

FAMILY STRUCTURE, PSYCHOSOCIAL FACTORS, AND CARDIOVASCULAR
RISK FACTORS IN THE NHLBI CARDIA STUDY

by

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ABSTRACT

Title of Thesis: Family Structure, Psychosocial Factors, and Cardiovascular Risk Factors
in the NHLBI CARDIA study

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Introduction This study examined relationships among biological and psychological cardiovascular risk factors and marital and family status among young and middle-aged adults. It was hypothesized that risk factors would differ as a function of marital and parental status.

Methods 5112 (male = 2327, female = 2785) participants (mean age = 35 years at baseline) in the NHLBI CARDIA study were assessed for age, race, sex, smoking status, BMI, marital and parental status, BP, cholesterol, and depression at 3 time points over 15 years. Linear mixed models and hierarchical regressions were used in analyses.

Independent variables were marital status, parental status, and time since divorce.

Dependent variables were BP, cholesterol, and depression scores.

Results Married and cohabitating men had lower SBP and depression scores than other marital status categories. Married or cohabiting women had increased LDL, and lower HDL and SBP. Cohabiting after divorce but not after widowhood, and having children were related to decreased BP and depression for men, and increased DBP and

cholesterol for women. Few interactions between parental and marital status were found. Time since divorce was not related to cardiovascular risk factors for men or women.

Conclusions Family structure is related to heart health risk, however relationships vary by sex and risk factor. Consideration of family structure variables may help identify individuals at risk and suggest interventions for risk reduction.

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CHAPTER 1: Introduction

Coronary artery disease (CAD) is the leading cause of death in the United States (US) (1). Additionally, the health care costs associated with CAD and heart failure (HF) are high, estimated to be over \$177 billion in 2011 (1). Therefore, the study of the contributing factors involved in the development of CAD may ultimately lead to development of more effective and cost efficient treatments for patients. There are many behavioral risk factors for CAD including smoking, diet, and exercise (31).

In addition, psychosocial risk factors are involved in the development and progression of CAD (e.g. 12, 30, 31, 46). Psychosocial factors can include depression (30), stress (31), and other emotional factors and also social support and systems (12). One important support system is the structure of the family unit. The family unit may be one of the most direct long term sources of social support. Therefore the focus of the proposed study is to examine relationships among social support systems, specifically family structure and various risk factors for CAD.

CORONARY ARTERY DISEASE PATHOPHYSIOLOGY

CAD is a disease of the arteries that surround the heart. Commonly, CAD is caused by fatty deposits in the endothelium of arteries that collect and harden (31). These deposits can obstruct the artery such that blood flow is impaired and blood pressure and heart function are negatively impacted (31). In addition, the fatty deposits can rupture and tear or block the artery leading to stroke and heart failure. Heart failure is a symptomatic condition that occurs when the heart is no longer able to pump blood through the body (31). One contributing factor to the development of these fatty plaques is cholesterol, a

non-soluble lipid which exists in our blood. There are two types of cholesterol, low-density lipoprotein (LDP) that is considered unhealthy cholesterol and increases risk of developing heart problems (58) and high-density lipoprotein which actually protects against heart failure (49).

Hypertension (high blood pressure) is also a biological marker of disease and disease progression (e.g. 35). When blood pressure is consistently elevated, this is called hypertension (8). Blood pressure can be elevated due to increased blood flow from the heart, increased viscosity of blood or decreased diameter of blood vessels. Hypertension can be either primary or secondary, where primary hypertension has no known cause and is not due to a disease state and secondary hypertension is due to a disease state or medication (8). Secondary hypertension accounts for approximately 5-8% of hypertension cases (2). Many factors influence the development of hypertension, although it has no specific cause. Some of these factors include diet and exercise as well as psychosocial factors such as stress. Hypertension is associated with increased risk for heart failure and chronic heart failure (35). Hypertension can be used as a biological marker of increased risk for future heart events. Additionally, there are several behavioral risk factors associated with CAD.

BEHAVIORAL RISK FACTORS

Behavioral risk factors for the development and maintenance of CAD include dietary practices and the beliefs or attitudes associated with them, obesity, levels of physical activity, and tobacco use (31). Dietary practices including high sodium intake (2, 4, 8) and high trans fat diet (2) can increase the risk of hypertension, cardiovascular disease, and heart failure.

Behavioral risk factors for the development of CAD have been well studied. For example, Heo, Lennie, Moser, and Okoli, (26), examined dietary practices and perceived barriers to adherence to low sodium and low fat diets. Results indicated that while participants may have general knowledge that high fat, high sodium diets can have negative impacts on their health and HF symptoms, their knowledge of the specific impact of dietary practices was low. Additionally, social support provided in the form of knowledge and encouragement from not only medical professionals, but also significant others and children impacted these individual's adherence to heart healthy diets.

Along with diet, decreased physical activity and sedentary lifestyle is another risk factor for heart health and cardiovascular disease (2). For example, in one study (36), the fewer minutes spent in moderate to vigorous physical activity every day, the higher the risk reported for hypertension, body mass index (BMI), and obesity as well as cholesterol. Whether psychosocial factors such as the influence of family members or family structure were related to the inactivity observed in this study was not discussed.

Tobacco and alcohol consumption are both related to increased risk of heart health issues such as CAD (2, 28). They are directly associated with other risk factors such as obesity, inactivity, and lifestyle. Although tobacco is considered to have negative effects regardless of the level of smoking individual's engage in, alcohol has a more complicated relationship to heart health (28). With moderate alcohol consumption, that is, 2 or fewer drinks a day for men and one or fewer drinks a day for women may in fact be protective for heart health (28). However, excessive drinking (amounts over the above mentioned limits) act as risk factors for hypertension, CAD, arrhythmias, and HF (28). While there are multiple behavioral and biological risk factors and markers of disease,

psychological factors are also influential in the development and maintenance of heart diseases.

PSYCHOSOCIAL FACTORS IN THE DEVELOPMENT OF CAD AND CAD RISK FACTORS

In addition, a set of psychological factors are associated with many components of the CAD disease process (46). Specifically, factors such as stress, depression, and anxiety, as well as various facets of social support systems are considered to either increase or decrease the risk of developing CAD, as well as affect CAD mortality and morbidity. Social support systems are made up in part by family structure and include variables such as marital status, history of divorce, and caregiver status/having dependents, and are also related to increased risk and severity of cardiovascular health problems (21, 37, 43, 53). However, the interrelationships between family structure variables and heart health are complex and not well understood.

DEPRESSION

Depression is common in patients with CAD and is also predictive of an increased risk of mortality (45). According to the DSM 5, symptoms of depression include changes in sleep patterns (over or under sleeping), changes in appetite and loss of interest, or taking pleasure in normal activities. Often this may mean patients are not exercising or engaging in activities which they find enjoyable and reduce stress or increase positive affect. A flat, depressed or irritable mood is also a common symptom (3). These symptoms can result in changes to the physiology of an individual, ultimately effecting health.

Psychosocial factors such as depression and stress influence the body in several ways. That is, the factors are involved in sympathetic nervous system activation and the stress response. Activation and deregulation of these systems, common in many

depressed patients, sets into action responses, including the immune and inflammation cascades, which subsequently increase the risk for the development of atherosclerotic plaques and for plaque rupture (45). Along with stress, depression is also associated with increased social isolation, decreased health behaviors, and family structure transitions (change in family structure such as divorce, marriage, birth of a child) (13). Further investigation into the influence of psychosocial variables including stress and depression as risk factors for the development of CAD and heart failure in combination with family structure variables is warranted.

SOCIAL SUPPORT

Evidence indicates that increased social support, in general, may decrease cardiovascular risk factors (15). Lower levels of social support, in both structure (the size of the support system) and function (what the support system provides for the individual) (18) have been associated with increased mortality rates in coronary heart disease patients (6). Specifically, the stress-buffering model proposes that social support acts as a buffer to limit the ways in which stressful situations impact individuals (18). That is, having a social support network that provides an outlet to discuss potentially stressful situations, or comfort when stress levels rise may limit, actively reduce, or protect the individual from the stress associated with various situations. In contrast to the stress buffering hypothesis, the direct or main effects hypothesis states that social support is always beneficial to individuals regardless of stress levels (10). That is, the more social support an individual has, the better off he or she is both physically and emotionally.

Social support is directly associated with CAD prognosis and heart disease progression in patients with CAD (33). Therefore, it is of interest to further examine the

specific psychosocial and physiological pathways by which social support affects CAD. The focus of the present study is on family structure as one index of social support.

FAMILY PROCESS IN CAD

Previous research has addressed the role the family plays in providing social support and reducing social isolation for individuals with chronic diseases such as chronic heart failure syndromes (19). The specific aspects of the family structure as a social support network discussed here are marital status, history of divorce and number of dependents. Marital status is further divided into several categories including married, divorced, single, living in a marriage-like relationship and widowed.

MARITAL STATUS

Marital status is one important index of social support in patients with CAD and HF. While it is well established that marriage is related to improved health the mechanism by which the relationship works is not well understood. The presence of a spouse or significant other may serve as a buffer to individuals in terms of how stressful they perceives certain situations and therefore could reduce the risk for development of and hospitalizations from CAD and HF events. Brummett and colleagues (7) found socially isolated patients with CAD to have an elevated risk for mortality and be significantly more likely to be unmarried. Similarly, Friedmann and colleagues (20) found social isolation to be predictive of mortality in HF outpatients, but did not examine the influence of marital status on these outcomes. Single, widowed and divorced women were also found to have increased risk factors for CAD compared to their married counterparts (21, 56). On the other hand, in another study, unmarried status was related to

increased risk, but not reported as predictive, for all cause mortality, and cardiovascular mortality (43).

Marital status is indicative of risk for overall health later in life (16). Venters et al. (56) found differences between men and women in the risk factors associated with marital status. Specifically, they found for men cholesterol was higher than for women in their sample. In addition men and women differed in their behavioral risk factors and risk of stroke across marital status in a study conducted by Maselko, Bates, Avendano, and Glymour, (39). Although this study looked specifically at stroke incidence as the final outcome, the risk factors used included BMI, hypertension, smoking status, heart disease, and other chronic cardiovascular condition, making these findings relevant to the current study. Based on the prior literature, looking at heart health risk factors by sex may lead to a clearer picture of the influence of family structure on heart health.

Besides sex, participants' ethnicity may also affect the relationship between family structure, psychosocial variables and heart health. According to Koball, Moiduddin, Henderson, Goesling, and Besculides (29), marriage may be indicative of worse health for African American women than African American men or men and women of other ethnicities. That is, all persons who marry are likely to increase in weight, which is a risk factor for the development of hypertension, diabetes, CAD, and HF (29). However, African American women are more likely to become obese following marriage than white or Hispanic men and women or African American men (29). These women are therefore placed at a higher risk for developing heart health issues later in life.

