistance. *American Journal of Epidemiology*, *104*, 107-123.
- Cattell, V. (2001). Poor people, poor places, and poor health: The mediating role of social networks and social capital. *Social Science & Medicine*, *52*, 1501-1516.
- Christenfeld, N., & Gerin, W. (2000). Social support and cardiovascular reactivity. *Biomedicine & Pharmacotherapy*, *54*, 251-257.
- Cobb, S. (1976). Social support as a moderator of life stress. *Psychosomatic Medicine*, *38*, 300-314.
- Cohen, S. (1988). Psychosocial models of the role of support in the etiology of physical disease. *Health Psychology*, *7*, 269-297.
- Cohen, S., Doyle, W.J., Skoner, D.P., Rabin, B.S., & Gwaltney, J.M. (1997). Social ties and susceptibility to the common cold. *JAMA*, *277*, 1940-1944.
- Cohen, S., & Hoberman, H.M. (1983). Positive events and social supports as buffers of life change stress. *Journal of Applied Social Psychology*, *13*, 99-125.

- Cohen, S., Kaplan, J.R., & Manuck, S.B. (1994). Social support and coronary heart disease: Underlying psychological and biological mechanisms. In S.A. Shumaker & S.M. Czajkowski (Eds.), *Social support and cardiovascular disease* (pp. 195-221). New York: Plenum Press.
- Cohen, S., & Manuck, S.B. (1995). Stress, reactivity, & disease. *Psychosomatic Medicine*, 57, 423-426.
- Cohen, S., Mermelstein, R., Kamarck, T., & Hoberman, H.M. (1983). Measuring the functional components of social support. In I. G. Sarason & B. R. Sarason (Eds.), *Social support: Theory, research, and application*. The Hague, Holland: Martinus Nijhoff.
- Cohen, S., & Syme, S.L. (1985). Issues in the study and application of social support. In S. Cohen & S.L. Syme (Eds.), *Social support and health*. New York: Academic Press.
- Cohen, S., & Wills, T.A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, 98, 310-357.
- Craig, F.W., Lynch, J.J., & Quartner, J.L. (2000). The perception of available social support is related to reduced cardiovascular reactivity in phase II cardiac rehabilitation patients. *Integrative Physiological and Behavioral Science*, 35, 272-283.
- Cutrona, C.E., & Cole, V. (2000). Optimizing support in the natural network. In S. Cohen, L.G. Underwood, & B.H. Gottlieb (Eds.), *Social support measurement and intervention* (pp. 278-308). New York: Oxford.

- Cutrona, C.E., & Russell, D.W. (1987). The provisions of social relationships and adaptation to stress. In W.H. Jones & D. Perlman (Eds.), *Advances in personal relationships* (Vol. 1, pp. 37-67). Greenwich, CT: JAI Press.
- Cutrona, C.E., & Russell, D.W. (1990). Type of social support and specific stress: Toward a theory of optimal matching. In B.R. Sarason, I.G. Sarason, & G.R. Pierce (Eds.), *Social support: An interactional view* (pp. 319-366). New York: Wiley.
- Dakof, G.A., & Taylor, S.E. (1990). Victims' perceptions of social support: What is helpful from whom? *Journal of Personality and Social Psychology*, *58*, 80-89.
- DiMatteo, M.R. (2004). Social support and patient adherence to medical treatment: A meta-analysis. *Health Psychology*, *23*, 207-218.
- Dracup, K. (1994). Cardiac rehabilitation: The role of social support in recovery and compliance. In S.A. Shumaker & S.M. Czajkowski (Eds.), *Social Support and Cardiovascular Disease* (pp. 333-353). New York: Plenum Press.
- Duncan, T.E., Duncan, S.C., & McAuley, E. (1993). The role of domain and gender-specific provisions of social relations in adherence to a prescribed exercise regimen. *Journal of Sport & Exercise Psychology*, *15*, 220-231.
- Duncan, T.E., & McAuley, E. (1993). Social support and efficacy cognitions in exercise adherence: A latent growth curve analysis. *Journal of Behavioral Medicine*, *16*, 199-218.
- Duncan, T.E., & Stoolmiller, M. (1993). Modeling social and psychological determinants of exercise behaviors via structural equation systems. *Research Quarterly for Exercise and Sport*, *64*, 1-16.

- Dunkel-Schetter, C., & Bennet, T.L. (1990). Differentiating the cognitive and behavioral aspects of social support. In B.R. Sarason & I.G. Sarason (Eds.), *Social support: An interactional view. Wiley series on personality processes* (pp. 267-296). New York: John Wiley and Sons.
- Durkheim, E. (1897). *Suicide*. (Reprinted in 1951) New York: Free Press.
- Everson, S.A., Lynch, J.W., Kaplan, G.A., Lakka, T.A., Sivenius, J., & Salonen, J.T. (2001). Stress-induced blood pressure reactivity and incident stroke in middle-aged men. *Stroke*, 32, 1263-1270.
- Fraser, S.N., Rodgers, W.M., Daub, B., & Black, B. (2005). *A test of a model of stress and multiple levels of social support on overcoming barriers to exercise in cardiac rehabilitation*. Manuscript in preparation.
- Fraser, S.N., Rodgers, W.M., Murray, T.C., & Daub, B. (2005). *The relationship between sociodemographic factors, social support and exercise tolerance in men attending cardiac rehabilitation*. Manuscript submitted for publication.
- Fraser, S.N., & Spink, K.S. (2002). Examining the role of social support and group cohesion in exercise compliance. *Journal of Behavioral Medicine*, 25, 233-249.
- Gerin, W., Pieper, C., Levy, R., Pickering, T.G. (1992). Social support in social interaction: A moderator of cardiovascular reactivity. *Psychosomatic Medicine*, 54, 324-336.
- Glass, T. (2000). Psychosocial interventions. In L.F. Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 267-305). New York: Oxford University Press.

- Glass, T.A., Mendes de Leon, C.F., Seeman, T.E., Berkman, L.F. (1997). Beyond single indicators of social networks: A LISREL analysis of social ties among the elderly. *Social Science & Medicine*, 44, 1503-1517.
- Gottdiener, J.S., Krantz, D.S., Howell, R.H., Hecht, G.M., Klein, J., Falconer, J.J., & Rozanski, A. (1994). Induction of silent myocardial ischemia with mental stress testing: Relation to the triggers of ischemia during daily life activities and to ischemic function severity. *Journal of the American College of Cardiology*, 24, 1645-1651.
- Gottlieb, B.H. (1998). Support groups. In H.S. Friedman (Ed.), *Encyclopedia of Mental Health* (Vol. 3, pp. 635-648). San Diego, CA: Academic Press.
- Gottlieb, B.H. (2000). Selecting and planning support interventions. In S. Cohen, L.G. Underwood, & B.H. Gottlieb (Eds.), *Social support measurement and intervention* (pp. 195-220). New York: Oxford.
- Health Canada (1999). *Toward a healthy future: Second report on the health of Canadians*. Available online:  
<http://www.hcsc.gc.ca/hppb/phdd/report/toward/eng/report.html>
- Helgeson, V.S., & Cohen, S. (1996). Social support and adjustment to cancer: Reconciling descriptive, correlational, and intervention research. *Health Psychology*, 15, 135-148.
- Helgeson, V.S., Cohen, S., Schultz, R., & Yasko, J. (2000). Group support interventions for women with breast cancer: Who benefits from what? *Health Psychology*, 19, 107-114.

- Hemingway, H., & Marmot, M. (1999). Psychosocial factors in the aetiology and prognosis of coronary heart disease: Systematic review of prospective cohort studies. *British Medical Journal*, *318*, 1460-1467.
- Herd, J.A. (1991). Cardiovascular response to stress. *Physiological Reviews*, *71*, 305-330.
- Hilmert, C.J., Kulik, J.A., & Christenfeld, N. (2002). The varied impact of social support on cardiovascular reactivity. *Basic and Applied Social Psychology*, *24*, 229-240.
- House, J.S., Robbins, C., & Metzner, H.L. (1982). The association of social relationships and activities with mortality. *American Journal of Epidemiology*, *116*, 123-140.
- House, J.S., Umberson, D., Landis, K. (1988) Structures and processes of social support. *Annual Review of Sociology*, *14*, 293-318.
- Kamarck, T.W., Everson, S.A., Kaplan, G.A., Manuck, S.B., Jennings, J.R., Salonen, R. et al. (1997). Exaggerated blood pressure responses during mental stress are associated with enhanced carotid atherosclerosis in middle-aged Finnish men: findings from the Kuopio Ischemic Heart Disease Study. *Circulation*, *96*, 3842-3848.
- Kamarck, T.W., Jennings, J.R., Pogue-Geile, M., & Manuck, S.B. (1994). A multidimensional measurement model for cardiovascular reactivity: Stability and cross-validation in two adult samples. *Health Psychology*, *13*, 471-478.
- Kamarck, T.W., Manuck, S.B., & Jennings, J.R. (1990). Social support reduces cardiovascular reactivity to psychological challenge: A laboratory model. *Psychosomatic Medicine*, *52*, 42-58.

- Kamarck, T.W., & Lavallo, W.R. (2003). Cardiovascular reactivity to psychological challenge: Conceptual and measurement considerations. *Psychosomatic Medicine*, 65, 9-21.
- Kaplan, G.A., Wilson, T.W., Cohen, R.D., Kauhanen, J., Wu, M., & Salonen, J.T. (1994). Social functioning and overall mortality: prospective evidence from Kuopio ischemic heart disease risk factor study. *Epidemiology*, 5, 495-500.
- Kawachi, I., & Berkman, L. (2000). Social cohesion, social capital, and health. In L.F. Berkman & I. Kawachi (Eds.), *Social epidemiology* (pp. 174-190). New York: Oxford University Press.
- Kawachi, I., Colditz, G.A., Ascherio, A., Rimm, E.B., Giovannucci, E., Stampfer, M.J., Willet, W.C. (1996). A prospective study of social networks in relation to total mortality and cardiovascular disease in men in the USA. *Journal of Epidemiology & Community Health*, 50, 245-51.
- Kelsey, R.M. (1994). Habituation of cardiovascular reactivity to psychological stress: Evidence and implications. In J. Blascovich & S. Kaplan (Eds.), *Cardiovascular reactivity to psychological stress & disease*. Washington, DC: American Psychological Association.
- Kessler, R.C., Price, R.H., Wortman, C.B. (1985). Social factors in psychopathology. *Annual Review of Psychology*, 36, 531-572.
- Kiecolt-Glaser, J.K., McGuire, L., Robles, T.R., & Glaser, R. (2002). Emotions, morbidity, and mortality: New perspectives from psychoneuroimmunology. *Annual Review of Psychology*, 53, 83-107.

- Knox, S.S., & Unväs-Moberg, K. (1998). Social isolation and cardiovascular disease: An atherosclerotic pathway? *Psychoneuroendocrinology*, *23*, 877-890.
- Kop, W.J., Gottdiener, J.S., Patterson, S.M., & Krantz, D.S. (2000). Relationship between left ventricular mass and hemodynamic responses to physical and mental stress. *Journal of Psychosomatic Research*, *48*, 79-88.
- Kral, B.G., Becker, L.C., Blumenthal, R.S., Aversano, T., Fleisher, L.A., Yook, R.M. et al. (1997). Exaggerated reactivity to mental stress is associated with exercise induced myocardial ischemia in an asymptomatic high-risk population. *Circulation*, *96*, 4246-4253.
- Krantz, D.S., Kop, W.J., Santiago, H.T., & Gottdiener, J.S. (1996). Mental stress as a trigger for myocardial ischemia and infarction. *Cardiology Clinics*, *14*, 271-287.
- Krantz, D.S., & Manuck, S.B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. *Psychological Bulletin*, *96*, 435-464.
- Krantz, D.S., Sheps, D.S., Carney, R.M., Natelson, B.H. (2000). Effects of mental stress in patients with coronary artery disease. *JAMA*, *283*, 1800-1802.
- Krause, N., & Borawski-Clark, E. (1995). Social class differences in social support among older adults. *The Gerontologist*, *35*, 498-508.
- Kravdal, O. (2001). The impact of marital status on cancer survival. *Social Science and Medicine*, *52*, 357-368.
- Krieger, N. (2000). Discrimination and health. In L.F.Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 35-75). New York: Oxford University Press.



- Krieger, N., & Sidney, S. (1996). Racial discrimination and blood pressure: The CARDIA study of young black and white adults. *American Journal of Public Health, 86*, 1370-1378.
- Landrine, H., Richardson, J.L., Klonoff, E.A., & Flay, B. (1994). Cultural diversity in the predictors of adolescent cigarette smoking: The relative influence of peers. *Journal of Behavioral Medicine, 17*, 3331-3346.
- Lepore, S.J. (1998). Problems and prospects for the social support-reactivity hypothesis. *Annals of Behavioral Medicine, 20*, 257-269.
- Linden, W., Stossel, C., & Maurice, J. (1996). Psychosocial interventions for patients with coronary artery disease: a meta-analysis. *Archives of Internal Medicine, 156*, 745-752.
- Link, B.G., & Phelan, J. (1995). Social conditions as fundamental causes of disease. *Journal of Health and Social behavior, extra issue*, 80-94.
- Lund, R., Due, P., Modvig, J., Holstein, B.E., Damsgaard, M.T., & Anderson, P.K. (2002). Cohabitation and marital status as predictors of mortality-an eight year follow-up study. *Social Science & Medicine, 55*, 673-679.
- Lynch, J.W., Everson, S.A., Kaplan, G.A., Salonen, R., & Salonen, J.T. (1998). Does low socioeconomic status potentiate the effects of heightened cardiovascular responses to stress on the progression of carotid atherosclerosis? *American Journal of Public Health, 88*, 389-394.
- Malcolm, A.T., & Janisse, M.P. (1991). Additional evidence for the relationship between Type A behavior and social support in men. *Behavioral Medicine, 17*, 131-134.

- Malfatto, G., Facchini, M., Sala, L., Branzi, G., Bragato, R., & Leonetti, G. (1998). Effects of cardiac rehabilitation and beta-blocker therapy on heart rate variability after first acute myocardial infarction. *American Journal of Cardiology*, *81*, 834-840.
- Manuck, S.B., Kasprovicz, A.L., Monroe, S.M., Larkin, K.T., & Kaplan, J.R. (1989). Psychophysiologic reactivity as a dimension of individual differences. In N. Schneiderman, S.M. Weiss, & P.G. Kaufmann (Eds.), *Handbook of research methods in cardiovascular behavioral medicine* (pp. 365-382). New York: Plenum.
- Manuck, S.B., Olsson, G., Hjemdahl, P., & Rehnqvist, N. (1992). Does cardiovascular reactivity to mental stress have prognostic value in postinfarction patients? A pilot study. *Psychosomatic Medicine*, *54*, 102-108.
- Marmot, M. (2000). Multilevel approaches to understanding social determinants. In L.F. Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 349-367). New York: Oxford University Press.
- McAuley, E., Courneya, K.S., Rudolph, D.L., & Lox, C.L. (1994). Enhancing exercise adherence in middle-aged males and females. *Preventive Medicine*, *23*, 498-506.
- Meichenbaum, D., & Turk, D.C. (1987). *Facilitating treatment adherence*. New York: Plenum Press.
- Newcomb, M.D. (1990). What structural equation modeling can tell us about social support. In B.R. Sarason, I.G. Sarason, & G.R. Pierce (Eds.), *Social support: An interactional view* (pp. 23-63). New York: Wiley.

- Nishine, E.O., Cole, C.R., Blackstone, E.H., Pashkow, F.J., & Lauer, M.S. (2000). Heart rate recovery and treadmill exercise scores as predictors of mortality in patients referred for exercise ECG. *JAMA*, 284, 1392-1398.
- Oka, R.K., King, A.C., & Young, D.R. (1995). Sources of social support as predictors of exercise adherence in women and men ages 50 to 65 years. *Women's Health: Research on Gender, Behavior, and Policy*, 1, 161-175.
- O'Reilly, P. (1988). Methodological issues in social support and social network research. *Social Science and Medicine*, 26, 863-873.
- Orth-Gomer (1994). International epidemiological evidence for a relationship between social support and cardiovascular disease. In S.A. Shumaker & S.M. Czajkowski (Eds.), *Social Support and Cardiovascular Disease* (pp.97-117). New York: Plenum Press.
- Orth-Gomer, K., & Johnson, J.V. (1987). Social network interaction and mortality. A six year follow-up study of a random sample of the Swedish population. *Journal of Chronic Disease*, 40, 949-957.
- Pappas, G., Queen, S., Hadden, W., & Fisher, G. (1993). The increasing disparity between socioeconomic groups in the United States, 1960 and 1986. *New England Journal of Medicine*, 329, 103-109.
- Rodriguez, M., & Cohen, S. (1998). Social support. In H. Friedman (Ed.), *Encyclopedia of Mental Health*. New York: Academic Press.
- Rosengren, A., Orth-Gomer, K., Wedel, H., & Wilhelmsen, L. (1993). Stressful life events, social support, and mortality in men born in 1933. *British Medical Journal*, 307, 1102-1105.

- Rozanski, A., Blumenthal, J.A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*, 2192-2217.
- Rüddel, H, Langewitz, W., Schächinger, H., Schmieder, R., & Schulte, W. (1988). Hemodynamic response patterns to mental stress: Diagnostic and therapeutic implications. *American Heart Journal, 116*, 617-627.
- Sarason, B.R., Sarason, I.G., & Gurung, R.A.R. (1997). Close personal relationships and health outcomes: A key to the role of social support. In S. Duck (Ed.), *Handbook of personal relationships* (2nd ed., pp. 547-573). New York: John Wiley & Sons.
- Sarason, B.R., Shearin, E.N., Pierce, G.R., & Sarason, I.G. (1987). Interrelations of social support measures: Theoretical and practical implications. *Journal of Personality & Social Psychology, 52*, 813-832.
- Sarason, I.G., Levine, H.M., Basham, R.B., & Sarason, B.R. (1983). Assessing social support: The Social Support Questionnaire. *Journal of Personality and Social Psychology, 44*, 127-139.
- Sarason, I.G., Sarason, B.R., Brock, D.M., & Pierce, G.R. (1996). Social support: Current status, current issues. In C.D. Spielberger & I.G. Sarason (Eds.), *Stress and Emotion: Anxiety, Anger, and Curiosity, Vol 16. Series in Stress and Emotion* (pp. 3-27).
- Sarason, I.G., Sarason, B.R., & Pierce, G.R. (1989). Social support, personality, and performance. *Journal of Applied Sport Psychology, 2*, 117-127.

- Schoenbach, V., Kaplan, B.H., Fredman, L., & Kleinbaum, D.G. (1986). Social ties and mortality in Evans County, Georgia. *American Journal of Epidemiology*, 123, 577-591.
- Schwartz, A.R., Gerin, W., Davidson, K.W., Pickering, T.G., Brosschot, J.F., Thayer, J.F., Christenfeld, N., & Linden, W. (2003). Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine*, 65, 22-35.
- Spiegel, D., Kraemer, H.C., Bloom, J.R., & Gottheil, E. (1989). Effect of psychosocial treatment on survival of patients with metastatic breast cancer. *Lancet*, 2, 888-891.
- Stansfeld, S.A. (1999). Social support and social cohesion. In M.Marmot & R.G. Wilkinson (Eds.), *Social determinants of health* (pp.155-178). New York: Oxford.
- Statistics Canada (1999). *Statistical report on the health of Canadians*. Available online: <http://www.hc-sc.gc.ca/hppb/phdd/report/stat/eng/report.html>
- Strike, P.C., & Steptoe, A. (2003). Systematic review of mental stress-induced myocardial ischemia. *European Heart Journal*, 24, 690-703.
- Sundquist, K., Lindstrom, M., Malstrom, M., Johansson, S.-E., & Sundquist, J. (2004). Social participation and coronary heart disease: a follow-up study of 6900 women and men in Sweden. *Social Science & Medicine*, 58, 615-622.
- Syme, S.L. (2001). Understanding the relationship between socioeconomic status and health: New research initiatives. J.A. Auerbach & B.K. Krimgold (Eds.), *Income, Socioeconomic Status, And Health: Exploring the Relationships* (pp.12-15).

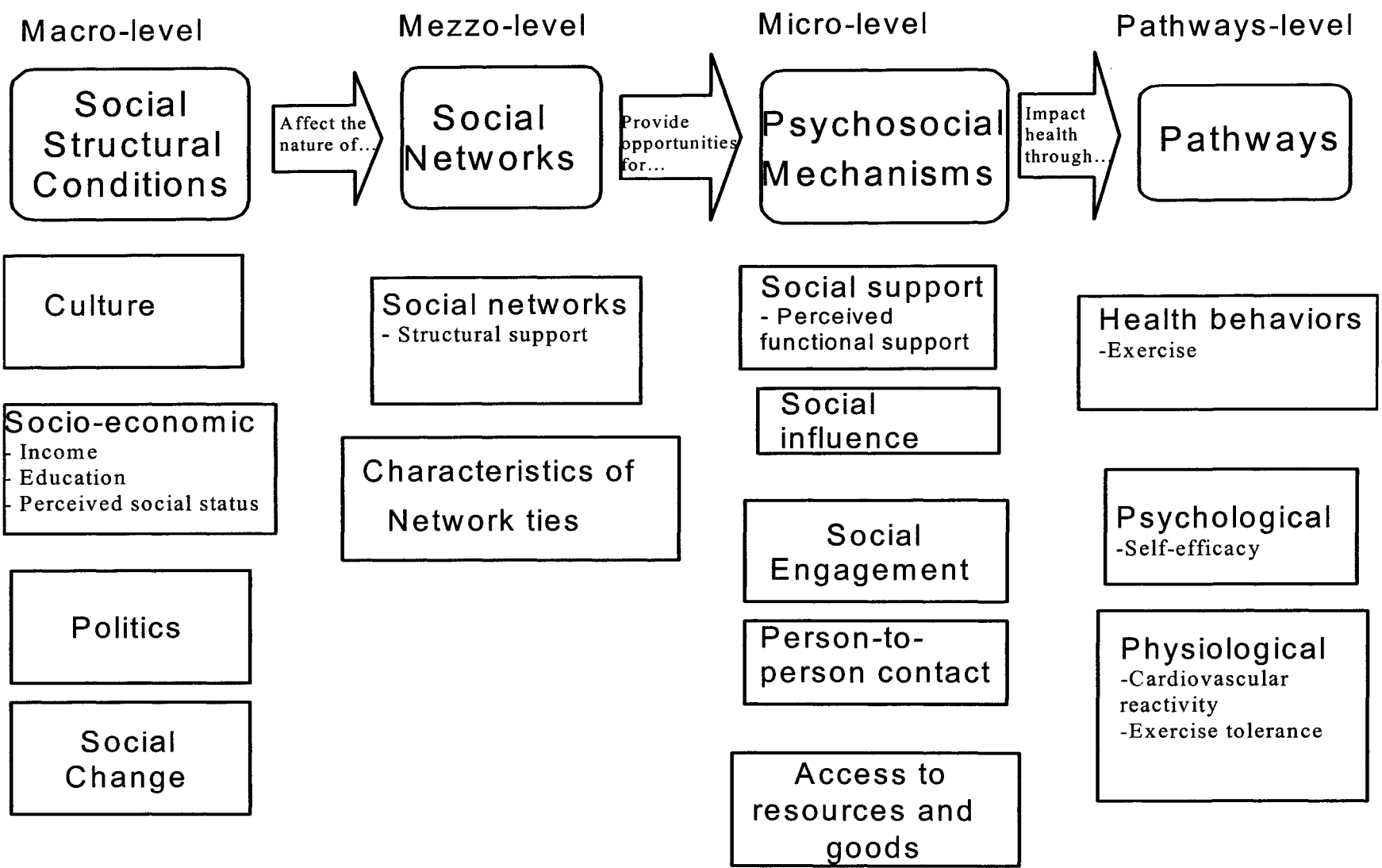
- Taylor, S.E., & Aspinwall, L.G. (1993). Coping with chronic illness. In L. Goldberger & S. Breznitz et al. (Eds.), *Handbook of stress: Theoretical & clinical aspects* (2nd ed., pp. 511-531). New York: Free Press.
- Thoits, P.A. (1995). Stress, coping, and social support processes: Where are we? What next? *Journal of Health and Social Behavior* (extra issue), 53-79.
- Trieber, F.A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical disease states. *Psychosomatic Medicine*, 65, 46-62.
- Turner, J.R. (1994). *Cardiovascular reactivity and stress: Patterns of physiological response*. New York: Plenum.
- Turner, R.J., & Marino, F. (1994). Social support and social structure: A descriptive epidemiology. *Journal of Health and Social behavior*, 35, 193-212.
- Uchino, B.N., Cacioppo, J.T., & Kiecolt-Glaser, J.K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin*, 119, 488-531.
- Uchino, B.N., & Garvey, T.S. (1997). The availability of social support reduces cardiovascular reactivity to acute psychological stress. *Journal of Behavioral Medicine*, 20, 15-27.
- Uden, A-L., Orth-Gomer, K., & Elofsson, S. (1991). Cardiovascular effects of social support in the work place: Twenty-four-hour-ECG monitoring of men and women. *Psychosomatic Medicine*, 53, 50-60.

- Vogt, T.M., Mullooly, J.P., Ernst, D., Pope, C.R., & Hollis, J.F. (1992). Social networks as predictors of ischemic heart disease, cancer, stroke and hypertension: Incidence, survival, and mortality. *Journal of Clinical Epidemiology*, 45, 659-666.
- Waldstein, S.R., Siegel, E.L., Lefkowitz, D., Maier, K.J., Pelletier Brown, J.R., Obuchowski, A.M. et al. (2004). Stress-induced blood pressure reactivity and silent cerebrovascular disease. *Stroke*, 35, 1294-1298.
- Weiss, R.S. (1974). The provisions of social relationships. In Z. Rubin (Ed.), *Doing Unto Others* (pp.17-26). Englewood Cliffs, NJ: Prentice Hall.
- Williams, D.R. (2001). Race and health: Trends and policy implications. In J.A. Auerbach & B.K. Krimgold (Eds.), *Income, socioeconomic status, and health: Exploring the relationships* (pp. 67-85). Washington, DC: National Policy Association.
- Williams, R.B., Barefoot, J.C., Califf, R.M., Haney, T.L., Saunders, W.B., Pryor, D.B., et al. (1992). Prognostic importance of social and economic resources among medically treated patients with angiographically documented coronary artery disease. *JAMA*, 267, 520-524.
- Yen, I.H., & Syme, S.L. (1999). The social environment and health: A discussion of the epidemiological literature. *Annual Review of Public Health*, 20, 287-308.

Figure caption

*Figure 1-1.* Berkman and Glass (2000) framework for examining social influences on health. Social factors range from upstream social demographic influences down to more proximal psychological/behavioral and physical influences on health.





### Figure caption

*Figure 1-2.* Relationships examined in Fraser, Rodgers, Murray, et al. (2005) within the Berkman and Glass (2000) framework. Upstream social status factors were related to social networks. Both were related to a prognostic indicator among cardiac patients. Arrows represent hypothesized and tested relationships in Fraser, Rodgers, Murray, et al.

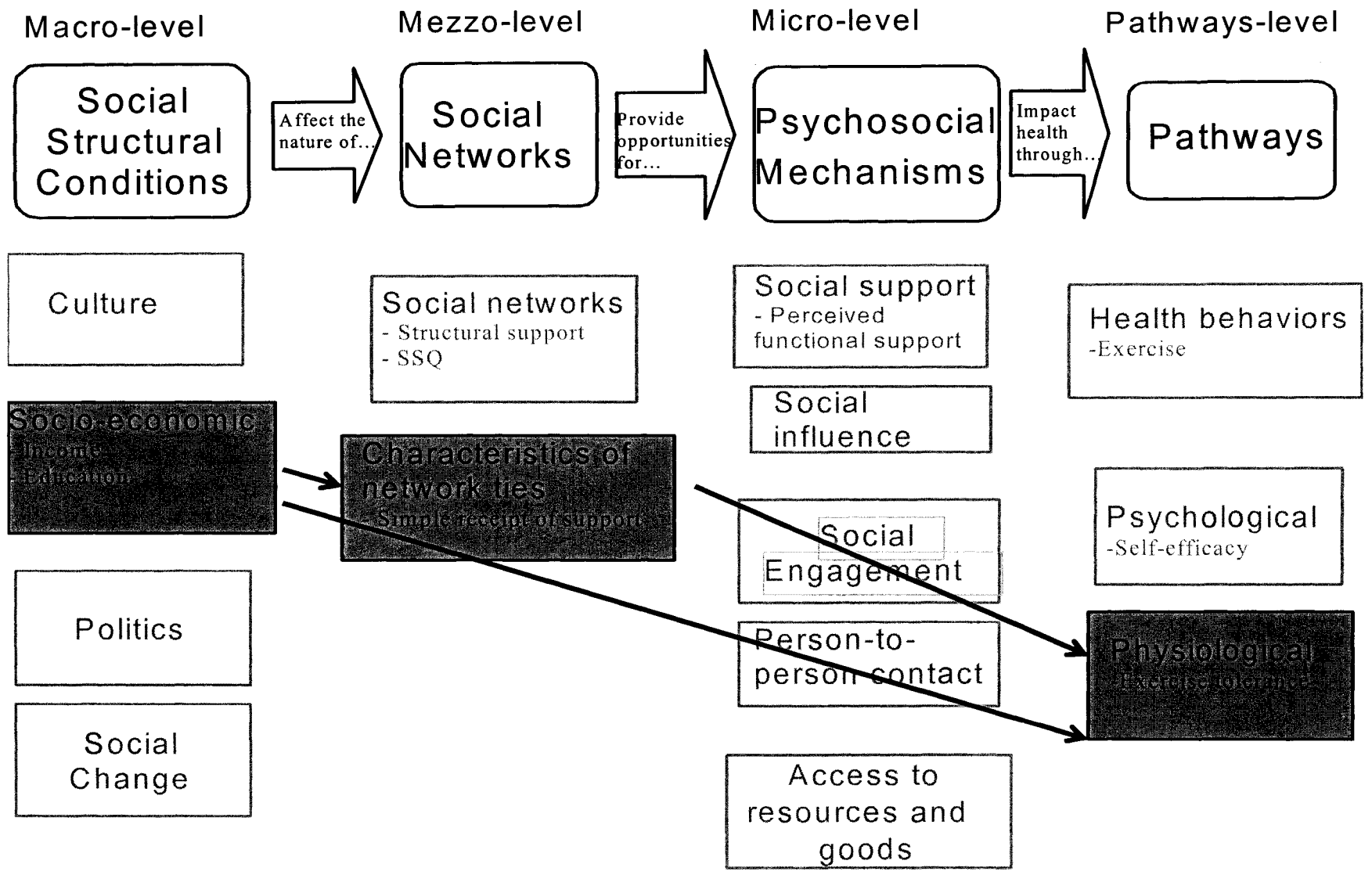


Figure caption

*Figure 1-3.* Relationships examined in Fraser, Rodgers, Daub, et al. (2005) within the Berkman and Glass (2000) framework. Results showed that social support at the micro-level was related to psychological pathway variables. Arrows represent hypothesized and tested relationships in Fraser, Rodgers, Daub, et al.

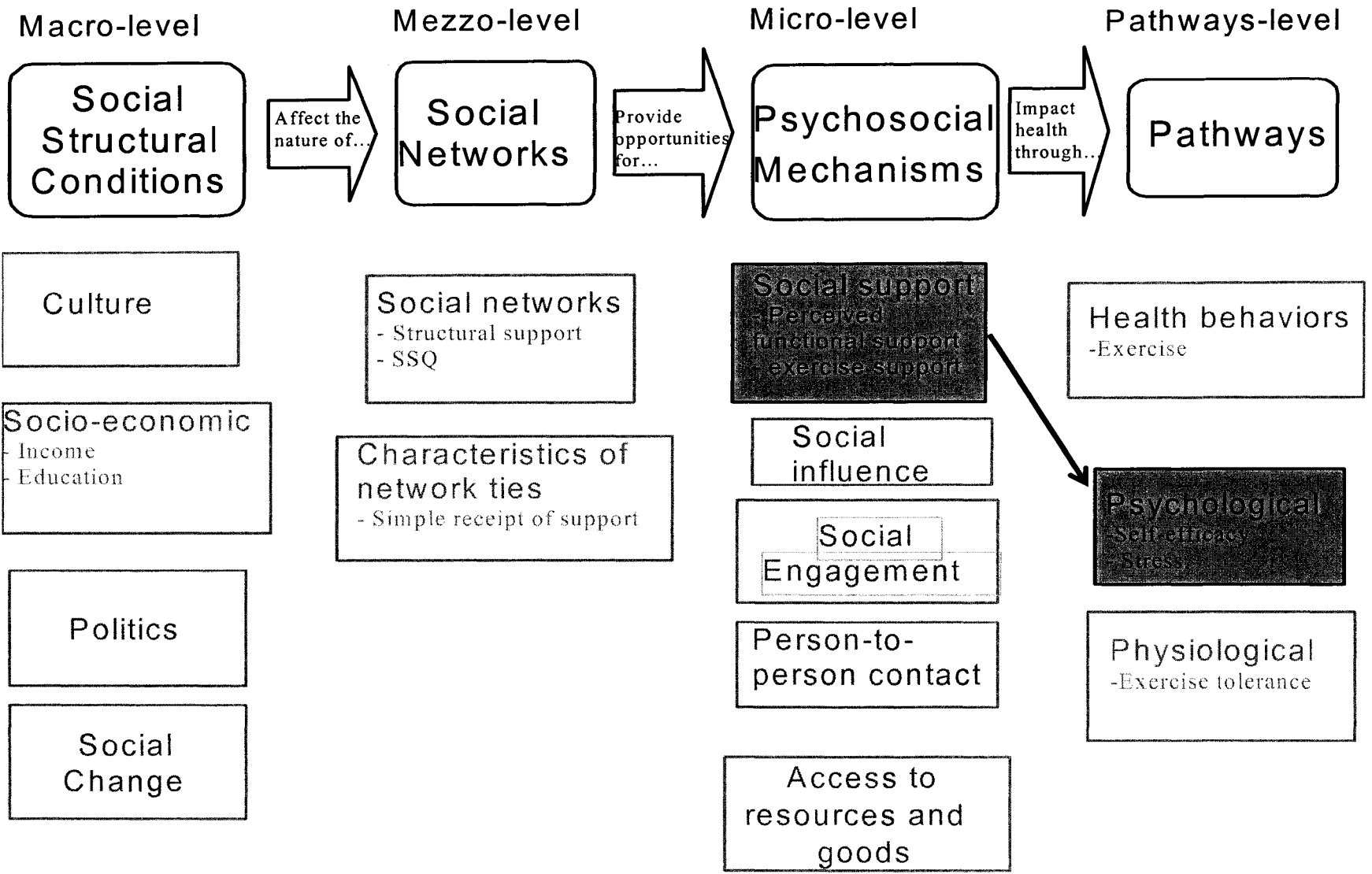
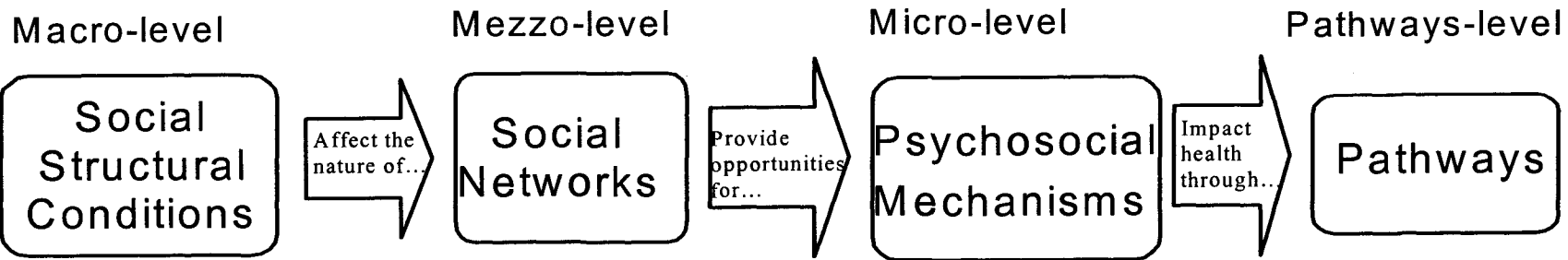


Figure caption

*Figure 1-4.* Relationships examined in Study 1 within the Berkman and Glass (2000) framework. Upstream social demographic factors are thought to lead to larger social networks, which should both be related to more social support at the micro level. Arrows represent hypothesized relationships tested in Study 1 of this dissertation.