The conflicting results of investigations involving the effects of marital status on health and CAD prognosis highlight the need for a further and more detailed examination

of the specific role of marital status. Marital status may act as a buffer by providing functional social support that protects individuals from participating in risk increasing behaviors (e.g. unhealthy diet and exercise) which increase risk for negative health outcomes. Sex and ethnic and racial differences also impact development of CAD (16, 29). In addition, the literature suggests that divorce has more negative consequences than being single or living alone.

DIVORCE STATUS

Divorce may be an important index of the degree of social support in patients with CAD. Compared to being married, being divorced also increases cardiovascular risk in women (43) and men (53). Divorce may be associated with particular psychosocial factors (e.g. hostility) making it particularly important in the investigation of factors linking stress and CAD outcomes (e.g. 52, 54). In addition to limiting social support, prior divorce may indicate personality factors, such as hostility, which are associated with increased social isolation and mortality (7).

TIME SINCE DIVORCE

In addition to relationships between divorce and psychosocial risk factors for CAD, time since divorce is also associated with CAD risk factors and outcomes (16). For women, although increased numbers of lifetime divorces are associated with decreased overall health, these effects appear to dissipate over time and when various risk factors and SES are controlled for (16). Interestingly, the effects of just one divorce on women's mortality risk appear to remain stable over time (16). In one study, for men, all associations between divorce and mortality risk were higher for the first four years after a divorce (16).

Associations between number of divorces and mortality disappeared after taking into account

other risk factors. These risk factors included health behaviors such as taking medications, exercising regularly, and health status. This suggests that examining more closely the influence of divorce on the development of CAD risk factors may be important.

Besides investigating history of divorce and overall health, previous authors have discussed divorce and remarriage or cohabitation effects on health (e.g., 17, 33, 40). Specifically, Mastakaasa's study explored divorce and remarriage or cohabitation resulting in evidence suggesting that individuals who divorce and subsequently remarry or cohabitate within a year have better overall well-being than those individuals who divorce and do not remarry or cohabitate within that time frame (40). This study investigated subjective well-being, however, but not physical health outcomes. In their research, Lee et. al. examined BMI, physical activity, and smoking behavior in association with marital status in women (32). These authors found that while divorce was associated with varying levels of health behaviors (e.g., BMI decreased but smoking behavior increased), remarriage was associated with increased health behaviors (32). Additionally, similar conflicting patterns have been found in studies involving health behaviors such as alcohol intake, vegetable consumption, physical activity, and patterns of divorce and remarriage in a sample of men (17). Overall, however, it appears that divorce may be associated with negative influences on health behavior while subsequent remarriage related to an increase in health behaviors. Further investigating the relationships among divorce and subsequent remarriage or cohabitation in heart health is warranted to clarify the role of these types of transitions to overall risk.

Additionally, the effects of time are not well understood in the negative influences of family structure transitions such as divorce or cohabitation without marriage. For example, it may be that there is an increasing protective effect of additional social support members (e.g.

spouse) and increasing negative effect of divorce over time, in which case it would be expected that the physical health of members of these two groups would continue to become more disparate over time. Another explanation would be that the health effects of these family structure variables are time limited in which case one would expect the differences between groups to remain stable or to decrease over time. Meadows, McLanahan, and Brooks-Gunn (42) examined the transitions in family structure of parents in the first through fifth years after their children's birth and found evidence for time limited influence of these types of variables in self-rated health of mothers and fathers (41). However, they used only self-rated general health and a limited time span not looking specifically at heart health. Focusing on long term effects of these variables on specific heart health variables which also develop slowly over decades is important. Given the unique psychosocial characteristics that may be associated with individuals reporting a history of divorce, separating this group from the general marital status of unmarried for further investigation is warranted.

DEPENDENTS

A much neglected aspect of family structure in the cardiovascular literature is the influence of children and parenting on cardiovascular health. The presence of children may serve as sources of stress for both mothers and fathers (44). Additionally, familial stressors originating from both spouses and children are associated with angina pectoris (37) and worsened CAD outcomes (57). Besides children being a source of stress, they require years of caregiving by parents/guardians. Although research on children, heart health risk factors, and CAD and HF may be somewhat minimal, caregiving effects on health are not. For instance, it has been demonstrated that caregiving is associated with higher levels of depression, anxiety, and stress (55). However, physiological outcomes

associated with the presence of children make the study of dependents and heart health risk factors complicated.

Physiological outcomes associated with having children have been examined in various studies. Holt-Lunstad, Birmingham, Howard, and Thoman (25) found that parenthood, in particular motherhood, was a protective factor for cardiovascular function (ambulatory blood pressure) in that parenthood was related to lower ambulatory blood pressure. Additionally, Hewitt, Baxter, and Western (24) found that for men, having preschool aged children negatively impacted health while having a preschool aged child had protective functions for women's health. These authors also report that women who worked less than full time and had children under aged 18 in the house reported less health concerns, then other women in the study. Additionally, Simons, Simons, Friedlander, and McCallum found that childless women had increased all cause mortality when compared to women with one, two, three, four, five or six or more children (51). While the stress associated with having children may be a long term stressor involved in CAD outcomes, there also seems to be some protective factors associated with having young children.

Given the contradictory findings associated with children's influence on parents health, it is important to continue to investigate and clarify these relationships. One possibility is to examine the intersection of parenting with marital status. Meadows, McLanahan, and Brooks-Gunn (42) found that mothers who are either stably cohabiting, stably single, begin a cohabiting or marital relationship or experience multiple family structure transitions within the first year after giving birth report lower overall health scores than do mothers who remain stably married during this time. Except for mothers

who stay cohabitating, begin cohabitating or marry after cohabitating with the child's father, these mothers also report more mental health issues during that time. Overall, stability verses family structure change following the birth of a child appear to result in better physical and mental health outcomes for mothers. How these relationships would play out in regard to heart health in the long term remains to be investigated. It also is unclear from this paper how these changes compare to women who do not have children experiencing similar family structure changes and how all of these relationships manifest in men/fathers, or various ethnic groups' heart health outcomes.

As with family structure variables involving significant others, those involving children may be buffering, have direct effects, and be time framed. In fact, it may be that the pros and cons listed above balance the impact of having children out in regards to cardiovascular health while the interaction of these influences with transitions in relationship status drive the health costs and benefits for parents. That is, they may either magnify or limit the influence of family structure on cardiovascular health. Further longitudinal investigations of the relationships among family structure variables, psychosocial factors and cardiovascular health outcomes are warranted.

RATIONALE FOR CURRENT STUDY AND CARDIA DATA SET

Several aspects of an individual's social network have been shown to influence overall health and heart in particular (6, 15, 18, 33). Specifically, family structure and transitions in that structure such as marriages, divorces, deaths and births have been shown to influence overall health and heart health outcomes (7, 16, 17, 20, 32, 37, 40, 42, 43, 53). However, there is a less literature as to the influences these family structure transitions have on heart health and risk factors for heart diseases. The influence of

family structure transitions over time has also rarely been studied in relation to heart health. Investigating these relationships is vital not only to establishing clearer descriptions of at risk groups, but also in informing interventions that include consideration of the individual risk factors associated with family structure. In order to study these variables, the CARDIA data set will be examined.

The CARDIA data set includes a sample of 5,112 participants who had data collected at each time point (1985, 1987, 1990, 1992, 1995, 2000, 2005, and 2010). The data includes survey questionnaires regarding psychosocial factors, demographic information, and physical functioning. The sample includes male and female subjects aged 18 – 30 years at the time of initial data collection. The sample was randomly selected from Birmingham AL; Minneapolis, MI; Chicago, IL; and Oakland, CA and balanced regarding sex, age, race, and education to The current study will utilize this data set to answer the following hypotheses.

AIMS AND HYPOTHESIS

Aim 1: Examine the relationships of marital status and sex to cardiovascular and psychosocial risk factors.

Hypothesis 1a: Widowed individuals will show greater risk than divorced individuals who in turn will show greater risk than single individuals who in turn will show higher risk than married and cohabiting individuals. Biological risk factors will include elevated blood pressure, higher LDL, and lower HDL, and psychosocial risk factors will include depression. These relationships will vary by sex.

Hypothesis 1b: Divorced individuals will show higher risk compared to individuals who are divorced and cohabiting. These relationships will vary by sex.

Hypothesis 1c: Individuals who are widowed or separated will show greater risk compared to those who are widowed or separated and cohabiting. These relationships will vary by sex.

Aim 2: Examine the relationships among time since divorce and cardiovascular and psychosocial risk factors.

Hypothesis 2: For those reporting divorce status, biological and psychosocial risk factors will initially increase and then decrease by years since divorce. These relationships will vary by sex.

Aim 3: Examine the relationships among marital status and children in the house on cardiovascular and psychosocial risk factors.

Hypothesis 3a: Having children will increase risk in all marital status categories compared to those who report having no children.

CHAPTER 2: Methods

All analyses in the present study utilized the CARDIA publicly accessible dataset. Methods of the CARDIA study have been published extensively (e.g., 23, 34, 47, CARDIA website: <http://www.cardia.dopm.uab.edu/>, 2013). Briefly, the CARDIA study was conducted in order to study cardiovascular risk factor progression in young adults. Following is a brief description of methods used in the CARDIA study relevant to the current study.

PARTICIPANT RECRUITMENT

Participants were recruited via telephone and in person requests from four locations around the United States; Birmingham AL; Minneapolis, MI; Chicago, IL; and Oakland, CA. Inclusion criteria were that the participants were aged 18-30 years at the time of recruitment, and had a permanent address in one of the above addresses. Exclusion criteria included having another household member already enrolled in the study, being blind, deaf, or pregnant at the time of first exam, able to complete all parts of the exam including the exercise tests and questionnaires. The sample selected was predominantly Caucasian and African American and balanced regarding sex, age, race, and education. During recruitment investigators explicitly excluded recruiting Hispanic individuals in order to allow for an all Caucasian and African American sample for comparisons between these two races (CARDIA website: <http://www.cardia.dopm.uab.edu/>; 2013). All participants provided written informed consent. The total initial sample consisted of 5,115 participants at baseline (1985); however 3 participants withdrew consent for use of their data leaving a total of 5,112 participants in the sample. Follow ups were conducted in 1987, 1990, 1992, 1995, 2000, 2005, and 2010. Given the

changes to protocol and exam components over the course of these follow ups, the current study will utilize data from the 1995, 2000, and 2005 follow ups for analyses. These three time points contain the most consistent variables for use in the analyses below. From this point on baseline shall refer to the 1995 follow up, follow up 1 to the 2000 follow up, and follow up 2 to the 2005 follow up. The sample size at each of the above follow ups were 3,950, 3,672, and 3547, respectively.

FOLLOW UP PROCEDURES

As part of each follow up period, participants came to the CARDIA center and underwent several tests and filled out several forms. Specifically, during the follow ups, participants blood pressure was taken, height and weight measurements taken, blood drawn for determining lipid levels and several demographic and psychosocial questionnaires completed. The height and weight measurements obtained during these follow ups were used to determine the participants' body mass index (BMI). Following are descriptions of the blood pressure, blood draw procedures as well as collection procedures for the demographic and psychosocial information obtained at each follow up.

BIOLOGICAL RISK FACTORS

Blood Pressure

Both diastolic and systolic blood pressure were measured in millimeters of mercury (mmHg) early at each follow up before participants had undergone testing, physical exam, or filled out questionnaires to reduce the risk of stress influencing the results. In addition, participants were asked to rest quietly for 5 minutes prior to their blood pressure being taken. Three measurements were obtained over the course of 10-15 minutes by a trained technician using a Hawksley random zero sphygmomanometer at

baseline and follow up 1, with the second and third measurements being averaged for use in analyses. At follow up 2 the blood pressure measurements were taken using the safer, OmRON HEM907XL with the same procedures and the second and third measurements being averaged for use in analyses.

Cholesterol

LDL and HDL cholesterol were examined at each of the three time points in the same manner. Individuals were asked not to eat or exercise vigorously in the 12 hours prior to their blood draw. Blood was drawn in a seated position, with the tourniquet never being used for more than 2 minutes at a time. Blood was drawn following blood pressure monitoring but before any physical or exercise exam. Following the blood draw, vials were labeled and placed in a freezer within 90 minutes. Lipid levels at milligrams per deciliter of blood (mg/dL) were analyzed at three sites, Northwest Lipid Laboratory, Linco Research Inc. and University of Minnesota and all blood draws and analyses were conducted by trained technicians.

PSYCHOSOCIAL AND DEMOGRAPHIC VARIABLES

Demographic variables including age, sex, household income, race, marital status and parental status were obtained using a demographic form designed for use in the CARDIA study. In addition, this form inquired about the participants' smoking status. This form was provided at each of the three time points used in the study. Marital status included married, divorced, single- never married, cohabitating and a combined term for widowed, separated or other. Baseline measurements included assessing the time since individuals reported being married, divorced, and cohabitating. Additionally, at baseline only, were participants who reported being divorced or widowed questioned as to

whether they were also cohabitating in a marriage like relationship. Parental status was determined by asking whether participants had any children or step children.

The CARDIA study assessed for several psychosocial variables including depression, job strain, social support and social network. Depression was assessed at all three time points using the Center for Epidemiological Studies Depression Scale (CES-D). This 20 item self report scale measures mood over the past week and results in an overall score ranging from 0 – 60. The CES-D has been shown to have high internal consistency ($\alpha \geq 0.85$), and concurrent validity against other validated scales of depression syndromes (48).

The Karasek Job Strain scale is a 15 item self report measure which asks about the demandingness of participants' work environment, the amount of control, flexibility the participants' experience in that environment and how they cope with the demands of the environment among other things. The scales provides overall scores for the psychological demandingness and control of the work environment both scores ranging from 12-48 and dichotomized into high and low categories for analyses in the current study. This scale was administered at baseline only. This measure has demonstrated strong predictive ability of cardiovascular symptoms and deaths (27).

Social support was measured at follow up 1 and 2 only using items taken from the MacArthur Network on Successful Midlife Development Survey of the Quality of Midlife in the US. The 8 variables used to measure social support were used to create 2 subscales. One measuring emotional social support from friends and family and one measuring demands/criticism from friends and family. Previous literature using these

scales from the CARDIA data set report alpha levels of 0.83 for the four items on the support scale and alpha levels of 0.73 on the demands/criticism scale (6).

Social Network was measured at follow up 1 and 2 using the Berkman Social Network Index. Specifically, the item assessing the number of friends and relatives participants had been in contact with over the past month was used to assess the size of the social network they were actively engaged with. This measure has been successfully used to measure social support, specifically in relation to mortality (5).

STATISTICAL ANALYSES

Statistical Assumptions

Variables were examined to determine if they met assumptions for performing proposed statistical analyses and basic descriptive statistics were obtained for sample characteristics. There was a slight skew in regards to several of the outcome variables including the physiologic and psychological variables. Specifically, these variables had a some positive outliers. For one variable, depression, scores were log transformed to account for this issue in analyses. While this was an appropriate solution for the CES-D scores, log transforming the physiologic variables led to an overcompensation of the variables. In addition, the physiologic variables do not lend themselves to multiplicative log transformations given the difficulty in establishing the meaning of a true zero point on this type of variable. For this reason, and because the large size of the sample used these variables were left without transformation for the analyses. A significant Leven's test among several of the marital status categories indicated violation of the assumptions of homoscedasticity. Again, transforming the data was considered however, doing so did not change the pattern of results and as explained above, overcompensated for the

difficulties in the sample. Additionally, given the large sample size, some variance is expected. Overall, the data presented some slight problems. However, after examining the concerns in the data set, the issues did not appear serious enough to warrant the use of non-parametric tests, which would have limited the sensitivity and generalizability of the results found; and the results are considered valid given the above considerations. All analyses controlled for age, race, household income, BMI, and smoking status.

Analysis Plan

To examine the relationship between marital status and risk factors, multilevel repeated measures models were used. Although the hypotheses do not make specific predictions about the impact of time on the variables in questions, a repeated measures model was warranted for several reasons. First, cardiovascular diseases are progressive and as such, the risk factors associated with the development of cardiovascular diseases also develop over time. It is important to be able to comment on any consistent pattern evidenced in the relationship of the variables examined over time, even though there is not a specific prediction regarding directionality or relationships. Additionally, prior literature regarding the patterns of the relationships among marital status categories and risk factors over time is lacking. Although, the prior literature does not lend itself to support a hypothesis in one direction or another regarding time, it is important to examine the patterns over time in this sample to assess for consistent or inconsistent patterns.

Throughout the analyses, significance levels for pairwise comparisons were adjusted using the Bonferroni correction to account for multiple comparisons. Where significant interaction terms were identified, follow-up ANOVAS were conducted to assess for the specific nature of these interactions. In addition, to assess for the

relationship of parental status on the above relationships, multilevel repeated measures models were again used with the addition of the parental status variables. Significance levels for pairwise comparisons were adjusted using the Bonferroni correction to account for multiple comparisons. To investigate significant 2 and 3 way interactions, follow-up ANOVAS were conducted.

The use of a multilevel model was warranted for several reasons. First, the overall sample size was 5115, however at each of the three time points used in the current analyses variations existed in the number of participants with data. Specifically, there were 3,950 participants at the baseline measurement, 3,672 at the first follow up measurement, and 3,547 at the second follow up. There were quite a few missing data points at each time point which would influence the ability of a repeated measures ANOVA to provide the most complete results, given that the participants with missing data would have been dropped from the analyses. The multilevel model accommodates for missing data by using predicted data points based off the data provided as a whole. The multilevel model is therefore a more accurate and sophisticated model for such analyses. In addition, unlike a repeated measures ANOVA, the multilevel model allows for variation in the independent variables (e.g., marital status category) over time. Specifically, because a new measurement for each independent and dependent variable is included in the model at each time point, the model takes into account changes in the independent variables over time. The multilevel model is an appropriate model for use with the hypotheses 1a and 3 in this project and presents several benefits above and beyond a repeated measures ANOVA.

To investigate the relationship among cohabitation and cardiovascular risk factors in participants reporting divorced or widowed status at baseline a series of one –way ANOVAS were conducted using baseline variables only. Lastly, to investigate the relationship between time since divorce and cardiovascular risk factors, regression analyses were conducted again using baseline variables only.

Given the variation among men and women of marital status and cardiovascular risk factors, for each outcome variable, men and women were compared using the mixed model developed for the present studies analysis and described below. These analyses were conducted first examining main effects for sex, and then including interactions between sex and time and sex and marital status. Several sex by time interactions and all main effects for sex were significant for each outcome variable. These analyses revealed that in this sample, as well as in the literature, there are differences between men and women’s patterns of risk. The analyses used in this project included several 2-way as well as 3-way interactions. Including sex as an independent variable in the model testing the hypothesis was not done due to the likelihood of difficulty interpreted results. Therefore, all analyses were conducted separately for men and women.

Exploratory analyses were conducted to determine if several psychosocial variables, job strain, social support, and social network might relate to or explain some of the above relationships considered. First a series of one-way ANOVAS was conducted to determine the relationships among these psychosocial variables and the marital status variables investigated at all time points available. If available at all three time points, these variables were entered into the multilevel models as covariates and re run to assess for changes in the pattern of results for marital status and cardiovascular risk factors.

Power Analysis

Calculations of power and sample size for multilevel models are rarely straightforward (50). One approach is to conduct thousands of simulations using constructed data sets that contain similar elements to the data set being used in the study analyses (50). For straightforward, repeated measures multilevel designs, a simpler alternative is to estimate sample size based on the sample size needed for a repeated measures ANOVA design. Multivariate tests in repeated measures ANOVA are equivalent to tests in a multilevel model for repeated measures when complete data are available for each subject and when the within-subject covariance is modeled using an unstructured covariance matrix. Some participants in this study have missing data for one or more time points; however, as long as the number of complete observations in the data set exceeds the required sample size for a repeated measures ANOVA, the study should have sufficient power. The observed power of the study will be even greater after the addition of participants with one or more missing values. In order to calculate the power needed for the current study G*power 3 was used. Assuming an effect size $f^2 = 0.25$, with an alpha of 0.05, a target power of 0.85, 10 groups, and 3 measurements, the estimated sample size needed for a repeated measures ANOVA was 200. Given that the total sample used in the current study well exceeds this number, and even the number of complete cases exceeds 200 at a total of 2,972, the study is adequately powered.

CHAPTER 3: Results

SAMPLE CHARACTERISTICS

The sample used in analyses consisted of 5,112 participants, 2,327 of which were male (45.5%) and 2,785 of which were female (54.5%). Demographics are presented separately for males and females because all analyses were conducted separately for males and females. The average age of participants in this sample at baseline was 35 years for both men and women. Of the men in this sample, 1,157 (49.7%) were African-American and 1,170 (50.3%) were Caucasian. Of the females, 1,480 (53.1%) were African –American and 1,305(46.9%) were Caucasian. For both men and women, the majority of individuals were married at all three time points. However, for women this number increased over time while for men, this number increased from baseline to follow up 1 then decreased again to follow up 3. For women, BMI increased at every time point whereas for men BMI increased from baseline to time one and then decreased again to time 2. For additional details on the sample demographics see tables 1 and 2.