Culture

Socio-economic  
- Income  
- Education  
- Subjective social status

Politics

Social Change

Social networks  
- Structural support

Characteristics of network ties  
- Simple receipt of support

Social support  
- Perceived  
- functional support

Social influence

Social Engagement

Person-to-person contact

Access to resources and goods

Health behaviors  
- Exercise

Psychological  
- Self-efficacy

Physiological  
- Exercise tolerance

Figure caption

*Figure 1-5.* Relationships examined in Study 2 within the Berkman and Glass (2000) framework. Micro-level social support is thought to be related to psychological pathway variables further down, which are related to physical pathway responses. Arrows represent hypothesized relationships tested in Study 2 of this dissertation.



Macro-level

Social Structural Conditions

Culture

Socio-economic  
- Income  
- Education

Politics

Social Change



Mezzo-level

Social Networks

Social networks  
- Structural support  
- SSQ

Characteristics of network ties  
- Simple receipt of support



Micro-level

Psychosocial Mechanisms

Social support  
- Perceived  
- functional support  
- SBI

Social influence

Social Engagement

Person-to-person contact

Access to resources and goods



Pathways-level

Pathways

Health behaviors  
- Exercise

Psychological  
- Self-efficacy  
- Stress  
- Anxiety

Physiological  
- Cardiovascular  
- Reactivity

## 2. STUDY 1

### Sociodemographic correlates of social networks and social support among people attending cardiac rehabilitation

Social support has been linked to prognosis for heart patients. That is, those reporting more social support typically survive longer (e.g., Berkman, Leo-Summers, & Horwitz, 1992; Williams et al., 1992), and function better (cf. Dracup, 1994; Fraser, Rodgers, Murray, & Daub, 2005). Yet, if social support is useful for heart patients it is important to understand what gives rise to social support. In other words, to understand how social support affects health in patients it is important to also understand the determinants of social support among those patients.

To this end, Berkman and Glass (2000) have developed a conceptual framework that outlines multiple levels of social influence on health (see Figure 1-1). The framework suggests that upstream macro and mezzo-level factors affect downstream micro and pathway-level factors. Thus, socioeconomic factors at the macro level are thought to influence the nature of one's social network, a mezzo-level factor. These network characteristics may include the size of one's network of family, friends, and other contacts, for example. Frequency of contact with network members is another example of an aspect of the social network. The nature of one's social network, or structural support (Cohen, 1988), should in turn influence the micro-level factor functional social support. Functional support (Cohen) is typically thought of as the resources available from social relationships. These may be in terms of emotional support, the sense of belonging, or more tangible in the form of resources such as money or transportation, for example. These

micro level factors, finally, should influence the more proximal pathways to health such as health behaviors and/or prognostic health determinants such as blood pressure.

Socioeconomic status (SES) is thought to be one of the most important determinants of health (Syme, 2001). Berkman and Glass (2000) have identified socioeconomic factors as a macro-level factor. For example, socioeconomic factors identified by Berkman and Glass include inequality, labor market structure, and poverty. However, it is apparent that these facets of socioeconomics can vary a great deal. For example, poverty may be identified as an individual factor, such as an individual's annual income. Conversely, labor market structure is a much higher-level factor perhaps applying to the level of a province or country. However, within a given city it would be expected that labor market structure might be fairly consistent. In other words, all Edmontonians would be subject to similar labor laws with the same minimum wage.

In a recent paper, Berkman and her colleagues (i.e., Sorensen et al., 2003) have outlined a conceptual model of how social context influences health behaviors. Characteristics such as age, social class, and gender are identified as factors shaping one's social context. In other words, some individual characteristics are representative of social status. Thus, these factors are viewed as upstream of other factors, like social networks and self-efficacy. This view is consistent with arguments of Grzywacz and Marks (2001) who adopt an ecological perspective in their examination of the influence of social inequalities on exercise behavior. Grzywacz and Marks outline an ecological model, similar to that of Sorensen et al., in which social status characteristics such as age, gender, education and income are furthest upstream giving rise to the characteristics of one's

community, work and family. Revenson (2003) similarly argues that these demographic factors fall within the sociocultural context in her ecological model.

In considering higher level effects on health the question of whether SES leads to poor health or if people with poor health somehow develop poor SES arises. The strongest evidence for this association comes from several large-scale longitudinal cohort studies like the Whitehall studies (e.g., Ferrie, Martikainen, Shipley, & Marmot, 2005; Marmot & Shipley, 1996). The studies consistently show that social inequalities are prospectively related to morbidity and mortality. Ferrie et al. recently reported on multiple follow-ups of economic difficulties. The authors examined economic difficulties from 1985-1988 (phase 1), 1989-1990 (phase 2), and 1992-1993 (phase 3) and examined the relationship to coronary events up to 1997-1999. Those reporting the most economic difficulties in all 3 phases were 2.5 times more likely to have non-fatal heart attacks and 2.8 times more likely have some other coronary event than those with the fewest economic difficulties. Further, the authors argue that this relationship is graded. That is, as the number of economic difficulties increases, the number of coronary events increases. Other studies have conducted long term follow-ups of socioeconomic position and health at multiple time points with similar results. For example, Lynch, Kaplan and Shema (1997) examined economic data from 1965, 1974, and 1983 to see the effect on physical, cognitive and social psychological functioning in 1994. Lynch et al. found that those who reported economic hardship at the most time points (i.e., in 1965, 1974, and/or 1983) had significantly poorer physical, psychological and cognitive health in 1994. Further, the authors specifically examined the issue of reverse causation (i.e., ill health leading to economic hardship) and found little evidence for this alternative explanation of the data.

Using the same data, Beebe-Dimmer et al. (2003) found that childhood and adult SES predicted 31-year mortality among women. Thus, the best evidence suggests that those with the lowest SES experience the highest morbidity and mortality rates later on. Those with the longest sustained economic hardship appear to be the worst off, but even early economic hardship appears to have a lasting effect.

Socioeconomic status has also been linked to social support, as seen in Fraser et al. (2005) and as implied by the Berkman and Glass (2000) framework. However, there is little consensus on how to conceptualize and operationalize SES (Bradley & Corwyn, 2002; Mishra, Ball, Dobson, Byles, & Warner-Smith, 2002; Steptoe & Marmot, 2002). Nevertheless, income, education, and subjective social status have been identified as useful conceptualizations of SES in previous studies (e.g., Adler, Epel, Castellazzo, & Ickovics, 2000; Ostrove, Adler, Kuppermann, & Washington, 2000; Singh-Manoux, Adler, & Marmot, 2003). While education and income have been identified previously, the notion of subjective social status is somewhat novel.

Subjective social status represents one's perception of his/her relative standing in society. Rossi and Berk (as cited in Oakes and Rossi, 2003) have argued that such a measure is perhaps a better representation of SES since it might be a better reflection of societal norms than more objective measures of SES. Further, the inclusion of individual level factors like age and gender seem to be useful when examining the notion of sociodemographic characteristics that are related to SES (cf. Grzywacz & Marks, 2001; Sorensen et al., 2003). Oakes and Rossi (2003) argue that such "fixed endowments of an actor" (p. 776) are an essential component of SES representing *human capital*. This approach to assessing social status or SES has recently been used by Adler et al. (2000),

Ostrove et al. (2000), Singh-Manoux et al. (2003), and Operario, Adler and Williams (2004). Ostrove et al. found subjective social status was related to traditional indicators of SES such as education and household income among pregnant Caucasian, Chinese, Latina, and African American women. Interestingly, subjective social status was more strongly related to income and education than income and education were related to each other. For example, the correlation between subjective social status and household income was .60, whereas the correlation between household income and education was only .30 among white women. This demonstrates some convergent validity in that subjective social status was related to other indices of SES. Subjective social status also demonstrated some predictive validity. Specifically, subjective social status was correlated with self-rated health. Regression analyses showed that subjective social status made unique contributions to the explanation of subjective health in white and Chinese women.

In another study of Caucasian women, Adler et al. (2000) found that subjective SES was related to a composite score of traditional SES factors and was meaningfully correlated with health indicators. That is, subjective social status was related to physical and psychological health indicators in the same way that more traditional SES factors have been related to health, supporting its predictive validity. For example, subjective social status was negatively related to resting heart rate, chronic stress, and pessimism. Regression analyses showed that subjective social status uniquely explained variance in heart rate, sleep latency, and several psychological health indices (e.g., chronic stress, pessimism, control over life) after controlling for objective SES and negative affect, adding some evidence for the construct validity of subjective social status.

Singh-Manoux et al. (2003) examined the association between subjective social status and traditional SES indicators in a large sample (N=6981) of men and women. The authors found that subjective social status was associated with other measures of SES such as employment grade, household income, and education, representing a high level of congruence between subjective and objective SES. Further, subjective status was a powerful predictor of health status. For example, those with lower subjective social status had higher rates of angina and diabetes. Singh-Manoux et al. concluded that this method of assessing subjective social status is a fair representation of other SES factors representing a cognitive averaging by participants that is not determined by psychological bias.

Fraser et al. (2005) found that socioeconomic factors and social support had an enduring impact on exercise tolerance among heart patients regardless of objective exercise behavior in CR. Those with more income and younger individuals had better exercise tolerance upon entry into CR, and 14 weeks and 9 months later. Further, it was found that those with higher SES also reported more social support. This finding was relevant both theoretically and practically. Theoretically, these results showed that a macro-level factor (income) was associated with mezzo-level factors (social network support), and that both SES and social support were prospectively related to a physical pathway to health: exercise tolerance. Practically, these results show that people with higher SES have more social support and both of these are related to exercise tolerance.

Within the Berkman and Glass (2000) framework social networks at the mezzo-level should be affected by SES factors. Specifically, higher SES should lead to a greater social network in terms of size, number of contacts and variety of different kinds of

contacts (Bradley & Corwyn, 2002). Previous research (e.g., Turner & Marino, 1994), and the framework, suggest that a larger network, or more structural social support, should give rise to more functional social support, which Berkman and Glass (2000) place at the micro-level. Thus, with a larger social network the functional aspects of support should be more readily available. However, this cascading mechanism from SES to social networks to social support has not been specifically examined among cardiac rehabilitation patients.

Thus, there is a need to further examine the relationships from higher-level to lower-level social factors. It is unclear what SES factors are related to one's social network and how SES factors and one's social network relate to different functional kinds of social support in cardiac patients. It may be that more education leads to a larger social network. A larger social network should provide more opportunities for emotional support than a smaller network. However, the relationship between income, education and other aspects of SES and social network characteristics is unclear. For example, might income eliminate the influence of a small social network on perceptions of tangible support? In sum, there is a need to examine how macro-level factors affect mezzo-level factors and how these macro and mezzo-level factors affect the micro-level social support factor.

The purpose of this study was to examine the macro and mezzo-level determinants of social support within the framework of Berkman and Glass (2000) among cardiac rehabilitation patients. This study examined the hypothesized cascade of influence from the macro to mezzo to micro-levels. Thus, it was thought that socioeconomic factors would affect the nature of one's social network, which would provide more opportunities for one's support needs to be satisfied. Specifically, higher socioeconomic status (i.e., income, education, and social status) was hypothesized to lead to larger social networks



(i.e., larger number of people in the network, and a more diverse network), which would lead to higher perceptions of the availability of functional forms of support (i.e., appraisal, tangible and belonging support).

### *Method*

#### *Participants*

In order to examine these relationships 33 women (mean age = 63.4, *SD* = 10.0) and 122 men (mean age = 60.3, *SD* = 11.3) were sequentially recruited from the regional cardiac rehabilitation program. This proportion of men and women is typical of this cardiac rehabilitation program and other programs in North America (e.g., Fraser et al., 2005, Blackburn et al., 2000; King et al., 2001; Missik, 2001). In this sample, the most common reasons for referral into this cardiac rehabilitation program were a recent angioplasty (61.3%), myocardial infarction (43.2%), bypass surgery (27.1%), and/or valve surgery (13.5%). The most common comorbidities in this sample included high cholesterol (65.2%), high blood pressure (53.5%), and arthritis/joint problems (36.1%).

The cardiac rehabilitation program is an individually tailored comprehensive approach with a focus on exercise. The basic program is 8 weeks in length but can be adjusted based on the needs and the progress of the patient as determined by the physician, nurses, and exercise specialists. Patients also had the option of attending a number of weekly educational classes on topics such as sexual health after a cardiac event, stress management, nutrition, and cardiac risk factor management. All patients were eligible to participate in the study.

#### *Measures*

*Sociodemographic factors.* Age, sex, marital status, and education were drawn from the patients' medical charts. Marital status was coded as "married" or "common-law" (1) or "single", "separated", "divorced", or "widowed" (0). Education was categorized as "less than high school" (1), "high school" (2), and "post-secondary" (3).

*Income* was assessed by asking participants to report their average annual household income before taxes. The 5 categories are reported in Table 2-1.

*Subjective social status.* Participants were shown a drawing of a 10 rung ladder and asked to place an 'X' on the rung that best described where they thought they stood on the ladder with respect to others in society. Operario et al. (2004, p. 242) reported adequate 6-month test-retest reliability of subjective social status. Thus, this method of assessing SES appears to capture traditional SES factors and is related to other constructs as would be expected theoretically. However, the ladder also appears to have the added advantage of being less invasive in terms of gathering personal information such as income and education. Finally, to reiterate the comments of Rossi and Berk (as cited in Oakes and Rossi, 2003) this particular approach may be a more representative indicator of SES since it might be a better reflection of societal norms than more objective measures of SES.

*Social networks* was assessed by Social Network Index (SNI; Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997). The SNI is an indicator of general structural social support. Participants were asked to complete 12 questions concerned with how many people they talked with at least every two weeks. The SNI distinguishes between network diversity and network size. Network diversity assesses the number of social roles one has

such as a spouse, a child, or a friend, for example. Network size refers to the number of people in one's social network.

The instrument seems to capture relevant aspects of one's social network (see for example Brissette, Cohen, & Seeman, 2000) including important roles such as, spouse, parent, children, other family and friends, for example. The instrument also captures the relative size of one's network and what domain the various network connections are from.

Cohen et al. (1997) have provided some predictive validity evidence for the SNI in their study of the common cold. Those with the most social ties were less susceptible to developing the common cold after introduction of a rhino-virus in experimental conditions. Further, a larger social network was a significant independent predictor of developing a cold after controlling for other relevant factors such as smoking, alcohol consumption, exercise, and sleep, for example. Cohen (1991) also provides cross sectional evidence that greater social network size was related to less smoking, alcohol consumption, and more exercise, sleep, better diet, and improved affect and self-esteem. These relationships between the SNI and these other constructs are consistent with the anticipated effects of social networks on health as seen in the above literature review offering some construct validity evidence.

*Social support* was assessed with the 12-item Interpersonal Support Evaluation List (ISEL; Cohen & Hoberman, 1983). The ISEL has been used previously with patients in this program and has exhibited adequate internal consistency reliability (Nunnally & Bernstein, 1994) with subscale scores ranging from .65 to .78. The ISEL is a 12-item scale with Likert-type ratings ranging from "definitely false" (1) to "definitely true" (4) assessing 3 different types of functional support. The three kinds of support are emotional,

tangible and belonging support. In this sample internal consistencies were: .50 for tangible, .73 for belonging, and .71 for appraisal. Nunnally (1967) originally argued that internal consistencies in the range of .50-.60 were adequate for initial research investigations, but increased the minimal recommendation to .70 (Nunnally, 1978). Thus, these values are acceptable for belonging and appraisal support, but low for tangible support. The results for tangible support should therefore be considered with caution, acknowledging sizeable attenuation of observed bivariate correlations between tangible support and other variables. However, these internal consistencies should be interpreted in light of the rather small number of items per scale. That is, with only 4 items per scale the internal consistencies would likely not be comparatively as large as longer scales unless each item was practically identical in content to other items in the scale. Further, internal consistency estimates are considered 'lower-bound' estimates (Onwuegbuzie & Daniel, 2002). Thus, the true reliability may be higher than the point estimate. Finally, in the case of tangible support, the value of .50 was not significantly different ( $F(150, 450) = .594, p > .10$ ) from the commonly accepted value of .70 (see Fan & Thompson, 2001, p. 523).

Cohen, Mermelstein, Kamarck, and Hoberman (1985) reported selected psychometric properties of the original ISEL, which contains more items than the ISEL-12 and an extra subscale, self-esteem support. Correlations with other support measures available at the time and the ISEL were .30 with the Moos family environment scale, .46 with number of close friends, and .42 with number of close relatives. However, Cohen et al. noted that the ISEL was developed to be conceptually different from existing measures, at the time, to capture functional social support. The ISEL subscales were related to self-esteem in a predictable manner. Specifically, the self-esteem subscale of the original ISEL

was related to the Rosenberg self-esteem scale with a correlation of .74, but the other subscales were only correlated at between .14 and .32 demonstrating some convergent and discriminant validity. Further discriminant validity evidence was offered where social desirability was not related to any of the ISEL subscales. Social support subscales were related to psychological symptoms in a predictable manner. For example, the ISEL subscales were correlated with depression as assessed by the Center for Epidemiological Studies depression (CES-D) scale ranging from -.29 to -.53. Total ISEL scores were correlated with the CES-D at -.52 and with the Kobassa psychological symptoms scale at -.57. The ISEL scores also predicted changes in physical symptomatology. Further, internal consistencies across 3 samples ranged from .70 to .82, .62 to .73, .73 to .78, and .73 to .81 for the subscales of the original ISEL. Two-day test-retest correlations ranged from .67 to .84 for the subscales and .87 for the total score. Six-week test retests in a different sample ranged from .63 to .69 for the subscales and .70 for the total scale score. Finally, subscale scores were correlated ranging from about .30 to .50, depending on the sample. For example, correlations between subscales ranged from .40 to .61 in a community smoking cessation study. One sample demonstrated high inter-factor correlations ranging from .44 to .81. This however, was not the norm among 9 different samples. The authors noted that the subscales should not be completely independent since multiple support functions are often available from the same people or from similar people within one's network.

Previous research in this cardiac program (Fraser et al., 2005) suggests that the subscales are relatively independent but related with correlations between subscales ranging from .57 to .59. Further, the subscales were meaningfully placed within a nomological network (Cronbach & Meehl, 1955), where the subscales were related

negatively to stress, for example, as predicted by social support theorizing with correlations ranging from  $-.23$  to  $-.37$ . Also, social support was related to self-efficacy with correlations ranging from  $.12$  to  $.20$ .

Green and Rodgers (2001) examined social support among low-income mothers using the ISEL-12. As part of the study Green and Rodgers examined the factor structure of the ISEL-12 and found an acceptable model fit for the 3-factor structure. Inter-factor correlations showed the subscales were related but relatively independent with correlations ranging between  $.48$  and  $.52$  at baseline and  $.58$  to  $.66$  one year later. The ISEL subscales were also meaningfully related to other theoretically relevant constructs in the manner expected. For example, correlations between stress and the social support subscales ranged from  $-.25$  to  $-.30$  at baseline and from  $-.35$  to  $-.38$  1 year later. Social support subscales were related to mastery/self-efficacy with correlations ranging from  $.22$  to  $.38$  and from  $.39$  to  $.46$  at baseline and 1 year later, respectively. Brookings and Bolton (1988) conducted a confirmatory factor analysis among college students that supported a three-factor solution with a higher-level general support factor.

### *Procedures*

Participants were sent the questionnaire package containing questions about income, social support, social networks and subjective social status along with the usual mailing of orientation materials. An information letter was sent along with the questionnaire package explaining the purpose of the study and inviting participants to participate in the study. Participants returned the completed package at their scheduled orientation session where they were given the opportunity to ask any questions they may have had.

## *Analyses*

An ordinary least squares approach to multiple regression was employed (i.e., coefficients based on minimizing sum of squares) rather than an iterative approach such as maximum likelihood approaches (Cohen, Cohen, West, & Aiken, 2003; Kleinbaum, Kupper, Muller, 1988). Specifically, hierarchical regression analyses were used to test the hypothesis that upstream factors were related to downstream factors. Thus, analyses examined how sociodemographic factors were related to one's social network in terms of network diversity and network size. Next, regression analyses examined how the sociodemographic factors and social networks were related to overall social support. Last, analyses examined the influence of sociodemographic factors and social networks on the three aspects of support.

After a significant regression equation was found, interpretation of results included examining the beta coefficients then examining the structure coefficients. Structure coefficients ( $r_{sc}$ ) were examined since regression analysis is considered part of the general linear model approach to data analyses (Cohen et al., 2003). Courville and Thompson (2001) suggest that interpretation of both beta coefficients and structure coefficients is therefore required in regression analyses (cf. Nunnally & Bernstein, 1994, p 191-192; Onwuegbuzie & Daniel, 2003). The advantage of structure coefficients is that, unlike beta coefficients, they are not affected by small relationships among independent variables. Even small relationships among independent variables can potentially mask relationships among some independent variables and the dependent variable. That is, one variable may arbitrarily be credited with a significant beta over another, despite similar bivariate relationships between both independent variables and the dependent variable (Courville &

Thompson, 2001). A structure coefficient was considered meaningful if it was greater than or equal to .30 in magnitude.

## *Results*

### *Data screening*

Data were screened for missing and unusual values by examining the means, ranges, and frequencies of responses to each survey item (Tabachnik & Fidell, 2001). Unusual values were those with ranges that were outside of possible response options. Missing and unusual values were checked with the original data and replaced with the appropriate value or confirmed as missing.

Some participants did not respond to all of the items on the ISEL, but the rate of missing data ranged from 2-8 missing values per item. For example, 8 responses were missing for item 4, and 2 responses were missing for item 9. For a sample of 155 people this was considered very low (cf. Tabachnik & Fidell, 2001). Missing data on each item was therefore replaced with the mean of the participant's responses for other items on the particular subscale (cf. Tabachnik & Fidell, 1996, p. 64). For example, if item 4 was missing, it was replaced by the mean of items 2, 6, and 11 (after the appropriate reverse scoring).

Data was also missing from 18.7% of participants on the question of income. Since this was considered a rather large proportion of the sample and since income was a critical variable, these people were eliminated from the analyses. A discriminant function coefficient with all of the study variables as predictors did not discriminate between those who did or did not report income. However, univariate F-tests suggested that those not reporting income were older ( $p < .01$ ) and had slightly less education ( $p < .01$ ).



Outliers were examined through typical procedures examining box-plots, histograms, z-scores, and ranking of the highest and lowest values of the study variables. Although consensus is lacking in terms of what constitutes an outlier and what procedures should follow the identification of an outlier, Osborne and Overbay (2004) suggest these typical procedures offer an acceptable starting point for examining outliers. The only variables raising concerns were the network measures. Specifically, network size ranged from 0 to 56, where the top 3 values in terms of size were 56, 51 and 40. These values were checked to determine if they were accurate and legitimate values. Since they were correct, they were not mindlessly eliminated. Instead, since outliers posed a potential source of bias, follow-up regression analysis was used to examine the potential influence of particular cases to identify any outliers in the multivariate analysis. Specifically, Cook's D and leverage values were calculated. Looking at the highest and lowest values, there were no cases identified as particularly influential. All Cook's D values were well below 1 and the leverage values were all low and distributed with a positive skew as expected (Kleinbaum et al., 1988). Thus, the impact of any potential outliers was thought to be minimal.

#### *Regression assumptions*

Data were assessed to see if they met some of the basic assumptions of linear regression analyses. There are many assumptions of regression analyses that need to be met in order to make inferences from a sample to a population. Common assumptions include 1) existence of Y (dependent variable) for any value of X (independent variable), 2) independence of the observations of Y and error of Y, 3) Y is linear function of the independent variables, 4) Y and the error of Y are normally distributed (for any X), 5)

constant variance in Y for any X (homoscedasticity), correct specification of independent variables (relevant variables included, irrelevant variables excluded), and 6) perfectly reliable measurement. Many of these assumptions are robust with respect to moderate violations however (Berry, 1993; Cohen et al., 2003; Kleinbaum et al., 1988; Osborne & Waters, 2002). Assumptions surrounding the normality of the independent variables (X's) and dependent variable (Y) are not strictly required except for inference making.

However, Osborne and Waters suggest that the requirement for the normal distribution of errors is particularly robust (see also Cohen et al.) and Kleinbaum et al. (1988) suggest that only serious departures from the normality in Y (i.e., the dependent variable in a regression analysis) are considered worrisome. In terms of homoscedasticity (constant variation of residuals), Kleinbaum et al. suggest that mild violations have a minor impact on results and Cohen et al. suggest that only a large degree of heteroscedasticity will have any effect (see also Osborne & Waters, 2002). Further, the effect is not to bias the regression coefficients, but to bias standard errors and consequently impact on confidence interval estimation and statistical tests.

The proper identification of the model and the independence of observations assumptions are accounted for in study design. In this study, the variables selected are determined from a review of the literature. However, since the study also sought to identify important determinants of social support, it would not be possible to suggest that relevant variables were not excluded. Further, there is no guarantee that all relevant variables were included given the goals of this study. However, as Cohen et al. (2003) note, it is not always clear what variables should and should not be included (see also Thompson, 1999). In terms of independence of observations, each dependent variable

represents information from one participant at one point in time. Cohen et al. (2003) suggest that data with different clusters (or groups) and repeated measures data are likely sources of violations of independence of observation. Since data was collected from a fairly homogeneous group (i.e., all patients in cardiac rehabilitation) at only one point in time, observations were considered independent.

Given the above discussion several assumptions were actively tested for severe violations. Specifically, normality, linearity, reliability of measures and homoscedasticity assumptions were tested. These were chosen since they seemed to be least controllable with respect to study design (cf. Osborne & Waters, 2002), they are generally possible to examine in most current statistical software packages, and these assumptions seem to be the most commonly mentioned (see for example Cohen et al., 2003; Kleinbaum et al., 1988; Osborne & Waters, 2002; Stevens, 2002; Tabachnik & Fidell, 1996, 2001).

#### *Examining assumptions*

Assumptions of normality of continuous variables were first tested by visually inspecting the distribution of variables, cumulative, or normal, probability plots (P-P plots) and by examining skewness and kurtosis (Osborne & Fidell, 2002; Kleinbaum et al., 1988). Examination of the histograms showed that age, network diversity, network size and SES ladder were fairly normally distributed. Total social support, and social support subtypes ranged in negative skewness from -1.127 to -.598. Examination of P-P plots, however, revealed no major departures from normality (Kleinbaum et al. acknowledge that this is necessarily a subjective and qualitative evaluation and Tabachnik & Fidell [2001] suggest that P-P plots are more useful than examining histograms, p. 75).

Skewness values reached a maximum of 1.08 in magnitude and kurtosis reached a highest

value of 2.47. Number of people in the network had the highest skewness and kurtosis, both being positive and not large enough to warrant any concerns. Skewness values for social support ranged from -.77 to -.104. Tabachnik and Fidell suggest that with samples over 100 underestimation of variance associated with positive kurtosis disappears. Skewness is thought to only have a minor impact in large samples like this one and skewness might not make much of an impact anyway according to Stevens (2002).

Regression diagnostic procedures were used to further examine the issue of normality in terms of examination of residuals with total social support and social support subtypes used as the dependent variables. That is, residuals were plotted against different values of X for all independent variables and different support subtypes. Compared to the univariate distribution of total social support, the standardized residuals had a very normal (i.e., Gaussian) looking distribution. The normal P-P plot looked very good as well. Similarly, residuals were nicely distributed when social support subtypes were used as dependent variables.

An advantage of examining the various plots of residuals is that information about the linearity and homoscedasticity assumptions can be obtained in addition to information about normality (Cohen et al., 2002). Residual plots showed that relationships were likely linear. In other words, there appeared to be no systematic relationship between the residuals and any independent variables. The data points were distributed roughly equally above and below 0 with no apparent slope or curvilinear relationship. Residual analyses is thought to be a preferred method of examining the linearity assumption especially if multiple regression is to be the ultimate analysis of choice (Osborne & Watters, 2002; cf. Cohen et al. 2003; Tabachnik & Fidell, 2001). Thus, keeping in mind the general

robustness of regression analysis with respect to violations of the normality assumption, there appeared to be no major violations of normality.

Residual plots also offer some support for the assumption of homoscedasticity. Again, keeping in mind the robustness of regression analysis, the plots offered no evidence of major departures from homoscedasticity. That is, plotting residuals versus standardized values of the various independent variables showed that variability was relatively constant throughout the range of values of the independent variables.

In terms of perfectly reliable measurement, internal consistency estimates were reported where relevant. Of course, some measurement error is assumed to exist for all measures including age and gender, for example. Age and gender were double checked by getting this data from 2 separate sources. Any discrepancies were resolved by examining the medical charts. Issues of reliability were addressed in the measures section.

### *Descriptive results*

Table 2-1 reports demographics, comorbidity, and admitting diagnosis of the participants. As seen in Table 2-1 a large proportion of the participants were men (78.7%) reflecting the typical composition of cardiac rehabilitation programs in North America (e.g., Missik, 2001). Table 2-1 also shows that the most common procedures/events prior to CR were angioplasty, a heart attack (MI), or bypass surgery. Men and women differed in terms of comorbidities, with a significantly higher proportion women having high blood pressure ( $\chi^2(1) = 4.40, p < .05$ ). Interestingly, 27.1% of the sample reported a household income greater than \$80,000 per year, and only 18.7% of the sample declined to report their income. However, men and women differed in terms of income distribution ( $\chi^2(4) = 12.69, p < .05$ ). Table 2-1 shows that most women report incomes in the lower range (\$40-

59,999 and below) of the income distribution, whereas most men report incomes in the upper range (\$40-59,999 and above). A significantly larger proportion of men than women were married ( $\chi^2(1) = 22.91, p < .001$ ).

Table 2-2 reports descriptive statistics of the study variables and the correlations among study variables is reported in Table 2-3. Men reported being higher on the subjective social status ladder, and men reported more total social support and more appraisal, tangible and belonging support.

#### *Regression analyses predicting social networks*

Table 2-4 shows the results of the regression predicting social network diversity from sociodemographic variables. Overall the sociodemographic factors explained 22% ( $p < .001$ ) of the variance in network diversity. Only subjective social status and income had significant betas ( $ps < .05$ ). Specifically, those with higher incomes and those who reported being high on the social ladder reported having higher network diversity. Age and marital status did not quite reach statistical significance, but the structure coefficients were  $-.53$  and  $.58$ , respectively. Thus, older individuals reported lower diversity and married individuals reported more diversity. Both betas and structure coefficients show that education and gender did not contribute to the regression equation.

The results of the regression predicting social network size are reported in Table 2-5. The sociodemographic variables explained 21% ( $p < .001$ ) of variance in network size. Both subjective social status and income were positively related to network size ( $ps < .05$ ). Education was not quite significantly ( $p = .067$ ) negatively related to network size. The structure coefficients, however, suggest little relationship between education and network size. Age, gender and marital status all have structure coefficients greater than  $.30$

suggesting they make contributions to explaining network size. Age was negatively related to network size, whereas men and married individuals reported larger networks.

#### *Regression analyses predicting functional social support*

The first regression examined the relationship between sociodemographic factors and social networks and total social support. As seen in Table 2-6, sociodemographic factors were entered first, followed by social networks. Sociodemographic factors explained 25% of the variance in social support and the significant addition of social networks ( $p = .037$ ) resulted in a regression equation explaining 28% of variance in social support. At step 1, only subjective social status and marital status had significant betas, where higher social status was related to more support and married individuals had more support. At the second step social networks added significant variability, but the betas were not significant. The network size was not quite significant ( $p = .073$ ). Structure coefficients confirmed that marital status and subjective social status were relevant contributors to the explanation of social support. However, the structure coefficients suggest that those with higher income and men reported more social support. Further, the impact of network size ( $r_{sc} = .63$ ) was comparable to subjective social status ( $r_{sc} = .70$ ), and network diversity ( $r_{sc} = .56$ ) contributed as well.

The results of the regression examining appraisal support is reported in Table 2-7. Step 1 shows the sociodemographic variables explained 16% of variance in appraisal support and step 2 shows the addition of social networks results in an equation explaining 19% of variance. The significance of the addition of social networks was  $p = .055$ . At step 1 and 2, the only significant beta was subjective social status, positively related to appraisal support. At step 1 being married or common-law was not quite significantly ( $p =$

.068) related to more appraisal support. However, examination of the structure coefficients shows a pattern of relationships similar to total support. Specifically, the variables with the largest structure coefficients were subjective social status ( $r_{sc} = .81$ ) and network diversity ( $r_{sc} = .61$ ) and size ( $r_{sc} = .59$ ). Further, being male, married and having higher income appear to be related to more appraisal support.

The results of the regression examining belonging support is reported in Table 2-8. Results show that the sociodemographic variable explain 22% ( $p < .001$ ) of variability in belonging support and that social networks do not significantly add to the explanation of belonging support. Both subjective social support and marital status were significantly related to belonging support at both steps. Those who were married or common-law or reported higher social status reported more belonging support. Results of the regression determining belonging support showed that marital status ( $r_{sc} = .77$ ) and subjective social status ( $r_{sc} = .64$ ) had the largest structure coefficients. However, network diversity and size contribute the next most to the equation with structure coefficients of .56 and .58, respectively. Being male or having higher income was also associated with more belonging support.

Finally, the results of the regression examining tangible support is reported in Table 2-9. Step 1 shows the sociodemographic variables explained 17% ( $p < .001$ ) of variance and that the social networks added significant variability ( $p = .027$ ) with a final equation explaining 21% of variability in tangible support. At step 1 and 2, subjective social status and marital status had significant beta weights, where married/common-law individuals or those higher in social status has more tangible support. At step 2, network size was significantly positively related to tangible support. Structure coefficients confirm



that network size ( $r_{sc} = .63$ ), subjective social status ( $r_{sc} = .58$ ) and marital status ( $r_{sc} = .67$ ) were the most important predictors of tangible support. However, being male, having higher income, or more diversity was also related to more tangible support.

### *Discussion*

This study to examined correlates of social support among patients enrolled in cardiac rehabilitation. The results showed that certain sociodemographic factors were related to social network size and diversity. Second, social network characteristics were related to different aspects of functional social support.

#### *Social network characteristics*

These results support the idea that sociodemographic factors influence the nature of one's social network. Both the diversity of one's network and the overall size of one's network were related to sociodemographic factors. Beta coefficients for income and subjective social status were both significant for network diversity and network size. Structure coefficients (and correlations) suggest that age and marital status also contributed. Specifically, for both network diversity and size older people had smaller networks and married people had larger networks. This effect was larger for diversity as seen in the larger structure coefficients. For network size, it appeared that men reported larger networks based on the structure coefficient. The results differ from Mickelson and Kubzansky (2003) who found that women reported more contact with family and friends than men. However, compared to Mickelson and Kubzansky the results from this study are from an older Canadian sample in cardiac rehabilitation composed primarily of men. Further, the measure of friend and family contact in Mickelson and Kubzansky's study was less specific than the SNI, with only 2 questions assessing extent of contact with 1)

friends and 2) family. However, another explanation might be the relationship between age and marital status and social networks. In this sample, women were considerably older and were less likely to be married (55% women married vs. 90% men married). Thus, the fact that men reported larger and more diverse networks might reflect their younger age and higher likelihood of being married. Generally these results support previous research and theorizing suggesting that sociodemographic factors affect social network size. For example, Turner and Marino (1994) found that people with higher income and education had more contact with network members. In this case, income and social status were related to having a larger and more diverse network. These results expand on previous research examining sociodemographic influences on social networks.

Interestingly, these results show that both income and subjective social status contributed to the explanation of network size and diversity. While somewhat correlated, each contributed to the regression equations with significant beta weights. This is interesting since it was not clear if the shared variation between income and subjective social status would mask the impact of one of these factors over the other (cf. Courville & Thompson, 2001). The results here support the notion of assessing social status subjectively to gain additional insight into the question of how sociodemographic factors might affect health supporting previous research. These results also showed that subjective social status was more important than education in explaining network diversity and size. These results expand on the notion that social status is related to social network contact by showing the importance of perceived social status.

#### *Social support functions*

In terms of explaining total social support, subjective social status and marital status were significant sociodemographic correlates of social support. Structure coefficients showed that men and those with higher income also reported more support. Adding network diversity and size added significantly to the explanation of support but only size of network was close to significant. The lack of significance may have been due to inadequate power since the beta was .24 and the *p*-value was .073. Assuming the size of this effect is stable, a larger sample size would have enabled significant detection of this effect. Another problem could be that network diversity and size were too closely related to each contribute significant betas. Similarly, the strong and stable relationships between income, subjective social status and social networks would result in attenuating the relationship between social networks and social support. Nevertheless, both diversity and size had sizable structure coefficients and were both positively related to total social support.

Thus, people with larger and more diverse networks reported more overall social support. This is consistent with previous research generally showing more support from larger networks. For example, Seeman and Berkman (1988) found that availability of support was related to network size, number of contacts, and number of close ties. In general these results show that sociodemographic factors and social networks were related to total social support. However, these results also show that accounting for social networks results in subjective social status remaining related to social support. Again, being male and married also was related to social support. This might reflect the older age of the women and the fact that fewer women were married. That is, the relationship between gender and support might be confounded by the fact that the women in this

program were generally older and fewer women were married. Future research needs to address whether social support per se is important for women's health, if age and marital status are critical demographic influences on health, or if social support is important in combination with age and marital status.