Table 1. Demographics for males

Demographic variables for males in sample						
Variable	Level					
Smoking	Non smoker		<i>Ex-smoker</i>		Smoker	
Baseline	1017(43.7%)		237(10.2%)		495(21.3%)	
Follow up 1	980(42.1%)		247(10.6%)		388(16.7%)	
Follow up 2	936(40.2%)		259(11.1%)		323(13.9%)	
Marital Status	Married	Divorced	Never Married	MLR	Widowed/Other	
Baseline	869(37.3%)	171(7.3%)	497(21.4%)	133(5.7%)	77(3.3%)	
Follow up 1	916(39.4%)	161(6.9%)	371(15.9%)	105(4.5%)	62(2.7%)	
Follow up 2	899(38.6%)	171(7.3%)	267(11.5%)	116(5%)	76(3.3%)	
Parental Status	Yes parent		No not parent			
Baseline	1047(45%)		702(30.2%)			
Follow up 1	1117(48%)		499(21.4%)			
Follow up 2	1122(48.2%)		405(17.4%)			
Income	<5k-15.9k	16k-24.9k	25k-34.9k	35k-49.9k	50k-74.9k	75k+
Baseline	247(10.6%)	190(8.2%)	273(11.7%)	327(14.1%)	350(15%)	347(14.9%)
Follow up 1	130(5.6%)	86(3.7%)	152(6.5%)	240(10.3%)	329(14.1%)	660(28.4%
Follow up 2	132(5.7%)	63(2.7%)	91(3.9%)	171(7.3%)	301(12.9%)	756(32.5%)
Body Mass Index	Min	<i>Max</i>	Mean(SD)		<i>N</i>	
Baseline	19.27	43.13	27.08(4.74)		1738	
Follow up 1	20.13	43.71	28.19(4.97)		1615	
Follow up 2	19.84	44.86	28.83(5.2)		1528	

Table 2. Demographics for females

Demographic variables for females in sample						
Variable	Level					
Smoking	Non smoker		<i>Ex-smoker</i>		Smoker	
Baseline	1257(45.1)		411(14.8%)		509(18.3%)	
Follow up 1	1212(43.5%)		417(15%)		419(15%)	
Follow up 2	1214(43.6%)		422(15.2%)		360(12.9%)	
Marital Status	Married	Divorced	Never Married	MLR	Widowed/Other	
Baseline	1037(37.2%)	316(11.3)	510(18.3%)	169(6.1%)	139(5%)	
Follow up 1	1041(37.4%)	312(11.2)	427(15.3%)	143(5.1%)	121(4.3%)	
Follow up 2	1054(37.8%)	327(11.7)	359(12.9%)	142(5.1%)	123(4.4%)	
Parental Status	Yes parent		No not parent			
Baseline	1461(52.5)		711(25.5)			
Follow up 1	1524(54.7)		520(18.7)			
Follow up 2	1555(55.8)		448(16.1)			
Income	<5k-15.9k	16k-24.9k	25k-34.9k	35k-49.9k	50k-74.9k	75k+
Baseline	405(14.5%)	219(7.9%)	334(12%)	412(14.8%)	429(15.4%)	365(13.1%)
Follow up 1	212(7.6%)	154(5.5%)	190(6.8%)	331(11.9%)	465(16.7%)	672(24.1%)
Follow up 2	226(8.1%)	125(4.5%)	153(5.5%)	269(9.7%)	369(13.2%)	836(30%)
Body Mass Index	Min	Max	Mean(SD)	N		
Baseline	18	52.37	27.76(7.24)	2145		
Follow up 1	18.65	51.76	29.08(7.47)	2012		
Follow up 2	18.79	51.70	29.69(7.37)	2000		

AIM 1

Hypothesis 1a

To investigate Hypothesis 1a, that widowed individuals will show greater risk than divorced individuals who in turn will show greater risk than single individuals, who in turn will show higher risk than married and cohabiting individuals, a multilevel repeated measures model was used. This model was run separately for males and females to investigate if male and females would demonstrate differing relationships among marital status and risk factors. As described earlier, significant interactions were further investigated using ANCOVAs. All analyses controlled for race, age, BMI, household income, and smoking status.

Blood Pressure

Results of the model indicated that for males, there was a significant main effect of time $F(2, 1931.876) = 53.768, p < .001$ and a significant main effect of marital status $F(4, 3954.364) = 2.431, p < .05$. In addition, there was a significant time by marital status interaction on systolic blood pressure (SBP) $F(8, 2032.444) = 2.189, p < .05$. This interaction was further examined using one way ANCOVAs at each of the three time points to assess where the marital status variables varied and how. Results indicate that at baseline, married men ($M = 112.896, SE = .445$) had lower SBP than never married ($114.792, SE = .544$) and divorced men ($M = 115.325, SE = .853$). Men in a marriage-like relationship ($M = 114.236, SE = .963$) and who were widowed ($M = 113.254, SE = 1.247$) did not differ significantly from any other marital status at baseline. At follow up 1, married men ($M = 115.725, SE = .663$) had lower SBP than divorced ($M = 118.606, SE = 1.164$) or widowed men ($M = 119.704, SE = 1.881$) while never married men ($M =$

115.299, SE = .779) had lower SBP than divorced and widowed men (see figure 1). Men in a marriage-like relationship (M = 117.651, SE = 1.434) did not differ from any other marital status at follow up 1. Finally, at follow up 2, there was no significant difference between any of the marital status' for men in this sample. In addition, in terms of the covariates, there was a significant main effect of race $F(1, 1846.160) = 56.088, p < .001$ with African American males reporting higher SBP (M = 119.054, SE = .427) compared to Caucasian males (M = 115.213, SE = .433).

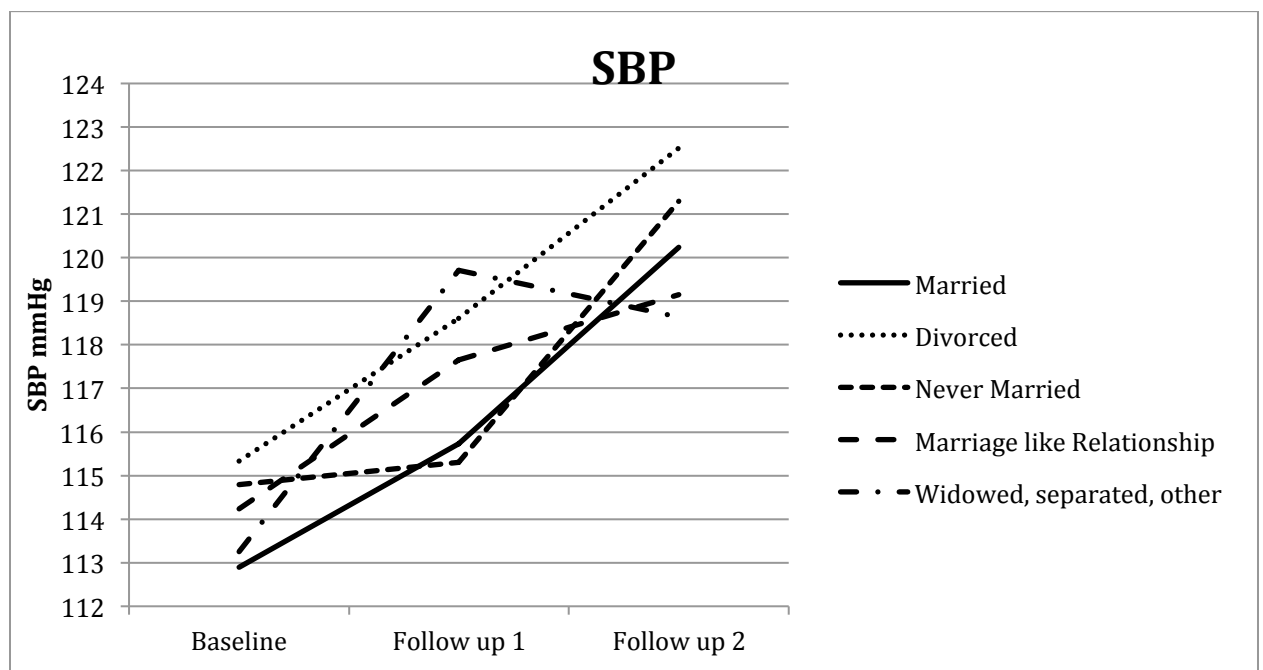


Figure 1. Time by Marital status interaction for males and SBP.

For women on the other hand, there was no time by marital status interaction but instead significant main effects of both time $F(2, 2194.945) = 103.937, p < .01$ and marital status $F(4, 4942.174) = 3.325, p < .05$. Pairwise comparisons indicate that each time point was significantly different from each other time point for females SBP. Specifically, baseline SBP was lower (M = 107.968, SE = .331) than follow up 1 SBP (M = 112.201, SE = .401) and baseline and follow up 1 SBP were both lower than follow up

2 SBP ($M = 114.252$, $SE = .460$) (see figure 2). For female's for marital status pairwise comparisons indicate that married females had lower SBP ($M = 110.480$, $SE = .379$) than widowed females ($M = 112.769$, $SE = .687$) (see figure 3). Women who reported being divorced ($M = 111.174$, $SE = .495$), never married ($M = 110.888$, $SE = .498$) or in a marriage-like relationship ($M = 112.057$, $SE = .637$) did not differ significantly from any other marital status in their SBP levels. In addition, in terms of the covariates, there was a significant main effect of race $F(1, 2324.575) = 142.207$, $p < .001$ with African American females reporting higher SBP ($M = 114.603$, $SE = .395$) compared to Caucasian females ($M = 108.344$, $SE = .416$).