In terms of the specific aspects of social support, subjective social status was consistently related to higher support. That is, those reporting higher social status had more appraisal, belonging, and tangible support available to them. In fact, subjective social status had the only significant beta in the equation examining appraisal support (having a person to talk to about problems). However, network diversity and size were the next most important predictors, with income and marital status making meaningful contributions as well. That is, those with more diverse and larger networks had more appraisal support available. These results however suggest that one's subjective social status is important in determining the availability of someone to talk to about one's problems and that having more people in one's network also helps to have someone available to talk to. These results compare favorably with Mickelson and Kubzansky (2003) who found that people reporting more income and education reported more emotional support. The finding that social networks were related to more support is consistent with Seeman and Berkman (1988) who found that having more social ties was related to more social support.

Belonging support refers to the availability of people to engage in social activities with. The sociodemographic factors most important for this type of support were subjective social status and marital status. Thus, higher social status and being married seemed to be important in determining if one had people available to socialize with. In

terms of network characteristics, diversity and size of network did not have significant beta coefficients, but their structure coefficients were the next largest. Thus, more diversity and larger size were somewhat related to more belonging support. This is consistent with Berkman and Glass' (2000) notion that knowing more people should increase one's opportunities, in this case to socialize. Income and gender appeared to be related to belonging support as well. More income and being male was associated with increased reported belonging support. Again, the men in this sample were younger than women and for the most part were married. Simply being married might provide more of a sense of belonging. This is interesting since marital status did not contribute to network size to the extent that marital status contributed to support. That is, marital status seemed to have a direct effect on belonging support, whereas for network size or diversity marital status did not directly contribute.

Finally, tangible support refers to the availability of material aid. Higher subjective social status and being married were related to more tangible support. Being male and having higher social status also contributed to reporting more tangible support. Unlike appraisal and belonging support, social networks were significantly related to tangible support. Specifically, network size had a significant beta that was larger than any other variable. The structure coefficient was also large suggesting that network size was a key contributor to feeling one had tangible support available. This relationship is consistent with Berkman and Glass' (2000) argument that having more people in one's network should theoretically create more opportunities to meet this particular support need.

Overall these results support the idea that social networks influence downstream functional social support. However, the results here also showed that certain

sociodemographic factors were critical in explaining certain downstream social factors like social support types. In fact, select upstream factors affected functional social support both directly and indirectly. Interestingly subjective social status was consistently related to each aspect of social support. Evidently this aspect of sociodemographics is related to one's perceptions of available support. A plausible explanation for this comes from the Berkman and Glass (2000) model which suggests that upstream factors (i.e., social status) influence the nature of one's social network (i.e., network size), which influences opportunities to meet support needs. However, social status remained significant even after the addition of social networks. Arguably, there is a slight yet consistent attenuation of this relationship when looking at the beta coefficients once network characteristics are added. Nevertheless, social status appears to have a direct impact on social support types. Another explanation might be that those with higher social status should theoretically have a support system with a similar social status, since upstream factors affect the nature and composition of the network. Thus, higher social status acquaintances should have more freedom to socialize, be more able to provide material aid, and be more available to talk to. These aspects of one's network (e.g., extent of contact, homogeneity) were not addressed here, but this could explain why social status was so strongly related to support irrespective of network diversity and size. These results seem to confirm other research showing that sociodemographic factors influence lower level social factors (e.g., Mickelson & Kubzansky, 2003).

Another interesting finding was that marital status was more important for belonging and tangible support than for appraisal support. Appraisal support refers to the availability of someone to talk to, and being married seems to provide a guarantee that

someone should be available. Yet, social status and more diverse and larger networks were more important for appraisal support. Seeman and Berkman (1988) found that having a spouse was important for emotional support, but for those without a spouse a confidant was important. This might be a consideration in this data since the only variability around marital status was among women. That is, people might be obtaining these support needs from others in the social network. However, being married was most important for belonging support and arguably as important for tangible support as network size. Belonging support was related to network size and diversity as well, but being married seemed to ensure that someone was available to socialize with. In order to meet one's tangible support needs it seemed to be helpful to be married, but having a larger network also helped one feel that aid was available if required. This needs to be investigated in future research since most men were married anyway. So it may be that the marriage results simply reflect gender differences.

A number of limitations affect the generalizability of these findings. First, this study was conducted with a sample of cardiac patients attending rehabilitation and not everyone who attended the CR program chose to participate in this research. The primary concern is how these results might generalize to other heart patients. Since most heart patients eligible for CR do not end up at CR, it is not apparent if these results will generalize. Related, in this study most participants were men. The small number of women in this study made it necessary to examine men and women at the same time. Gender was entered into the regression equations gaining some insight into potential gender effects. However, ideally women and men should be examined separately since there is reason to believe that they experience heart disease differently (Tobin et al., 1987)

and respond differently to interventions (Jiang, Glassman, Krishnan, O'Connor, & Califf, 2005). A further problem is that a far larger proportion of patients are men than women. The proportion of men and women in this sample is typical of the composition of CR programs in North America (Blackburn et al., 2000; Grace et al., 2002; King et al., 2001; Missik, 2001). Nevertheless, the lack of women limits generalizability and future research needs to examine gender differences further and should take extra efforts to recruit women.

Another limitation is the poor reliability of the tangible social support subscale. This low level of reliability likely attenuated the bivariate relationships among the variables in this study. The effect on the multivariate relationships is unknown. Thus, the results for tangible support should be interpreted with caution until they can be replicated. Future research could examine the psychometric properties of this social support scale further. It may be necessary to add items or to use a longer support scale to improve reliability. However, the results might be sample specific.

These results offer support for the idea that higher-level social factors influence social factors downstream. The general suggestion that sociodemographic factors influence social network factors is supported here, as well as the idea that network characteristics influence social support functions downstream, supporting the framework of Berkman and Glass (2000). However, these results highlight some specific ways in which certain sociodemographics factors might be related to network characteristics. These results also suggest how specific support functions might be influenced by upstream network characteristics and sociodemographic factors directly.



## References

- Adler, N.E., Epel, E., Castellazzo, G., & Ickovics, J. (2000). Relationship of subjective and objective social status with psychological and physiological functioning: Preliminary data in healthy white women. *Health Psychology, 19*, 586-592.
- Beebe-Dimmer, J.L., Lynch, J.W., Turrell, G., Lustgarten, S., Ragnathan, T., & Kaplan, G.A. (2004). Childhood and adult socioeconomic conditions and 31-year mortality risk in women. *American Journal of Epidemiology, 159*, 481-489.
- Berkman, L.F., & Glass, T. (2000). Social integration, social networks, social support, and health. In L.F. Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 137-173). New York: Oxford University Press.
- Berkman, L.F., Leo-Summers, L., & Horwitz, R.I. (1992). Emotional support and survival after myocardial infarction: A prospective, populations-based study of the elderly. *Annals of Internal Medicine, 117*, 1003-1009.
- Berry, W.D. (1993). *Understanding regression assumptions*. Newbury Park: Sage.
- Blackburn, G.G., Foody, J.M., Sprecher, D.L., Park, E., Apperson-Hanson, C., & Pashkow, F.J. (2000). Cardiac rehabilitation participation patterns in a large, tertiary care center: Evidence for selection bias. *Journal of Cardiopulmonary Rehabilitation, 20*, 189-195.
- Bradley, R.H., & Corwyn, R.F. (2002). Socioeconomic status and child development. *Annual Review of Psychology, 53*, 371-399.
- Brissette, I., Cohen, S., & Seeman, T.E. (2000). Measuring social integration and social networks. In S. Cohen, L. G. Underwood, & B. H. Gottlieb (Eds.), *Social support*

*measurement and intervention: A guide for health and social scientists*. New York: Oxford.

- Brookings, J.B., & Bolton, B. (1988). Confirmatory factor analysis of the Interpersonal Support Evaluation List. *American Journal of Community Psychology, 16*, 137-147.
- Cohen, J., Cohen, P., West, S.G., & Aiken, L.S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah, NJ: Erlbaum.
- Cohen, S. (1988). Psychosocial models of the role of support in the etiology of physical disease. *Health Psychology, 7*, 269-297.
- Cohen, S. (1991). Social supports and physical health: Symptoms, health behaviors and infectious disease. In A.L. Greene, M. Cummings, & K.H. Karraker (Eds.), *Life-span developmental psychology: Perspectives on stress and coping* (pp. 213-234). Hillsdale, NJ: Erlbaum.
- Cohen, S., Doyle, W.J., Skoner, D.P., Rabin, B.S., & Gwaltney, J.M. (1997). Social ties and susceptibility to the common cold. *JAMA, 277*, 1940-1944.
- Cohen, S., & Hoberman, H.M. (1983). Positive events and social supports as buffers of life change stress. *Journal of Applied Social Psychology, 13*, 99-125.
- Cohen, S., Mermelstein, R., Kamarck, T., & Hoberman, H.M. (1983). Measuring the functional components of social support. In I. G. Sarason & B. R. Sarason (Eds.), *Social support: Theory, research, and application*. The Hague, Holland: Martinus Nijhoff.

- Courville, T., & Thompson, B. (2001). Use of structure coefficients in published multiple regression articles:  $\beta$  is not enough. *Educational and Psychological Measurement*, *61*, 229-248.
- Cronbach, L.J., & Meehl, P.E. (1955). Construct validity in psychological test. *Psychological Bulletin*, *52*, 281-302.
- DiMatteo, M.R. (2004). Social support and patient adherence to medical treatment: A meta-analysis. *Health Psychology*, *23*, 207-218.
- Dracup, K. (1994). Cardiac rehabilitation: The role of social support in recovery and compliance. In S.A. Shumaker & S.M. Czajkowski (Eds.), *Social Support and Cardiovascular Disease* (pp. 333-353). New York: Plenum Press.
- Fan, X., & Thompson, B. (2001). Confidence intervals about score reliability coefficients, please: An *EPM* guidelines editorial. *Educational and Psychological Measurement*, *61*, 517-531.
- Ferrie, J.E., Martikainen, P., Shipley, M.J., & Marmot, M.G. (2005). Self-reported economic difficulties and coronary events in men: evidence from the Whitehall II study. *International Journal of Epidemiology*, *34*, 640-648.
- Fraser, S.N., Rodgers, W.M., Murray, T.C., & Daub, B. (2005). *The relationship between sociodemographic factors, social support and exercise tolerance in men attending cardiac rehabilitation*. Manuscript submitted for publication.
- Grace, S.L., Abbey, S., Shnek, Z., Irvine, J., Franche, R.L., Stewart, D.E. (2002). Cardiac Rehabilitation II: Referral and Participation. *General Hospital Psychiatry*, *24*, 127-134.

- Green, B.L., & Rodgers, A. (2001). Determinants of social support among low-income mothers: A longitudinal analysis. *American Journal of Community Psychology, 29*, 419-441.
- Grzywacz, J.G., & Marks, N.F. (2001). Social inequalities and exercise during adulthood: Toward an ecological perspective. *Journal of Health and Social Behavior, 42*, 202-220.
- Jiang, W., Glassman, A., Krishnan, R., O'Connor, C.M., & Califf, R.M. (2005). Depression and ischemic heart disease: What have we learned so far and what must we do in the future? *American Heart Journal, 150*, 54-78.
- King, K.M., Humen, D.P., Smith, H.L., Phan, C.L., & Teo, K.K. (2001). Predicting and explaining rehabilitation attendance. *Canadian Journal of Cardiology, 17*, 291-296.
- Kleinbaum, D.G., Kupper, L.L., & Muller, K.E. (1988). *Applied regression analysis and other multivariable methods*. Boston: PWS-KENT Publishing Company.
- Lynch, J.W., Kaplan, G.A., & Shema, S.J. (1997). Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. *New England Journal of Medicine, 337*, 1889-1895.
- Marmot, M.G., & Shipley, M.J. (1996). Do socioeconomic differences in mortality persist after retirement? 25 year follow up of civil servants from the first Whitehall II study. *British Medical Journal, 313*, 1177-1180.
- Mickelson, K.D., & Kubzansky, L.D. (2003). Social distribution of social support: The mediating role of life events. *American Journal of Community Psychology, 32*, 265-281.

- Mishra, G.D., Ball, K., Dobson, A.J., Byles, J.E., & Warner-Smith, P. (2002). Which aspects of socio-economic status are related to health in mid-ages and older women? *International Journal of Behavioral Medicine*, 9, 263-285.
- Missik, E. (2001). Women and cardiac rehabilitation: accessibility issues and policy recommendations. *Rehabilitation Nursing*, 26, 141-147.
- Nunnally, J.C. (1967). *Psychometric theory*. New York: McGraw-Hill.
- Nunnally, J. C. (1978). *Psychometric theory* (2nd ed.). New York: McGraw-Hill.
- Nunnally, J.C., & Bernstein, I. (1994). *Psychometric theory* (3rd ed.). New York: McGraw-Hill.
- Oakes, J.M., & Rossi, P.H. (2003). The measurement of SES in health research: Current practice and steps toward a new approach. *Social Science & Medicine*, 56, 769-784.
- Onwuegbuzie, A.J., & Daniel, L.G. (2002). A framework for reporting and interpreting internal consistency reliability estimates. *Measurement and Evaluation in Counseling and Development*, 35, 89-103.
- Onwuegbuzie, A.J., & Daniel, L.G. (2003). Typology of analytical and interpretational errors in quantitative and qualitative educational research. *Current Issues in Education* [On-line], 6(2). Retrieved December 6, 2005 from <http://cie.ed.asu.edu/volume6/number2/>
- Operario, D., Adler, N.E., & Williams, D. (2004). Subjective social status: Reliability and predictive utility for global health. *Psychology & Health*, 19, 237-246.

- Osborne, J. W., & Overbay, A. (2004). The power of outliers (and why researchers should ALWAYS check for them). *Practical Assessment, Research, and Evaluation*, 9(6). Retrieved December 6, 2005 from <http://pareonline.net/getvn.asp?v=9&n=6>.
- Osborne, J.W., & Waters, E. (2002). Four assumptions of multiple regression that researchers should always test. *Practical Assessment, Research & Evaluation*, 8(2). Retrieved January 10, 2006 from <http://PAREonline.net/getvn.asp?v=8&n=2>.
- Ostrove, J.M., Adler, N.E., Kuppermann, M. & Washington, A.E. (2000). Objective and subjective assessments of socioeconomic status and their relationship to self-rated health in an ethnically diverse sample of pregnant women. *Health Psychology*, 19, 614-618.
- Revenson, T.A. (2003). Scenes from a marriage: Examining support, coping, and gender within the context of chronic illness. In J. Sullis & K.A. Wallston (Eds.), *Social psychological foundations of health and illness* (pp. 530-559). Malden, MA: Blackwell.
- Seeman, T.E., & Berkman, L.F. (1988). Structural characteristics of social networks and their relationship with social support in the elderly: Who provides support. *Social Science & Medicine*, 26, 737-749.
- Singh-Manoux, A., Adler, N.E., Marmot, M.G. (2003). Subjective social status: its determinants and its association with measures of ill-health in the Whitehall II study. *Social Science & Medicine*, 56, 1321-1333.
- Sorensen, G., Emmons, K., Hunt, M.K., Barbeau, E., Goldman, R., Peterson, K. et al. (2003). Model for incorporating social context in health behavior interventions:

- applications for cancer prevention for working-class multiethnic populations. *Preventive Medicine*, 37, 188-197.
- Steptoe, A., & Marmot, M. (2002). The role of psychobiological pathways in socioeconomic inequalities in cardiovascular disease risk. *European Heart Journal*, 23, 13-25.
- Stevens, J.P. (2002). *Applied multivariate statistics for the social sciences* (4<sup>th</sup> ed.). Mahwah, NJ: LEA.
- Syme, S.L. (2001). Understanding the relationship between socioeconomic status and health: New research initiatives. J.A. Auerbach & B.K. Krimgold (Eds.), *Income, Socioeconomic Status, And Health: Exploring the Relationships* (pp.12-15).
- Tabachnik, B.G., & Fidell, L.S. (1996). *Using multivariate statistics* (3<sup>rd</sup> ed.). New York: Harper Collins.
- Tabachnik, B.G., & Fidell, L.S. (2001). *Using multivariate statistics* (4<sup>th</sup> ed.). Needham Heights, MA: Allyn and Bacon.
- Thompson, B. (1999). Five methodological errors in educational research: A pantheon of statistical significance and other faux pas. *Advances in Social Science Methodology*, 5, 23-86.
- Tobin, J.N., Wassertheil-Smoller S., Wexler, J.P., Steingart, R.M., Budner, N., Lense, L. et al. (1987). Sex bias in considering coronary bypass surgery. *Annals of Internal Medicine*, 107, 19-25.
- Turner, R.J., & Marino, F. (1994). Social support and social structure: A descriptive epidemiology. *Journal of Health and Social behavior*, 35, 193-212.

Williams, R.B., Barefoot, J.C., Califf, R.M., Haney, T.L., Saunders, W.B., Pryor, D.B., et al. (1992). Prognostic importance of social and economic resources among medically treated patients with angiographically documented coronary artery disease. *JAMA*, 267, 520-524.



Table 2-1

*Demographic and medical information of women and men*

Demographic variable	Women (n = 33)		Men (n = 122)	
	n	%	n	%
Marital Status				
Married/Common law	18	54.5	110	90.2
Education				
Less than HS	5	15.2	19	15.6
High-school	12	36.4	48	39.3
Post secondary	14	42.4	52	42.6
Missing	2	6.1	3	2.5
Income				
<\$20,000	5	15.2	6	4.9
\$20-39,999	9	27.3	19	15.6
\$40-59,999	5	15.2	22	18.0
\$60-79,999	2	6.1	16	13.1
≥\$80,000	3	9.1	39	32.0
missing	9	27.3	20	16.4
Admitting diagnosis				
Myocardial infarction	14	42.4	53	43.4
Bypass surgery	5	15.2	37	30.3
Valve	6	18.2	15	12.3
Angioplasty	19	57.6	76	62.3
Co morbidities				
Arthritis/joint	14	42.4	42	34.4
Asthma/bronchitis	6	18.2	19	15.6
High Blood pressure	23	69.7	60	49.2
Diabetes	11	33.3	22	18.0
High Cholesterol	21	63.6	80	65.6

Note: comorbidities and admitting diagnoses are not cumulative

Table 2-2

*Means and standard deviations of study variables among women and men*

Demographic variable	Women ( <i>n</i> = 33)		Men ( <i>n</i> = 122)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age	63.42 <sub>a</sub>	9.98	60.33 <sub>a</sub>	11.28
Subjective SS	5.48 <sub>a</sub>	1.74	6.75 <sub>b</sub>	1.54
SNI-diversity	5.33 <sub>a</sub>	1.90	5.96 <sub>a</sub>	2.02
SNI-size	15.36 <sub>a</sub>	10.15	17.74 <sub>a</sub>	8.56
ISEL-Appraisal	3.05 <sub>a</sub>	.72	3.48 <sub>b</sub>	.56
ISEL-Belonging	3.02 <sub>a</sub>	.76	3.51 <sub>b</sub>	.51
ISEL-Tangible	3.21 <sub>a</sub>	.61	3.55 <sub>b</sub>	.47
ISEL-Total	3.09 <sub>a</sub>	.64	3.51 <sub>b</sub>	.43

Note: SNI: Social Network Index; ISEL: Interpersonal Support Evaluation List. Means in the same row with different subscripts differ significantly at  $p < .001$ .

Table 2-3

*Means, standard deviations, and correlations among study variables*

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7	8	9	10
1. Age	60.99	11.06										
2. Education	2.28	0.73	-.28**									
3. Gender			-.12	-.01								
4. Income	3.41	1.38	-.38**	.31**	.31**							
5. Martial Status			-.16*	.00	.38**	.37**						
6. Subjective SS	6.50	1.65	.03	.26**	.31**	.49**	.17*					
7. SNI-diversity	5.83	2.00	-.26**	.13	.13	.45**	.26**	.31**				
8. SNI-size	17.23	8.94	-.19*	.04	.11	.45**	.11	.33**	.73**			
9. ISEL-Appraisal	3.39	0.62	.07	-.04	.28**	.18*	.27**	.40**	.20*	.19*		
10. ISEL-Belonging	3.40	0.61	-.02	-.00	.33**	.23*	.40**	.33**	.23**	.24**	.60**	
11. ISEL-Tangible	3.48	0.52	-.08	-.05	.27**	.22*	.30**	.29**	.11	.24**	.64**	.68**

Note: SNI: Social Network Index; ISEL: Interpersonal Support Evaluation List

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

Table 2-4

*Regression analysis predicting social networks diversity from demographic characteristics*

Variable	<i>R</i>	$R^2_{adj}$	$\beta$	<i>p</i>	$r_{sc}$
Age	.51	.22	-.18	.061	-.53
Gender			-.09	.341	.26
Education			-.04	.668	.28
Income			.25	.026	.87
Marital status			.17	.075	.58
SubjectSS			.21	.041	.62

Note:  $F(6, 112) = 6.555, p < .001$ ;  $r_{sc}$  = structure coefficient; SubjectSS = subjective social status.

Table 2-5

*Regression analysis predicting social network size from demographic characteristics*

Variable	<i>R</i>	$R^2_{adj}$	$\beta$	<i>p</i>	$r_{sc}$
Age	.50	.21	-.13	.164	-.40
Gender			-.02	.861	.35
Education			-.17	.066	.07
Income			.35	.003	.90
Marital status			.00	.965	.38
SubjectSS			.23	.028	.68

Note:  $F(6, 112) = 6.359, p < .001$ ;  $r_{sc}$  = structure coefficient; SubjectSS = subjective social status.

Table 2-6

*Regression analysis predicting total social support from demographic characteristics and social networks*

Variable	<i>R</i>	<i>R</i> <sup>2</sup> <sub>adj</sub>	$\beta_1$	<i>p</i>	$\beta_2$	<i>p</i>	<i>r</i> <sub>sc</sub>
Step 1	.54	.25					
Age			-.07	.471	-.04	.698	-.07
Gender			.08	.357	.09	.329	.51
Education			-.12	.195	-.08	.395	-.04
Income			-.08	.473	-.16	.153	.43
Marital status			.31	.001	.31	.001	.68
SubjectSS			.39	<.001	.34	.001	.70
Step 2	.57	.28					
SNI-diversity					.00	.992	.56
SNI-size					.24	.073	.63

Note:  $F_{\text{Step 1}}(6, 112) = 7.485, p < .001$ ;  $F_{\text{Step 2}}(8, 110) = 6.707, p < .001$ ;  $R^2 \Delta F(2, 110) = 3.407, p = .037$ ; *r*<sub>sc</sub> = structure coefficient; SubjectSS = subjective social status.

Table 2-7

*Regression analysis predicting appraisal social support from demographic characteristics and social networks*

Variable	<i>R</i>	<i>R</i> <sup>2</sup> <sub>adj</sub>	$\beta_1$	<i>p</i>	$\beta_2$	<i>p</i>	<i>r</i> <sub>sc</sub>
Step 1	.45	.16					
Age			.06	.546	.10	.313	.20
Gender			.04	.669	.06	.545	.41
Education			-.07	.446	-.05	.591	-.02
Income			-.05	.642	-.13	.293	.35
Marital status			.18	.068	.15	.126	.47
SubjectSS			.40	<.001	.34	.002	.81
Step 2	.50	.19					
SNI-diversity					.17	.240	.61
SNI-size					.09	.539	.59

Note:  $F_{\text{Step 1}}(6, 111) = 4.772, p < .001$ ;  $F_{\text{Step 2}}(8, 109) = 4.451, p < .001$ ;  $R^2 \Delta F(2, 109) = 2.977, p = .055$ ; *r*<sub>sc</sub> = structure coefficient; SubjectSS = subjective social status.

Table 2-8

*Regression analysis predicting belonging social support from demographic characteristics and social networks*

Variable	<i>R</i>	<i>R</i> <sup>2</sup> <sub>adj</sub>	$\beta_1$	<i>p</i>	$\beta_2$	<i>p</i>	<i>r</i> <sub>sc</sub>
Step 1	.51	.22					
Age			-.08	.393	-.06	.569	-.14
Gender			.09	.339	.09	.314	.53
Education			-.07	.458	-.04	.693	.02
Income			-.09	.448	-.15	.191	.49
Marital status			.34	<.001	.34	.001	.77
SubjectSS			.32	.002	.27	.010	.64
Step 2	.53	.23					
SNI-diversity					.18	.184	.56
SNI-size					.01	.910	.58

Note:  $F_{\text{Step 1}}(6, 112) = 6.429, p < .001$ ;  $F_{\text{Step 2}}(8, 110) = 5.449, p < .001$ ;  $R^2 \Delta F(2, 110) = 2.124, p < .124$ ; *r*<sub>sc</sub> = structure coefficient; SubjectSS = subjective social status.

Table 2-9

*Regression analysis predicting tangible social support from demographic characteristics and social networks*

Variable	<i>R</i>	<i>R</i> <sup>2</sup> <sub>adj</sub>	$\beta_1$	<i>p</i>	$\beta_2$	<i>p</i>	<i>r</i> <sub>sc</sub>
Step 1	.46	.17					
Age			-.14	.153	-.13	.179	-.19
Gender			.07	.460	.06	.542	.48
Education			-.17	.068	-.12	.208	-.14
Income			-.06	.592	-.14	.254	.42
Marital status			.26	.007	.30	.002	.67
SubjectSS			.31	.004	.27	.010	.58
Step 2	.51	.21					
SNI-diversity					-.22	.112	.40
SNI-size					.37	.008	.63

Note:  $F_{\text{Step 1}}(6, 112) = 5.028, p < .001$ ;  $F_{\text{Step 2}}(8, 110) = 4.893, p < .001$ ;  $R^2 \Delta F(2, 110) = 3.746, p = .027$ ; *r*<sub>sc</sub> = structure coefficient; SubjectSS = subjective social status.

### 3. STUDY 2

#### Social support and cardiovascular reactivity to anticipation of a treadmill test

Social support has been identified as an important contributor to overall health. This relationship has been established among clinical and non-clinical populations (see Berkman & Glass, 2000 for a recent review). Social support is also thought to affect recovery among post heart attack patients resulting in increased survival and fewer recurrent events (Berkman & Glass, 2000; Berkman, Leo-Summers, & Horwitz, 1992; Dracup, 1994).

In order to understand how social support influences health, Berkman and Glass (2000) have proposed a conceptual framework that describes multiple levels of social influence on health (see Figure 1-1). This framework outlines how higher-level (or macro-level) social factors such as socioeconomic status may influence lower-level (or micro-level) social factors such as social support and how these might affect health.

These pathways may involve various psychosocial variables. For example, previous research in this series of studies (Fraser, Rodgers, Daub, & Black, 2005 ; see Figure B-2, Appendix B) examined how micro-level factors (i.e., social support) impacted psychological pathways and found that stress and social support influenced self-efficacy for overcoming barriers to cardiac rehabilitation participation among heart patients. Barrier self-efficacy is thought to influence behavior (e.g., Blanchard, Rodgers, Courneya, Daub, & Knapik, 2002a), so self-efficacy and behavior might represent one possible pathway through which social support may affect health. That is, the pathway level variables between social support and a specific health indicator might include self-efficacy mediating the influence of stress and social support on health behavior.

However, behavior is probably not the only pathway through which social support might affect health. Uchino, Cacioppo, and Kiecolt-Glaser (1996) note that behavior is probably insufficient to explain the effects of social support on health. There may be other psychosocial mechanisms at play. One hypothesized mechanism has been termed the reactivity hypothesis (e.g., Lepore, 1998). This hypothesis states that a heightened psychological response to a psychosocial stressor can result in an excessive cardiovascular response, known as cardiovascular reactivity (CVR), which can be detrimental to health. For heart patients, such a response may even trigger myocardial ischemia or a myocardial infarction (e.g., Gullette et al., 1997; Krantz, Kop, Santiago, & Gottdiener, 1996). Social support is thought to attenuate the heightened response to such stressors. This has been proposed as a possible explanation for the protective effects of social support in terms of cardiovascular health (Lepore). Within the Berkman and Glass (2000) framework, this mechanism from the micro-level to psychological and physical pathways has yet to be examined in cardiac patients.

Stress has been identified as an important pathway variable in understanding the influence of social support on health (Cohen, Kaplan, & Manuck, 1994). That is, social support is thought to attenuate or buffer the health impact of stress and stressful situations (Christenfeld & Gerin, 2000; Greenwood, Muir, Packham, & Madeley, 1996). The stress may be a chronic or background stressor (Gump & Matthews, 1999), such as living in poverty, or the stressor may be more acute, such writing an exam. It is argued that a person with adequate support would experience less distress in such situations than someone with no support. The person with no support would then be more susceptible to the negative consequences of the situation such as an increase in anxiety, negative affect



and the physiological consequences of this arousal (i.e., increased heart rate, blood pressure, and exposure to stress hormones). In a review of the effect of background stressors on responses to acute stressors, Gump and Matthews found that background stressors were associated with impaired recovery from acute stressors. That is, people who experience generalized stress do worse in acute stress situations.

Moving further down the proposed pathway, an emotional or affective consequence of stress, anxiety, may be a potential mediator of the effects of stress on health (see Kubzansky & Kawachi, 2000; cf. Pedersen & Denollet, 2003). Ewart (1995) suggests that negative affective experiences contribute to low self-efficacy for exercise. In other words, the stress experienced by heart patients would be manifested by the affective experience of anxiety, which should lead to a negative impact on their confidence to exercise and to overcome barriers that may impede their ability to exercise. The availability of social support might attenuate the experience of arousal by affecting interpretations of the impact of chronic and/or acute stress. In other words, those with more social support should perceive less stress overall and consequently experience less anxiety related to their heart condition and have more self-efficacy to exercise.

Another factor that might contribute to the experience of a stressor, especially within the context of cardiac rehabilitation is the patient's view of his or her health. Idler and Benyamini (1997) suggest that self-rated health captures a broad array of relevant health information including not just medical status but other factors related to health, such as cognitive appraisals based on one's behaviors, social resources, symptoms, family history, and severity of current illness. Further, the authors argue that self-rated health is important and "an individual's health status cannot be assessed without it" (p. 34). Thus,

self-rated health seems to add subjective information from the view of the patient that could impact on the appraisal of the cardiac rehabilitation context.

Melchior, Berkman, Niedhammer, Chea, and Goldberg (2003) used self-rated health as a dependent variable in a large 12 month prospective study. The construct of self-rated health was related to several social constructs in a manner consistent with theory. Specifically, those with more functional support reported better self-rated health status. Cross sectional analysis showed that poor self-rated health was related to overweight status, depressive symptoms, no alcohol consumption or excessive alcohol consumption, and smoking status, as would be expected. Thus, self-rated health also correlated with other predictors of poor health.

Stress and anxiety may also manifest themselves more acutely among heart patients as mental-stress responses. These responses have been linked to poorer prognosis and silent ischemia. For example, Manuck, Olsson, Hjemdahl, & Rehnqvist (1992) found that patients with a recurrent coronary event exhibited higher systolic blood pressure (SBP) and diastolic blood pressure (DBP) responses to a mental stress task they participated in 39 to 64 months previously. Gottdiener et al. (1994) found that coronary artery disease (CAD) patients who experienced ischemia during a laboratory mental stressor also experienced ischemia during normal sedentary daily activities. Blumenthal et al. (1995) examined mental stress-induced ischemia in patients with CAD who had previously experienced exercise-induced ischemia. The authors found that patients who experienced mental stress-induced ischemia in the laboratory were more likely to experience ischemia during daily living and from daily mental stress than those who did not experience the mental stress-induced ischemia in the laboratory setting. One reason for

the poorer prognosis among more reactive patients may be that mental stressors can induce a silent myocardial ischemia both in the laboratory and during daily activities (e.g., Gullette et al., 1997; Krantz et al., 1996).

In a recent review of the literature, Strike and Steptoe (2003) concluded that “one third to one half of patients with CAD” (p. 12) experienced silent myocardial ischemia as a result of mental stress, regardless of the severity of the patient’s CAD (cf. Krantz et al., 1996). They further note that the mental stressors were not severe. The stressors, such as a speech task or mental arithmetic, were similar to daily challenges typically encountered by individuals.

#### *Medication effects on CVR*

Since it is common for cardiac patients to be on several medications designed to influence cardiovascular responses, it is important to understand how these medications might affect cardiovascular responses to mental stressors. Common medications for cardiac patients that might influence cardiovascular responses include calcium channel blockers, ACE (angiotensin-converting enzyme) inhibitors, beta-blockers, alpha-blockers, and diuretics. The following sections examine some of the most common medications and their potential effects on mental stress responses.

In terms of cardiovascular reactivity, a few studies have reported the role of medications in potentially attenuating the effects of acute stress. Spence, Munoz, Huff, and Tokmakjian (2000) argue that there is little known about how these medications might affect cardiovascular responses to mental stressors. The studies available seldom examine the role of medications on CVR to a mental stressor, per se. Rather, medications are usually used to explore how mental stress might affect certain pathways to CVR (e.g.,

withdrawal of vagal tone, increase sympathetic activity, etc.) or medications are considered potential confounds. For example, Strike and Steptoe noted that no drugs have consistently blocked the effect of mental stressors on ischemia. Also, Craig, Lynch, and Quartner (2000) examined the role of social support in attenuating CVR to a mental stressor among cardiac patients. Craig et al. found notable CVR responses despite patients being on medications (beta-blockers, vasodilators, anti-arrhythmics, diuretics, anxiety/depression drugs) designed to dampen these effects. Dosages were not included, but patients were presumably taking dosages prescribed by their physicians to affect resting heart rate (HR) and blood pressure (BP). Thus, CVR responses differed between patients with high vs. low social support even though both groups had similar medication profiles. However, the exact medication effects on CVR, per se, were not examined.

#### *Calcium channel blockers*

Malhotra, Kumari, and Pandhi (2001) conducted a placebo-controlled double-blinded study of the effects of calcium channel blockers (5 mg amlodipine and 4 mg lacidipine) on mental stress induced blood pressure and heart rate changes. Both hypertensive patients and normotensive volunteers were included in the study. A cold stressor and a handgrip exercise task were used to induce changes in blood pressure. Results showed that among normotensive participants resting blood pressures and heart rate was not affected by the medications, whereas blood pressures were reduced in hypertensive subjects. Both stressors induced a significant increase in blood pressure and heart rate in both groups of participants. However, in both groups of participants the medications had no impact on the magnitude of the rise in blood pressure for both cold stress and isometric exercise.

Gebara et al. (1996) examined the effects of a mental stressor (mental arithmetic followed by Stroop color test) and a cold pressor task on hemodynamic factors in patients with mild to moderate hypertension taking a calcium channel blocker, verapamil. In a double-blinded randomized placebo crossover design, patients received either a placebo or verapamil (starting at 240 mg daily) for 4 weeks. Dosages of both the placebo and verapamil were increased at 2 weeks if blood pressure was not reduced adequately. Results showed that verapamil reduced resting blood pressures and mental and cold stress induced increases in blood pressures. However, there were no differences in the magnitude of blood pressure response to any of the stressors between the verapamil and control group. Verapamil had no effect on baseline or stress induced changes in heart rate.

Spence et al. (2000) examined the effects of another calcium channel blocker, amlodipine, on hemodynamic responses to mental stress (Stroop color test) among patients with mild to moderate hypertension. In a double-blind randomized placebo crossover trial, patients were given 4 weeks of either a placebo or amlodipine (starting at 5mg daily) following a 4 week run-in period. Results showed that resting and mental stress SBP and DBP were reduced following the use of amlodipine. Resting total peripheral resistance (TPR) was lower following the use of amlodipine and resting and mental stress HR were different between the placebo and treatment conditions. Although BP values decreased at rest and during the stress task, the magnitude of the blood pressure responses did not differ between the placebo and amlodipine conditions.

#### *Beta-blockers*

Benschop et al. (1994) examined the role of beta-adrenergic blockade (40 mg propranolol) on immunologic responses to mental stress in healthy men. The mental

stressor involved an evaluative procedure testing reaction time to two tasks involving identifying a stimulus and memorizing and identifying letters. Benschop et al. also examined cardiovascular responses to identify if responses were primarily sympathetic. Results showed that the magnitude of responses was similar between the two tasks. Further, no changes in heart rate were observed in the propranolol group but blood pressure (both SBP and DBP) responses were not different between the placebo and treatment group. As a result, total peripheral resistance increased from baseline in the treatment group but not in the control group, whereas cardiac output decreased in the treatment group and increased in the control group. These results are consistent with Nicotero, Beamer, Moutsos, and Shapiro (1968) who examined the effect of up to a 150 mg daily dose of propranolol on responses to a cold stressor among hospitalized hypertensives. The authors found no effect of propranolol on blood pressure reactivity to the stressor.

Bairey, Krantz, DeQuattro, Berman, and Rozanski (1991) conducted a double-blinded randomized examination of beta-blockade (100 mg metoprolol twice daily) on ischemia induced by mental stress and exercise. The authors examined changes in HR, SBP, and DBP from rest to peak change during exercise (graded bicycle exercise test) and mental stressors (mean of math, stroop task, and speech tasks). Results showed no difference between resting SBP and peak SBP during mental stress between placebo and metoprolol groups. Diastolic blood pressure was lower in the treatment group at rest but was not different during the mental stressor. Heart rate was lower between treatment and control groups, but the magnitude of change was not different between rest and mental

stress. Interestingly, mental stress induced ischemia occurred at HRs much lower than ischemia induced by exercise (81 vs. 123 beats/min.).

Jain, Burg, Soufer, and Zaret (1995) examined the effects of mental stress testing on left ventricular (LV) function in patients with stable angina who remained on all prescribed medications. The authors found that LV dysfunction induced by mental stress was related to a 3-fold increase in risk of a cardiac event 2-years later. The only other factors to predict a future cardiac event were patients taking beta-blockers and a low HR. Specifically, those patients taking beta-blockers were far more likely to experience or develop an adverse cardiac event. The authors suggest that beta-blockers “may be ineffective or even detrimental in the setting of mental stress” (p. 34). The reason is that hemodynamic responses between exercise and mental stress are probably different. Jain et al. suggest that exercise triggers largely an epinephrine response whereas mental stress results in mostly a norepinephrine response. Thus, mental stress may trigger an alpha-adrenergic response that is unopposed. However, studies of alpha-adrenergic blockade show little effect on reactivity (e.g., Ring et al., 2000).

François, Cahen, Gravejat, and Estrade (1984) specifically examined the effects of beta-blockade on pressor responses to physical and mental stress among hypertensive patients and a control group. Patients completed the physical (dynamic bicycle and static handgrip exercises) and mental stressor (mental arithmetic and color identification) tasks after 2 weeks of taking a placebo and again after almost 2 months on 100 mg per day of atenolol. Doses were increased/decreased as necessary in order to control resting BP. The results showed that resting HR, SBP, and DBP were different between the hypertensives and the controls before treatment and between hypertensives before and after treatment.

Thus, atenolol successfully reduced resting BP and HR for the hypertensives. Results of the pressor responses showed that BP, mean arterial pressure (MAP), and HR increased among all participants during the mental tasks and the physical tasks. However, before treatment the hypertensives showed greater rises in SBP, DBP and MAP during the color stress and grip strength tasks, and greater rises in DBP and HR in the mental arithmetic task. After treatment there were no differences in SBP, DBP, MAP, or HR responses to the handgrip or mental stress task among the hypertensive patients. For the color stress task there were no differences except that the HR response was lower after medication than before. For the bicycle task only SBP and HR were different after medication, both attenuated. The authors concluded that atenolol could not reduce the blood pressure responses to stress except for dynamic exercise stress, where the magnitude of the response was reduced but not eliminated.

Waal-Manning and Bolli (1980) also examined the effects of atenolol on responses to stressors among mildly hypertensive patients. All patients were given 8 weeks of placebo and 8 weeks of atenolol in random order. Dosage started at 50mg twice daily and was adjusted to control BP. Other medications were not withdrawn and included diuretics and one patient on prazosin. The dosages for these were not reported nor were any potential interactive or combined effects. Nevertheless, resting BP (reduction in SBP was not significant while standing) and HR were reduced while on atenolol. Similar to the results of François et al. (1984), HR and BP values while engaged in the physical and mental tasks were lower while on atenolol. However, there were no differences in the magnitude of the changes for SBP, DBP, and HR, except for a slight attenuation of SBP and HR in the bicycle exercise task. Interestingly, while on atenolol, baseline values of BP



while sitting on the bicycle were significantly higher than when sitting at a table. These results suggest that atenolol simply lowers resting BP and HR values, and hence task related BP and HR values, but has no effect on the magnitude of BP and HR response to mental stress and isometric exercise, and possibly a slight attenuation of the effect of dynamic exercise.

In a review of 59 studies Mills and Dimsdale (1991) noted that blood pressure responses to stress were unaffected by beta-blockers, while heart rate responses were reduced. Specifically, the authors reviewed the effects of 8 beta-blocking drugs according to three drug classifications: beta<sub>1</sub> selective and nonselective, ISA (intrinsic sympathomimetic activity) and no ISA (low or high), and solubility (lipophilic and hydrophilic). Dosage was reported where available and had no effect on CVR. However, nonselective blockers were associated with reduced heart rate reactivity more often than selective blockers. Further, Mills and Dimsdale found no difference in cardiovascular responses (heart rate, SBP, DBP reactivity) between hypertensive and normotensive individuals. Thus, it appears that heart rate responses, but not blood pressure responses, to a psychological stressor are reduced with beta-blockade. The authors conclude that the sympathetic activation of psychological stress may not be enough for the beta-blockade to dampen the effect of blood pressure responses as is sometimes seen in SBP responses to exercise, for example.

#### *ACE-inhibitors*

Dimsdale, Mills, Ziegler, Leitz, and Nelesen (1992) specifically examined the influence of an ACE inhibitor (25 mg captopril twice daily) on the magnitude of blood pressure responses to mental stress (mental arithmetic with distractions and reading a

disturbing newspaper story out loud) in a double-blind randomized study of normotensive and hypertensive patients. Results showed that the ACE inhibitor reduced the resting blood pressures (SBP and DBP) of hypertensive men more than normotensive men, but overall the magnitude of reactivity of the treatment group was the same as the control group. Normotensive patients had bigger increases in heart rate, but there were no differences in the magnitude of responses between the treatment and control group. These results are consistent with the results of Allen, Shykoff, and Izzo (2001) showing that ACE inhibitors do not blunt HR and BP responses to stress.

Cardillo, Mores, Motolese, and Folli (1994) examined the effects of another ACE inhibitor, benazepril, on CVR to mental arithmetic, handgrip, and cycle ergometry testing in a placebo-controlled, within patient double-blind study. Patients with hypertension were given a placebo, 10 mg, or 20 mg doses of benazepril once daily for 2 weeks. Results showed a decrease in resting SBP and DBP and no differences in HR between the treatment groups and the placebo group. There were no differences between the two treatment groups on resting BP or HR values. Similar to Dimsdale et al. (1992), there were no differences in BP or HR responses to the mental stressor. In other words, there was no difference between the placebo condition and the treatment conditions (regardless of dosage) in the magnitude of SBP and DBP and HR responses to the mental stressor. Interestingly, there were no differences in the magnitude of BP responses in the handgrip condition and both doses of benazepril induced increased SBP responses compared to the placebo in the cycle ergometry task. The 20 mg dose also resulted in a marked increase in HR compared to placebo in the cycle ergometry condition.

Schmieder et al. (1996) examined the effects of the ACE inhibitor cilazapril on hemodynamic responses to stress (30 minutes of reaction time tasks) among normotensives and those with essential hypertension. Participants in the double-blinded randomized placebo crossover study were given 1 week of either a placebo or 25mg daily of cilazapril separated by a 2 week washout period between treatments. Among both normotensives and hypertensives on placebo or cilazapril, the mental stressor resulted in increased MAP, HR, CO, and stroke volume. Among normotensives there were no differences (MAP, HR, CO, SV, or TPR) between placebo and cilazapril at rest or during stress. Among hypertensives MAP at rest and during stress was lower among those on cilazapril than placebo, but the magnitude of the difference was the same. Further, there were no other differences between placebo or cilazapril among hypertensives at rest or during stress. The authors conclude, "ACE inhibition had little effect on systemic haemodynamics" (p. 1205).

Kahan and Eliasson (1999) examined the long term effects of ACE inhibition on hemodynamic responses to stress (Stroop color task and cold pressor test) among mild to moderate hypertensive patients. In this double-blind randomized placebo crossover study, patients received either a placebo or ramipril (starting at 5 mg daily) for 6 weeks. After the crossover period, all patients then received the ramipril for the following 6 months, where dosages were adjusted where necessary. Results showed that 6 weeks of ramipril reduced resting SBP and DBP with no effect on HR. These effects remained consistent at 6 months. Typically, mental stress SBP and DBP levels were lower in the ramipril condition. However, at 6 weeks there were no differences in the magnitude of the changes, and at 6 months SBP was slightly, but significantly, attenuated (exact figures are not

reported, but Figure 2, p. 1191, seems to show about 25 mmHg change for placebo vs. 19 mmHg change for 6 months of ramipril). Hemodynamic responses to the cold pressor showed that ramipril reduced mental stress SBP and DBP. However, there was no difference in BP responses to the cold pressor at 6 weeks or at 6 months. In other words, the magnitude of the changes was not different between placebo and treatment conditions. Thus, the authors found that 6 months of ramipril slightly attenuated the SBP response to the Stroop task.

#### *Alpha-blockers*

Mancia, Ferrari, Gregorini, Ferrari et al. (1980) examined the effects of an alpha-blocker (prazosin 2-5 mg 3 times per day) on several cardiovascular markers of autonomic control at rest and in response to dynamic and isometric exercise, a cold stressor, and increases/decreases in carotid sinus baroreceptor activity. Patients with essential hypertension, not taking any antihypertensive medications, completed the 4 tasks before and after a 10-15 day treatment period. Treatment doses were increased until BP was considered controlled. After treatment, mean arterial pressure (MAP) was reduced, but HR and cardiac output (CO) did not change. Thus, the authors conclude that the drug affected BP by reducing total peripheral resistance (TPR). Dynamic exercise caused large increases in HR and CO, large decreases in TPR, and significant increases in MAP. There were no difference in the responses of these variables to exercise before and after treatment. However, MAP and TPR were lower after treatment. The responses to the cold pressor and isometric exercise were very similar to each other, with increases in MAP and HR. According to the authors, both CO and TPR seemed to cause the pressure increase in the isometric condition, whereas the pressure increase in the cold pressor condition was likely

caused by an increase in TPR. The treatment did not affect these responses since there were no differences in the magnitude of changes in these variables to the two stressors. Further, only MAP and TPR were lower after treatment than before treatment (both pre-task and on-task). Finally, the authors found that increased baroreceptor activity led to decreased MAP and HR, whereas a decrease in baroreceptor activity led to an increase in MAP and HR. The authors conclude that the decrease in pressure with increased baroreceptor activity was due to reduced TPR and CO, whereas the increase in pressure from decreased baroreceptor activity was due to an increase in TPR. Interestingly, HR and BP responses to increased or decreased baroreceptor activity was not affected by the treatment. Further, TPR and CO responses influencing changes in MAP were also unaffected by the treatment.

Mancia, Ferrari, Gregorini, Bianchi et al. (1980) conducted a study of the effects of a different alpha-blocker (methyldopa starting at 500 mg every 12 hours) on resting and hemodynamic responses to physical stress (dynamic and isometric exercise, cold pressor, and baroreceptor activity increase/decrease). Dosage was increased for the essential hypertensive patients until there was a constant reduction in arterial pressure. In line with their other study of prazosin (Mancia, Ferrari, Gregorini, Ferrari et al. 1980), the authors found that methyldopa reduced MAP and CO at rest, but had no effect on HR or CO. Responses to dynamic exercise were the same as in Mancia, Ferrari, Gregorini, Ferrari et al. with increases in MAP, HR, CO and decreased TPR, and methyldopa had no effect on the magnitude or pattern of these responses. Similarly, hemodynamic responses to isometric exercise were unaffected by the medication. However, in addition to increased MAP and HR, CO also increased with isometric exercise, whereas CO did not increase in

Mancia, Ferrari, Gregorini, Ferrari et al. Responses to the cold pressor were unaffected by methyldopa as well. Methyldopa only slightly decreased the MAP increase associated with decreased baroreceptor activity.

Ring et al. (2000) examined the effects of alpha-adrenergic blockade (1 mg doxazosin) on immunological responses to mental and physical stressors among men and women. The authors found only a limited effect on cardiovascular responses to a mental arithmetic task. Specifically, heart rate was faster in the placebo condition, but there was no task/treatment interaction. In other words, the magnitude of the response was not affected by doxazosin. In terms of SBP and DBP reactions, these were not different among the placebo group and the group taking the alpha-adrenergic blockade during the arithmetic task. Interestingly, there were no interaction (task/treatment) or treatment effects for any of the cardiovascular variables for an 8 minute submaximal exercise test condition.

#### *Medication comparison studies*

Rüddel, Langewitz, Schächinger, Schmieder, and Schulte (1988) compared CVR to mental stress (mental arithmetic) among hypertensives and normotensive patients or volunteers. Hypertensive patients were assigned to one of four medications types: beta-blocker (160 mg daily oxprenolol), antiadrenergic agent without selective receptor blockade (clonidine 37.5 µg twice daily), calcium channel blocker (20 mg daily nitrendipine), or an ACE inhibitor (10 mg daily enalapril). Dosages were increased as necessary over the 3-6 month treatment period with a goal of controlling hypertension. At baseline (before medication), hypertensives had higher SBP and DBP than normotensives, and higher SBP reactivity to the mental stressor. Baseline HR at rest and during the

stressor were not different among the groups. After taking medications, the four hypertensive treatment groups had the same reduced resting BPs. Resting HR was lower among those taking the beta-blocker, and highest among those taking the calcium channel blocker. However, none of the antihypertensive medications had any effect on SBP reactivity to the mental stressor. The effects on HR were inconsistent. The beta-blocker reduced (but did not eliminate) the HR reactivity, the calcium channel blocker increased HR reactivity, and HR reactivity remained unchanged by the other two medications.

Delamater et al. (1989) specifically examined cardiovascular reactivity to a stressor (role playing and naturalistic interactions) among normotensives and hypertensive patients taking beta-blockers or diuretics as prescribed by their physicians. Specific drug and dosage information was not reported. The authors found that hypertensives had higher SBP and DBP than normotensives, but HR, SBP, and DBP reactivity did not differ between the normotensives and hypertensives on medications to treat hypertension. Further, there were no differences in CVR between hypertensives on diuretics or beta-blockers suggesting diuretics affected blood pressure responses to the mental stressors in a manner similar to the effect of beta-blockers.

Schneider et al. (1989) examined BP responses during mental (mental arithmetic with noise) and physical stress (cycling exercise) with hypertensive men on one of four different kinds of medications: antiadrenergic agent without selective receptor blockade (75 µg clonidine daily), beta-blocker (160 mg daily oxprenolol), calcium channel blocker (20 mg daily nitrendipine), or an ACE inhibitor (5 mg daily enalapril). Dosages were increased as necessary to ensure that BP was controlled over a 6 month period. Stress responses were compared before and after the introduction of the medication. The results

showed that resting BP was reduced in all four treatment groups. Diastolic BP was not different among the groups, whereas SBP was lowest in those treated with clonidine, and highest in those treated with enalapril, as measured in the outpatient clinic. However, prior to the mental and physical stress testing there were no differences among the treatment groups in SBP or DBP. Further, the mental stressor produced the same BP responses among the four treatments groups. The physical stress resulted in greater increases in SBP than the mental stressor among all treatment groups, but the clonidine group showed the greatest SBP responses (55 mmHg), whereas the oxprenolol group showed the lowest increase (30 mmHg). The clonidine group also displayed the highest DBP response to physical stress. Although there were no differences in SBP or DBP responses to the mental stressor, the authors were able to show that the patients treated with enalapril and nitrendipine increased their BP through an increase in HR and stroke volume, and cardiac output, and a decrease in total peripheral resistance. Conversely, patients on oxprenolol experienced a reduced stroke volume, along with slight increases in HR and total peripheral resistance.

Bateman, Dean, Mucklow, Bulpitt, and Dollery (1979) compared the effects of a placebo (2 pills daily), atenolol (100 mg daily plus placebo), chlorthalidone (25 mg daily plus placebo), or both atenolol (100 mg) and chlorthalidone (25 mg) on resting HR, BP and HR and BP responses to exercise (isometric hand grip and bicycle geometry) and a mental stressor (mental arithmetic) in a randomized double blinded crossover study. The three medication conditions were effective in reducing SBP and DBP, and the presence of atenolol reduced resting HR. The increase in BP in response to isometric exercise or mental stress did not differ among the 4 treatment groups. The SBP response to bicycle



exercise was lower in those taking atenolol alone or in combination 2 or 4 hours post-dose, but there were no difference 24 hours post-dose.

Eliasson, Kahan, Hylander, and Hjemdahl (1987) examined HR and BP responses to mental (color word task) and physical stressors (cold pressor immersion of hand in ice water, and orthostatic stress of 10 minutes standing) among mild to moderate hypertensive patients randomly assigned 6 months of treatment with one of 2 beta-blockers (metoprolol starting at 100 mg once to twice daily, or propranolol starting at 80mg twice daily), or a diuretic (hydrochlorothiazide 25mg once to twice daily to start). Dosages were double after 2 months if DBP was not reduced to 95 mmHg or less. Resting SBP and DBP were reduced similarly among all three treatment groups. The mental stressor induced large and rapid increases in SBP and DBP among all three treatment groups. The two beta-blockers reduced task related SBP and DBP, whereas the diuretic only decreased SBP. However, the magnitude of BP changes on medication was not different from BP responses before treatment. Heart rate responses were reduced compared to pre-treatment but were not eliminated. Further, there were no differences between treatment conditions in terms of the magnitude of BP responses. Similar results were seen for the BP responses to the cold pressor. The two beta-blockers reduced the HR response to standing, but the diuretic had no effect. All three treatments reduced SBP and DBP levels during standing, but none of the treatments reduced the magnitude of change (i.e., increase) in SBP or DBP while standing.

Lasser, Nash, Lasser, Hamill, and Batey (1989) conducted a randomized, placebo-controlled study of the effects of three drugs on BP control, reactivity (Pac Man with encouragement and Stroop task with time pressure and encouragement) and cognitive

function. After a washout period, the patients were assigned to a placebo or one of three drug conditions: alpha-adrenergic blocker (starting at 2 mg daily prazosin), beta-blocker (starting at 40 mg daily propranolol), or a diuretic (starting at 25 mg daily hydrochlorothiazide; HCTZ). Dosages were increased as necessary until BP was controlled or side effects became limiting. The three drugs were generally effective in reducing BP. In terms of reactivity, the prazosin group had lower HR responses compared to the placebo group. However, this group also had the highest baseline heart rate after treatment. There were no other differences between groups on any of the other CVR variables (i.e., SBP and DBP) in response to the mental stressors.

#### *Medications in combination*

Benight et al. (1997) examined the effects of mental stressors (anger and desperation recall) on myocardial perfusion between men with CHD and healthy controls. While the authors' hypothesis regarding myocardial perfusion was not supported, the authors found that SBP, DBP and HR responses to the mental stressors did not differ between the two groups. One confound of their study with respect to the investigation of myocardial perfusion was that the patients were "not taken off their vasoactive medications (e.g., nitrates, calcium channel blockers, and beta-antagonists" (p. 142). Unfortunately, the exact medications and dosages were not reported.

Grosse, Bianchi, Diaz Puertas de Grosse, Iglesias, and Coviello (1987) conducted a study of the effects of hypertensive medications on BP reactivity to psychological stressors. The authors compared reactions among 3 groups: normotensives, moderately hypertensive, and severe hypertensives. The normotensive group was not taking any antihypertensive medication, whereas the moderately hypertensive patients were taking

both thiazides and beta-blockers, and the severe hypertensives were taking clonidine (an alpha-blocker) in addition to thiazides and beta-blockers. Although dosages were not reported, the hypertensive patients had been taking the medications for a minimum of six months with acceptable responses (i.e., reduced/normal resting BP) to the medications. The results of the psychological stressor showed that the normotensive comparison group had the smallest BP responses (24.2 mmHg SBP; 11.2 mmHg DBP), the moderately hypertensive group had higher BP responses (33.2 mmHg SBP; 17.0 mmHg DBP), and the severe hypertensives had the highest BP responses (42.6 mmHg SBP; 23.5 mmHg DBP). This study is consistent with others showing that antihypertensive medication reduced resting blood pressure, but shows that the severity of hypertension may be related to the severity of the BP response to stress, despite multiple medications.

Thomas et al. (1992) examined CV responses of cardiac patients talking and engaging in an exercise test. Patients in the study were taking a variety of antihypertensive medications including beta-blockers, calcium channel blockers and diuretics. Medications were prescribed by the individual patient's physician and dosages were not reported. Results showed no differences in HR or BP reactivity between patients taking medications or not. The authors noted that the "antihypertensive medications, [beta]-blockers, and calcium channel blockers did not block the HR and BP increases either while the patients were talking or during exercise" (p. 70).

In a study of cardiovascular reactivity to mental stress (time pressured anagram task) in Swedish women, Weidner et al. (2001) found no differences in cardiovascular measures between heart patients taking medications (beta-blockers, calcium blockers, ACE inhibitors) and those not taking medications, or compared to a matched group of

controls. Beta-blocker use was examined separately and it was found that those on beta-blockers had lower resting heart rates compared to the other groups, but the reactivity (magnitude of change from baseline to stressor) of blood pressure and heart rate were not different. However, patients had lower resting heart rates than controls whether or not they were on beta-blockers. Since these women were sampled from hospitals, dosages and medication selections were not under control of the investigators and were not reported.

Jacot-des-Combes, Brunner, Waeber, Porchet, and Biollaz (1984) examined the effect of no treatment, beta-blockers, diuretics, or a combination of both beta-blockers and diuretics on the variability of ambulatory blood pressure among male and female hypertensive patients. Medication doses were prescribed with the goal of reducing resting blood pressure (propranolol dose of at least 160mg/70 kg of body weight and hydrochlorothiazide dose of at least 25mg/70 kg of body weight). Mean SBP and DBP did not differ between the 4 groups. Variability of blood pressure was examined and the authors found no differences among the four groups with SBP responses ranging from 41.4 to 50.6 mmHg, and DBP responses ranging from 30.1 to 34.4 mmHg. This study is consistent with others showing a lack of effect of medications on BP reactivity but includes a treatment condition with two drugs, and shows that outside of the laboratory, these two medications were not successful in dampening BP responses. However, it is unknown if the BP responses were to behavioral or psychological stressors.

Velasco et al. (1978) examined the effects of a beta-blocker (propranolol) on reactivity to the cold pressor among patients with essential hypertension also using the vasodilator hydralazine in a cross-over design. Hydralazine dosages were increased every 15 minutes until BP reached normal levels. Propranolol dosages were issued every

6-8 hours for 1 week. Results showed that hydralazine reduced resting BP and increased resting HR. Propranolol alone reduced resting BP and HR, and in combination with hydralazine reduced BP with no reduction in HR overall. Propranolol did not change the BP responses to the cold pressor, but did block the HR response.

*Miscellaneous medications*

Fauvel, Najem, Maakel, Pozet, and Laville (1998) examined the effect of a one-month intervention with the selective imidazoline receptor agonist moxonidine (.4 mg per day) on CVR responses and sodium handling to mental stress (Stroop word task) among hypertensive men and women. The authors employed a placebo-controlled double-blinded crossover study. As with other antihypertensive medications moxonidine was found to reduce resting and stress BPs. Resting heart rate was unaffected by moxonidine. Again, as with other antihypertensives, BP reactivity was unaffected by the medication. However, HR reactivity was unaffected by medication as well.

Straznicky, Howes, Lam, and Louis (1995) examined the effects of a lipid lowering agent (40 mg per day pravastatin) on cardiovascular reactivity in patients with mild hypertension in a randomized placebo-controlled double-blinded study. The authors compared the reactivity induced by a cold pressor test (2 min. hand immersion in ice-water), isometric exercise (handgrip at 30% max), and drug induced reactivity (norepinephrine and angiotensin II). They found no differences in blood pressure and heart rate responses between the pravastatin and placebo group for the cold pressor and handgrip test. However they did find reduced DBP, but not reduced SBP, reactivity among those patients with drug-induced reactivity.

While not exhaustive, this review conforms to the conclusions of Dimsdale et al. (1992) that while the many antihypertensive medications successfully lower resting BP, none of them “block the BP response to challenge, be it exercise or behavioral stress” (p. 213; cf. Muir, Burton, & Lawrie, 1969). That is, medications do not entirely eliminate BP responses to these task, but these responses might be attenuated. More specifically, these medications tend to affect cardiovascular responses by lowering resting and task related BP values, with differential effects on heart rate. Although some studies show a slight attenuation of the BP responses to mental stressors, the above discussion shows that the *magnitude* of BP responses generally does not differ whether one is on medications or not. Further, the responses might vary depending on the specific kind of stressor, or they might not. However, the mental stressors involved in the medication/pharmacological studies tend to be limited in scope and degree of strength as stressors (e.g., Stroop task, reaction time, mental arithmetic, cold pressor) compared to the kinds of stressors examined in the CVR research, such as social conflict, anticipation, public speaking, etc; tasks thought to lead to greater CVR responses. Nevertheless, it seems that a variety of medications will either slightly attenuate or have no effect on the magnitude of blood pressure responses to a mental stressor, but clearly the medications do not block these responses. Thus, a SBP response seems to be unaffected by medications and is likely to be similar in magnitude whether a person is medicated or not. So compared to HR and DBP, a patient’s SBP should display the largest and most consistent increase after encountering a mental stressor. Heart rate and DBP might change, but the effect is inconsistent across studies. Therefore, if HR and DBP do change after a mental stressor it is not clear if these effects are attenuated or not.

Allen et al. (2001) examined the influence of social support (randomization to pet ownership) on cardiovascular responses to mental stress (speech or mental arithmetic task) among hypertensive men and women before and after taking antihypertensive medication (20 mg per day lisinopril). The authors found that the ACE inhibitor therapy lowered resting blood pressure with no changes in HR. Both groups had significant cardiovascular responses to mental-stress after 1 month. However, after 6 months the medication only group had higher cardiovascular reactivity than the group with the social support intervention. Although resting blood pressure was lower, there were no differences in the magnitude of blood pressure responses before and after the introduction of lisinopril. Further, HR responses were unaffected by the medication. Interestingly, differences between home and workplace resting blood pressure were examined before and after the introduction of medication and social support. The results showed that workplace blood pressures were significantly higher than home blood pressure before and after the medication and social support intervention. These results are similar to those of Craig et al. (2000) in that significant CVR responses were observed among patients on medications and social support dampened the CVR response. In summary, medications do not eliminate CVR responses, and probably don't attenuate SBP responses to a mental stressor. Further, even in patients on medications, social factors appear to dampen CVR effects.

However, among heart patients, little research has actually examined the role of social support in attenuating reactive responses. Craig et al. (2000) note that it is surprising that the relationship between social support and CVR has not been studied in cardiac patients since the influence of CVR on prognosis is strongest among patients with

CAD (e.g., Blumenthal et al., 1995; Kamarck et al., 1997; Lynch, Everson, Kaplan, Salonen, & Salonen, 1998). Given the relationship between CVR and prognosis and the relationship between social support and CVR it is important to determine if social support might affect the health of heart patients through this potential mechanism which has received little attention to date.

Thus, the purpose of this study was to examine how micro-level social support affects psychological and physical pathways as shown in Figure 1-5. Specifically, this study examined the role of social support in attenuating the CVR of heart patients. It was thought that higher perceptions of social support would lead to lower perceptions of stress and anxiety, which would influence self-efficacy to exercise and acute stress responses. Understanding how social support might affect CVR can offer practical and theoretical mechanisms of intervention.

It was hypothesized that social support would attenuate reported stress and anxiety and that both social support and anxiety would affect CVR. Specifically, social support should be negatively related to stress and anxiety. Social support should be positively related to self-efficacy and stress and anxiety should be negatively related to self-efficacy. Social support should be negatively related to CVR so that patients with more social support should have smaller changes in cardiovascular parameters from resting to anticipation. Further, more self-efficacy for exercise was thought to be related to lower CVR responses. Potential gender differences were explored based on the suggestion that stress responses between men and women should be examined separately (Taylor et al., 2000).

### *Method*



### *Participants*

Patients were recruited from the Northern Alberta Cardiac Rehabilitation Program (NACRP). These patients included 71 men ranging in age from 35 to 84 years (mean age = 60.1, *SD* = 9.9) and 27 women ranging in age from 37 to 79 years (mean age = 59.9, *SD* = 11.8) drawn from those attending CR. However, only 45 men and 17 women provided resting HR and BP values and pre-exercise HR and BP values. The proportion of men and women in this study is typical of this particular cardiac rehabilitation program and other programs in North America (e.g., Blackburn et al., 2000; Blanchard et al. 2002a; King, Humen, Smith, Phan, & Teo, 2001; Missik, 2001). In this sample, the most common events responsible for referral to cardiac rehabilitation were angioplasty (56.6%), myocardial infarction (47.5%), and/or bypass surgery (36.7%). The cardiac rehabilitation program is an individually tailored comprehensive approach with a focus on exercise. Patients also had the option of attending a number of weekly educational classes on topics such as sexual health after a cardiac event, stress management, nutrition, and cardiac risk factor management.

### *Measures*

*Blood pressure* at rest was measured with a standard mercury sphygmomanometer. The mercury sphygmomanometer is considered to be the 'gold standard' for indirect blood pressure measurement (Jones, Appel, Sheps, Roccella, & Lenfant, 2003). Three measures of blood pressure were taken with one minute intervals in between each measurement. Resting blood pressure was the mean of these three readings. Since standing blood pressure is used diagnostically, the attending physician or exercise specialist measured standing pre-exercise blood pressure. Pre-exercise (anticipation) standing blood pressure

was measured with an aneroid sphygmomanometer after one and three minutes of standing.

*Heart rate* at rest was inferred from pulse rate which was measured manually by counting the radial pulse for 30 seconds and recorded in beats per minute (bpm). Foss and Keteyian (1998) suggest that pulse rate and heart rate are the same for most people. However, Hwu, Coates, and Lin (2000) found an average 2.16 bpm difference between 30 second radial pulse counts and an ECG measure. Sharpley and Gordon (1999) found 95% of differences between ECG and earlobe pulse were between -4.36 and 3.64 beats per minute. There appears to be strong agreement between pulse rate and heart rate, but there is likely some error in pulse rate compared to a standard ECG measurement of heart rate. Resting heart rate was the mean of the three measures. Pre-exercise heart rate was assessed from readings taken continually by the 12-lead ECG and was the HR at the time BP was measured. Thus, the highest HR was taken as the anticipation measure.

*Patient History Questionnaire* (PHQ). The PHQ was a standard part of the patient's record. The PHQ contains disease and cardiac event history, work and family history and medication history.

*Self-rated health* was assessed since a negative appraisal of one's illness may influence the patient's response to the exercise tolerance test as seen in Fraser, Rodgers, Murray, and Daub (2005) of this series of studies. Participants responded on a 5-point Likert type scale that asked, "in general would you say your health is" and response options were 1(*excellent*), 2(*very good*), 3(*good*), 4(*fair*), and 5(*poor*). This was reverse scored so that a higher score reflected better self-rated health. This method of assessing self-rated health has been used in a variety of large-scale studies (e.g., Melchior et al.,

2003; Ross & Wu, 1995; Walters, McDonough, & Strohschein, 2002) demonstrating a consistent association between poor self-rated health and mortality (Idler & Benyamini, 1997).

Idler and Benyamini (1997) reviewed 27 community based studies from around the world to examine the relationship between self-rated health and mortality. The authors found that 23 of the studies reviewed demonstrated a powerful and consistent relationship between self-rated health and survival/mortality after accounting for traditional risk factors. Thus, the authors conclude that this particular method of assessing one's health is "valid in predicting a substantively significant outcome" (p. 31), mortality. This offers predictive validity support for the instrument.

*Barrier self-efficacy for exercise* was assessed with the scale use previously (Fraser, Rodgers, Daub et al., 2005). This scale was developed by Blanchard et al. (2002a) and asked the patient how confident he/she was to exercise in the face of barriers to exercise. The items are meant to capture barriers encountered by cardiac patients and was developed in consultation with actual patients who had participated in this particular CR program, adding to the strength of inferences drawn from scores obtained from this instrument in this sample. Participants were provided a list of 16 obstacles that might interfere with their exercise and were asked to indicate how confident they were that they could exercise on a day when they experienced that obstacle. Responses ranged from 1 (*not at all confident*) to 10 (*very confident*). Some of the obstacles were "bad weather", "too much work", and "minor health problems." In the present study the internal consistency reliability was  $\alpha = .96$ .

Blanchard et al. (2002a) found that barriers efficacy had good internal consistency ( $\alpha = .86$ ) in a similar sample of cardiac patients including men and women. Also, barrier self-efficacy was related to adherence in CR, as expected. In a different sample of cardiac patients from the same CR program Blanchard, Rodgers, Courneya, Daub & Knapik (2002b) examined changes in barrier self-efficacy over time. Internal consistencies ranged from .64 to .87 over the four assessment periods. Barrier self-efficacy remained stable from 3-5 weeks before CR and immediately before CR. Further, barrier self-efficacy improved after the completion of the CR program. Changes (increases) in barrier self-efficacy over the course of the CR program were related to better adherence, increased vigor, and reduced anxiety. These observations are what would be expected based on theory (Blanchard et al., 2002b) adding support for the construct validity of barriers efficacy.

In a different sample (Fraser, Rodgers, Daub, et al., 2005) of these patients this instrument has demonstrated adequate internal consistency (.92). Further, scores were related to other constructs in meaningful ways. For example, those reporting less confidence to overcome barriers also reported more stress and less support for exercise. Given the recent development of this particular instrument, other validity information is lacking.

*Social support* was assessed with the Interpersonal Support Evaluation List (ISEL-12; Cohen & Hoberman, 1983). The ISEL is a 12-item Likert type scale ranging from 1(*definitely false*) to 4(*definitely true*) assessing three different types of functional support. The three kinds of support are appraisal, tangible and belonging support. Detailed validity information is reported in Chapter 2. The ISEL has been used previously with patients in

this program exhibiting adequate internal consistencies (e.g., Fraser, Rodgers, Daub, et al.) with subscale alphas ranging from .65 to .78. The alphas in this sample were .65, .68, and .82, for appraisal, tangible, and belonging support, respectively.

*Stress* was assessed with the short form of the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). The short form of the PSS is a 4-item Likert-type scale assessing general levels of stress. This measure assesses the person's perception of his/her ability to meet demands. Participants responded to questions about their thoughts and feelings over the last month. Examples include "In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?" and "In the last month, how often have you felt confident in your ability to handle your personal problem?" Responses ranged from 0(*never*) to 4(*very often*). Scale scores were computed as mean item responses for the items comprising the particular subscale. The internal consistency reliability for this sample was  $\alpha = .83$ .

Cohen, Kamarck, and Mermelstein (1983) noted that the PSS was developed as a global measure of perceived stress, as opposed to objective stress, to account for the cognitive component of the stress experience. Aside from the conceptual contribution to the creation of the PSS, the authors also provided other validity and reliability information for the PSS based on two college samples and a community sample. Internal consistencies ranged from .84 to .86 across the three samples. The four-item version was used in a community sample and had an internal consistency of .72 with a six-week test-retest reliability of .55. Two-day test-retest reliability was .85 in a college sample, and six-week test-retest was .55 in a community sample. According to the authors these values represent the relative short-term stability of perceived stress that is expected to change over longer

periods of time (e.g., 1-2 months). The PSS demonstrated small to moderate correlations with objective life events (list of stressful life event) ranging from .17 to .39, and higher correlations with the reported impact of those events ranging from .24 to .49. Thus the PSS demonstrated some relative independence from objective life events, which does not account for the cognitive component of stress, but was related to a similar construct, the impact of life events. The authors also compared number and impact of events and PSS scores in their correlation with depressive symptoms. The PSS was more highly related to depressive symptoms with correlations from .65 to .76 in the college samples compared to correlations of .29 and .33 between depressive symptoms and impact of life events and correlations of .18 and .14 between number of life events and depressive symptoms. In terms of physical symptoms, the PSS had correlations ranging from .52 to .70 across the three samples. Impact and number of life events ranged from .23 to .51 in their correlations with physical symptoms. These patterns of correlations support the predictive and concurrent validity of the PSS as an indicator of stress and also confirm the theoretical notion that perception of stress is more important than objective stress adding to the construct validity of perceived stress. The PSS was also related to maintenance of reduced smoking and social anxiety, where higher stress scores were related to more anxiety and poorer maintenance to smoking reduction.

Psychometric properties of the PSS were examined further in a probability sample in United States adults (mean age = 42.8) by Cohen and Williamson (1988). This study included a factor analysis of the PSS that suggested that a 10-item version of the PSS was as good as the original 14-item version. Further, the 4-item version was determined to be useful as well. In fact, the 4-item version was less ambiguous in terms of the results of the

factor analysis. Specifically, the factor analysis of 14 item version indicated a two-factor solution based on the positive and negative wording of items, as did the analysis of the 10 items version. The analysis of the four-item version contained one factor.