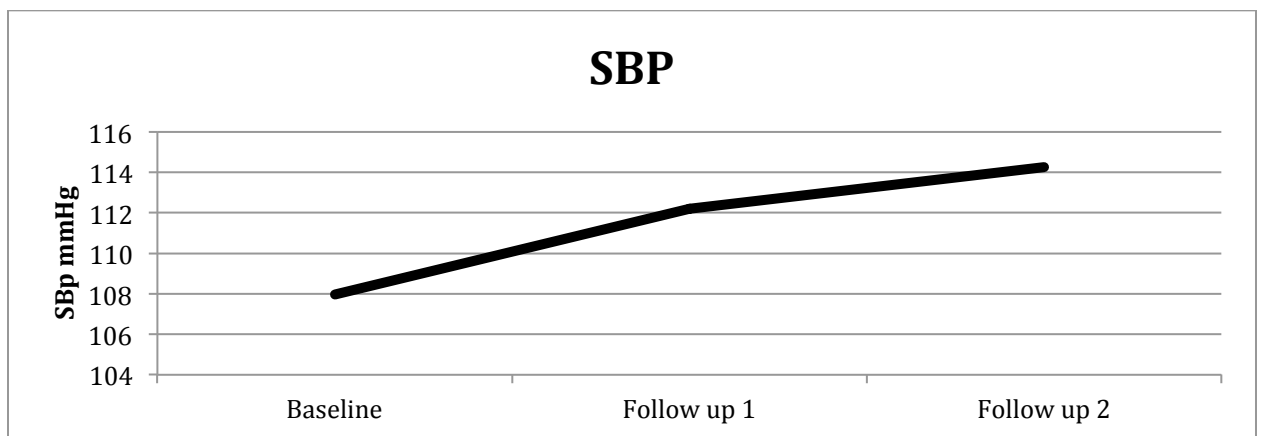


Figure 2. Main effect of Time on SBP in females

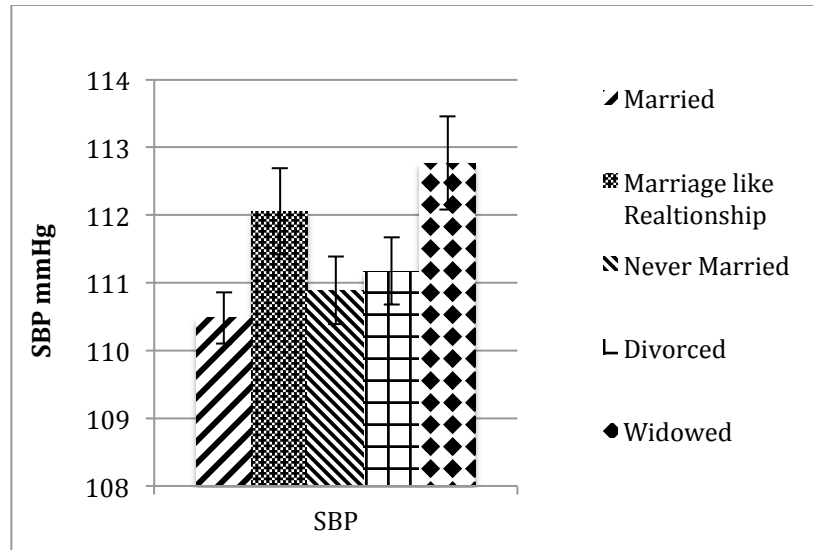


Figure 3. Main effect of Marital Status on SBP in females

For diastolic blood pressure (DBP), there was no interaction between time and marital status or main effect of marital status for males. However there was a main effect of time $F(2, 1970.853) = 26.895, p < .01$ indicating that, baseline DBP ($M = 75.564, SE = .316$) was lower than follow up 2 ($M = 77.273, SE = .415$) and higher than follow up 3 ($M = 73.943, SE = .388$) DBP while follow up 2 was higher than both baseline and follow up 3 (see figure 4). In addition, in terms of the covariates, there was a significant main effect of race $F(1, 1805.804) = 46.053, p < .001$ with African American males reporting higher DBP ($M = 77.026, SE = .348$) compared to Caucasian males ($M = 74.161, SE = .353$). Results indicated a dissimilar pattern of DBP for women. While there was no interaction between time and marital status or main effect of marital status there was a significant main effect of time $F(2, 2287.233) = 11.550, p < .01$. However, pairwise comparisons indicated that DBP at baseline ($M = 71.077, SE = .266$) was lower than both follow up 2 ($M = 72.646, SE = .323$) and follow up 3 ($M = 72.094, SE = .336$) while follow up 2 and 3 did not differ significantly (see figure 5). In addition, in terms of

the covariates, there was a significant main effect of race $F(1, 2320.234) = 135.070, p < .001$ with African American females reporting higher DBP ($M = 74.225, SE = .297$) compared to Caucasian females ($M = 69.607, SE = .341$).

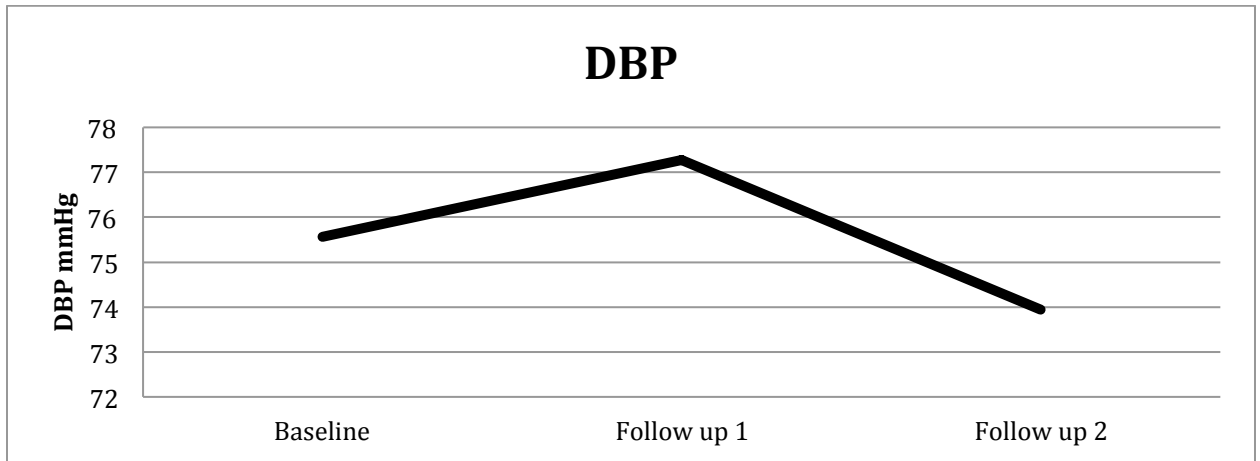


Figure 4. Main effect of Time for DBP in males

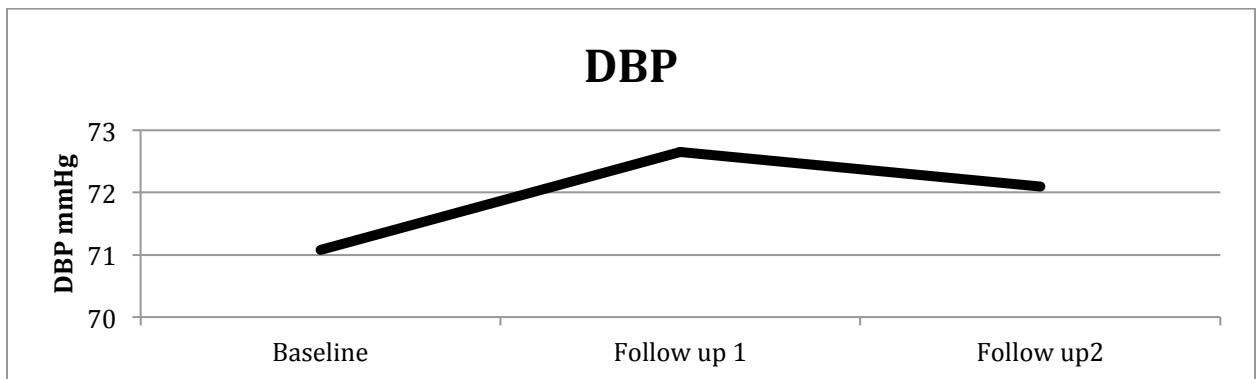


Figure 5. Main effect of Time for DBP in females

Cholesterol Fractions

For LDL cholesterol, results indicated that for men there was only a main effect of time ($F(2, 1820.600) = 9.466, p < .01$) and no main effect of marital status or an interaction between time and marital status. Pairwise comparisons indicate that for the main effect of time baseline LDL ($M = 112.897, SE = 1.046$) and follow up 1 LDL ($M = 115.056, SE = 1.185$) were both higher than follow up 2 LDL ($M = 109.585, SE = 1.238$) but not significantly different from each other (see figure 6). In addition, in terms of the

covariates, there was a significant main effect of race $F(1, 1857.561) = 6.364, p < .05$ with Caucasian males reporting higher LDL ($M = 114.409, SE = .1.194$) compared to African American males ($M = 110.616, SE = 1.187$).

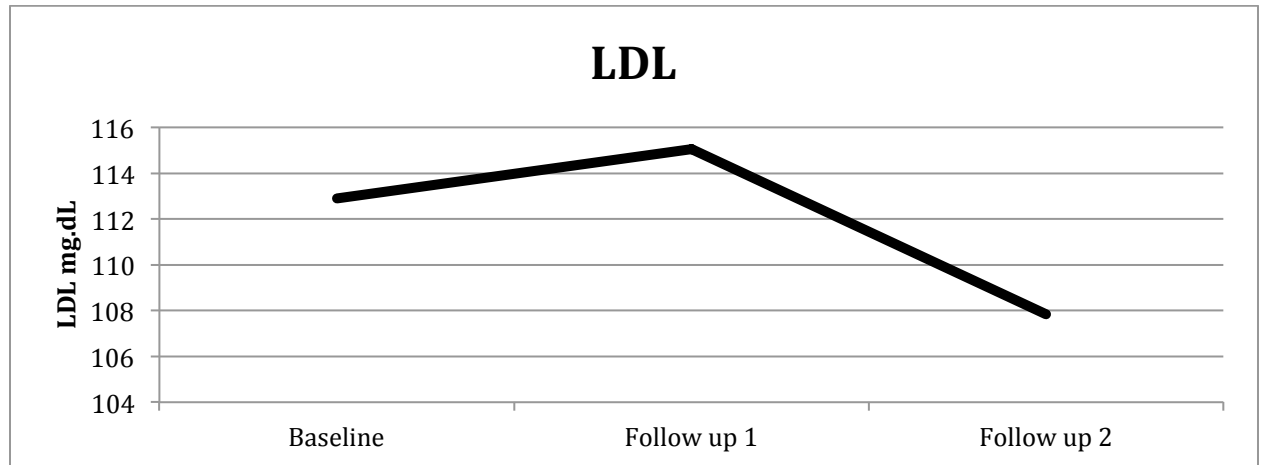


Figure 6. Main effect of time for LDL in males

For women, there was no significant interaction between time and marital status. However, there was a significant main effect of marital status which remained significant ($F(4, 4912.772) = 3.205, p < .05$) when the interaction term (time by marital status) was removed from the model. Additionally, following removal of the interaction term from the model, there was a significant main effect of time ($F(2, 2096.366) = 3.421, p < .05$). Pairwise comparisons for marital status indicate that women who were in a marriage-like relationship ($M = 110.029, SE = 1.231$) had higher LDL than both single and never married women ($M = 106.182, SE = 1.042$) and widowed women ($M = 104.905, SE = 1.319$) (see figure 7). Women who reported being either married ($M = 106.671, SE = .821$) or divorced ($M = 106.601, SE = 1.017$) did not significantly differ in LDL levels from any other marital status groups. Additionally, none of the pairwise comparisons for the main effect of time were significant (see figure 8). Lastly, in terms of covariates, for women there was no main effect of race.