Similar to Cohen et al. (1983), Cohen and Williamson (1988) also gathered construct validity evidence by examining relationships between PSS scores and other indicators of stress as well as relationships between other meaningful constructs. Compared to Cohen et al., perceived stress was related to number of life events and the impact of those events in a similar magnitude. That is, the correlation between number of events and the PSS was .30 and the correlation between negative impact of events and PSS was -.27. The overall impact of events, however, was only correlated with the PSS at -.10.

Cohen and Williamson (1988) also reported correlations between perceived stress and self-rated health and health care use. As expected those reporting more stress also reported poorer self-rated health, more serious and nonserious illness symptoms, more flu symptoms, and more health care use. Finally, life dissatisfaction was related to more stress, and those reporting more stress reported considering seeking help for emotional or personal reasons.

This scale has been used with this CR program previously with an internal consistency of .75 (Fraser, Rodgers, Daub, et al., 2005). Scores on the PSS in this sample were related in theoretically meaningful ways. Specifically, higher stress was related to less social support and poorer self-efficacy.

*Anxiety* was assessed with the anxiety subscale of the Hospital Anxiety and Depression Scale (HADS-A; Zigmond & Snaith, 1983). The anxiety subscale examines

how anxious the patient has been feeling over the previous week. Sample items include “I can sit at ease and feel relaxed” and “I feel tense or ‘wound up’”. Participants responded on a 4-point Likert type scale ranging from 0 to 3. The validity of the HADS has been demonstrated previously in patients with noncardiac chest pain (Kuijpers et al., 2003) and in a recent literature review (Bjelland, Dahl, Haug, & Neckelmann, 2002). Bjelland et al. found 71 studies reporting some aspect of validity for the HADS. Nineteen studies of factor structure were found and 11 confirmed a 2-factor structure (anxiety and depression). Across fifteen studies internal consistencies ranged from .68 to .93 for the anxiety subscale and .67 to .90 for the depression subscale (HADS-D). Discriminant validity was examined by looking at the correlations between the two scales. Across 21 studies correlations between depression and anxiety subscales ranged from .40 to .74. Several studies reported relationships between other representative measures of depression and anxiety. Six studies reported correlations between the Beck depression inventory (BDI) and the HADS-D, which ranged from .62 to .73. Correlations between the HADS-A and Spielberger’s State-Trait Anxiety measure varied from .64 to .81 across six studies. Correlations between the symptom checklist-90 anxiety subscale and the HADS-A ranged from .49 to .73 across two studies, whereas the symptom checklist-90 depression subscale was correlated at .69 with the HADS-D in the two studies. Across four studies, the Montgomery-Ashberg depression rating scale was correlated with the HADS-A from .37 to .44 and with the HADS-D from .62 to .80 demonstrating some concurrent and discriminant validity. Similar correlations were found between the HADS-A and the clinical anxiety scale (.69 to .75) across three studies but low correlations were found between the HADS-A and the Hamilton anxiety scale (subscale score correlations from



.34 to .44) in one study. The studies reviewed by Bjelland et al. included samples of the general population, clinical samples (e.g., post-stroke, breast cancer, general medical, out-patient), and psychiatric samples, of various ages. Samples were from a variety of countries including Dutch, Swedish, Spanish, and Canadian, for example. The authors concluded that the HADS had generally similar properties regardless of the sampled population.

Kuijpers et al. (2003) examined selected validity of the HADS for diagnosing depression and anxiety in patients with chest pain. The results of the study showed that patients with a score greater or equal to 8 on either HADS subscale were likely to be diagnosed as depressed or as having panic disorder (73.3% vs. 3.9%) as determined by the Mini International Neuropsychiatry Interview (Sheehan et al., 1998). The internal consistency reliability in this sample was  $\alpha = .84$ .

### *Procedures*

Participants were sent a questionnaire package along with other orientation material as a part of their usual participation in CR. Participants were asked to complete the attached survey and to return it to the orientation session, which all new CR patients attend. Thus, patient characteristics and psychosocial variables were assessed prior to the exercise tolerance test.

The patient's exercise stress test occurred within one to two weeks of the orientation session. Patients were asked to refrain from consuming caffeinated beverages, tobacco products, and exercising 3 hours prior to the exercise test. Patients were asked not to consume alcohol for 12 hours prior to the exercise test. Upon arrival to the program, patients were brought to a separate room and rested quietly in the sitting position with the

blood pressure cuff on the non-dominant arm at heart level. Participants were left alone in the room for 5 minutes after which resting systolic and diastolic BP were measured 3 times one minute apart (cf. American College of Sports Medicine, 2000) to obtain resting values, with resting heart rate measured in between blood pressure measures. Systolic and diastolic BP (standing on the treadmill) were assessed immediately prior to beginning the participant's exercise stress test after one and three minutes of standing to ensure the participant did not suffer from orthostatic hypotension (an abnormal drop in SBP  $\geq 20$  mmHg from rest to standing most common among the elderly; Bradley, & Davis, 2003; Luukinen, Koshi, Laippala, & Airaksinen, 2004), a contraindication to participating in cardiac rehabilitation (American College of Sports Medicine, p. 167). It is normal for BP to drop when posture changes from a supine to a standing position, however BP values typically return within two heartbeats (Luukinen et al.; Silverthorn, 1998). These changes are usually the same for hypertensive individuals as well (Pickering & Blank, 1989) although not necessarily for older hypertensives (e.g., Cleophas, Grobowski, Niemeyer, Mäkel, van der Wall, 2002). However, hypotension associated with postural change among older hypertensives can be reversed with the use of beta-blockers (Cleophas et al.). This anticipation of the exercise test while standing was considered to be both a novel and naturalistic stressor (cf. Everson, Kaplan, Goldberg, & Salonen, 1996; Steptoe & Marmot, 2002; Thomas et al., 1992) thought to provoke variable CVR responses (Kamarck & Lovallo, 2003). Further, it was thought that exposure to an exercise stress test would be perceived as evaluative (cf. Hilmert, Kulik, Christenfeld, 2002), particularly stressful (i.e., physical exertion after a recent cardiac event or surgery), challenging, and perhaps threatening among CR patients. Threat and challenge have been identified as other

important characteristics of a psychosocial stressor (Herd, 1991). Further, the anticipation component has recently been identified as a key psychological stressor (Feldman, Cohen, Hamrick, & Lepore, 2004).

The difference between the mean resting BP's and HR and pre-exercise/anticipation (cf. Everson et al., 2001; Kamarck et al., 2000; Turner, 1994) BPs and HR were considered the CVR. Difference scores are thought to be reliable indicators of CVR (Kamarck & Lovallo, 2003; Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991) and are commonly used as indicators of cardiovascular response in the pharmacology, medical, and psychological literature cited above. The exercise stress test was conducted as a normal part of the CR program prior to beginning rehabilitative exercise. The attending physician and/or exercise technician recorded supine heart rate and BPs as well as the pre-exercise BP and heart rates immediately prior to the exercise test while the patient was standing on the treadmill.

### *Analyses*

Resting HR and BP values were compared to HR and BP values immediately prior to the exercise tolerance test. Repeated measures MANOVA (time by gender) was used to determine if there were differences between resting and pre-exercise HR and BP values. Gender differences were also explored with the repeated measures MANOVA. Partial eta-square ( $\eta^2_{\text{partial}}$ ) values are reported as effect sizes, which represent the proportion of variance in a dependent variable (i.e., HR, BP) explained by a factor (i.e., time or gender) (Cohen, Cohen, West, & Aiken, 2003; Pierce, Block, & Aguinis, 2004).

Difference scores (Ragossa, 1995) were used to calculate the magnitude of CVR among the CR patients (cf. Everson et al., 1996; Gump, Matthews, & Raikkonen, 1999;

Kamarck et al., 2000; Turner, 1994). In other words, average resting values were compared to peak values during the stressful period (cf. Kamarck, Jennings, Pogue-Geile, & Manuck, 1994). Thus, CVR scores for each variable were calculated as: reactivity score = peak cardiovascular parameter – mean cardiovascular parameter. For example,  $SBP\text{-reactivity} = SBP_{\text{peak}} - SBP_{\text{mean}}$ .

Given the relatively small sample, the relationship between CVR scores and psychosocial variables were examined by correlations. Further, to improve power, a relaxed  $p$ -value ( $p < .10$ ) was employed (Cohen, 1990; Stevens, 2002) and reported in examining the correlations. One-sided  $t$ -tests for correlations were conducted based on the hypothesized direction of relationships. More importantly, however, effect sizes (i.e., correlations) were interpreted based on their sizes and not on  $p$ -values per se. This is in line with Cohen (1990) who argued that measures of effect size are “the primary product of research inquiry” (p. 1310) and not  $p$ -values.

## *Results*

### *Data screening*

Data were screened for missing and unusual values by examining the descriptive statistics including means, ranges, and frequencies of responses to each survey item (Tabachnik & Fidell, 2001). Unusual values were those with ranges that were outside of possible response options. Missing and unusual values were checked with the original data and replaced with the appropriate value or confirmed as missing.

Missing data was a problem for the physiological data in this study. The total sample size was 98 people and HR and BP data was only available for 62 people. Thus, in reporting results with HR and BP, data are only reported for these people and no attempts

were made to estimate or replace missing data. The main reasons for missing data on HR and BP are that the participant did a stress test elsewhere prior to coming to the rehabilitation program. In terms of survey data, there was some missing data for different items. In the cases where there was data missing for only a few survey items, missing data on each item was replaced with the mean of the participant's responses for other items on the particular subscale (cf. Tabachnik & Fidell, 1996, p. 64). For example, with the ISEL if item 4 was missing, it was replaced by the mean of items 2, 6, and 11 (after the appropriate reverse scoring). Some participants did not answer any questions for a particular survey scale or subscale. This data was left as unreported. Full data for the various surveys was calculated for 87-90 people for the multiple item surveys and 83 people answered the question about self-rated health status. Since this data was used in correlation analyses only, data were deleted pairwise. In other words, the correlations between self-rated health and any other survey variable has a maximum sample size of 83, whereas the correlation between anxiety and depression is based on a sample of 89. Similarly, the correlations between HR, BP and survey variables are limited to the number of participants who provided data on HR and/or BP.

Data were also checked for outliers, since outliers can have a profound affect on results and outliers can help identify potential errors in data entry (Osborne & Overlay, 2004). Typical procedures including examining histograms, z-scores, box-plots, and scatterplots were used to identify potential outliers. The survey variables were all within possible ranges. The biggest concern in terms of outliers was for HR change, where the highest value from rest to pre-exercise was 40bpm. The next highest value was 20bpm. The value of 40 was associated with a z-score of 4.95, the next closest z-score in

magnitude was -2.25. Thus, this value was double checked. Notes taken during data collection indicated potential problems with obtaining an accurate radial pulse at rest and this value was considered to be potentially unreliable. Therefore resting HR data for this person was not included in the following analyses. Elimination of this value resulted in little change in average resting HR in the sample. The correlations between change in HR from rest to pre-exercise and other variables changed markedly for the relationship between self-efficacy and self-rated health and HR-change among men with elimination of the outlier. Examination of the histogram and box-plot showed that the highest value of peak pre-exercise DBP was curiously high. The value of 118 was double checked and found to be inaccurate. This data entry error was therefore corrected.

#### *Analysis assumptions*

Several general analysis assumptions were checked and specific assumptions for a repeated measures analysis and correlations are separately reported below. Cohen et al. (2003) suggest that no assumptions are necessary to calculate or interpret correlation (or regression) coefficients (p.41). However, useful inferences about the population can be drawn when assumptions are met. Further, Cohen et al. note that moderate violations of these assumptions have little impact on error of inferences since these statistics are fairly robust (see also Kleinbaum, Kupper, Muller, 1988). Nevertheless, various assumptions were tested for severe violations.

Normality of the variables is a general requirement for correlational type analyses (repeated measure being included), and so the normality of the variables was examined. Typical techniques such as examining distributions, box-plots, skewness, kurtosis, and normal probability plots (P-P plots) were used after the examination of outliers as

mentioned above. Histograms showed that anxiety, stress, and social support subtypes were skewed. The skew was in the direction indicating that people tended to report being low in stress and anxiety and high in support. Skewness values were checked and the highest values for skew were resting SBP (1.39) and belonging support (-1.13). These variables also had the most kurtosis with values of 3.72 for resting SBP and 1.10 for belonging support. The normal probability plots were examined based on recommendations by Kleinbaum et al (1988) and Tabachnik and Fidell (2001) who suggest that P-P plots are probably more useful than histograms. These plots revealed no serious violations or departures from normality. Thus, normality was thought to be possibly violated to a small extent in these variables. Follow-up regression diagnostics, reported below, were employed to further examine the extent of this violation.

#### *Repeated measures MANOVA*

Specific assumptions for repeated measures analysis include independence of observations, multivariate normality, and equality of group variances. Observations in this study were based on separate participants. Thus, the research design accounted for independence of observations. Further, data were collected from a fairly homogeneous group so that likelihood of clustering was minimized (cf. Cohen et al., 2003). Multivariate normality was assessed by examining each variable for univariate normality. Stevens (2002) argues that this is usually sufficient, and he further argues that much nonnormality comes from outliers. Thus, outliers were also checked. However, Stevens argues that repeated measures ANOVA and MANOVA are fairly robust with respect to violations of the assumption of multivariate normality. Nevertheless, both normality and outlier checks are reported above. Finally, Box's test was used to examine equality of group variances.

Specifically, equality of variances between men and women were tested by using gender as a grouping variable in a discriminant function analysis with all study variables and independent variables. Results showed that the covariance matrices did not differ remarkably between men and women.

#### *Correlational analyses*

The assumptions for correlation analyses are similar to regression analyses (cf. Cohen et al., 2003), and violations of these assumptions generally only cause problems when they are severe. Assumptions about normality of variables were tested above. Assumptions surrounding perfection of reliability of measurement are reported in the measures section below. Linearity and homoscedasticity were checked by examining scatterplots among the variables. Further, regression analysis was used with the change measures as dependent variables to see how the residuals were distributed for the independent variables.

Scatterplots suggested that there were no apparent nonlinear relationships and that homoscedasticity was not severely violated. Follow-up residual analysis showed that the residuals (of the dependent variables HR, SBP, DBP, and MAP-change) were all approximately normally distributed. Further, the residual plots showed that data was spread around a mean of zero with no discernible pattern. That is, there was no indication of a possible nonlinear or curvilinear relationship. To be sure, the patterns were not perfect, but there were clearly no serious violations and no patterns to suggest nonlinear relationships. Any violations were therefore thought to be minor (keeping in mind the comment by Kleinbaum et al. that this process is necessarily qualitative). Also, data



seemed to be fairly homoscedastic with respect to a full range of residuals being represented throughout the range of independent variables.

### *Sample characteristics*

Patient characteristics are reported in Table 3-1. Typical of this program, most patients were married (74.7%) and most smoked prior to their heart event (74.7%). Almost half of patients were attending CR for a heart attack (47.5%), more than half had angioplasty (56.6%) as part of their treatment, and 36.7% had bypass surgery. The most common comorbidities were hypercholesterolemia (61.6%), hypertension (58.6%) and arthritis/joint problems (37.4%), typical of other samples from this program. Women had higher rates of arthritis/joint problems ( $\chi^2(1) = 7.86, p < .01$ ) and stomach problems ( $\chi^2(1) = 6.11, p < .05$ ) than men. Table 3-2 shows descriptive statistics for women and men on the psychosocial study variables. Women and men did not significantly differ in any of the psychosocial variables except for anxiety. Women reported significantly more anxiety than men,  $F(1, 97) = 6.11, p = .015$ .

### *Cardiovascular responses*

Heart rate and blood pressure responses at baseline and pre-exercise are shown in Figure 3-1. Mean arterial pressure (MAP) was calculated as  $MAP = DBP + 1/3(SBP - DBP)$ . Results of a repeated measures MANOVA (time by gender) showed that HR, SBP, DBP, and MAP all increased significantly from resting to peak pre-exercise. There were significant main effects for time ( $\eta^2_{\text{partial}} = .72$ ) and gender ( $\eta^2_{\text{partial}} = .23$ ) but there were no significant interactions. Significant large effect sizes (Cohen, 1992) for the cardiovascular changes over time were:  $\eta^2_{\text{partial}} = .57$  for HR,  $\eta^2_{\text{partial}} = .65$  for SBP,  $\eta^2_{\text{partial}} = .23$  for DBP, and  $\eta^2_{\text{partial}} = .54$  for MAP. Only SBP and MAP were significantly different between men

and women with effect sizes of  $\eta^2_{\text{partial}} = .17$  and  $\eta^2_{\text{partial}} = .12$ , respectively. Table 3-3 reports specific cardiovascular variable measurements at rest and pre-exercise for both men and women.

#### *Correlates of cardiovascular reactivity*

Table 3-4 reports the correlations among the psychosocial variables and the CVR variables for men and women. Reactivity scores were calculated as the change from resting to anticipation for HR, SBP, DBP, and MAP. These reactivity scores were correlated with the psychosocial variables. Given the small sample, correlations significant at the  $p < .10$  level were identified in addition to the usual  $p < .05$  and  $p < .01$  levels. Additionally, relevant effect sizes were reported despite potentially being non-significant (Cohen, 1990). As seen in Table 3-4 there were several interesting correlations among CVR variables and psychosocial variables, but there is a clear lack of power.

#### *HR-reactivity*

The largest correlations among CVR variables and the psychosocial variables for women was between HR-reactivity and self-efficacy ( $r = -.61$ ), anxiety ( $r = .47$ ), and self-rated health ( $r = -.51$ ). Specifically, low self-efficacy and self-rated health and more anxiety was associated with a larger change in heart rate from rest to anticipation. The correlation between stress and HR-reactivity was nonsignificant but the effect size ( $r = .29$ ) supports the hypothesis that stress should be related to more reactivity. These relationships were all in the hypothesized directions. The correlations between total social support ( $r = -.34$ ), appraisal ( $r = -.40$ ) and belonging social support ( $r = -.39$ ) and HR-reactivity would be considered medium effect sizes (Cohen, 1992), significant at  $p < .10$ . Thus, more social support is associated with less HR-reactivity. These correlations are in

the hypothesized direction and support the hypothesis that social support should attenuate CVR responses.

For men, the correlations between HR-reactivity and total social support ( $r = .25$ ), appraisal support ( $r = .34$ ) and tangible support ( $r = .24$ ) showed that more support was associated with a bigger change from rest to anticipation. This result was in the opposite direction of what was hypothesized. Self-efficacy was negatively related to HR-reactivity ( $r = -.22$ ) as hypothesized and self-rated health was positively related to HR-reactivity opposite of the hypothesized direction ( $r = .31$ ).

#### *SBP-reactivity*

In terms of SBP-reactivity among women, the relationships between anxiety and SBP-reactivity and self-rated health and SBP-reactivity were  $r = .40$  and  $r = .42$  respectively, at  $p < .10$ . That is, more anxiety was related to higher SBP-reactivity, as hypothesized, and better self-rated health was related to higher SBP-reactivity, opposite of the hypothesis. The other notable correlation was between SBP-reactivity and self-efficacy which was  $r = -.24$ . Higher self-efficacy related to lower SBP-reactivity supported the hypothesized relationship. In terms of social support, the associations with SBP-reactivity were generally small ranging from  $r = .16$  to  $r = .23$ . These were in the opposite direction of the hypotheses.

In men, belonging support ( $r = -.22$ ) and self-efficacy ( $r = -.25$ ) were related to SBP-reactivity. More support and higher self-efficacy were related to lower SBP-reactivity. These relationships were in the hypothesized directions. Other psychosocial variables were not significantly related to SBP-reactivity in men, and the effect sizes were all small.

### *DBP-reactivity*

None of the psychosocial variables were significant and the effect sizes were small. In both men and women, none of the psychosocial variables was significantly related to DBP-reactivity and all of the effect sizes were less than  $r = .20$  in magnitude.

### *MAP-reactivity*

Among women, mean arterial pressure had a medium effect size with anxiety ( $r = .28$ ) and self-rated health ( $r = .31$ ). Higher anxiety associated with larger reactivity supported the hypothesis, but higher self-rated health with higher reactivity was opposite of what was hypothesized. No other psychosocial variables were related to MAP-reactivity for women. In men, all of the effect sizes between MAP-reactivity and the psychosocial variables were non-significant and small (all  $r$ 's  $\leq .15$  in magnitude).

### *Psychological correlates*

Although this study lacked the power to more formally examine the paths from social support to CVR (i.e., through structural equation modeling or hierarchical regression analysis), the correlations in Table 3-4 provide some evidence for the hypotheses above.

For both men and women, social support was related to anxiety and stress in the hypothesized direction. That is, more social support was associated with less stress and less anxiety. For women, this relationship was fairly stable for stress and social support. That is, stress was correlated with the various aspects of social support from  $r = -.48$  to  $r = -.50$ . For men, belonging support had the highest relationship to stress ( $r = -.72$ ), and tangible support had the lowest ( $r = -.54$ ). Similarly, anxiety was related to social support facets in women from  $r = -.31$  to  $r = -.39$ . For men the correlations between anxiety and

support ranged from  $r = -.36$  for tangible support to  $r = -.57$  for belonging support. These results support the hypothesis that support should lead to less stress and anxiety.

In terms of self-efficacy, for both men and women more anxiety and stress was related to less self-efficacy. This is consistent with what was hypothesized. Further, more social support was related to more self-efficacy. However, this relationship was weaker than the relationship observed between stress and self-efficacy, and between stress and support. Thus, the variables thought to be more distal from each other were less related than variables thought to be more proximal (cf. Wilson, Rodgers, Fraser, & Murray, 2004). This pattern of relationships arguably supports the hypothesis that more social support should lead to less stress and this should then lead to higher self-efficacy (cf. Fraser, Rodgers, Daub, et al., 2005). Similarly, the pattern of correlations suggests that social support is less related to anxiety than stress. Thus, the hypothesis that social support might influence anxiety by first impacting on stress has some support.

### *Discussion*

The main purpose of this study was to examine the potential for social support to attenuate the cardiovascular response to the anticipation of an exercise tolerance test in cardiac rehabilitation patients. Another purpose was to examine the role of self-rated health, stress, anxiety, and self-efficacy on CVR. This study therefore examined downstream relationships at the micro and pathways level of the Berkman and Glass (2000) model.

The anticipation period appeared to elicit significant cardiovascular responses. It was not clear if DBP and HR changes would be significant based on the review of the medication literature previously. It was expected that changes in SBP responses among

patients would be significant, however, despite the varied medications. Effect sizes were large for all CVR variables, and all cardiovascular changes from rest to anticipation were significant. Women had higher resting and anticipation SBP and MAP than men, but there were no interaction effects. Thus, men and women responded to the stressor in essentially the same manner.

Overall these results suggest that the micro level factor social support might be related to the physiological pathway CVR in heart patients attending CR. Importantly, if these results are replicated in larger samples, there appears to be differences in the kinds of social support and psychological factors that affect different CVR parameters among men and women. Thus, CVR responses might have different psychosocial determinants for men and women. Further, different aspects of CVR might be more related to psychosocial determinants than other aspects.

#### *Psychosocial correlates of CVR*

In terms of social support effects on CVR, the largest effects among women were seen for social support attenuating HR-change. Specifically, more appraisal support and belonging support was related to smaller HR-change from resting to anticipation. This result seems to confirm previous findings in the social support literature suggesting that appraisal (or emotional) support is generally adaptive. Further, belonging support is thought to be particularly important for women. Taylor et al. (2000) argue that in times of stress women benefit from befriending. These results show that more support was related to less HR-reactivity. Interestingly, HR-reactivity was also related to anxiety. Women who reported more anxiety showed larger changes in HR from rest to anticipation. Further, those women with more support also reported less anxiety. Self-efficacy and self-rated

health were negatively related to HR-reactivity. That is, women who were more confident in their ability to overcome barriers to exercise and women who reported better health, had lower changes in HR from rest to anticipation. Both self-rated health and self-efficacy were negatively related to anxiety and positively related to social support. These results partly replicate those of Fraser, Rodgers, Daub, et al. (2005). That is, the correlations suggest that social support might attenuate anxiety and might increase self-efficacy. If these results can be replicated, they extend Fraser, Rodgers, Daub, et al. by showing that these psychological processes then impact upon HR-reactivity. Unfortunately the small sample here does not allow for mediational analysis to test this explanation.

For men, HR-reactivity was also related to appraisal support, but the relationship with belonging support was small and non-significant. In men, higher HR-reactivity was also related to more tangible support. The relationships between reactivity and appraisal and tangible support were contrary to expectations. It is not clear why HR-reactivity was positively associated with support in men. It is possible that the support men received, or at least the support that was salient after the heart event, was more controlling than helpful. In terms of appraisal support and tangible support, it might be that these men had noticed these kinds of support being offered to them. Dakof and Taylor (1990) found that among cancer patients emotional/appraisal support could at times be perceived as helpful or misguided. Whether having more support subjects one to more good and bad forms of support, or if this support was interpreted as misguided is not known for this study. Future research might benefit from clarifying the patient's interpretation of support. Another explanation is that support was more salient for men who were worse off, and therefore needed support. For example, patients are likely to receive support from multiple sources

such as the physician, nurse, and family. These multiple sources may provide more support to patients who are sicker, and the source of this support (friend, physician, spouse) may influence whether the support is interpreted as misguided or helpful (cf. Dakof & Taylor, 1990).

Self-efficacy and self-rated health were significantly related to HR-reactivity in men and women. For women, self-efficacy and self-rated health were the largest psychosocial predictors of HR-reactivity. In men, the relationships were smaller, and in the opposite direction. That is, more self-efficacy and better self-rated health was associated with less HR-reactivity in women and more in men. These relationships need to be explored in future research. If these results can be replicated it might highlight an important difference in the way men and women respond to stressors (cf. Taylor et al., 2000). However, it might also reflect a different interpretation of the stressor. This study did not address how the situation was perceived, so future research might try to determine if men and women differ in terms of their appraisal of the situation as threatening, evaluative and challenging (Herd, 1991). Herd argues that these characteristics of a situation can maximize a stress response. In this study, it is not clear if the situation provoked a stress response because the exercise test was perceived as potentially challenging, threatening or evaluative. If this information were available it might explain some of the gender differences observed here.

In terms of SBP-reactivity, the effect sizes for social support among women were in the small to medium range (Cohen, 1992). However, anxiety and self-rated health were positively related to SBP-reactivity. That is, more anxiety and higher self-rated health was related to higher change in SBP. Whereas higher SBP-reactivity with more anxiety is



consistent with the hypothesized relationship, the positive association with self-rated health is opposite of what was expected. It is not clear why better self-rated health was related to greater SBP-reactivity. However, it might lie in how the situation is perceived. Clearly, the situation resulted in significant increases in SBP, more than in any other aspect of CVR. Yet, it is not clear why the anticipation elicited this effect. It was presumed that situation was challenging and threatening (Herd, 1991). However, perhaps women with better health were concerned that the test might prove them wrong. In other words, it may be that the situation is perceived as a threat since it might undermine the views of the women that they are relatively healthy. This explanation requires further investigation since HR did change rather dramatically in the hypothesized direction in women with lower self-rated health.

For men, SBP-reactivity was related to self-efficacy and social support in a manner consistent with the hypotheses. More self-efficacy and more belonging support was related to less SBP-reactivity. This is consistent with the idea that social support should attenuate SBP responses. Further, this supports the role of self-efficacy in attenuating the appraisal of a stressful situation. The results here also show that social support is related to more self-efficacy. Thus, future research with larger samples should test the pathway from support to self-efficacy and SBP-reactivity further. Finally, interpreted in light of Fraser, Rodgers, Murray, et al. (2005) and Blanchard et al. (2002a) these results offer support for the idea that social support can impact on self-efficacy and show how this might affect physical processes in heart patients.

For women and men, DBP-reactivity was not significantly related to any psychosocial variables and the effect sizes were generally small. The results of MAP

mirror those of DBP-reactivity. One exception was that higher self-rated health was related to more MAP-reactivity in women. However, this probably reflected the rather large effect seen between SBP-change and self-rated health.

#### *Relationships among psychosocial variables*

In terms of the relationships among the psychosocial variables, results generally support the hypothesized relationships. Social support subtypes were related to less stress and less anxiety and more self-efficacy. A closer look at the pattern of relationships showed that, generally, social support was more highly related to stress than to anxiety and self-efficacy. This pattern is what one would expect if social support influenced self-efficacy and anxiety through stress (cf. Wilson et al., 2004). If these results are replicated in larger samples they offer support for Fraser, Rodgers, Daub, et al. (2005) and extend those findings to a physiological indicator, CVR. Further, evidence of weaker relationships between upstream factors and factors further downstream offers support for Berkman and Glass (2000) framework at this level. Specifically, this supports the suggestion that social support influences downstream pathways among psychological and physiological processes.

Another interesting finding with respect to the relationships among social psychological variables is the difference in the sizes of correlations between men and women. For men, social support types had a very similar relationship to self-efficacy ranging from .27 to .30. For women, correlations ranged from .30 to .44, where belonging support had the highest correlation with self-efficacy. However, in terms of stress, the correlations with social support types ranged from -.48 to -.50 in women, and from -.54 to -.72 for men, where the highest correlation with stress was belonging support. Thus,

belonging support might be important for men and women for different reasons. That is, sense of belonging seems to influence self-efficacy in women more than in men.

Belonging support seems to influence feelings of stress more in men than in women.

### *Limitations*

This study had a number of limitations which impact on the generalizability of the findings and the confidence in the results. First, this sample was small and gender differences were difficult to assess accurately. That is, the severely limited power meant that many medium to large effect sizes for women were not statistically significant. Nevertheless, the effects sizes here can offer insight for future research to help target appropriate sample sizes.

Second, of the 97 participants recruited into this study, only 17 women and 45 men completed the physiological assessments. Thus, it is not clear how this subset of individuals represents the patients in this CR program, or cardiac rehabilitation patients more generally. For example, some of the patients recruited into this sample did not provide physiological data because they performed their stress tests at some other location. Thus, people from outside of Edmonton, or people with a different course of disease may not be represented here. Related, this sample was composed of patients who were referred and at least showed up for some aspect of the CR program. It is not clear how heart patients in general would compare to this sample. For example, there are known differences between those who do and do not show up, or who are not referred, to CR. In fact, most people eligible for CR do not attend or are not referred to CR. Thus, perhaps less than 70% of eligible heart patients would not be represented (cf. King et al., 2001).

Future research should try to sample heart patients who do not attend CR to see how they would compare to patients who attend CR.

Another limitation concerns the lack of control in a field study like this one. Whereas some research (Myers & Valdivieso, 2003) shows that a resting blood pressure measure taken by a research assistant is closer to an ideal measure of resting blood pressure (i.e., ambulatory resting blood pressure) than a physician or specialist (i.e., white coat effect), the readings here are likely not an optimal representation of resting BP. Future research could use an automated device so that BP readings could be taken with no one present with the patient. In terms of resting measures, a radial pulse measure was used instead of a direct measure of heart rate. Although pulse measures are generally very close to heart rate (e.g., Sharpley & Gordon, 1999), an error of a few beats per minute in resting heart rate could impact on the size of the cardiovascular reactivity observed.

Other influences on resting cardiovascular measures include time of day effects and medication effects. These two factors might interact in this study since participants were tested at any time of day from morning to afternoon. Blood pressure varies throughout the day normally (Pickering, 1990), but with these patients medication effects could change as time passes between doses. Thus, there may be blood pressure differences if a patient was tested early vs. late in the morning due to typical changes in BP as well changes associated with the clearance of the medication.

A related limitation is the measurement of anticipatory cardiovascular measures. The BP measures were taken in the exercise testing lab by the attending physician or the exercise technologist. There is likely some variability in BP measurement technique among the physicians and the exercise technologist. Further, it may be that the physicians

elicited cardiovascular responses in the patients different from that of the exercise technologist.

Equipment differences between resting and pre-exercise blood pressures could potentially add a source of error. In this study, the mercury sphygmomanometer was used for resting BP measures, but an aneroid device was used to measure pre-exercise BP. Whereas the mercury device is thought to be the 'gold standard' for indirect BP measurement, the aneroid device has been criticized as being inaccurate. Canzanello, Jensen, and Schwartz (2001) found that properly calibrated aneroid devices were within 0.5 mmHg of a digital pressure device calibrated with a mercury sphygmomanometer. However, they also point out that most mercury devices are easier to maintain than aneroid devices. Thus, it is conceivable that the calibration of the aneroid device was not maintained to the same extent of the mercury device. Further, the actual use of the aneroid device is different from the use of the mercury device. That is, the aneroid device is based on a spring system and numbers are read like reading a hand on a clock, whereas the mercury device is influenced by gravity and involves reading numbers next to a vertical column of mercury. Fortunately, the aneroid manometer was the device consistently used by the physicians and exercise technologist in the exercise testing lab. Nevertheless, future research should use a more consistent measure of BP measurement, such as an automated device for both resting and pre-exercise blood pressures.

Finally, a better estimate of peak cardiovascular responses could be obtained if more frequent blood pressure measures were taken. It is probably the case that the peak BP values recorded here were underestimates of the true peak value. Since two readings were taken two minutes apart, it seems likely that at some point outside of these two

measurements, HR and BP values would be higher. This could be examined with an automated device measuring second by second BP measures. The different testers could also have an inexplicable influence on cardiovascular reactions to the testing situation. Specifically, all of the physicians were men, whereas the exercise technologist was female. The gender of the tester may have had an influence on how the men and/or women may have reacted in this situation.

These results generally suggest that HR-reactivity is related to certain psychosocial variables with key differences between men and women. For men, appraisal and tangible support was related to more HR-reactivity, and for women more anxiety and low self-efficacy and self-rated health were related to larger HR-reactivity. For women it appears that appraisal support might attenuate HR responses but for men HR responses might be exaggerated. Systolic BP responses differ in their influences between men and women as well. For women, social support seems less important than self-efficacy and self-rated health, whereas for men belonging and self-efficacy are important. However, these relationships might indicate a pathway from social support to self-efficacy to HR and SBP-reactivity. If future research can confirm these findings it would add support to the idea that social support, upstream of psychological and physiological pathways, has an influence on the specific psychophysiological processes between self-efficacy and reactivity in CR patients. Generally, however, these results are consistent with the relationships outlined by Berkman and Glass (2000) in this group of CR patients.

## References

- Allen, K., Shykoff, B.E., & Izzo, J.L. (2001). Pet ownership, but not ACE inhibitor therapy, blunts home blood pressure responses to mental stress. *Hypertension*, 38, 815-820.
- American College Of Sports Medicine (2000). *ACSM's guidelines for exercise testing and prescription* (6th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- Bairey, C.N., Krantz, D.S., DeQuattro, V., Berman, D.S., & Rozanski, A. (1991). Effect of beta-blockade on low heart rate-related ischemia during mental stress. *Journal of the American College of Cardiology*, 17, 1388-1395.
- Bateman, D.N., Dean, C.R., Mucklow, J.C., Bulpitt, C.J., and Dollery, C.T. (1979). Atenolol and chlorthalidone in combination for hypertension. *British Journal of Clinical Pharmacology*, 7, 357-363.
- Benight, C.C., Segall, G.M., Ford, M.E., Goetsch, V.L., Hays, M.T., & Taylor, C.B. (1997). Psychological stress and myocardial perfusion in coronary disease patients and healthy controls. *Journal of Psychosomatic Research*, 42, 137-144.
- Benschop, R.J., Nieuwenhuis, E.E.S., Tromp, E.A.M., Godaert, G.L.R., Ballieux, R.E., & van Doornen, L.J.P. (1994). Beta adrenergic blockade: Effects of beta-adrenergic blockade on immunologic and cardiovascular changes induced by mental stress. *Circulation*, 89, 762-769.
- Berkman, L.F., & Glass, T. (2000). Social integration, social networks, social support, and health. In L.F.Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 137-173). New York: Oxford University Press.

- Berkman, L.F., Leo-Summers, L., & Horwitz, R.I. (1992). Emotional support and survival after myocardial infarction: A prospective, populations-based study of the elderly. *Annals of Internal Medicine, 117*, 1003-1009.
- Bjelland, I., Dahl, A.A., Haug, T.T., & Neckelmann, D. (2002). The validity of the Hospital Anxiety and Depression Scale: An updated literature review. *Journal of Psychosomatic Research, 52*, 69-77.
- Blackburn, G.G., Foody, J.M., Sprecher, D.L., Park, E., Apperson-Hanson, C., & Pashkow, F.J. (2000). Cardiac rehabilitation participation patterns in a large, tertiary care center: Evidence for selection bias. *Journal of Cardiopulmonary Rehabilitation, 20*, 189-195.
- Blanchard, C.M., Rodgers, W.M., Courneya, K.S., Daub, B, Knapik, G. (2002a). Does barrier efficacy mediate the gender-exercise adherence relationship during phase II cardiac rehabilitation? *Rehabilitation Psychology, 47*, 106-120.
- Blanchard, C.M., Rodgers, W.M., Courneya, K.S., Daub, B, Knapik, G. (2002b). Self-efficacy and mood in cardiac rehabilitation: Should gender be considered? *Behavioral Medicine, 27*, 149-160.
- Blumenthal, J.A., Jiang, W., Waugh, R.A., Frid, D.J., Morris, J.J., Coleman, E., et al. (1995). Mental stress-induced ischemia in the laboratory and ambulatory ischemia during daily life. *Circulation, 92*, 2102-2108.
- Bradley, J.G., & Davis, K.A. (2003). Orthostatic hypotension. *American Family Physician, 68*, 2393-2398.



- Canzanello, V.J., Jensen, P.L., & Schwartz, G.L. (2001). Are aneroid sphygmomanometers accurate in hospital and clinical settings? *Archives of Internal Medicine*, *161*, 729-731.
- Cardillo, C., Mores, N., Motolese, M., & Folli, G. (1994). Effects of benazepril on stress testing blood pressure in essential hypertension. *The American Journal of Cardiology*, *73*, 368-373.
- Christenfeld, N., & Gerin, W. (2000). Social support and cardiovascular reactivity. *Biomedicine & Pharmacotherapy*, *54*, 251-257.
- Cleophas, T.J., Grobowsky, I., Niemeyer, M.G., Mäkel, W.M., van der Wall, E.E. (2002). Paradoxical pressor effects of  $\beta$ -blockers in standing elderly patients with mild hypertension. *Circulation*, *105*, 1669-1671.
- Cohen, J. (1990). Things I have learned so far. *American Psychologist*, *45*, 1304-1312.
- Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*, 155-159.
- Cohen, J., Cohen, P., West, S.G., & Aiken, L.S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah, NJ: Erlbaum.
- Cohen, S., & Hoberman, H.M. (1983). Positive events and social supports as buffers of life change stress. *Journal of Applied Social Psychology*, *13*, 99-125.
- Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*, 385-396.
- Cohen, S., Kaplan, J.R., & Manuck, S.B. (1994). Social support and coronary heart disease: Underlying psychological and biological mechanisms. In S.A. Shumaker

- & S.M. Czajkowski (Eds.), *Social support and cardiovascular disease* (pp. 195-221). New York: Plenum Press.
- Cohen, S., & Williamson, G.M. (1988). Perceived stress in a probability sample of the United States. In S. Spacapan & S. Oskamp (Eds.), *The social psychology of health* (pp. 31-67). Newbury Park, CA: Sage.
- Craig, F.W., Lynch, J.J., & Quartner, J.L. (2000). The perception of available social support is related to reduced cardiovascular reactivity in phase II cardiac rehabilitation patients. *Integrative Physiological and Behavioral Science*, 35, 272-283.
- Dakof, G.A., & Taylor, S.E. (1990). Victims' perceptions of social support: What is helpful from whom? *Journal of Personality and Social Psychology*, 58, 80-89.
- Delamater, A.M., Taylor, C.B., Schneider, J., Allen, R., Chesney, M., & Agras, W.S. (1989). Interpersonal behavior and cardiovascular reactivity in pharmacologically-treated hypertensives. *Journal of Psychosomatic Research*, 33, 335-345.
- Dimsdale, J.E., Mills, P., Ziegler, M., Leitz, K., & Nelesen, R. (1992). Converting enzyme inhibition and blood pressure reactivity to psychological stressors. *Hypertension*, 20, 210-213.
- Dracup, K. (1994). Cardiac rehabilitation: The role of social support in recovery and compliance. In S.A. Shumaker & S.M. Czajkowski (Eds.), *Social Support and Cardiovascular Disease* (pp. 333-353). New York: Plenum Press.
- Eliasson, K., Kahan, T., Hylander, B., & Hjemdahl, P. (1987). Responses to mental stress and physical provocations before and during long term treatment of hypertensive

- patients with  $\beta$ -adrenoceptor blockers or hydrochlorothiazide. *British Journal of Clinical Pharmacology*, 24, 1-14.
- Everson, S.A., Kaplan, G.A., Goldberg, D.E., & Salonen, J.T. (1996). Anticipatory blood pressure response to exercise predicts future high blood pressure in middle-aged men. *Hypertension*, 27, 1059-1064.
- Everson, S.A., Lynch, J.W., Kaplan, G.A., Lakka, T.A., Sivenius, J., & Salonen, J.T. (2001). Stress-induced blood pressure reactivity and incident stroke in middle-aged men. *Stroke*, 32, 1263-1270.
- Ewart, C.K. (1995). Self-efficacy and recovery from heart attack: Implications for a social cognitive analysis of exercise and emotion. In J.E. Maddux (Ed.), *Self-efficacy, adaptation, and adjustment: Theory, research, and application* (pp. 203-226). New York: Plenum Press.
- Fauvel, J.P., Najem, R., Maakel, N., Pozet, N., & Laville, M. (1998). Effects of moxonidine on stress-induced peak blood pressure and renal function: A randomized, double-blind, placebo-controlled crossover study. *Journal of Cardiovascular Pharmacology*, 32, 495-499.
- Feldman, P.J., Cohen, S., Hamrick, N., & Lepore, S.J. (2004). Psychological stress, appraisal, emotion and cardiovascular responses in a public speaking task. *Psychology & Health*, 19, 353-369.
- Foss, M.L., & Keteyian, S.J. (1998). *Fox's physiological basis for exercise and sport* (6th ed.). Boston: McGraw-Hill.

- François, B., Cahen, R., Gravejat, M.F., & Estrade, M. (1984). Do beta blockers prevent pressor responses to mental stress and physical exercise. *European Heart Journal*, 5, 348-353.
- Fraser, S.N., Rodgers, W.M., Daub, B., & Black, B. (2005). *A test of a model of stress and multiple levels of social support on overcoming barriers to exercise in cardiac rehabilitation*. Manuscript in preparation.
- Fraser, S.N., Rodgers, W.M., Murray, T.C., & Daub, B. (2005). *The relationship between sociodemographic factors, social support and exercise tolerance in men attending cardiac rehabilitation*. Manuscript submitted for publication.
- Gebara, O.C.E., Jimenez, A.H., McKenna, C., Mittleman, M.A., Xu, P., Lipinska, I. et al. (1996). Stress-induced hemodynamic and hemostatic changes in patients with systemic hypertension: Effect of verapamil. *Clinical Cardiology*, 19, 205-211.
- Gottdiener, J.S., Krantz, D.S., Howell, R.H., Hecht, G.M., Klein, J., Falconer, J.J., & Rozanski, A. (1994). Induction of silent myocardial ischemia with mental stress testing: Relation to the triggers of ischemia during daily life activities and to ischemic function severity. *Journal of the American College of Cardiology*, 24, 1645-1651.
- Greenwood, D.C., Muir, K.R., Packham, C.J., & Madeley, R.J. (1996). Coronary heart disease: A review of the role of psychosocial stress and social support. *Journal of Public Health Medicine*, 18, 221-231.
- Grosse, A., Bianchi, J.M., Diaz Puertas de Grosse, C.S., Iglesias, G.E., & Coviello, A. (1987). Vascular reactivity of hypertensive patients treated with thiazides, beta

- blockers and clonidine during a psychological experimental stress situation. *Hypertension*, *9*, 526.
- Gullette, E.C., Blumenthal, J.A., Babyak, M., Jiang, W., Waugh, R.A., Frid, D.J., et al. (1997). Effects of mental stress on myocardial ischemia during daily life. *JAMA*, *277*, 1521-1526.
- Gump, B.B., & Matthews, K.A. (1999). Do background stressors influence reactivity to and recovery from acute stressors? *Journal of Applied Social Psychology*, *29*, 469-494.
- Gump, B.B., Matthews, K.A., & Raikkonen, K. (1999). Modeling relationships among socioeconomic status, hostility, cardiovascular reactivity, and left ventricular mass in African American and White children. *Health Psychology*, *18*, 140-150.
- Herd, J.A. (1991). Cardiovascular response to stress. *Physiological Reviews*, *71*, 305-330.
- Hilmert, C.J., Kulik, J.A., & Christenfeld, N. (2002). The varied impact of social support on cardiovascular reactivity. *Basic and Applied Social Psychology*, *24*, 229-240.
- Hwu, Y.-J., Coates, V.E., & Lin, F.-Y. (2000). A study of the effectiveness of different measuring times and counting methods of human radial pulse rates. *Journal of Clinical Nursing*, *9*, 146-152.
- Idler, E.L., & Benyamini, Y. (1997). Self-rated health and mortality: A review of twenty-seven community studies. *Journal of Health and Social Behavior*, *38*, 21-37.
- Jacot-des-Combs, B., Brunner, H.R., Waeber, B., Porchet, M., & Biollaz, J. (1984). Blood pressure variability in ambulatory hypertensive patients: Effects of  $\beta$ -blocking agents and/or diuretics. *Journal of Cardiovascular Pharmacology*, *6*, 263-266.

- Jain, D., Burg, M., Soufer, R., & Zaret, B.L. (1995). Prognostic implications of mental stress-induced silent left ventricular dysfunction in patients with stable angina pectoris. *The American Journal of Cardiology*, *76*, 31-35.
- Jones, D.W., Appel, L.J., Sheps, S.G., Roccella, E.J., & Lenfant, C. (2003). Measuring blood pressure accurately: New and persistent challenges. *JAMA*, *289*, 1027-1030.
- Kahan, T., & Eliasson, K. (1999). The influence of long-term ACE inhibitor treatment on circulatory responses to stress in human hypertension. *American Journal of Hypertension*, *12*, 1188-1194.
- Kamarck, T.W., Eranen, J., Jennings, J.R., Manuck, S.B., Everson, S.A., Kaplan, G.A., et al. (2000). Anticipatory blood pressure responses to exercise are associated with left ventricular mass in Finnish men. *Circulation*, *102*, 1394-1399.
- Kamarck, T.W., Everson, S.A., Kaplan, G.A., Manuck, S.B., Jennings, J.R., Salonen, R. et al. (1997). Exaggerated blood pressure responses during mental stress are associated with enhanced carotid atherosclerosis in middle-aged Finnish men: findings from the Kuopio Ischemic Heart Disease Study. *Circulation*, *96*, 3842-3848.
- Kamarck, T.W., Jennings, J.R., Pogue-Geile, M., & Manuck, S.B. (1994). A multidimensional measurement model for cardiovascular reactivity: Stability and cross-validation in two adult samples. *Health Psychology*, *13*, 471-478.
- Kamarck, T.W., & Lovallo, W.R. (2003). Cardiovascular reactivity to psychological challenge: Conceptual and measurement considerations. *Psychosomatic Medicine*, *65*, 9-21.

- King, K.M., Humen, D.P., Smith, H.L., Phan, C.L., & Teo, K.K. (2001). Predicting and explaining rehabilitation attendance. *Canadian Journal of Cardiology*, 17, 291-296.
- Kleinbaum, D.G., Kupper, L.L., & Muller, K.E. (1988). *Applied regression analysis and other multivariable methods*. Boston: PWS-KENT Publishing Company.
- Krantz, D.S., Kop, W.J., Santiago, H.T., & Gottdiener, J.S. (1996). Mental stress as a trigger for myocardial ischemia and infarction. *Cardiology Clinics*, 14, 271-287.
- Kubzansky, L.D., & Kawachi, I. (2000). Affective states and health. In L.F. Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 213-241). New York: Oxford University Press.
- Kuijpers, P.M.J.C., Denollet, J., Lousberg, R., Wellens, H.J.J., Crijns, H., Honig, A. (2003). Validity of the hospital anxiety and depression scale for use with patients with noncardiac chest pain. *Psychosomatics*, 44, 329-335.
- Lasser, N.L., Nash, J., Lasser, V.I., Hamill, S.J., & Batey, D.M. (1989). Effects of antihypertensive therapy on blood pressure control, cognition, and reactivity: A placebo controlled comparison of prazosin, propranolol, and hydrochlorothiazide. *The American Journal of Medicine*, 86(Suppl. 1B), 98-103.
- Lepore, S.J. (1998). Problems and prospects for the social support-reactivity hypothesis. *Annals of Behavioral Medicine*, 20, 257-269.
- Llabre, M.M., Spitzer, S.B., Saab, P.G., Ironson, G.H., & Schneiderman, N. (1991). The reliability and specificity of delta versus residualized change as measures of cardiovascular reactivity to behavioural challenges. *Psychophysiology*, 28, 701-711.

- Luukinen, H., Koski, K., Laippala, P., & Airaksinen, K.E.J. (2004). Orthostatic hypertension and the risk of myocardial infarction in the home-dwelling elderly. *Journal of Internal Medicine*, 255, 486-493.
- Lynch, J.W., Everson, S.A., Kaplan, G.A., Salonen, R., & Salonen, J.T. (1998). Does low socioeconomic status potentiate the effects of heightened cardiovascular responses to stress on the progression of carotid atherosclerosis? *American Journal of Public Health*, 88, 389-394.
- Malhotra, S., Kumari, S., & Pandi, P. (2001). Effect of calcium antagonists on stress-induced rise in blood pressure and heart rate: a double-blind, placebo-controlled study. *International Journal of Clinical pharmacology & Therapeutics*, 39, 19-24.
- Mancia, G., Ferrari, A., Gregorini, L., Bianchini, C., Terzoli, L., Leonetti, G., et al. (1980). Methyldopa and neural control of circulation in essential hypertension. *The American Journal of Cardiology*, 45, 1237-1243.
- Mancia, G., Ferrari, A., Gregorini, L., Ferrari, M.C., Bianchini, C., Terzoli, L., et al. (1980). Effects of prazosin on autonomic control of circulation in essential hypertension. *Hypertension*, 2, 700-707.
- Manuck, S.B., Olsson, G., Hjemdahl, P., & Rehnqvist, N. (1992). Does cardiovascular reactivity to mental stress have prognostic value in postinfarction patients? A pilot study. *Psychosomatic Medicine*, 54, 102-108.
- Melchior, M., Berkman, L.F., Niedhammer, I., Chea, M., Goldberg, M. (2003). Social relations and self-reported health: a prospective analysis of the French Gazel cohort. *Social Science & Medicine*, 56, 1817-1830.



- Mills, P., & Dimsdale, J.E., (1991). Cardiovascular reactivity to psychological stressors: a review of the effects of beta-blockade. *Psychosomatics*, 32, 209-220.
- Missik, E. (2001). Women and cardiac rehabilitation: accessibility issues and policy recommendations. *Rehabilitation Nursing*, 26, 141-147.
- Muir, A.L., Burton, J.L., & Lawrie, D.M. (1969). Circulatory effects at rest and exercise of clonidine, an imidazoline derivative with hypotensive properties. *The Lancet*, 294, 181-185.
- Myers, M.G., & Valdivieso, M.A. (2003). Use of an automated blood pressure recording device, the BpTru, to reduce the “white coat effect” in routine practice. *American Journal of Hypertension*, 16, 494-497.
- Nicotero, J.A., Beamer, V., Moutsos, S.E., & Shapiro, A.P. (1968). Effects of propranolol on the pressor response to noxious stimuli in hypertensive patients. *The American Journal of Cardiology*, 5, 657-666.
- Osborne, J. W., & Overbay, A. (2004). The power of outliers (and why researchers should ALWAYS check for them). *Practical Assessment, Research, and Evaluation*, 9(6). Retrieved December 6, 2005 from <http://pareonline.net/getvn.asp?v=9&n=6>.
- Pedersen, S.S., & Denollet, J. (2003). Type D personality, cardiac events, and impaired quality of life: a review. *European Journal of Cardiovascular Prevention and Rehabilitation*, 10, 241-248.
- Pickering, T.G. (1990). *Ambulatory monitoring and blood pressure variability: Part 2*. London, Ontario: Science Press.

- Pickering, T.G., & Blank, S.G. (1989). The measurement of blood pressure. In N. Schneiderman, S.M. Weiss, & P.G. Kaufmann (Eds.), *Handbook of research methods in cardiovascular behavioral medicine* (pp. 69-79). New York: Plenum.
- Pierce, C.A., Block, R.A., & Aguinis, H. (2004). Cautionary note on reporting eta-squared values from multifactor ANOVA designs. *Educational and Psychological Measurement, 64*, 916-924.
- Ragosa, D. (1995). Myths and methods: "Myths about longitudinal research" plus supplemental questions. In J.M. Gottman (Ed.), *The analysis of change* (pp. 3-66), Mahwah, NJ: LEA.
- Ring, C., Harrison, L.K., Winzer, A., Carroll, D., Drayson, M., & Kendall, M. (2000). Secretory immunoglobulin A and cardiovascular reactions to mental arithmetic, cold pressor, and exercise: Effects of alpha-adrenergic blockade. *Psychophysiology, 37*, 634-643.
- Ross, C.E., & Wu, C-l. (1995). The links between education and health. *American Sociological Review, 60*, 719-745.
- Rüddel, H, Langewitz, W., Schächinger, H., Schmieder, R., & Schulte, W. (1988). Hemodynamic response patterns to mental stress: Diagnostic and therapeutic implications. *American Heart Journal, 116*, 617-627.
- Schmieder, R.E., Bahr, M., Langewitz, W., Rüddel, H., Schächinger, H., & Schulte, W. (1989). Efficacy of four antihypertensive drugs (clonidine, enalapril, nitrendipine, oxprenolol) on stress blood pressure. *The American Journal of Cardiology, 63*, 1333-1338.

- Schmieder, R.E., Schobel, H.P., Gatzka, C.E., Hauser, W., Dominiak, P., Mann, J.F.E. et al. (1996). Effects of angiotensin converting enzyme inhibitor on renal haemodynamics during mental stress. *Journal of Hypertension*, 14, 1201-1207.
- Sharpley, C.F., & Gordon, J.E. (1999). Differences between ECG and pulse when measuring heart rate and reactivity under two physical and two psychological stressors. *Journal of Behavioral Medicine*, 22, 285-301.
- Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Amorim, P., Janavs, J., Weiller, E., et al. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *The Journal of Clinical Psychiatry*, 59(Suppl. 20), 22-33.
- Silverthorn, D.U. (1998). *Human physiology: An integrated approach*. Upper Saddle River, NJ: Prentice Hall.
- Spence, J.D., Munoz, C., Huff, M.W., & Tokmakjian, S. (2000). Effect of amlodipine on hemodynamic and endocrine response to mental stress. *American Journal of Hypertension*, 13, 518-522.
- Stephoe, A., & Marmot, M. (2002). The role of psychobiological pathways in socioeconomic inequalities in cardiovascular disease risk. *European Heart Journal*, 23, 13-25.
- Stevens, J.P. (2002). *Applied multivariate statistics for the social sciences* (4<sup>th</sup> ed.). Mahwah, NJ: LEA.
- Straznicky, N.E., Howes, L.G., Lam, W., & Louis, W.J. (1995). Effects of *pravastatin* on cardiovascular reactivity to norepinephrine and angiotensin II in patients with

- hypercholesterolemia and systemic hypertension. *American Journal of Cardiology*, 75, 582-586.
- Strike, P.C., & Steptoe, A. (2003). Systematic review of mental stress-induced myocardial ischemia. *European Heart Journal*, 24, 690-703.
- Tabachnik, B.G., & Fidell, L.S. (1996). *Using multivariate statistics* (3<sup>rd</sup> ed.). New York: Harper Collins.
- Tabachnik, B.G., & Fidell, L.S. (2001). *Using multivariate statistics* (4<sup>th</sup> ed.). Needham Heights, MA: Allyn and Bacon.
- Taylor, S.E., Klein, L.C., Lewis, B.P., Gruenewald, T.L., Gurung, R.A.R., & Updegraff, J.A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review*, 107, 411-429.
- Thomas, S.A., Freed, C.D., Friedman, E., Stein, R., Lynch, J.J., & Rosch, P.J. (1992). Cardiovascular responses of patients with cardiac disease to talking and exercise stress testing. *Heart & Lung*, 21, 64-73.
- Turner, J.R. (1994). *Cardiovascular reactivity and stress: Patterns of physiological response*. New York: Plenum.
- Uchino, B.N., Cacioppo, J.T., & Kiecolt-Glaser, J.K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin*, 119, 488-531.
- Velasco, M., Romero, E., Bertoncini, H., Urbina-Quitana, A., Guevara, J., & Hernandez-Pieretti, O. (1978). Effect of propranolol on sympathetic nervous activity in hydralazine-treated hypertensive patients. *British Journal of Clinical Pharmacology*, 6, 217-220.

- Waal-Manning, H., & Bolli, P. (1980). Atenolol v placebo in mild hypertension: Renal, metabolic and stress antipressor effects. *British Journal of Clinical Pharmacology*, 9, 553-560.
- Walters, V., McDonough, P., & Strohschein, L. (2002). The influence of work, household structure, and social, personal and material resources on gender differences in health: an analysis of the 1994 Canadian National Population Health Survey. *Social Science & Medicine*, 54, 667-692.
- Weidner, G., Kohlmann, C-W., Horsten, M., Wamala, S.P., Schenck-Gustafsson, K., Hogbom, M., et al. (2001). Cardiovascular reactivity to mental stress in the Stockholm female coronary risk study. *Psychosomatic Medicine*, 63, 917-924.
- Wilson, P.M., Rodgers, W.M., Fraser, S.N., & Murray, T.C. (2004). Relationships between exercise regulations and motivational consequences in university students. *Research Quarterly for Exercise and Sport*, 75, 81-91.
- Zigmond, A.S., & Snaith, R.P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, 67, 361-370.

Table 3-1

*Demographic characteristics of women and men*

Demographic variable	Women ( <i>n</i> = 27)		Men ( <i>n</i> = 71)	
	<i>n</i>	%	<i>n</i>	%
Education				
Less than high-school	3	11.1	11	15.5
High-school	11	40.7	20	28.2
Some post secondary	11	40.7	35	49.3
Missing	2	7.4	5	7.0
Married				
Yes	18	66.7	56	78.9
Smoker before event				
Yes	20	74.1	56	78.9
Admitting diagnosis to CR				
Myocardial infarction	17	63.0	31	43.7
Bypass surgery	3	11.1	32	45.1
Angioplasty	14	51.9	45	63.4
Co morbidities				
Arthritis/joint	16	59.3	22	31.0
Asthma/bronchitis	4	14.8	6	8.5
High Blood pressure	15	55.6	46	64.8
Diabetes	5	18.5	16	22.5
High Cholesterol	16	59.3	48	67.6
Stomach	11	40.7	13	18.3

Note: Admitting diagnosis and comorbidities are not cumulative.

Table 3-2

*Means and standard deviations of psychosocial variables for women and men*

Psychosocial variable	Women ( <i>n</i> = 27)		Men ( <i>n</i> = 71)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age	59.89	11.81	60.07	9.95
Anxiety	8.12	3.64	5.95	3.74
Stress	2.27	.79	2.16	.86
ISEL-Total	3.27	.62	3.33	.61
ISEL-Appraisal	3.26	.66	3.26	.66
ISEL-Tangible	3.36	.66	3.39	.67
ISEL-Belonging	3.19	.73	3.32	.72
Self-efficacy	5.83	1.97	6.36	2.11
Self-rated health	2.95	1.02	3.20	.89

Note: ISEL: Interpersonal Support Evaluation List.

## Figure Caption

Figure 3-1.

Cardiovascular changes from baseline to anticipation (pre-exercise). All changes from resting to pre-exercise are significant ( $p < .001$ ).

Note: HR: heart rate in beats per minute; SBP: systolic blood pressure in mmHg; DBP: diastolic blood pressure in mmHg; MAP: mean arterial pressure in mmHg.



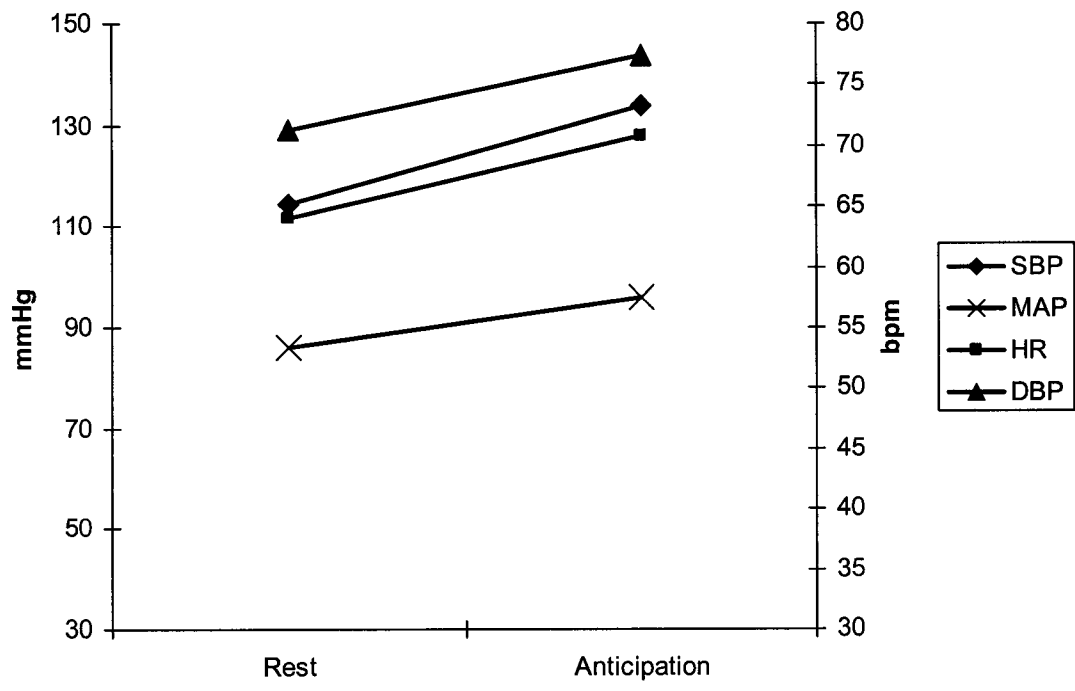


Table 3-3

*Means, standard deviations and ranges of mean resting and peak pre-exercise HR, BP, and MAP for men and women*

Variable	Women (n = 17)			Men (n = 45)		
	<i>M</i>	<i>SD</i>	range	<i>M</i>	<i>SD</i>	range
HR-rest	67.7	10.4	52.0-92.0	62.5	11.1	44.0-94.7
HR-preex	73.9	11.7	57.0-99.0	69.6	13.4	47.0-106.0
SBP-rest**	124.7	16.8	109.0-180.0	110.7	14.3	93.0-160.0
SBP-preex**	146.1	16.1	112.0-182.0	129.4	22.5	94.0-178.0
DBP-rest	74.3	12.6	50.7-93.0	70.2	10.3	51.3-96.0
DBP-preex	78.5	16.1	58.0-104	75.9	12.7	50.0-108.0
MAP-rest*	91.1	10.9	72.3-113.3	83.9	10.3	66.0-113.8
MAP-preex*	102.5	12.2	84.7-123.3	93.7	13.5	66.0-120.7

Note: HR-rest = resting heart rate; HR-preex = pre-exercise heart rate; SBP-rest = resting systolic blood pressure; SBP-preex = pre-exercise systolic blood pressure; DBP-rest = resting diastolic blood pressure; DBP-preex = pre-exercise diastolic blood pressure; MAP-rest = resting mean arterial pressure; MAP-preex = pre-exercise mean arterial pressure.

\* $p < .05$ , \*\* $p < .01$  for differences between gender

Table 3-4

*Correlations among psychosocial predictors and cardiovascular changes from resting to anticipation for men and women*

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Age	-	.03	-.01	.08	.01	.09	.07	-.03	-.04	-.10	.06	-.12	-.05
2. Anxiety	-.17	-	.72***	-.49***	-.41***	-.36***	-.57***	-.46***	-.38***	-.12	.16	.00	.09
3. Stress	-.39**	.52***	-	-.65***	-.57***	-.54***	-.72***	-.47***	-.34***	-.19	.01	-.06	-.04
4. ISEL-total	.31*	-.39**	-.54***	-	.90***	.87***	.92***	.31***	.12	.25*	-.09	.11	.03
5. ISEL-Appraisal	.33**	-.39**	-.50***	.88***	-	.66***	.76***	.27**	.03	.34**	-.06	.18	.10
6. ISEL-Tangible	.26	-.31*	-.48***	.90***	.67***	-	.68***	.30**	.09	.23*	.03	.18	.14
7. ISEL-Belonging	.25	-.36**	-.48***	.93***	.73***	.76***	-	.30**	.29**	.12	-.22*	-.04	-.15
8. Self-efficacy	.25	-.57***	-.42**	.41**	.30*	.37**	.44**	-	.30**	.22*	-.25*	.19	.01
9. Self-rated health	.29*	-.55***	-.52***	.63***	.52***	.51***	.67***	.81***	-	.31**	-.05	-.13	-.12
10. HR-change	-.20	.47**	.29	-.34*	-.40*	-.18	-.39*	-.61**	-.51**	-	.17	.26**	.27**
11. SBP-change	-.34*	.40*	.13	.20	.17	.16	.23	-.24	.42*	.34*	-	.19	.68***
12. DBP-change	-.05	.10	.02	.01	.02	-.06	-.07	-.04	.14	.03	.61***	-	.85***
13. MAP-change	-.21	.28	.08	.10	.10	.04	.16	-.16	.31	.20	.88***	.91***	-

Note: ISEL: interpersonal support evaluation list; HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure. Correlations above the diagonal are for men and correlations below the diagonal are for women.

\* $p < .10$ , \*\* $p < .05$ , \*\*\* $p < .01$

#### 4. DISCUSSION

The goal of this series of studies was to examine the ways in which social support impacted on the health of cardiac rehabilitation patients. This goal was undertaken by considering social support within a broader framework of social influences on health, as outlined by Berkman and Glass (2000). This broad framework provided some potential links to explore so that determinants of social support and effects of social support could be examined. Specifically, this framework places social support within the context of upstream social factors, such as socioeconomic factors (macro level) and the structure of one's social network (mezzo level), and downstream factors, where social support resides at the micro level, with psychological, behavioral, and physical pathways furthest downstream, most proximal to health.

As background to the dissertation studies, sociodemographic determinants of social support as well as their effects on a health indicator were examined. This study allowed an initial examination of the cascade of social influences outlined in Berkman and Glass (2000). The cascade from macro level (sociodemographic factors) to mezzo level (social networks) to physiological pathways (exercise tolerance) was examined. Fraser, Rodgers, Murray, and Daub (2005) found that the prognostic indicator exercise tolerance was affected by both social networks and sociodemographic factors. Limitations surrounding the scope of measurement of social networks and sociodemographic characteristics led to the first study of this dissertation. Study 1 attempted to more carefully measure relevant sociodemographic factors as well as social support and social networks.

Fraser, Rodgers, Daub, and Black (2005) examined social support at the micro level and the effects social support had on psychological pathways. Results showed that functional forms of social support influenced exercise specific social support. Further, higher levels of social support generally led to lower perceptions of stress, which lead to more confidence to overcome barriers to exercise among CR patients. Thus, this study found that social support influenced a specific pathway from stress to self-efficacy. This led to the second dissertation study which examined psychosocial consequences further. Specifically, Study 2 examined the role of social support and anxiety in cardiovascular reactivity.

Study 1 of the dissertation examined the cascade from macro level factors to mezzo level factors to social support at the micro level. Results showed that select sociodemographic factors were related to social network types (network size and diversity). Further, social networks were related to different functional forms of support. However, sociodemographic factors influenced functional forms of social support as well. That is, social networks did not appear to mediate the relationship between sociodemographic factors and social support functions in patients in cardiac rehabilitation. Rather select sociodemographic factors appeared to have a direct influence on social support. This study provides direction for future research into the effects of higher level social factors on health in cardiac rehabilitation patients by providing support for some of the potential pathways from sociodemographics, to social networks, and to social support suggested by the Berkman and Glass (2000) framework. These pathways should be tested in other populations to see where any differences might lie. Further, these pathways should be linked to psychological, behavioral, and physiological pathways to health.

Study 2 examined the influence of social support on psychological and physiological pathway level influences on health. Specifically, this study investigated changes in heart rate and blood pressure from rest to anticipation of a treadmill test and the psychosocial correlates of this change. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) all increased from resting to anticipation despite the patients taking numerous medications. Women and men only differed in terms of resting systolic blood pressure and resting MAP, as well as peak SBP and MAP. That is, the size of change from resting to anticipation did not differ between men and women. There was a lack of power to fully examine the pathways suggested from Fraser, Rodgers, Daub, et al. (2005) and Berkman and Glass (2000). Nevertheless, the size and direction of the correlations generally supported the hypotheses. Additionally, this data provides some guidelines for sample size determination in the future based on the size of the effects seen here.

Overall these dissertation studies benefited from examining the effect of social factors on health within the Berkman and Glass (2000) framework. The pathways from upstream to downstream factors were generally supported in this series of studies. Further, this series of studies identified some variables that may be critical for future research with cardiac rehabilitation patients. For example, income was a critical factor in terms of determining social networks and social support availability (Study 1) as well as the prognostic factor, exercise tolerance (Fraser, Rodgers, Murray, et al., 2005). In terms of psychological pathways, more social support was related to less stress/anxiety and more self-efficacy (Study 2, Fraser, Rodgers, Daub, et al., 2005). Further, the results of Study 2

showed that social and psychological pathways were potentially related to cardiovascular reactivity, a factor thought to be related to prognosis.

The results of these studies would be worth repeating in other clinical and nonclinical samples. Study 1 clarified some potential relationships identified in Fraser, Rodgers, Murray, et al. (2005) and suggested by Berkman and Glass (2000). Future research should replicate and integrate these studies. That is, the effects of sociodemographic factors on exercise tolerance seen in Fraser, Rodgers, Murray, et al. (2005) should be examined using the insights gained from Study 1. In particular, the examination of gender differences is a crucial component not available in Fraser, Rodgers, Murray, et al. Study 1 suggests that gender may be important in determining how social networks and social support are perceived. Whether or not this has any effect on exercise tolerance, or on how social factors affect exercise tolerance, could be examined in the future. Also, behavior was not included in this series of studies. It would be important to examine to what extent behavior might mediate the relationships between upstream factors and exercise tolerance. This is important since behavior is probably not the only pathway through which social factors affect health (Uchino, Cacioppo, & Kiecolt-Glaser, 1996).

Future research could also examine the results of Study 1 in light of the results of Study 2. That is, Study 2 examined social support influences on CVR. It would be important to see if the sociodemographic factors affecting social support, as seen in Study 1, have any effect on CVR. Gump, Matthews, and Räikkönen (1999) found that lower SES was associated with greater reactivity. Future research could examine if social networks or social support might mediate the relationship between SES and CVR. Further, CVR might explain the results of Fraser, Rodgers, Murray, et al. (2005). Thus, CVR to

anticipation of exercise might influence cardiovascular responses during an exercise test. Research examining cardiovascular responses to stressors has rarely examined how the cardiovascular system responds to a mental stressor *during* aerobic activity (for two notable exceptions see Rousselle, Blascovich, & Kelsey, 1995; Siconolfi, Garber, Baptist, Cooper, & Carleton, 1984). If mental stress responses during activity are at all additive, this response could have implications for the prognosis of heart patients (cf. Strike & Steptoe, 2003).

The results of these studies have identified potential key variables both upstream and downstream that might affect physiological pathways to health. Thus, the potential relevant social and psychological mediators observed in these studies could be examined further. In other words, it might be possible to examine the cascade of influence from macro, to mezzo, to micro and pathway factors. These studies found income and subjective social status as particularly important macro level factors affecting social networks in Study 1 and income influenced exercise tolerance in Fraser, Rodgers, Murray, et al. (2005). Social network size and diversity seemed to be important mezzo level factors influencing social support in Study 1, and a rudimentary network measure seemed to influence exercise tolerance in Fraser, Rodgers, Murray, et al. Social support at the micro level affected self-efficacy and stress in Fraser, Rodgers, Daub, et al. (2005) and Study 2. These 3 factors were related to CVR in Study 2. Thus, important variables from each level worth considering in future research include: income and subjective social status at the macro level, social network diversity and size at the mezzo level, social support at the micro level, and self-efficacy and stress at the pathways level. Both exercise tolerance and



CVR could be examined as dependent variables. These are not the only candidate variables to be examined, and surely others are relevant.