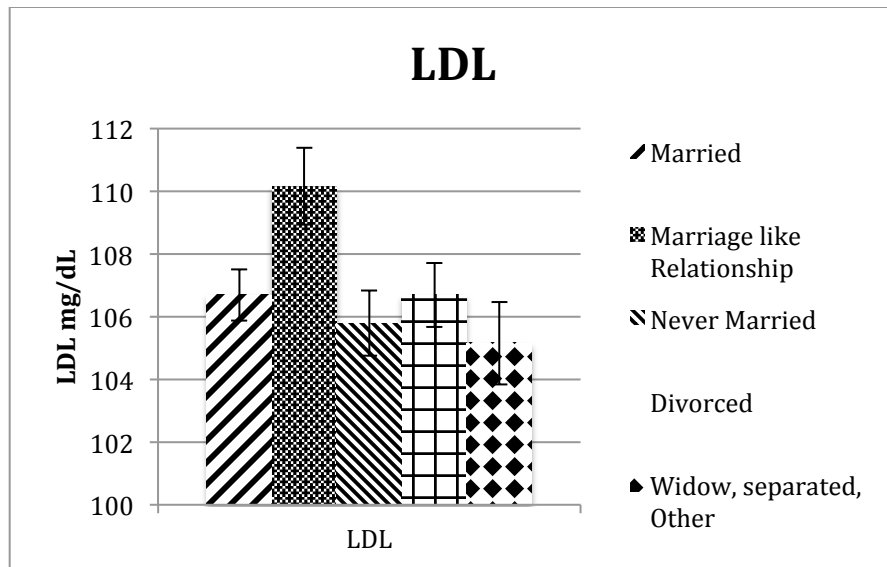


Figure 7. Main effect of marital status on LDL for females

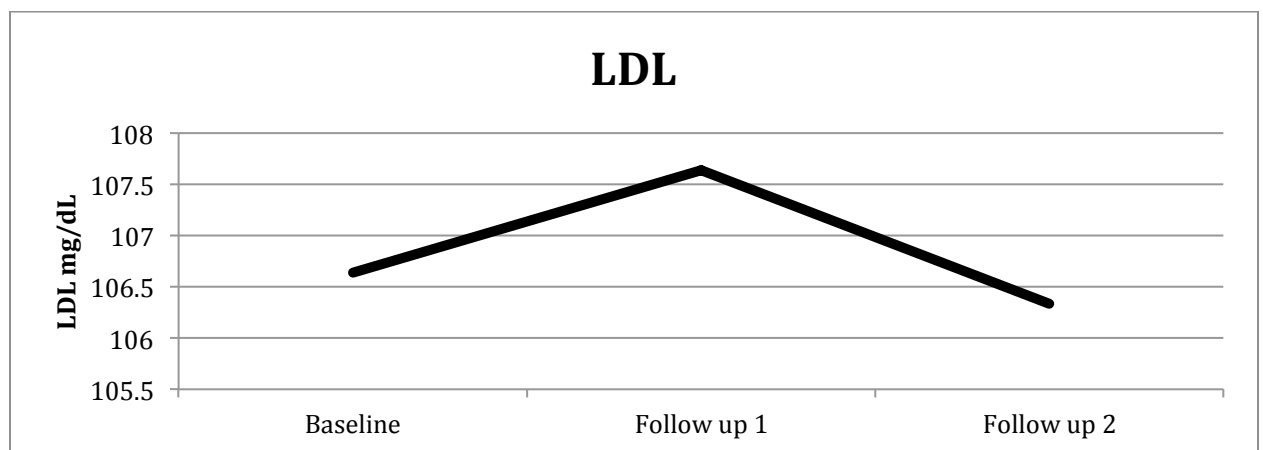


Figure 8. Main effect of Time on LDL for females

Results indicate that for men, there was no significant interaction between time and marital status or a main effect of marital status for HDL cholesterol. However there was a significant main effect of time $F(2, 1858.402) = 50.038, p < .01$ indicating that baseline HDL ($M = 45.627, SE = .380$) did not differ from follow up 1 ($M = 45.539, SE = .393$) HDL but both baseline and follow up 1 HDL were lower than follow up 2 HDL ($M = 49.033, SE = .441$) (see figure 9). With the removal of the time by marital status interaction from the model, the main effect of time remained and a marginal main effect

of marital status was revealed ($p = .055$). In addition, in terms of the covariates, there was a significant main effect of race $F(1, 1835.755) = 131.148, p < .001$ with African American males reporting higher HDL ($M = 50.002, SE = .446$) compared to Caucasian males ($M = 43.465, SE = .442$).

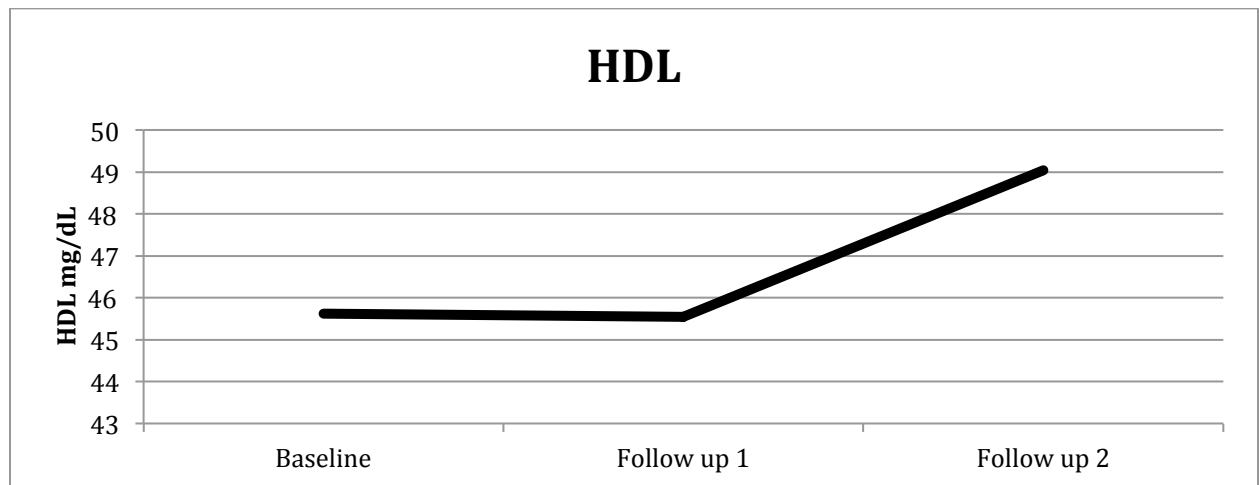


Figure 9. Main effect of time on HDL in males

For women on the other hand, there were significant main effects of both time ($F(2, 2146.09) = 126.747, p .001$) and marital status ($F(4, 4770.596) = 6.286, p < .001$) as well as a significant marital status by time interaction $F(8, 2205.408) = 2.224, p < .05$. This interaction was further examined using one way ANCOVAs at each of the three time points to assess the sources of the differences in HDL. Results of the above ANOVAS indicate that at baseline, females reporting a status of married ($M = 52.379, SE = .469$) had lower HDL than those reporting a marriage like relationship ($M = 54.697, SE = .995$), divorced ($M = 54.584, SE = .748$) and being single and never married ($M = 55.560, SE = .624$). Women who reported being widowed at baseline ($M = 54.338, SE = 1.106$) did not differ significantly in HDL from any other marital status. At follow up 1, those females reporting a status of married had lower HDL ($M = 53.508, SE = .600$) than

women reporting a status of single and never having married ($M = 55.994$, $SE = .715$) and being divorced ($M = 56.324$, $SE = .813$) while at follow up 2 women reporting being married ($M = 57.472$, $SE = .708$) or divorced ($M = 56.992$, $SE = .895$) had lower HDL than both those who were never married and single ($M = 59.813$, $SE = .903$) and those who were widowed ($M = 60.998$, $SE = 1.517$), but were not significantly different from each other (see figure 10). Women who reported living in a marriage like relationship ($M = 54.613$, $SE = 1.172$) or being widowed ($M = 54.780$, $SE = 1.287$) at follow up 1 did not significantly differ in HDL levels from any other marital status. At follow up 2, women who reported living in a marriage like relationship ($M = 58.077$, $SE = 1.355$) did not differ from any other marital status in HDL levels. In addition, in terms of the covariates, there was a significant main effect of race $F(1, 2383.404) = 13.541$, $p < .001$ with African American females reporting higher HDL ($M = 57.277$, $SE = .413$) compared to Caucasian females ($M = 55.198$, $SE = .431$).

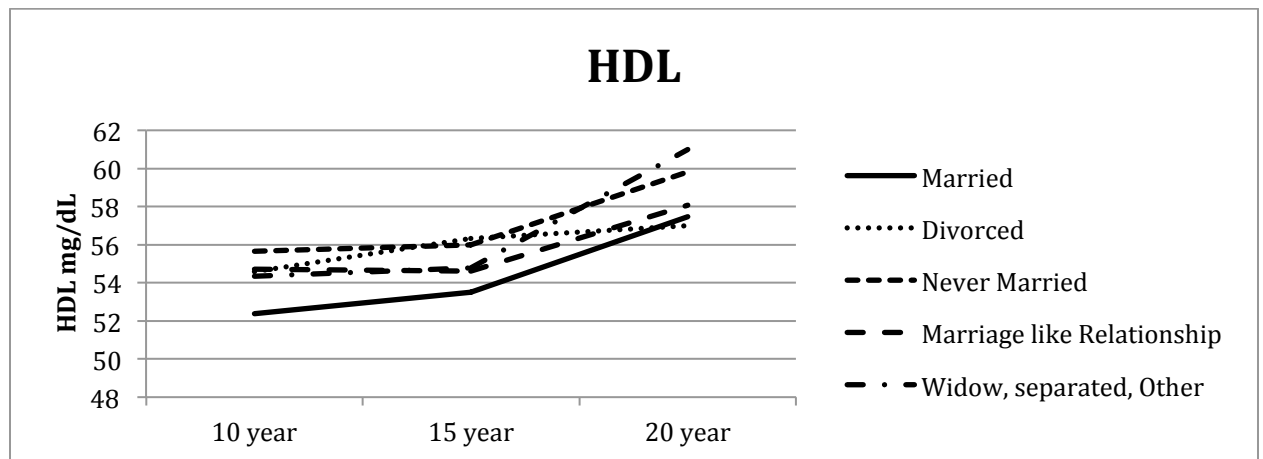


Figure 10. Interaction between time and marital status on HDL in females

Depression

For males, results of the multilevel model indicate that there was no interaction between time and marital status, but there was both a main effect of time ($F(2, 1949.388)$

= 5.923, $p < .01$) and marital status ($F(4, 4021.283) = 5.910, p < .01$). Pairwise comparisons indicate that for men, depression levels at baseline ($M = 9.311, SE = 1.03$) were significantly higher than at either follow up 1 ($M = 8.356, SE = 1.03$) or 2 ($M = 8.650, SE = 1.03$). However depression did not differ significantly between follow up time 1 or 2 for men (see figure 11). Results indicated that widowed men ($M = 10.209, SE = 1.05$) had higher depression scores than married men ($M = 8.053, SE = 1.03$), those in a marriage-like relationship ($M = 8.375, SE = 1.04$), and divorced ($M = 8.453, SE = 1.04$) men. Those reporting never having been married ($M = 8.851, SE = 1.03$) did not differ significantly from any other marital status categories (figure 12). In addition, in terms of the covariates, there was a significant main effect of race ($F(1, 1837.391) = 10.637, p < .01$) with African American males reporting higher depression scores ($M = 9.247, SE = 1.028$) compared to Caucasian males ($M = 8.299, SE = 1.028$).