In terms of some of the variables at the pathways level, stress, anxiety, and self-efficacy appeared to be valuable as consequences (Fraser, Rodgers, Murray, et al., 2005) of social support and as potential mediators between social support and physical processes (Study 2). These processes among psychological and physiological pathways should be examined further in the context of social support and social networks. In other words, the study of social support influences on CVR could account for determinants of social support as well. Additionally, behavior is an obvious component included in the pathways component of the Berkman and Glass (2000) model that was not studied here. Clearly behavior is the presumed consequence of the model in Fraser, Rodgers, Daub, et al. (2005), and behavior might partially mediate the relationship between social factors and CVR. This is especially relevant to the context of cardiac rehabilitation where exercise is the main treatment. Blumenthal et al. (2005) recently found that stress management had positive effects on the health of heart patients in a comparison of stress management, usual care, or exercise only. Since exercise is probably not the only pathway to CR success, future research could try to examine to what extent behavior and social psychological processes influence CVR.

Stress and self-efficacy were important in these studies, but they certainly could be examined in different ways. In Study 2 there was no indication of how the context was perceived other than by the cardiovascular reactions of the patients. Stress referred to thought and feeling over the last month, and self-efficacy was for overcoming barriers to exercise. Anxiety in this study was with reference to the week before attending CR.

However, it might be useful to know if the situation was perceived as threatening or challenging or just how anxious the patients were at that moment. Presumably those who had larger changes in HR and BP were more threatened or anxious standing on the treadmill. Also, self-efficacy to overcome barriers to exercise might be too general. Perhaps the self-efficacy to perform the behavior (i.e., exercise) is more relevant, or maybe self-efficacy to cope with fear of exercising would be worth exploring.

Future research might benefit from investigating the measurement of social support in this study further with cardiac patients. The shortened version of the Interpersonal Support Evaluation List (Cohen & Hoberman, 1983) was used in Study 1 and Study 2 based on the promising results of Fraser, Rodgers, Murray, et al. (2005). However, across these studies the internal consistency reliabilities were never particularly high, often, depending on the sample, on the cusp of the minimally acceptable reliability of  $\alpha = .70$  suggested by Nunnally (1978). Given the low number of items per scale (i.e., 4) it is not surprising that the reliabilities were generally not very high. In fact the numbers seen here are probably admirable given the items per scale. Nevertheless, the reliabilities were low enough that bivariate relationships among variables were surely attenuated (Cohen, Cohen, West & Aiken, 2003). Future research should investigate whether adding an item to each subscale has a positive effect on these reliabilities in other samples.

This initial examination of the Berkman and Glass (2000) framework in the current series of studies provides support for the idea that upstream and downstream factors can have an effect on the psychological and physical processes thought to influence health in cardiac rehabilitation patients. Placing social support within this framework allowed for

the generation of questions to examine the determinants and consequences of social support in CR patients. Social support was found to have many determinants theoretically upstream of social support, as well as consequences downstream of support. The information gleaned here can be used to further explicate how social factors can affect health in other samples of heart patients, and perhaps in people more generally.

## References

- Berkman, L.F., & Glass, T. (2000). Social integration, social networks, social support, and health. In L.F.Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 137-173). New York: Oxford University Press.
- Blumenthal, J.A., Sherwood, A., Babyak, M.A., Watkins, L.L., Waugh, R., Georgiades, A. et al. (2005). Effects of exercise and stress management training on markers of cardiovascular risk in patients with ischemic heart disease. *JAMA*, *293*, 1626-1634.
- Cohen, J., Cohen, P., West, S.G., & Aiken, L.S. (2003). Applied multiple regression/correlation analysis for the behavioral sciences (3rd ed.). Mahwah, NJ: Erlbaum.
- Cohen, S., & Hoberman, H.M. (1983). Positive events and social supports as buffers of life change stress. *Journal of Applied Social Psychology*, *13*, 99-125.
- Fraser, S.N., Rodgers, W.M., Daub, B., & Black, B. (2005). *A test of a model of stress and multiple levels of social support on overcoming barriers to exercise in cardiac rehabilitation*. Manuscript in preparation.
- Fraser, S.N., Rodgers, W.M., Murray, T.C., & Daub, B. (2005). *The relationship between sociodemographic factors, social support and exercise tolerance in men attending cardiac rehabilitation*. Manuscript submitted for publication.
- Gump, B.B., Matthews, K.A., & Räikkönen, K. (1999). Modeling relationships among socioeconomic status, hostility, cardiovascular reactivity, and left ventricular mass in African American and white children. *Health Psychology*, *18*, 140-150.
- Nunnally, J. C. (1978). *Psychometric theory* (2nd ed.). New York: McGraw-Hill.

- Rousselle, J.G., Blascovich, J., & Kelsey, R.M. (1995). Cardiorespiratory response under combined psychological and exercise stress. *International Journal of Psychophysiology*, 20, 49-58.
- Siconolfi, S.F., Garber, C.E., Baptist, G.D., Cooper, F.S., & Carleton, R.A. (1984). Circulatory effects of mental stress during exercise in coronary artery disease patients. *Clinical Cardiology*, 7, 441-444.
- Strike, P.C., & Steptoe, A. (2003). Systematic review of mental stress-induced myocardial ischemia. *European Heart Journal*, 24, 690-703.
- Uchino, B.N., Cacioppo, J.T., & Kiecolt-Glaser, J.K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin*, 119, 488-531.

APPENDIX A: Fraser, Rodgers, Murray, and Daub (2005)

The Enduring Impact of Social Factors on Exercise Tolerance in Men Attending Cardiac  
Rehabilitation<sup>1</sup>

Shawn N. Fraser, Wendy M. Rodgers, Terra C. Murray

Faculty of Physical Education and Recreation, University of Alberta

Bill Daub

Northern Alberta Cardiac Rehabilitation Program, Glenrose Hospital

Correspondence: Shawn N Fraser, Faculty of Physical Education and Recreation,  
University of Alberta, E-401 Van Vliet Center, Edmonton, Alberta, Canada T6G 2H9.  
Tel: 1 780 492 7424. E-mail: [shawn.fraser@ualberta.ca](mailto:shawn.fraser@ualberta.ca)

## Abstract

Social support is related to rehabilitative success after a cardiac event, but it is not clear how. Further, the determinants of social support among heart patients is unclear. This explored the influence of sociodemographic factors and social support on a prognostic indicator among cardiac patients, exercise tolerance. We also examined how sociodemographic factors were related to social support as well as the role of social support as a potential mediator between sociodemographic factors and exercise tolerance. A sample of 254 men referred to cardiac rehabilitation (CR) completed surveys upon entry into CR, after 14 weeks, and after 9 months. Sociodemographic factors and social support reported upon entry into the CR program were related to initial and post CR exercise tolerance, after controlling for admitting diagnoses, comorbid factors, smoking, and perceived severity of illness. Overall, 28% of variance in exercise tolerance was explained at baseline, 19% at 14 weeks, and 20% at 9 months. Specifically, older individuals had poorer exercise tolerance, whereas those with more income had better exercise tolerance. Social support was positively related to exercise tolerance at all three times. Older men reported less social support than younger men, and those with more income reported more social support. However, social support did not mediate the relationship between sociodemographic factors and exercise tolerance. Results support the potential use of broad social factors in examining determinants of prognostic factors for heart patients.

Keywords: social support; cardiac rehabilitation; socioeconomic; exercise tolerance; sociodemographic; mediation



## The Enduring Impact of Social Factors on Exercise Tolerance in Men Attending Cardiac Rehabilitation

The presence of social support and its functions have been identified as important markers for future success of those with cardiovascular disease (Everson-Rose & Lewis, 2005). For example, Berkman et al. (1992) found patients hospitalized for an acute myocardial infarction were 2.9 times more likely to die within only 6 months if they reported a lack of emotional support. Furthermore, in general, individuals reporting more social support and more access to social resources do better in terms of multiple indicators of morbidity, such as fewer recurrent events and better psychological health, as well as lower mortality (Berkman et al.). The extant literature clearly implicates social support as an important factor influencing health and prognosis. What is not clear is how social support affects health, and what determines one's social support (Berkman et al.).

Berkman and Glass (2000) have recently outlined a "conceptual model of how social networks impact health" (p. 144), which provides a strategy for studying the influence of broader social factors on biological factors and health. Berkman and Glass's model suggests that individuals with better socioeconomic status (SES) are likely to have larger social networks, and the individuals comprising those networks are likely to have more resources. Thus, the person is likely to both perceive themselves as 'supported' as well as have access to actual support if they are of higher SES. This support, it is argued, makes them more likely to engage in positive health behaviors which makes them, in turn, more likely to have a more desirable health outcome. Thus, social support is thought to mediate the relationship between SES and more proximal behavioral (e.g., exercise) and physiological determinants (e.g., blood pressure) of health.

These relationships remain unclear, however, because the influence of SES on social support or on proximal indicators of health including specific health outcomes (see also Suls & Rothman, 2004) has seldom been examined. Berkman and Glass (2000) argue that the study of social structural factors (e.g., SES) “is almost completely absent in studies of social network influences on health” (p. 144) and little attention has been given to the determinants of social support (House, Umberson, & Landis, 1988). This is consistent with Suls and Rothman’s recent comment that there have been limited explorations of the relationship between biological and social factors (cf. Everson-Rose & Lewis, 2005).

Social support is thought to be influenced by the broader social structure and context in which it is provided (Berkman & Glass, 2000). Part of this broader structure may include the individual’s marital status, age, household income, and education. This combination of socioeconomic factors (e.g., income and education) and demographic factors (e.g., age and marital status) and are sometimes collectively referred to in the literature as sociodemographic factors (cf. Boardman, 2004; Leonard et al., 2005). These factors are thought to make up part of the broader social structure underpinning support. However, the various sociodemographic factors (e.g., income, education, age) may be differently related to support and health outcomes in different populations, such as patients attending cardiac rehabilitation. For example, income might be a critical factor in rehabilitation contexts where the patient pays for services. Age might be more important for rehabilitation of progressive conditions, regardless of other sociodemographic factors.

Finally, it is important to study the prospective influence of a variety of social factors (e.g., support and sociodemographic factors) because some may not be suitable for

intervention (e.g., age, marital status, income, education). Therefore a thorough understanding of their long-term effects on health outcomes, and potential mediators, is important. Since the goal of cardiac rehabilitation (CR) is to reduce future cardiac risk it is important to determine the relationship of variables potentially suitable for intervention with pre-existing, unchangeable factors that are also related to cardiac outcomes.

*The social and clinical context of study:*

Cardiac rehabilitation is primarily a behavioral therapy focused on increasing both exercise behavior and exercise tolerance of patients so that patients can resume normal activities (American College of Sports Medicine, 2000). Whereas it has been demonstrated that higher levels of social support are associated with better CR outcomes, it remains unclear whether or not social support has an independent effect on specific physiological health indicators expected from CR. One prognostic indicator thought to be particularly important in cardiac rehabilitation is exercise tolerance. Previous research has found exercise tolerance to be a key marker for future success of patients with heart disease (Ghayoumi et al., 2002). For example, in a sample of men referred to exercise tolerance testing Prakash et al. (2001) found exercise tolerance to be one of the most important predictors of future mortality along with having congestive heart failure, myocardial infarction history and age. Another study found that among both men with cardiovascular disease and healthy men, exercise tolerance was the strongest predictor of mortality after adjusting for age (Myers et al., 2002).

*Exercise tolerance* is an indication of the cardiovascular system's capability to respond to increasingly difficult physical challenges and is primarily used as a diagnostic test to determine a patient's capacity, or tolerance, for exercise (Williams, 2001). It also is

used in order to diagnose coronary artery disease (CAD) and also at the onset of CR to provide an appropriate exercise prescription for the subsequent rehabilitative period (Lear et al., 1999), and to guide the patient's future physical activity. Exercise tolerance is tested, typically, by having patients walk on a treadmill with regular increases in speed and grade (steepness) until such time as the attending physician or the patient him or herself deem that the test must be stopped (American College of Sports Medicine, 2000). The exercise tolerance test is symptom limited for cardiac patients. That is, the attending physician, exercise technician, or the patient determines the cessation of the exercise test. Some of the factors indicating test cessation include: hypertensive response, abnormally high heart rate, chest pain, fatigue, leg cramps, ischemic response (ST elevation equal to or greater than 1.0 mm), or patient does not want to continue (Gibbons et al., 2002). Several of these responses such as chest pain, ischemic response, and heart rate and blood pressure elevation have also been observed as responses to a mental stressor at rest (Sheps et al. 2002) and during exercise (Rousselle et al., 1995; Siconolfi et al., 1984). Thus, it seems reasonable to hypothesize that performance on the exercise tolerance test can be influenced by both physiological and psychosocial factors (e.g., Everson et al., 2001). Consequently exercise tolerance is an important biological/health outcome that might be directly influenced by social psychological factors.

At least one study has specifically examined the role of social psychological factors on exercise tolerance previously. Ruo et al. (2004) examined the unique effect of depression on exercise tolerance in patients with coronary artery disease. The authors found that depressive symptoms were a significant independent predictor of poor exercise tolerance after controlling for a variety of other factors such as smoking, BMI, diabetes,

age, income, beta-blocker use and statin use, as examples. Ruo et al. did not specifically examine how sociodemographic factors, income and education, independently contributed to exercise tolerance, rather these factors were controlled for. However, the results showed those with higher income and education were more likely to have a normal exercise capacity, suggesting an important avenue for further research.

Therefore the purpose of the current study was to explore the importance of two types of social factors (sociodemographic factors and social support) in determining exercise tolerance of cardiac rehabilitation patients across multiple time points. Specifically, we were interested in determining how sociodemographic factors and social support related to exercise tolerance before the rehabilitation period and 14 weeks and nine months subsequent to the rehabilitation period. Second, we were interested in examining how sociodemographic factors were related to social support. Third, we were interested in examining the mediating role of social support between sociodemographic factors and exercise tolerance also over time.

It was hypothesized that sociodemographic factors (age, education and income) (cf. Grzywacz & Marks, 2001; Sorensen et al., 2003), would be associated with exercise tolerance and that social support would be associated with exercise tolerance at all three times. Specifically, age was thought to be associated with worse exercise tolerance, whereas income, education and social support should be positively related to exercise tolerance. Second, it was hypothesized that sociodemographic factors would be related to higher general perceptions of social support. Specifically, it was thought that older individuals would have less support and those with more income and education would have more support. We also thought that marital status would be related to more social

support. Last, it was hypothesized that social support would mediate the relationship between sociodemographic factors and exercise tolerance.

## Method

### *Participants*

Archival data from 438 male (mean age = 60.0, SD = 10.4) cardiac rehabilitation patients who attended the Regional Cardiac Rehabilitation Program from Jan 1995 to May 1999 was collected. One hundred and sixty seven individuals did not provide information on income. This resulted in a final sample of 254 men. Of these men, 237, 235, and 241 men completed the initial exercise test, 14 week test and 9 month test, respectively. Unfortunately, in CR programs in North America women generally make up less than 20% of CR participants (Blackburn et al., 2000; King et al., 2001; Missik, 2001). Due to the relatively small sample of women in this CR program, sex comparisons could not be made. Thus, the decision was made to exclude women from the analysis. Further, previous research suggests that there may be important sex differences between sociodemographic factors, social support and health outcomes (e.g., House et al., 1988; House et al., 1982; Mishra et al., 2002; Singh-Manoux et al., 2003). Further men and women may display different physiological responses. For example, baroflex control differences between men and women (Christou et al., 2005) could impact on indicators of exercise test cessation. Demographic characteristics of the current sample are presented in Table 1.

### *Measures*

*Sociodemographic* factors included age, income and education. These data were drawn from a profile of the patients' medical history. Age was assessed as a continuous variable based upon age at the CR program orientation. Household income before taxes

was assessed by seven income categories (see Table 1), with 38% of participants choosing not to respond.<sup>1</sup> Education was categorized from 1 to 3, where 1 = *less than grade 9*, 2 = *completed high school*, and 3 = *some college/university*. Marital status was included since it was thought to influence perceptions of support and was categorized as 1 = *married or common-law*, and 0 = *divorced, widowed, or separated*. While there is currently considerable debate over the conceptualization and measurement of sociodemographic status (e.g., Bradley & Corwyn, 2002, Steptoe & Marmot, 2002), we felt that these indicators represent key aspects of sociodemographic influences in recent models of social effects on health (cf. Suls & Rothman, 2004; Grzywacz & Marks, 2001; Sorensen et al., 2003).

*Social support.* Social support was derived from a summary index of questions from the patients' medical charts. Participants were asked to "rate the degree of support you feel that you receive from the following." Participants then placed a check mark beside 7 different sources of support (religious beliefs, immediate family, extended family, friends, co-workers, boss, and organizations/clubs) ranging from 1 (*none*), 2 (*a little*), 3 (*a moderate amount*) and 4 (*a great deal*). Missing items were treated as not applicable indicating a lack of a particular source. Items were summed and scores ranged from 3 to 29. Since this archival data was drawn from a clinical setting, there is limited reliability and validity information for this scale. Nevertheless, the internal consistency for the scale was .78 (standardized Cronbach's [1951] alpha). Further, the items have some face validity appearing to capture one's overall perception of social support.

*Perceived severity of illness* was assessed with a single item asking “how severe do you think your heart condition is?” Participants’ response choices were 1 (*mild*), 2 (*moderate*), or 3 (*severe*).

*Exercise tolerance*, or capacity, was assessed following the Bruce protocol for all patients at baseline, 14 weeks and 9 months. The Bruce protocol involves a graded exercise treadmill test with 7 three minute stages, with increases in incline and speed with subsequent stages (Hill & Timmis, 2002). The test was conducted in the presence of a cardiology technologist and a cardiologist. Exercise was terminated if the cardiology technologist or the cardiologist detected symptoms suggesting the cessation of exercise or the patient was unable or unwilling to continue (see Gibbons et al., 2002). The indicator of exercise tolerance in this study was exercise duration (Gibbons et al.) which was recorded in seconds. A longer exercise time indicates higher exercise tolerance. Exercise tests were completed prior to beginning the CR program and 14 weeks and 9 months after the initial test.

### *Procedures*

Ethics approval was obtained to examine the medical charts of all patients who had completed CR and three exercise tolerance tests. The information obtained from charts included past history of disease and comorbidities, social and demographic information, as well as admitting diagnosis. The format of the CR program was as follows: participants initially attended an orientation session where they provided sociodemographic information, responses to the above surveys, and medical history. Within one week patients completed their first exercise tolerance test. Patients then completed an average of 6 weeks of exercise based cardiac rehabilitation. The overall length of rehabilitation,



frequency and duration of exercise prescribed varied based on the patient's heart condition. These factors were adjusted in the interest of the patient's progress as determined by the cardiologist, nurses, and exercise specialists at the rehabilitation program. At fourteen weeks and nine months after orientation participants completed follow-up exercise tolerance tests.

### *Analyses*

Correlations were first examined to see the overall relationships between sociodemographic factors, social support and exercise tolerance. Second, a formal test of the hypothesis that social support mediated the relationship between sociodemographic factors and exercise tolerance involved testing three regression analyses (Baron & Kenney, 1986; Holmbeck, 1997). These regression analyses tested: a) the impact of sociodemographic factors on social support, b) the impact of sociodemographic factors on exercise tolerance, and c) the impact of sociodemographic factors in addition to social support on exercise tolerance. If the impact of sociodemographic factors on exercise tolerance are reduced or eliminated when social support is in the equation, mediation is supported.

Finally, in interpreting the overall relationships between sociodemographic factors, social support and exercise tolerance, as seen in the final regression analysis, both beta and structure coefficients were consulted. This was done since beta coefficients can be influenced by even small relationships among predictor variables, whereas correlations and structure coefficients are not (Courville & Thompson, 2001). Thus, a predictor variable with a non-significant beta weight may still be contributing meaningfully to the regression equation (cf. Brotman et al., 2005).

## Results

The correlations among sociodemographic factors, social support and exercise tolerance are reported in Table 2. The correlations generally confirmed our first hypothesis that sociodemographic factors and social support would be related to exercise tolerance. Specifically, age was negatively related to exercise tolerance at all three times, whereas income and social support were positively related to exercise tolerance at all three times. Education was positively related to exercise tolerance only at nine months. Marital status was not related to social support or any other variables.

### *Adjusted regression analyses testing mediation*

Hierarchical regression analyses were used to test the hypothesis that sociodemographic factors and social support contribute to exercise tolerance and the secondary hypothesis that social support would mediate the relationship between sociodemographic factors and exercise tolerance (Baron & Kenney, 1986; Holmbeck, 1997) at each time point.

Three regression analyses were conducted to examine the mediational hypotheses as outlined in Holmbeck (1997) for each time. As shown in Tables 3-5, the covariates were included in the model prior to testing the mediation (Holmbeck) and included the admitting diagnoses and comorbidities reported in Table 1, and perceived severity of illness. Perceived severity of illness was included to reduce the possibility that the patient's exercise tolerance was influenced by the patient's thoughts about their physical condition. That is, since the test can be stopped by the patient if he feels unwell, perceived severity of illness is likely to influence the test length (i.e., exercise tolerance) even if the patient and physician do not agree about the severity. Thus, a patient's test time may be

reduced simply because he was afraid to continue with the test. Categorical variables were all dummy coded as 1 or 0 to indicate the presence or absence, respectively, of a factor as suggested by Cohen et al. (2003).

Results of the mediation are shown in Tables 3-5. The first regression analysis tested for the effects of the sociodemographic factors on social support, the first step in establishing mediation. Table 3 shows that the sociodemographic factors were related to social support, explaining 14% of the variance in social support. Specifically, age was related to less social support and income was related to more social support. Marital status and education had nonsignificant beta weights. Thus, the first condition for mediation was met showing age and income were related to social support.

The second regression analyses tested for the effects of the sociodemographic factors on exercise tolerance, the second step in establishing mediation. Table 4 shows that the sociodemographic factors were significantly related to exercise tolerance at all 3 times explaining 28, 19, and 20 percent of variance at baseline, fourteen weeks and nine months, respectively. At each time, the social demographic factors added unique variance above the control factors, as reported in Table 4. Age was negatively related to exercise tolerance and income was positively related to exercise tolerance at all 3 times. Education and marital status were nonsignificant at all three times. Thus, in terms of age and education, the second step of mediation was supported.

Finally, the third regression analyses tested for the effects of sociodemographic factors and social support on exercise tolerance at each time, the final step in establishing mediation. If social support mediated the relationship between sociodemographic factors and exercise tolerance as hypothesized, then the regression coefficients for age and

income should no longer be significant with the inclusion of social support in the model. Table 5 shows that the addition of social support did not impact on the relationship between sociodemographic factors and exercise tolerance at any of the three times. That is, after controlling for social support, the beta coefficients for the sociodemographic factors remained similar in magnitude as in Table 4 and social support did not have a significant beta weight. Thus, the last condition of mediation failed. Social support did not mediate the relationship between sociodemographic factors and exercise tolerance.

Last, in order to understand the overall interpretation of the impact of sociodemographic factors and social support on exercise tolerance, we looked at the structure coefficients. Results showed that social support was positively related to the regression equation predicting exercise tolerance at all three times ranging in magnitude from 0.38 to 0.53. These magnitudes are comparable to that of income, suggesting social support has a similar impact on exercise tolerance, but likely shares variability. Similarly, education seems to have made a contribution at nine months with a structure coefficient of 0.35.

### *Summary of results*

Sociodemographic factors and social support were prospectively related to initial exercise tolerance, exercise tolerance at fourteen weeks, and nine months. A test of mediation showed that sociodemographic factors were related to social support, but that social support did not mediate the relationship between sociodemographic factors and exercise tolerance. Age was consistently negatively related to exercise times, whereas income and social support were consistently positively related to exercise times. Overall, age and income appeared to have the greatest impact on exercise tolerance. Education had

a small impact on social support and nine month exercise tolerance. Marital status was not related to any other variable.

### Discussion

The first purpose of this study was to examine the influence of two types of social factors, sociodemographic factors (age, marital status, income and education) and social support on exercise tolerance (a proximal indicator of health) of CR patients across nine months. Second we wanted to examine the relationship between sociodemographic factors and social support. Last, we sought to examine the potential mediating role of social support between sociodemographic factors and exercise tolerance.

Sociodemographic status and social support were related to exercise tolerance according to the zero-order correlations. Specifically, age was negatively related to test times at baseline, fourteen weeks and nine months. Income was positively related to test times at all three time points. Marital status and education were not related to test times, except for education at nine-months. Overall, these results support our hypothesis that sociodemographic status and social support are related to a proximal prognostic indicator of cardiac health. Both social support and sociodemographic variables appeared to contribute to initial exercise tolerance, independent of admitting diagnosis, comorbid factors, smoking and perceived severity of illness. That is, the results here suggest that exercise tolerance was not *solely* influenced by one's current health or perception of their illness. These results are consistent with previous research (Ruo et al., 2004) showing social psychological factors are related to exercise tolerance.

In the current study we also examined the relationship between the sociodemographic variables and social support. Regression analysis showed that

sociodemographic factors were related to the perceptions of social support. Specifically, age and income contributed to the explanation of social support. Marital status was not related to social support. However, a large proportion of men were married (87.9%) and therefore a lack of variability might have masked any potential relationship between marital status and the other study variables. Age was negatively related to social support, whereas income was positively related to social support. An examination of the structure coefficients showed that age and income were the most important predictors of social support and that education ( $r_{sc} = .35$ ) contributed meaningfully to perceptions of support. Thus, younger people and those with more income reported receiving more support. These results confirm previous research suggesting that sociodemographic characteristics influence social support (Cattell, 2001) but extend previous findings to a cardiac rehabilitation context.

Social support did not mediate the relationship between sociodemographic factors and exercise tolerance. After controlling for the influence of social support on exercise tolerance, the sociodemographic factors remained significant predictors of exercise tolerance and social support was no longer a significant predictor. This finding is surprising given the amount of literature both supporting a relationship of social support to health indicators and the current socio-environmental models proposing the sequential influences of “upstream” social factors – like sociodemographics – on “downstream” factors – like social support – and subsequently on the proximal health indicators – like exercise tolerance in CR patients. As Berkman and Glass (2000) have pointed out, whereas strong connections between sociodemographics and social support, between sociodemographics and a proximal health indicator, and between social support and a

proximal health indicator have been robustly established in the literature, the relationship of all three variables together has seldom been addressed. The results of our study reproduce the expected relationships but do not support any mediating function of the downstream factor between the upstream factor and the proximal health indicator in CR patients.

There are a number of possible explanations for this unexpected finding, but all would require additional research to confirm. Limiting comment only to our data, first, it appears that the previously observed relationships between sociodemographics and a proximal health indicator and social support and a proximal health indicator are not independent. Second, it appears that sociodemographics predict social support. Third, the addition of social support to the prediction of the proximal health indicator does not weaken the influence of the sociodemographic factors, but the zero-order effect of social support is eliminated, suggesting that the influence of sociodemographic factors is so robust that it cannot be offset even slightly by the more malleable variable, social support. One rather drastic interpretation is that the influence of relatively stable, longstanding social factors can not be overcome through social support. This interpretation, however, has to be tempered in view of the limited operationalization of social support in this study. This measure of social support arguably captures general structural aspects (Cohen & Wills, 1985) such as support from one's network (i.e., friends, family, co-workers, etc.), rather than specific functional aspects of support (e.g., information, tangible support). Berkman and Glass (2000) implied that structural support is upstream of functional support and should therefore impact on functional support. The current results do suggest that the general support from one's social network is unlikely to be a useful predictor of a

specific health indicator taken by itself. However, the literature concerning social support has abundant definitions and measures of different aspects of support. Narrowing down the many possible influences of different aspects of support on specific health indicators is an important step forward in specifying potentially effective targets for intervention, and information that is clinically relevant. The current results do not support simple assessments of the extent of the social network as a useful prognostic indicator of exercise tolerance in CR patients.

These results seem to offer limited support for recent models of social influences on health (e.g., Berkman & Glass, 2000). They support the idea that broad sociodemographic factors and social support may influence a specific prognostic indicator of cardiac health independent of perceived illness severity and comorbid factors among CR patients. Specifically, it appears that income and age were important sociodemographic factors influencing and indicator of cardiac health over time in these men. As pointed out, the influence of these variables was not mediated by social support. This is not to say, however, that other conceptualizations of social support or other social-cognitive variables might not mediate the influence of age and income.

Income might also be associated with other lifestyle behaviors that are disease relevant. Previous research has clearly indicated that lower income individuals are less physically active (Taylor, Baranowski, & Young, 1998) and generally suffer from higher rates of chronic disease (Lantz, House, Lepkowski, Williams, Mero, & Chen, 1998). However, lower SES has been linked to worse health regardless of physical activity levels (Lantz et al.). In the present study, the advantage of a higher income appears to have been maintained despite the behavioral intervention (i.e., cardiac rehabilitation).



Sociodemographic factors might influence exercise tolerance by providing the CR patient with the necessary resources to maintain exercise during and after the CR program. Future research should examine specifically how income might help these patients.

An interesting finding was that education was correlated to exercise tolerance only at nine months. Further, education was not a significant predictor of exercise tolerance in the regression analyses. For this particular sample of men attending CR, income and age appear to play a more important role than educational attainment in determining exercise tolerance. This finding is somewhat surprising given that previous research generally demonstrates the importance of education in relationship to health in general (Ross & Wu, 1995). However, after a heart event, having higher levels of income may be more important than education. That is, after a heart event, a lack of income might be more salient than a lack of education, since many patients might need to miss work to recover and attend CR. The chronic stress of low income might express itself with a greater stress response during the exercise test (cf. Matthews et al., 2001). This needs to be tested in future research with heart patients.

A limitation of the current study concerns the generalizability of the results. First, only those individuals who completed all three exercise tolerance tests were included for analyses. It is unclear how these results might hold for individuals who did not complete the second and third exercise tests. Second, this was a sample of men only. It would be useful to examine how these results might differ between men and women in the future. Finally, many patients were reluctant to provide their income. Although, we found no difference among those who did and did not report incomes in terms of other study

variables, future research is needed to demonstrate whether or not these results generalize and can be replicated.

Older individuals entering CR with low incomes, in particular, are likely to be in poorer health as indicated by lower exercise tolerance, and the social support reported here is unlikely to offset this disadvantage, even over a period of structured, supervised rehabilitation. Future research would do well to focus on the type of support needed by these individuals, and not to assume that all interventions will be equally effective in a given rehabilitation context, independent of the demographic characteristics of the individual rehabilitation patients. The present results clearly indicate that there is potential for sociodemographic factors to directly influence prognostic health indicators, regardless of the support available. There is a need, therefore, to determine what types of social and social-cognitive factors can offset the influence of these pervasive and unavoidable “upstream” factors in terms of influencing health indicators, or to firmly establish that they can not be offset and to reconsider what might be successful rehabilitation outcomes accordingly.

## References

- American College Of Sports Medicine (2000). *ACSM's guidelines for exercise testing and prescription* (6th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- Baron, R.B., & Kenney, D.A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical consideration. *Journal of Personality and Social Psychology, 51*, 1173-1182.
- Berkman, L.F., & Glass, T. (2000). Social integration, social networks, social support, and health. In L.F.Berkman & I. Kawachi (Eds.), *Social Epidemiology* (pp. 137-173). New York: Oxford University Press.
- Berkman, L.F., Leo-Summers, L., & Horowitz, R.I. (1992). Emotional support and survival after myocardial infarction: A prospective, populations-based study of the elderly. *Annals of Internal Medicine, 117*, 1003-1009.
- Blackburn, G.G., Foody, J.M., Sprecher, D.L., Park, E., Apperson-Hanson, C., & Pashkow, F.J. (2000). Cardiac rehabilitation participation patterns in a large, tertiary care center: Evidence for selection bias. *Journal of Cardiopulmonary Rehabilitation, 20*, 189-195.
- Boardman, J.D. (2004). Stress and physical health: the role of neighborhoods as mediating and moderating mechanisms. *Social Science & Medicine, 58*, 2473-2483.
- Bradley, R.H., & Corwyn, R.F. (2002). Socioeconomic status and child development. *Annual Review of Psychology, 53*, 371-399.
- Brotman, D.J., Walker, E., Lauer, M.S., & O'Brien, R.G. (2005). In search of fewer independent risk factors. *Archives of Internal Medicine, 165*, 138-145.

- Cattell, V. (2001). Poor people, poor places, and poor health: The mediating role of social networks and social capital. *Social Science & Medicine*, *52*, 1501-1516.
- Christou, D.D., Jones, P.P., Jordan, J., Diedrich, A., Robertson, D., & Seals, D.R. (2005). Women have lower tonic autonomic support of arterial blood pressure and less effective baroflex buffering than men. *Circulation*, *111*, 494-498.
- Cohen, J., Cohen, P., West, S.G., & Aiken, L.S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah, NJ: Erlbaum.
- Cohen, S., & Wills, T.A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, *98*, 310-357.
- Courville, T., & Thompson, B. (2001). Use of structure coefficients in published multiple regression articles:  $\beta$  is not enough. *Educational and Psychological Measurement*, *61*, 229-248.
- Cronbach, L.J. (1951). Coefficient alpha and internal structure tests. *Psychometrika*, *16*, 297-334.
- Everson, S.A., Lynch, J.W., Kaplan, G.A., Lakka, T.A., Sivenius, J., & Salonen, J.T. (2001). Stress-induced blood pressure reactivity and incident stroke in middle-aged men. *Stroke*, *32*, 1263-1270.
- Everson-Rose, S.A., & Lewis, T.T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health*, *26*, 469-500.
- Ghayoumi, A., Raxwal, V., Cho, S., Myers, J., Chun, S., & Froelicher, V.F. (2002). Prognostic value of exercise tests in male veterans with chronic coronary artery disease. *Journal of Cardiopulmonary Rehabilitation*, *22*, 399-407.

- Gibbons, R.J., Balady, G.J., Bricker, J.T., Chaitman, B.R., Fletcher, G.F., Froelicher, V.F. et al. (2002). ACC/AHA 2002 guideline update for exercise testing: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). American College of Cardiology Web site. Available at [www.acc.org/clinical/guidelines/exercise/dirIndex.htm](http://www.acc.org/clinical/guidelines/exercise/dirIndex.htm)
- Grzywacz, J.G., & Marks, N.F. (2001). Social inequalities and exercise during adulthood: Toward an ecological perspective. *Journal of Health and Social Behavior, 42*, 202-220.
- Hill, J., & Timmis, A. (2002). ABC of clinical electrocardiology: Exercise tolerance testing. *British Medical Journal, 324*, 1084-1087.
- Holmbeck, G.N. (1997). Toward terminological, conceptual, and statistical clarity in the study of mediators and moderators: Examples from the child-clinical and pediatric psychology literatures. *Journal of Consulting and Clinical Psychology, 65*, 599-610.
- House, J.S., Robbins, C., & Metzner, H.L. (1982). The association of social relationships and activities with mortality. *American Journal of Epidemiology, 116*, 123-140.
- House, J.S., Umberson, D., & Landis, K. (1988) Structures and processes of social support. *Annual Review of Sociology, 14*, 293-318.
- King, K.M., Humen, D.P., Smith, H.L., Phan, C.L., & Teo, K.K. (2001). Predicting and explaining rehabilitation attendance. *Canadian Journal of Cardiology, 17*, 291-296.

- Lantz, P.M., House, J.S., Lepkowski, J.M., Williams, D.R., Mero, R.P., & Chen, J. (1998). Socioeconomic factors, health behaviors, and mortality: Results from a nationally representative prospective study of US adults. *JAMA*, *279*, 1703-1708.
- Lear, S.A., Brozic, A., Myers, J.N., & Ignaszewski, A. (1999). Exercise stress testing: An overview of current guidelines. *Sports Medicine*, *27*, 285-312.
- Leonard, H., Petterson, B., De Klerck, N., Zubrick, S.R., Glasson, E., Sanders, R. et al., (2005). Association of sociodemographic characteristics of children with intellectual disability in Western Australia. *Social Science & Medicine*, *60*, 1499-1513.
- Matthews, K.A., Gump, B.B., & Owens, J.F. (2001). Chronic stress influences cardiovascular and neuroendocrine responses during acute stress and recovery, especially in men. *Health Psychology*, *20*, 403-410.
- Mishra, G.D., Ball, K., Dobson, A.J., Byles, J.E., & Warner-Smith, P. (2002). Which aspects of socio-economic status are related to health in mid-ages and older women? *International Journal of Behavioral Medicine*, *9*, 263-285.
- Missik, E. (2001). Women and cardiac rehabilitation: accessibility issues and policy recommendations. *Rehabilitation Nursing*, *26*, 141-147.
- Myers, J., Prakash, M., Froelicher, V., Do, D., Partington, S., & Atwood, J.E. (2002). Exercise capacity and mortality among men referred for exercise testing. *New England Journal of Medicine*, *346*, 793-801.
- Prakash, M., Myers, J., Froelicher, V.F., Marcus, R., Do, D., Kalisetti, D., et al. (2001). Clinical and exercise test predictors of all-cause mortality: Results from >6,000 consecutive referred male patients. *Chest*, *120*, 1003-1013.

- Ross, C.E., & Wu, C-L. (1995). The Links between Education and Health. *American Sociological Review*, 60, 719–745.
- Rousselle, J.G., Blascovich, J., & Kelsey, R.M. (1995). Cardiorespiratory response under combined psychological and exercise stress. *International Journal of Psychophysiology*, 20, 49-58.
- Ruo, B., Rumsfeld, J.S., Pipkin, S., & Whooley, M.A. (2004). Relation between depressive symptoms and treadmill exercise capacity in the Heart and Soul study. *American Journal of Cardiology*, 94, 96-99.
- Sheps, D., McMahon, R.P., Becker, L., Carney, R.M., Freedland, K.E., Cohen, J.D. et al. (2002). Mental stress-induced ischemia and all-cause mortality in patients with coronary artery disease: Results from the psychophysiological investigations of myocardial ischemia study. *Circulation*, 105, 1780-1784.
- Siconolfi, S.F., Garber, C.E., Baptist, G.D., Cooper, F.S., & Carleton, R.A. (1984). Circulatory effects of mental stress during exercise in coronary artery disease patients. *Clinical Cardiology*, 7, 441-444.
- Singh-Manoux, A., Adler, N.E., & Marmot, M.G. (2003). Subjective social status: its determinants and its association with measures of ill-health in the Whitehall II study. *Social Science & Medicine*, 56, 1321-1333.
- Sorensen, G., Emmons, K., Hunt, M.K., Barbeau, E., Goldman, R., Peterson, K., et al. (2003). Model for incorporating social context in health behavior interventions: applications for cancer prevention for working-class multiethnic populations. *Preventive Medicine*, 37, 188-197.

- Suls, J., & Rothman, A. (2004). Evolution of the biopsychosocial model: Prospects and challenges for health psychology. *Health Psychology, 23*, 119-125.
- Steptoe, A., & Marmot, M. (2002). The role of psychobiological pathways in socioeconomic inequalities in cardiovascular disease risk. *European Heart Journal, 23*, 13-25.
- Taylor, W.C., Baranowski, T., & Young, D.R. (1998). Physical activity interventions in low-income, ethnic minority, and populations with disability. *American Journal of Preventive Medicine, 15*, 334-343.
- Williams, M.A. (2001). Exercise testing in cardiac rehabilitation: Exercise prescription and beyond. *Cardiology Clinics, 19*, 415-431.



## Endnotes

<sup>1</sup>A version of this chapter has been submitted for publication.

<sup>2</sup>A discriminate function analysis was conducted with all of the independent variables except income as well as all three exercise tolerance times to determine if there was a difference between those reporting and not reporting their income. Results showed no multivariate difference ( $p = .71$ ) between those reporting and not reporting their income. Univariate analyses also showed no differences on any of the variables.

Table A-1

*Socio-demographic and comorbidities of men*

Demographic variable	<i>n</i>	%
Age		
30-39	14	3.2
40-49	68	15.5
50-59	115	26.3
60-69	167	38.1
70-79	71	16.2
≥ 80	2	0.5
Missing	1	0.2
Education		
Less than HS	76	17.4
High-school	154	35.2
Some post secondary	204	46.6
Missing	4	0.9
Household Income		
1. <10,000	11	2.5
2. 10-19,999	31	7.1
3. 20-29,999	56	12.8
4. 30-39,999	38	8.7
5. 40-49,999	39	8.9
6. 50-59,999	22	5.0
7. >60,000	74	16.9
Missing	167	38.1
Married		
Yes	385	87.9
No	53	21.1
Smoker before event		
Yes	340	77.6
No	98	22.4
Admitting diagnosis to CR		
MI	276	63.0
Bypass surgery	143	32.6
Angioplasty	130	29.7
Angiogram	291	66.4
Co morbidities		
Arthritis/joint	128	29.2
Asthma/bronchitis	44	10.0
High Blood pressure	144	32.9
Diabetes	41	9.4
High Cholesterol	180	41.1
Thyroid	13	3.0
Gallbladder	36	8.2
Stomach	82	18.7

Note: Admitting diagnoses and comorbidities percentages are not cumulative.

Table A-2

*Correlations among study variables*

Variable	1.	2.	3.	4.	5.	6.	7.
1. Age	-						
2. Income	-.30**	-					
3. Married	.09	.12	-				
4. Education	-.15*	.28**	-.05	-			
5. Social support	-.27**	.36**	.11	.16*	-		
6. Exercise tolerance 1	-.42**	.25**	.07	.12	.22**	-	
7. Exercise tolerance 2	-.30**	.26**	.09	.11	.27**	.77**	-
8. Exercise tolerance 3	-.34**	.27**	.04	.18*	.22**	.70**	.84**

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

Table A-3

*Adjusted regression examining effect of sociodemographic factors on social support*

Variable	<i>R</i>	$R^2_{adj}$	$\beta$	$r_{sc}$
Age	.46	.14	-.22*	-.59
Income			.29*	.78
Married			.10	.24
Education			.03	.35

Regression equation significant  $p < .001$ . Independent variables add 14% of variance over control factors.

Note: Adjusted Model controlling for smoking status before event, perceived severity of illness, comorbidities (see Table 1) and admitting diagnosis (see Table 1).

\* $p < .005$

Table A-4

*Adjusted regression analyses examining effect of sociodemographic factors on exercise tolerance*

Variable	Exercise test times											
	Baseline				14 weeks				9 months			
	$R^a$	$R^2_{adj}$	$\beta$	$r_{sc}$	$R$	$R^2_{adj}$	$\beta$	$r_{sc}$	$R$	$R^2_{adj}$	$\beta$	$r_{sc}$
Age	.58	.28	-.33**	-.74	.51	.19	-.18**	-.59	.51	.20	-.23**	-.67
Income			.15*	.43			.24**	.51			.20*	.53
Married			.04	.17			.04	.18			.00	.08
Education			-.01	.21			-.01	.22			.05	.35

Note: Adjusted Model controlling for smoking status before event, perceived severity of illness; comorbidities (see Table 1) and admitting diagnosis (see Table 1). Independent variables add 9, 10, and 11 % of variance over control factors at baseline, 14 weeks and 9 months, respectively.

<sup>a</sup>All regression equations significant  $p < .001$ .  $r_{sc}$  = structure coefficient

\* $p < .05$  \*\* $p < .01$ .

Table A-5

*Adjusted regression analyses examining mediated effect of social support between sociodemographic factors and exercise tolerance*

Variable	Exercise test times											
	Baseline				14 weeks				9 months			
	$R^a$	$R^2_{adj}$	$\beta$	$r_{sc}$	$R$	$R^2_{adj}$	$\beta$	$r_{sc}$	$R$	$R^2_{adj}$	$\beta$	$r_{sc}$
Support	.58	.28	.05	.38	.51	.19	.10	.53	.52	.20	.04	.42
Age			-.32**	-.72			-.16**	-.59			-.22**	-.65
Income			.14*	.43			.21**	.51			.19*	.52
Married			.03	.17			.03	.18			.00	.08
Education			-.01	.21			-.01	.22			-.01	.35

Note: Adjusted Model controlling for smoking status before event, perceived severity of illness; comorbidities (see Table 1) and admitting diagnosis (see Table 1). Independent variables add 14, 11, and 12 % of variance over control factors at baseline, 14 weeks and 9 months, respectively.

<sup>a</sup>All regression equations significant  $p < .001$ .  $r_{sc}$  = structure coefficient

\* $p < .05$  \*\* $p < .01$ .

APPENDIX B: Fraser, Rodgers, Daub, and Black (2005)

A test of a model of stress and multiple levels of social support influences on efficacy for  
overcoming barriers to cardiac rehabilitation.

Shawn N. Fraser and Wendy M. Rodgers

Faculty of Physical Education & Recreation, University of Alberta

Bill Daub, & Bill Black

Northern Alberta Cardiac Rehabilitation Program, Glenrose Rehabilitation Hospital

Corresponding author: Shawn N. Fraser, Faculty of Physical Education and Recreation, E-401 Van Vliet Center, University of Alberta, Edmonton, Alberta, Canada, T6G 2H9. Tel: 1 (780) 492-7424. e-mail: [shawn.fraser@ualberta.ca](mailto:shawn.fraser@ualberta.ca)

Acknowledgements: This research was supported by a grant awarded to Dr. Rodgers from the Canadian Institutes of Health Research.

Shawn N. Fraser is supported by a Doctoral dissertation fellowship from the Social Sciences and Humanities Research Council of Canada.

Not yet submitted.



## A Test Of A Model Of Stress And Multiple Levels Of Social Support Influences On Efficacy For Overcoming Barriers To Cardiac Rehabilitation.

Cardiovascular diseases (CVD) continue to be the number one killers in North America and account for about 1/3 of all deaths globally (World Health Organization, 2002). For those suffering from CVDs, exercise based cardiac rehabilitation has been shown to increase survival and reduce recurrent events (i.e., heart attacks; Gibbons & Clark, 2001). The influence of exercise on prognosis is thought to act through several mechanisms (see for example Ades, 2001) including improved lipid profile, body composition, exercise tolerance, and glucose tolerance, for example. Exercise based rehabilitation is thought to confer psychological benefits as well, including stress reduction (e.g., Blumenthal et al., 1997) and increases in self-efficacy (Ewart, 1995). Thus, compliance with a physician's instruction to both attend cardiac rehabilitation and to exercise after rehabilitation is critical for achieving and maintaining these protective benefits.

Despite the known benefits of rehabilitation, adherence tends to be poor over time. Evidence over the last 20 years suggests that about 50% of patients will cease rehabilitative exercise within 6 months regardless of the severity of the disease (Moore, Dolansky, Ruland, Pashkow, & Blackburn, 2003; Oldridge & Stoenfalk, 1984). Thus, there remains a need to further understand the influences of exercise behavior among heart patients.

One factor that has been shown to associate with patient adherence in a variety of domains is social support (DiMatteo, 2004). Social support generally refers to the presence of social relationships as well as the resources derived from these relationships.

Generally speaking, social support has been identified as a factor related to the maintenance of exercise behavior. Social support has been related to both recreational (Duncan, Duncan, & McAuley, 1993) and prescribed exercise behavior (Fraser & Spink, 2002). Duncan et al. found that a lack of social support predicted dropout from an aerobic based exercise program among middle-aged adults. Rovniak, Anderson, Winett, and Stephens (2002) found that social support was positively related to exercise behavior in university students. Among elderly women, O'Brien Cousins (1995) found that perceived social support was positively related to exercise behavior. In cardiac rehabilitation, social support has also been identified as an important determinant of compliance with exercise (Dracup, 1994). Moore et al. (2003) found that social support was related to exercise frequency and persistence after CR.

The characteristics of the social support are hypothesized to influence its resource value. Two important characteristics are the structure and function of support. The amount of contact with friends and families is an indicator of structural social support (Cohen, 1994) whereas, resources, such as emotional support and tangible support are considered functional forms of support (Cohen). Thus, structure is the potential for resources to be available. That is, persons with more contact with others have greater potential for certain resources, such as emotional support to be available. The function describes the nature of the support (the resource) provided by a particular source.

In addition to the structural and functional aspects of social support different levels of support (Berkman & Glass, 2000) are thought to be important. Levels refer to the degree of specificity of the support. Two levels of social support thought to be important to exercise are general and exercise specific support. Litt et al. (2002) found that exercise

specific social support was more important to the prediction of exercise behavior than general forms of support. However, higher level models such as Berkman and Glass' seem to suggest that higher general perceptions of support should give rise to higher specific forms of support. That is, the more developed the support structure of an individual, the higher the probability that a given resource or function will be available. For example, O'Brien Cousins (1995) reported that a larger family size was associated with more support for exercise. Thus, a higher-level factor, such as general support, should influence a specific lower level factor such as support for exercise.

One question that remains is how social support influences behavior. Social support is unlikely to directly influence the health behaviors of individuals. It is much more likely that the influence of social support is mediated by a person-level social cognitive variable such as self-efficacy (cf. Dracup, 1994). Self-efficacy has been shown to be robustly related to exercise behavior (Bandura, 1997; McAuley, 1992; Rodgers & Sullivan, 2001; Rodgers et al., 2003, 2003).

Since self-efficacy is, theoretically, most proximal to behavior, it can be hypothesized that exercise specific support should influence exercise self-efficacy. Bandura (1997) points out that social support for exercise should influence one's regulatory mechanisms for maintaining exercise, such as one's confidence for overcoming barriers that may impede exercise goals. Patients in cardiac rehabilitation face unique barriers, such as chest pain, as well as common barriers, such as time (Blanchard et al., 2002). Moore et al. (2003) found that barriers to exercise were important predictors of total amount of exercise among women after cardiac rehabilitation. Specifically, women who reported fewer barriers to exercise engaged in more total physical activity following

CR. Further, those who reported more self-efficacy for exercise also reported fewer barriers to exercise.

Blanchard et al. (2002) found that efficacy for overcoming barriers to exercise in cardiac rehabilitation was predictive of adherence to a CR program. Further, Blanchard et al. demonstrated that efficacy for overcoming barriers to CR tended to increase after a CR program. Thus, it seems that efficacy to overcome barriers to exercise in CR is important for exercise maintenance since it influences one's ability to actually overcome those barriers. One important function of social support might be in its influence on exercise self-efficacy.

Another potentially important role of social support might be its influence on stress. It has been observed that social support seems to attenuate stress (Cohen, Kaplan, & Manuck, 1994), something thought to be important during the first month after a cardiac event (e.g., heart attack, bypass surgery). Immediately following an event, heart patients tend to experience high levels of distress and depression as well as fear of another event (Ewart, Stewart, Gillilan, & Kelemen, 1986; Jones & West, 1996). In addition to the protective effect of social support with respect to stress, Ewart (1995) has noted the role of mood in shaping the efficacy beliefs of cardiac patients. Essentially, Ewart suggests that negative affective experiences contribute to low self-efficacy for exercise. In other words, the stress experienced by heart patients is thought to negatively impact on their confidence to exercise and to overcome barriers that may impede their ability to exercise. Thus, social support may be an important contributor to maintenance of exercise behavior by attenuating the stress experienced by heart patients and consequently attenuating the negative effects on self-efficacy. It can be expected that people who report more general

social support should report more exercise specific support, lower stress and higher self-efficacy for overcoming exercise barriers. However, the kinds of social support giving rise to efficacy beliefs and that might attenuate perceptions of life stress that sustain exercise behavior needs to be further examined.

### *Purpose*

The purpose of the present study was to examine the relationships among different levels of social support, and stress and self-efficacy for overcoming barriers to attending an exercise based cardiac rehabilitation program. First, it was thought that higher perceived availability of general forms of support would be associated with higher perceptions of support for exercise from spouse, family, and friends as well as reduced perceptions of stress. Second, it was hypothesized that support specific to exercise would be the most influential determinant of self-efficacy. Third, it was hypothesized that higher levels of stress would negatively impact on self-efficacy for overcoming barriers to exercise. This model is represented in Figure 1.

### Method

#### *Participants*

Participants were patients recruited between September 2001 and October 2002 from a regional Cardiac Rehabilitation Program, an outpatient cardiac rehabilitation program. The sample included 459 men (mean age = 58.7, SD = 10.5) and 138 women (mean age = 60.3, SD = 11.8), a typical ratio of men to women in CR settings (Blackburn et al., 2000; Caulin-Glaser, et al., 2001; King, Humen, Smith, Phan, & Teo, 2001; King, Humen, & Teo, 1999; Missik, 2001). Table 1 presents demographic, co-morbidity and past history of disease information for this sample.

## *Measures*

*Interpersonal Support Evaluation List* (ISEL; Cohen & Hoberman, 1983). General social support was assessed with the 12-item version of the ISEL. The ISEL uses a 4 point Likert-type scale assessing perceived availability of three dimensions of support, appraisal (or emotional), belonging, and tangible support. Participants are asked to indicate the extent to which several items are true or false about themselves. Responses range from 1 (*definitely false*) to 4 (*definitely true*), where a high score indicates more available support for a particular dimension. Subscale scores were calculated from mean item responses. Cronbach's (1951) alphas for this data ranged from .65-.77, indicating marginally acceptable reliabilities.

*Perceived Stress Scale* (PSS4; Cohen, Kamarck, & Mermelstein, 1983). The PSS is a 4-item Likert-type scale assessing general levels of stress. Participants respond to questions about their thoughts and feelings over the last months. Examples include "In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?" and "In the last month, how often have you felt confident in your ability to handle your personal problem?" Responses range from 0 (*never*) to 4 (*very often*). Scale scores were computed as mean item responses. Internal consistency for this data was adequate (alpha = .77).

*Social Support for Exercise Survey* (SSES; Sallis et al., 1987). Participants completed an adapted version of the Sallis et al.'s SSES to assess sources of support for exercise. The SSES is a 12-item Likert-type scale assessing support for exercise from family and friends. The version of the SSES used here assessed spouse, family and friend/co-worker support at baseline. Participants were asked to report how often the

different groups of people (spouse, family, friends) have engaged in a particular action related to their exercise behavior. The scale ranged from 0 (*never*) to 9 (*very often*). Examples include “exercised with me” and “gave me encouragement to stick with my exercise program.” Subscale scores were obtained from mean item responses. Internal consistencies for the scale in this sample were adequate (Cronbach’s alpha’s ranged from .93 to .94).

*Barriers efficacy* (Blanchard et al., 2002). Barriers to exercise participation were assessed with a 10-point Likert type scale. Participants were provided a list of 10 obstacles that might interfere with their exercise and were asked to indicate how confident they are that they could exercise on a day when they experienced that obstacle. The list of barriers was developed by Blanchard et al. (2002) from patients drawn from a previous sample of the current CR program. Responses ranged from 1 (*not at all confident*) to 10 (*very confident*). Some of the obstacles are “bad weather”, “too much work”, and “minor health problems.” Internal consistencies for this data were adequate (Cronbach’s alpha = .92).

### *Procedures*

Once referred to CR, participants were mailed the questionnaire package along with other orientation materials typically sent to patients prior to attending an orientation session at the cardiac rehabilitation program. The questionnaire package included a consent form, cover letter and information letter inviting the participants to complete the survey package and explaining the purpose of the study. The information letter asked the participants to bring the completed survey to the orientation session. When participants came to the orientation session with surveys that were not completed or with further questions about the research, the questions were addressed and the participants were

encouraged to bring back the completed surveys the next time they came back to the CR program. This usually occurred within 1 week of orientation when the participants would come back for blood work or an exercise tolerance test and before beginning rehabilitative exercise.

### *Data analysis*

#### *Measurement model*

Before testing the model depicted in Figure 1, the full measurement model was tested as proposed by Thompson (2000) and McDonald and Ho (2002). The full measurement model involves a confirmatory factor analysis of the latent constructs with items. Subscale scores for spouse, family, and friends were used as the indicators of the latent variable support for exercise, since data was incomplete at the item level. The level 2 model was used for general social support since it was unclear how each particular domain of general social support would relate to the other variables in the structural analysis (cf. McAuley et al., 2003). In other words, the latent factors belonging, tangible and appraisal support were thought to represent the broader latent factor social support.

#### *Structural model*

Based on the test of the underlying measurement model, the structural model in Figure 1 was tested. In other words, directional arrows were added linking general social support to stress, exercise specific support, and barriers efficacy. Directional arrows pointed from stress and exercise specific support to barriers efficacy. Finally, an arrow pointed from exercise specific support to stress. After initial estimation the non-significant paths were removed and the model was re-estimated as recommended by Jöreskog (1993).

## Results



### *Demographic and descriptive*

Participants' demographic characteristics are reported in Table 1. Table 2 reports descriptive statistics and correlations among study variables. Subscale scores were computed as mean item responses for ease of interpretation. As seen in Table 1 most participants were married and a large proportion had some post-secondary education. A larger proportion of men were married and had some post-secondary education than women. In terms of admitting diagnoses, a heart attack (MI), and angioplasty were fairly common and a little over ¼ of patients had bypass surgery. Men in women did not differ in terms of admitting diagnoses. A large proportion of patients had hypercholesterolemia hypertension and arthritis/joint problems. Women had significantly more comorbidities than men with higher rates of arthritis/joint problems, asthma/bronchitis, thyroid problems, and gallbladder problems.

### *Measurement model*

As recommended by Thompson (2000), the measurement model was tested first. The model had a reasonable fit as indicated by a variety of global fit indices reported in Table 3. Since the measurement model had a reasonable fit it was prudent to proceed to the test of the structural model (Thompson, 2000).

### *Structural Equation Model*

A structural equation model was used to test the hypothesized relationships shown in Figure 1. Figure 2 shows the results of the SEM with standardized path coefficients. Since there were a number of missing values, estimates were computed using the Full information Maximum Likelihood Estimation procedure in AMOS 4 (Arbuckle, 1999).

Fit indices reported in Table 3 revealed an acceptable model fit. As expected these values did not differ much from the measurement model, since the correlations used in the measurement model are mathematically indistinguishable from the directional arrows in the structural model. However, Figure 2 shows that the paths from general support to efficacy and from exercise support to stress were non-significant. Thus these paths were removed and the model was re-estimated. As shown in Table 2 the fit indices remained very similar. Thus, the removal of the nonsignificant paths was supported as seen in the minimal changes in the fit statistics.

### Discussion

The purpose of this study was to test the hypothesized relationships between two levels of social support, stress and self-efficacy to overcome barriers to exercise in cardiac rehabilitation. Results from the structural equation model generally supported the hypothesized relationships. Specifically, it was found that higher general perceptions of social support were related to less stress and more exercise specific support. Second, more exercise specific support for exercise was associated with higher efficacy for overcoming exercise barriers, while stress was associated with less efficacy to overcome exercise barriers.

Thus, it appears that general perceptions of social support among those entering cardiac rehabilitation are associated with less stress and more exercise specific support. In turn, less stress and more exercise specific support results in confidence to overcome potential barriers to exercise in cardiac rehabilitation. These results are important in the context of past research showing the influence that barriers to exercise and the confidence to overcome those barriers can have on the exercise behavior of heart patients. These

results suggest that social support and stress can play a major role in shaping one's expectations of their success for overcoming barriers to activity.

#### *General and exercise specific social support*

These results offer support for the notion that different levels of social support might make important and different contributions to psychological precursors of health behaviors. In this case, general support perceptions were related to more specific support for exercise, which impacted on self-efficacy for overcoming barriers to exercise, supporting the first and second hypotheses.

#### *Social support and stress*

General perceptions of social support were negatively related to stress, as predicted. That is, people reporting more social support reported lower levels of stress. This is consistent with previous research suggesting that social support should act to attenuate the psychological impact of stressful experiences. These results support the third hypothesis.

#### *Social support and self-efficacy*

Results support previous research suggesting that social support influences self-efficacy (Bandura, 1997; Rovniak et al., 2002). However, these results extend previous research by showing that higher forms of social support influence lower levels of support. In this case, general perceptions of support were associated with exercise specific perceptions of support. Thus, while general social support did not directly impact on self-efficacy it appears to affect self-efficacy through the mediator exercise specific support.

An alternative explanation for this finding is that exercise specific support and barriers efficacy are naturally related perhaps through past exposure to exercise. Thus, it

may be that those who have exercised in the past naturally feel more confident that they can exercise in the future (Bandura, 1997) and perceive that their exercise has been supported in the past. However, this explanation does not account for the influence of general social support on exercise specific support. Nevertheless, future research should include past exercise as well as a prospective design to help eliminate this as a possible explanation for the current results.

#### *Stress and self-efficacy*

Finally, these results support previous theory suggesting that negative affective experiences can negatively impact on self-confidence (e.g., Ewart, 1995). In this case, experiences of stress were associated with less self-efficacy to overcome barriers to exercise. Future research could benefit from expanding this pathway further.

#### *Future directions*

While interesting these results need to be replicated in future research adding a temporal sequence between social support, stress and barriers efficacy. Additionally, there is a need for research examining the influence of social support, self-efficacy and stress on exercise behavior during CR and after CR. It may be that initial perceptions of stress and barriers efficacy change as a result of cardiac rehabilitation. Further, one's perceptions of support before starting the CR program may be different from the support actually obtained during and after CR. Further, it is conceivable that the source of this support may change. For example, during the CR program the patient may rely upon support from other patients and from the exercise leaders. After leaving CR the people providing support will likely change. It may be important to identify how these sources change and if the usefulness of this support changes.

Finally, it is presumed that social support and self-efficacy should lead to greater adherence to the prescribed exercise. Thus, future research needs to investigate the impact of these psychosocial variables on adherence to exercise during and after CR as well as the health impacts of this relationship.

## References

- Arbuckle, J.L. (1999). *AMOS: Version 4.0*. Chicago, IL: Small Waters Corporation.
- Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.
- Berkman, L.F., & Glass, T. (2000). Social integration, social networks, social support, and health. In L.F. Berkman & I. Kawachi (Eds.), *Social epidemiology* (pp. 137-173). New York: Oxford University Press.
- Blackburn, G.G., Foody, J.M., Sprecher, D.L., Park, E., Apperson-Hanson, C., & Pashkow, F.J. (2000). Cardiac rehabilitation participation patterns in a large, tertiary care center: Evidence for selection bias. *Journal of Cardiopulmonary Rehabilitation, 20*, 189-195.
- Blanchard, C.M., Courneya, K.S., Rodgers, W.M., Fraser, S.N., Murray, T.C., Daub, B., & Black, B. (2003). Is the Theory of Planned Behavior a Useful Framework For Understanding Exercise During Phase II Cardiac Rehabilitation? *Journal of Cardiopulmonary Rehabilitation, 23*, 107-116.
- Blanchard, C.M., Rodgers, W.M., Courneya, K.S., Daub, B., Knapik, G. (2002). Does barrier efficacy mediate the gender / exercise adherence relationship during phase II cardiac rehabilitation? *Rehabilitation Psychology, 47*, 106-120.
- Caulin-Glaser, T., Blum, M., Schmeizl, R., Progerson, H.G., Zaret, B., Mazure, C.M. (2001). Gender differences in referral to cardiac rehabilitation programs after revascularization. *Journal of Cardiopulmonary Rehabilitation, 21*, 24-30.
- Cohen, S., & Hoberman, H.M. (1983). Positive events and social supports as buffers of life change stress. *Journal of Applied Social Psychology, 13*, 99-125.

- Cohen, S., Kamarck, T., Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 385-396.
- Cohen, S., Kaplan, J.R., & Manuck, S.B. (1994). Social support and coronary heart disease: Underlying psychological and biological mechanisms. In S.A. Shumaker & S.M. Czajkowski (Eds.), *Social Support and Cardiovascular Disease* (pp. 195-221). New York: Plenum Press.
- Cronbach, L.J. (1951). Coefficient alpha and internal structure tests. *Psychometrika, 16*, 297-334.
- DiMatteo, M.R. (2004). Social support and patient adherence to medical treatment: A meta-analysis. *Health Psychology, 23*, 207-218.
- Dracup, K. (1994). Cardiac rehabilitation: The role of social support in recovery and compliance. In S.A. Shumaker & S.M. Czajkowski (Eds.), *Social support and cardiovascular Disease* (pp. 333-353). New York: Plenum Press.
- Ewart, C.K. (1995). Self-efficacy and recovery from heart attack: Implications for a social cognitive analysis of exercise and emotion. In J.E. Maddux (Ed.), *Self-efficacy, adaptation, and adjustment: Theory, research, and application* (pp. 203-226). New York: Plenum Press.
- Ewart, C.K., Stewart, K.J., Gillilan, R.E., & Keleman, M.H. (1986). Self-efficacy mediates strength gains during circuit weight training in men with coronary artery disease. *Medicine and Science in Sports and Exercise, 18*, 531-540.
- Gibbons, L.W., & Clark, S.M. (2001). Exercise in the reduction of cardiovascular events: Lessons from epidemiological trials. *Cardiology Clinics, 19*, 347-355.

- Jones, D.A., & West, R.R. (1996). Psychological rehabilitation after myocardial infarction: multicentre randomized controlled trial. *British Medical Journal*, *313*, 1517-1521.
- Jöreskog, K.G. (1993). Testing structural equation models. In K.A. Bollen & J.S. Long (Eds.), *Testing structural equation models* (pp. 294-316). Newbury Park, CA: Sage.
- King, K.M., Humen, D.P., Smith, H.L., Phan, C.L., & Teo, K.K. (2001). Predicting and explaining rehabilitation attendance. *Canadian Journal of Cardiology*, *17*, 291-296.
- King, K.M., Humen, D.P., & Teo, K.K. (1999). Cardiac rehabilitation: The forgotten intervention. *Canadian Journal of Cardiology*, *15*, 979-085.
- Litt, M.D., Kleppinger, A., & Judge, J. (2002). Initiation and maintenance of exercise behavior in older women: Predictors from the social learning model. *Journal of Behavioral Medicine*, *25*, 83-97.
- McAuley, E., Jerome, G.J., Marquez, D.X., Elavsky, S., & Blissmer, B. (2003). Exercise self-efficacy in older adults: Social, affective, and behavioral influences. *Annals of Behavioral Medicine*, *25*, 1-7.
- McDonald R.P., & Ho, M-H.R. (2002). Principles and practice in reporting structural equation analyses. *Psychological Methods*, *7*, 64-82.
- Missik, E. (2001). Women and cardiac rehabilitation: accessibility issues and policy recommendations. *Rehabilitation Nursing*, *26*, 141-147.



- Moore, S.M., Dolansky, M.A., Ruland, C.M., Pashkow, F.J., & Blackburn, G.G. (2003). Predictors of women's exercise maintenance after cardiac rehabilitation. *Journal of Cardiopulmonary Rehabilitation*, 23, 40-49.
- O'Brien Cousins, S. (1995). Social support for exercise among elderly women in Canada. *Health Promotion International*, 10, 273-282.
- Rovniak, L.S., Anderson, E.S., Winett, R.A., & Stephens, R.S. (2002). Social cognitive determinants of physical activity in young adults: A prospective structural equation analysis. *Annals of Behavioral Medicine*, 24, 149-156.
- Sallis, J.F., Grossman, R.M., Pinski, R.B., Patterson, T.L., & Nader, P.R. (1987). The development of scales to measure social support for diet and exercise. *Preventive Medicine*, 16, 825-836.
- Thompson, B. (2000). The ten commandments of structural equation modeling. In L.G. Grimm & P.R. Yarnold (Eds.), *Reading and understanding MORE multivariate statistics*. (pp. 261-283). Washington, DC: American Psychological Association.
- World Health Organization (2002). Integrative management of cardiovascular risk: report of a WHO meeting, Geneva, 9-12 July 2002. WHO.

## Figure Caption

*Figure B-1*

Hypothesized model relating stress and general and specific social support to efficacy.

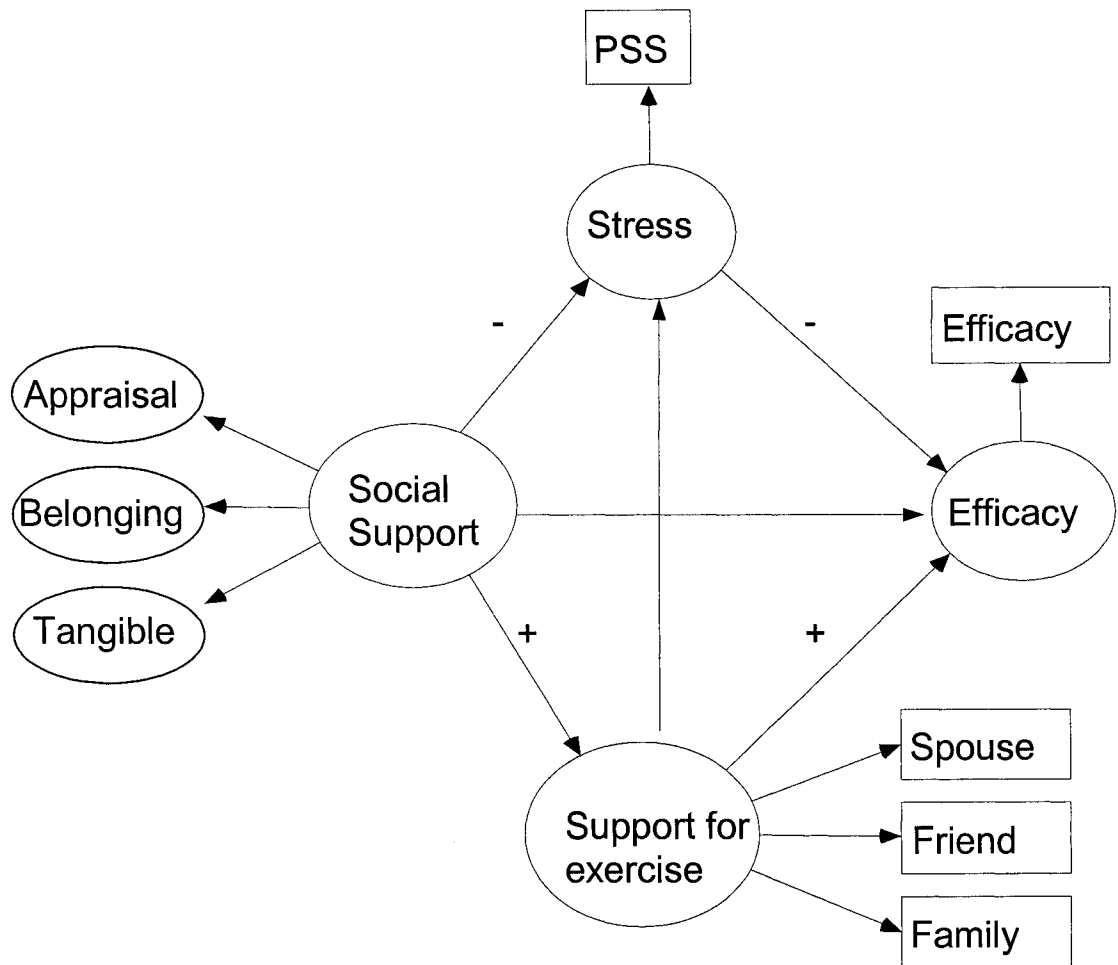


Table B-1

*Demographic characteristics of study sample*

Demographic variable	Sample			
	Women (n=138)		Men (n=459)	
	n	%	n	%
<b>Marital Status</b>				
Single	7	5.1%	32	7.0%
Married/Common law <sup>†</sup>	85	61.6%	369	80.4%
Divorced/separated**	22	15.9%	37	8.1%
Widowed <sup>†</sup>	23	16.7%	14	3.1%
Missing	2	1.4%	8	1.7%
<b>Education</b>				
Less than HS	18	13.0%	47	10.2%
High-school	53	38.4%	146	31.8%
Post-secondary*	60	43.5%	247	53.8%
Missing	7	5.1%	19	4.1%
<b>Admitting diagnoses</b>				
MI	73	52.9%	233	50.8%
Bypass surgery	35	25.4%	129	28.1%
Angioplasty	53	38.4%	203	44.2%
<b>Co morbidities</b>				
Arthritis/joint <sup>†</sup>	76	55.1%	162	35.3%
Asthma/bronchitis**	30	21.7%	48	10.5%
High Blood pressure	69	50.0%	235	51.2%
Diabetes	26	18.8%	96	20.9%
High Cholesterol	74	53.6%	273	59.5%
Thyroid <sup>†</sup>	23	16.7%	26	5.7%
Gallbladder <sup>†</sup>	27	19.6%	31	6.8%

Note: Percentages are not cumulative for admitting diagnosis or comorbidities.

\*p < .05. \*\*p < .01. <sup>†</sup>p < .001 between men and women by  $\chi^2$  difference test.

Table B-2

*Means, standard deviations, and correlations among study variables*

	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7	8
1. ISEL-Appraisal	3.22	0.72	(.77)							
2. ISEL-Belonging	3.21	0.66	.60	(.70)						
3. ISEL-Tangible	3.47	0.58	.58	.60	(.65)					
4. SSE-Spouse	4.98	2.35	.29	.31	.17	(.94)				
5. SSE-Family	4.09	2.38	.20	.22	.19	.61	(.94)			
6. SSE-Friends	3.61	2.32	.16	.18	.15	.48	.71	(.93)		
7. Stress	1.39	0.70	-.34	-.26	-.31	-.19	-.14	-.09	(.77)	
8. Efficacy	6.03	2.03	.18	.16	.21	.28	.22	.24	-.44	(.92)

Note: Standardized item alpha internal consistencies in parentheses. ISEL: Interpersonal Support Evaluation List; SSE: Social Support for Exercise. All correlations over .10 in magnitude are significant.

Table B-3

*Fit statistics for the structural equation models*

Model	$\chi^2 / df$	RMSEA	CFI	NFI	RFI	NNFI
Measurement model	3.861	.069	.974	.965	.958	.969
Structural model	3.861	.069	.974	.965	.958	.969
Final model	3.841	.069	.974	.965	.959	.969

*Note:* RMSEA = root mean square error of approximation; CFI = comparative fit index; NFI = normed fit index; RFI = relative fit index; NNFI = nonnormed fit index.

Figure Caption

*Figure B-2*

Model with standardized loadings.