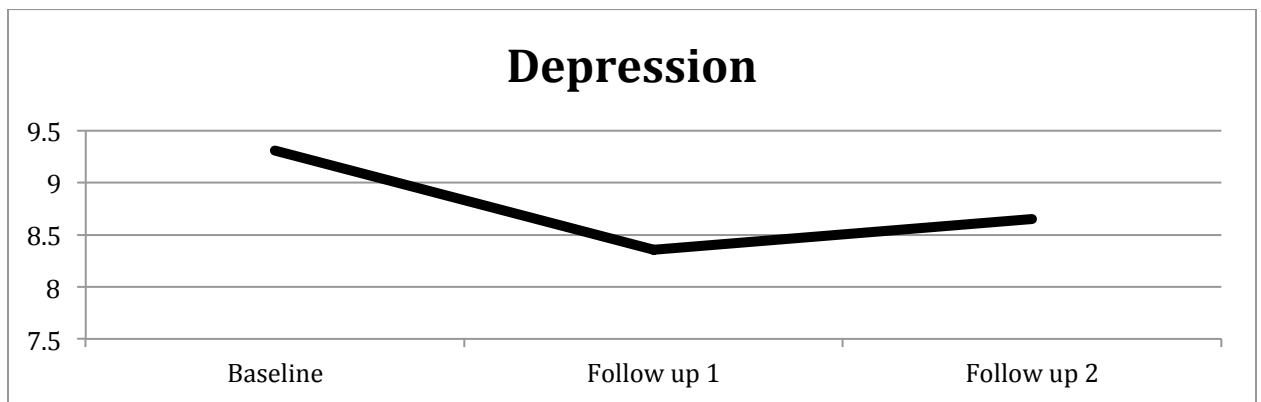


Figure 11. Main effect of time on depression scores for males

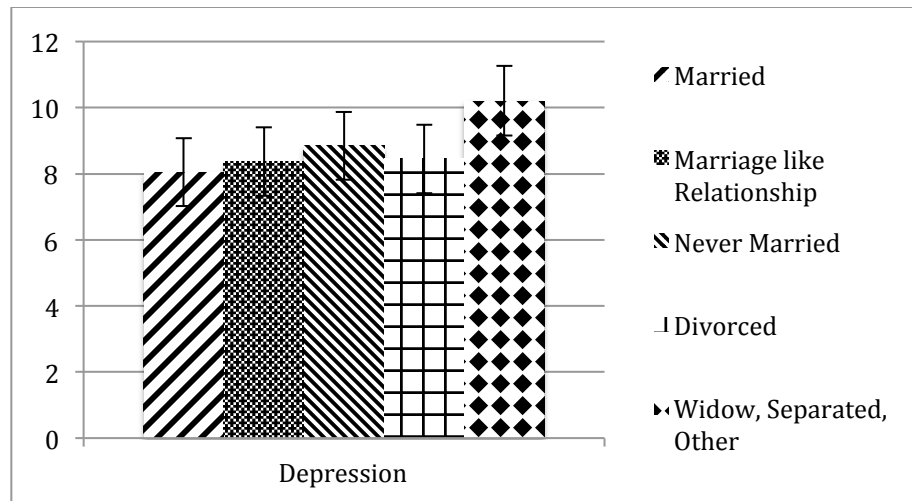


Figure 12. Main effect of marital status on male depression scores

For females, the pattern of results varied from those of the men. Specifically, there was no significant interaction between time and marital status or main effect of marital status. Instead, there was a significant main effect of time $F(2, 2298.458) = 10.695, p < .01$. Pairwise comparisons indicate that baseline depression scores ($M = 9.75$, $SE = 1.02$) were higher than both follow up 1 ($M = 8.65$, $SE = 1.03$) and follow up 2 ($M = 8.97$, $SE = 1.03$) while the difference in depression scores from follow up 2 to follow up 3 was not significant (see figure 13). In addition, in terms of the covariates, there was a significant main effect of race $F(1, 2344.638) = 14.580, p < .001$ with African American females reporting higher depression scores ($M = 9.705$, $SE = 1.023$) compared to Caucasian females ($M = 8.57$, $SE = 1.026$).

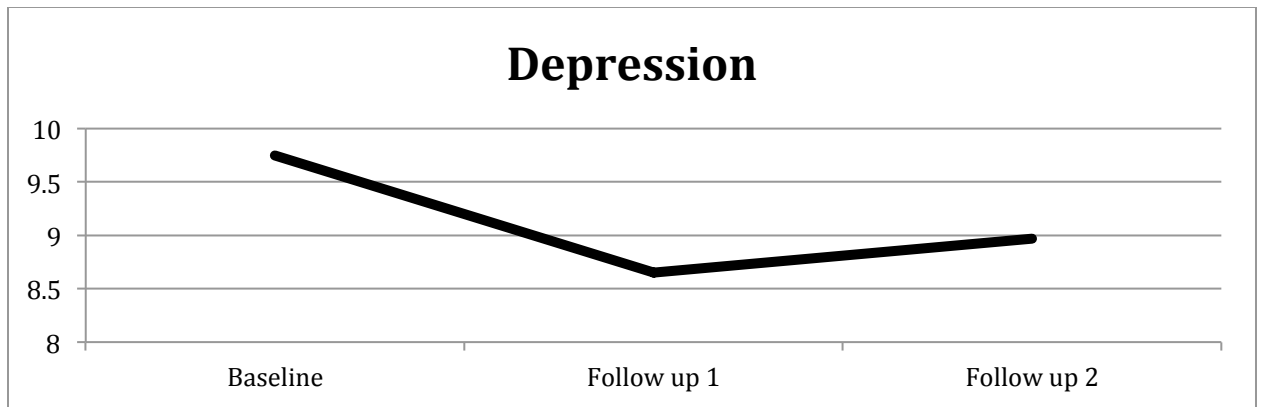


Figure 13. Main effect of time for depression scores in females

Hypothesis 1b

To investigate hypothesis 1b, that divorced individuals will show higher risk compared to individuals who are divorced and cohabiting, and that females will demonstrate a greater effects than males, a series of one way ANCOVAS were performed using baseline data for participants reporting a marital status of divorced at baseline. All analyses controlled for race, age, BMI, household income, and smoking status. Overall, this hypothesis was partially confirmed.

Blood Pressure

For men reporting divorce at baseline, there was a significant relationship between cohabitation and SBP $F(1, 152) = 9.966, p < .01$. Pairwise comparisons indicate that men who reported cohabitating had lower SBP (EMM = 110.136, SE = 1.979) compared to men who did not report cohabitating (EMM = 117.158, SE = 1.324) (see figure 14). For women reporting a marital status of divorced there was also a significant relationship between cohabiting and SBP $F(1, 283) = 4.552, p < .05$. Contrary to what men reported, results indicate that women who reported cohabitating had higher SBP (EMM = 110.293, SE = 1.760) compared to those who did not report cohabiting (EMM = 106.073, SE = 1.078) (see figure 15).

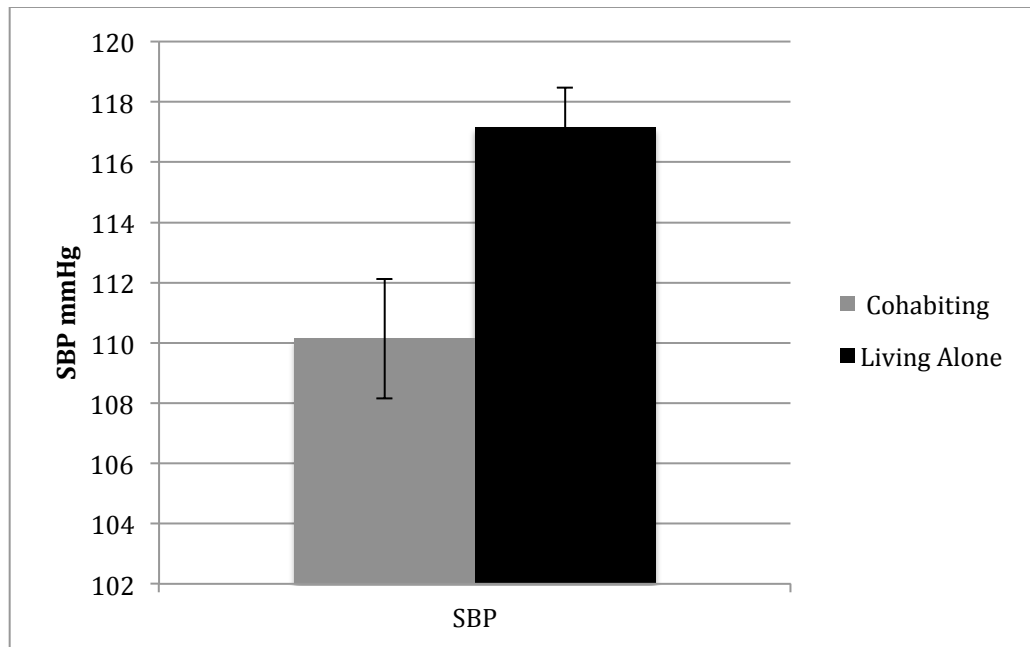


Figure 14. SBP in divorced men who live alone or cohabit at baseline

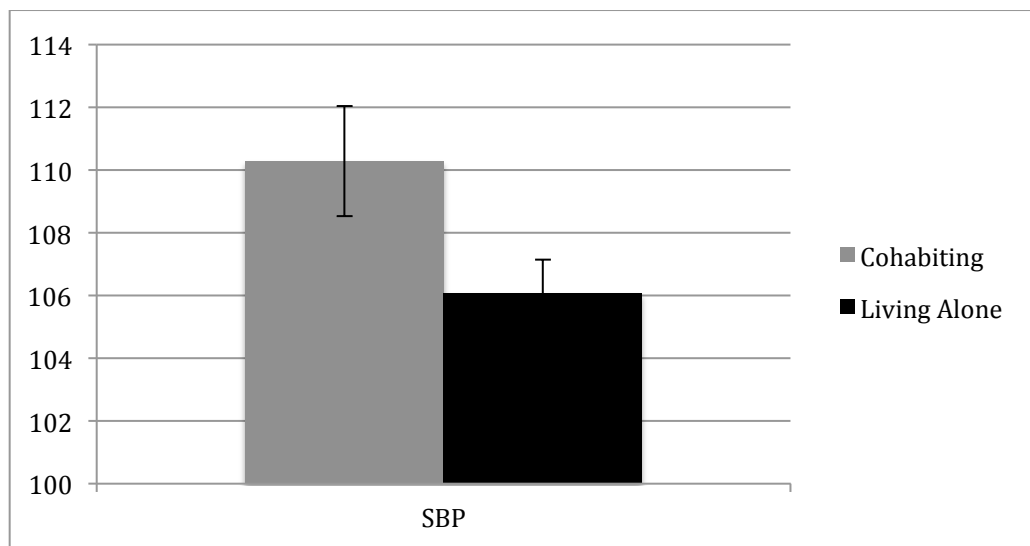


Figure 15. SBP in divorced women who live alone or cohabit at baseline

For DBP, in men only, in contrast to findings in women, there was a significant relationship between cohabiting $F(1, 152) = 13.540, p < .01$. In cohabiting men, DBP was lower (EMM= 70.353, SE = 1.728) compared to those who did not report cohabiting

($EMM = 77.501$, $SE = 1.157$) (see figure 16). There was no significant relationship between cohabitating and DBP for women.

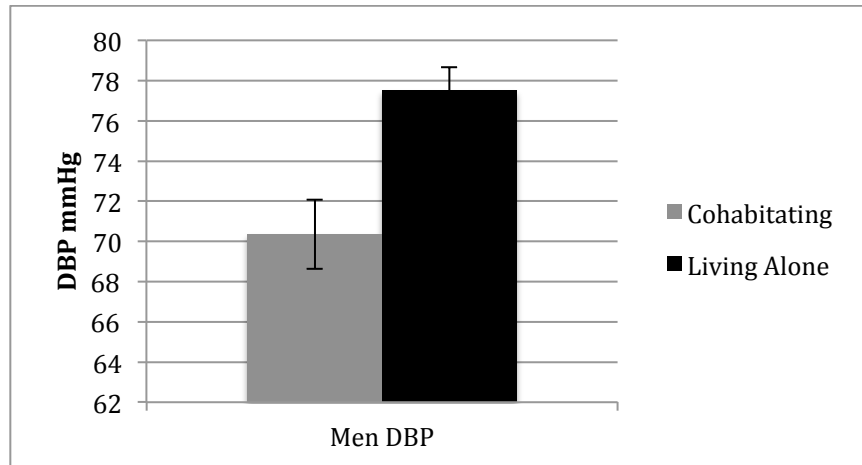


Figure 16. DBP in divorced men who live alone or cohabitate at baseline

Cholesterol Fractions

For both men and women who were divorced, there was no significant relationship between cohabitating and either LDL or HDL cholesterol.

Depression

For both men and women who were divorced, there was no significant relationship between cohabitating and CES-D depression scores.

Hypothesis 1c

To investigate hypothesis 1c, that widowed individuals will show higher risk compared to individuals who are divorced and cohabiting and that females will demonstrate a greater effects than males, a series of one way ANCOVAS were performed using baseline data for participants reporting a marital status of divorced at baseline. All analyses controlled

for race, age, BMI, household income, and smoking status. Overall, this hypothesis was not confirmed.

Blood pressure

For both men and women who were widowed, there was no significant relationship between cohabitating and either SBP or DBP.

Cholesterol Fractions

For women only, there was a significant relationship between cohabitating and LDL cholesterol $F(1, 116) = 5.294, p < .05$. Results indicate that for widowed women who reported cohabitating, their LDL was lower (EMM = 92.724, SE = 6.670) than for those who did not report cohabitating (EMM = 110.774, SE = 4.019) (see figure 17).

There was no significant relationship between cohabitating and LDL cholesterol for men who were of widowed. For both men and women who were widowed, there was no significant relationship between cohabitating and HDL cholesterol.

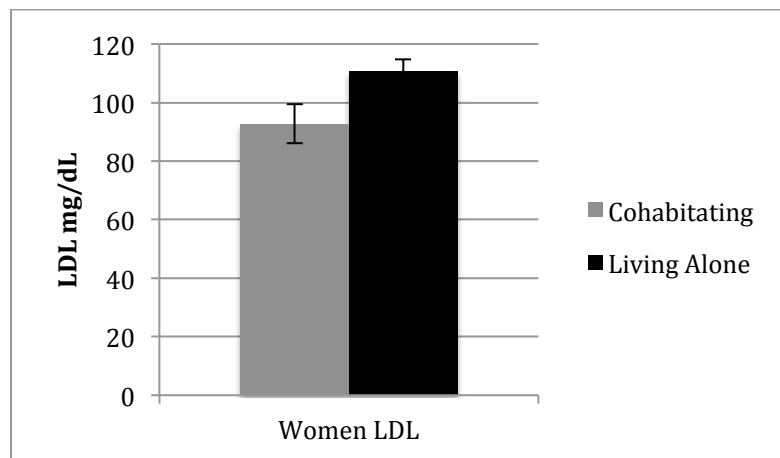


Figure 17. LDL in widowed women who cohabit or live alone at baseline

Depression

For both men and women who were widowed, there was no significant

relationship between cohabitating and CES-D depression scores.

AIM 2

Hypothesis 2

To investigate hypothesis 2, that for those reporting divorce status, biological variables and depression will initially increase and then decrease by years since divorce, hierarchical multiple regressions were run. The regressions were run using reported number of years since divorce for those participants reported being divorced at baseline. These regression were run separately for men and women. To assess if the above relationships varied by sex., age, BMI, smoking status, household income, and race, all were entered into step 1, and years since divorce was added in the second step for each risk factor (DBP, SBP, LDL, HDL, Depression). None of these analyses yielded any effect for years since divorce (see table 3, a-e).

Blood Pressure

Hierarchical multiple regression was used to assess the ability of years since divorce to predict DBP after controlling for age, smoking status, household income, BMI, and race. The covariates above were entered into step 1, explaining 13.3% of the variance in DBP for males. After entering years since divorce in step 2, the model explained 13.3% of the variance in DBP for males, a non significant change. For females, step 1 of the same model explained 17.8% of the variance in DBP. After the addition of time since divorce in step 2, the model explained 17.8% of the variance in DBP, a non significant change.

Hierarchical multiple regression was used to assess the ability of years since divorce to predict SBP after controlling for age, smoking status, household income, BMI,

and race. The covariates above were entered into step 1, explaining 16.6% of the variance in SBP for males. After entering years since divorce in step 2, the model explained 16.7% of the variance in SBP for males, a non significant change. For females, step 1 of the same model explained 16% of the variance in SBP. After the addition of time since divorce in step 2, the model explained 16% of the variance in SBP, a non significant change.

Cholesterol

Hierarchical multiple regression was used to assess the ability of years since divorce to predict LDL after controlling for age, smoking status, household income, BMI, and race. The covariates above were entered into step 1, explaining 8.5% of the variance in LDL for males. After entering years since divorce in step 2, the model explained 8.7% of the variance in LDL for males, a non significant change. For females, step 1 of the same model explained 6.8% of the variance in LDL. After the addition of time since divorce in step 2, the model explained 6.9% of the variance in LDL, a non significant change.

Hierarchical multiple regression was used to assess the ability of years since divorce to predict HDL after controlling for age, smoking status, household income, BMI, and race. The covariates above were entered into step 1, explaining 18.2% of the variance in HDL for males. After entering years since divorce in step 2, the model explained 18.5% of the variance in HDL for males, a non significant change. For females, step 1 of the same model explained 16.3% of the variance in HDL. After the addition of time since divorce in step 2, the model explained 16.9% of the variance in HDL, a non significant change.

Depression

Hierarchical multiple regression was used to assess the ability of years since divorce to predict depression scores after controlling for age, smoking status, household income, BMI, and race. The covariates above were entered into step 1, explaining 7.1% of the variance in depression for males. After entering years since divorce in step 2, the model explained 7.1% of the variance in depression for males, a non significant change. For females, step 1 of the same model explained 6.6% of the variance in depression. After the addition of time since divorce in step 2, the model explained 6.7% of the variance in depression, a non significant change.

Table 3a. Hierarchical Multiple Regression Analyses Predicting DBP From Marital Status With Men and Women

Male and Female Models				
Male			Female	
Predictor	ΔR^2	β	ΔR^2	β
Step 1	.133***		.178***	
Control variables ^a				
Step 2	.000	-.005	.000	-.014
DBP				
Total R^2	.133***		.178***	
n	171		314	

Note. ^aControl variables included age, race, smoking status, BMI, and income.

* $p < .05$. *** $p < .001$.

Table 3b. Hierarchical Multiple Regression Analyses Predicting SBP From Marital Status With Men and Women

Male and Female Models				
Male			Female	
Predictor	ΔR^2	β	ΔR^2	β
Step 1	.166***		.160***	
Control variables ^a				
Step 2	.001	-.044	.000	.006
SBP				
Total R^2	.167***		.160***	
n	171		314	

Note. ^aControl variables included age, race, smoking status, BMI, and income.

* $p < .05$. *** $p < .001$.

Table 3c. Hierarchical Multiple Regression Analyses Predicting LDL From Marital Status With Men and Women

Male and Female Models				
Male			Female	
Predictor	ΔR^2	β	ΔR^2	β
Step 1	.085*		.068***	
Control variables ^a				
Step 2	.002	.049	.002	.047
LDL				
Total R^2	.087*		.069***	
n	171		314	

Note. ^aControl variables included age, race, smoking status, BMI, and income.

* $p < .05$. *** $p < .001$.

Table 3d. Hierarchical Multiple Regression Analyses Predicting HDL From Marital Status With Men and Women

Male and Female Models				
Male		Female		
Predictor	ΔR^2	β	ΔR^2	β
Step 1	.182***		.163***	
Control variables ^a				
Step 2	.003	.061	.006	-.082
HDL				
Total R^2	.185***		.169***	
n	171		314	

Note. ^aControl variables included age, race, smoking status, BMI, and income.

* $p < .05$. *** $p < .001$.

Table 3e. Hierarchical Multiple Regression Analyses Predicting Depression From Marital Status With Men and Women

Male and Female Models				
Male		Female		
Predictor	ΔR^2	β	ΔR^2	β
Step 1	.071*		.066***	
Control variables ^a				
Step 2	.000	.001	.001	-.039
Depression				
Total R^2	.071 ^t		.067*	
n	171		314	

Note. ^aControl variables included age, race, smoking status, BMI, and income.

^t $p < .10$. * $p < .05$. *** $p < .001$.

AIM 3

Hypothesis 3

To investigate hypothesis 3 that having children will increase risk in all marital status categories compared to those who report having no children, multilevel repeated measures models were again used. The models were run separately for males and females to investigate if females would demonstrate greater effects of parental and marital status. Significant interactions were further investigated using ANCOVAS. All analyses controlled for race, age, BMI, household income, and smoking status. Only interactions with parental status will be reported for this aim. Interactions with marital status and time are reported previously.

Blood Pressure

For men, adding parental status as another variable to the multilevel model did not change the pattern of results regarding SBP with no significant three way interaction among time, marital status and parental status, 2 –way interactions involving parental status, or a main effect of parental status. The time by marital status interaction remained significant. In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model.

For women on the other hand, adding parental status to the model changed the pattern of results regarding SBP. Specifically, for women, the interactions among time, marital status and parental status, and among marital status and time or marital status and parental status and main effect of parental status were not significant. There was however a significant parental status by time interaction ($F(8, 2270.269) = 7.079, p < .01$), and the main effect of marital status was no longer significant. Follow-up ANCOVAs to

investigate this interaction indicated that at baseline and follow up 1, the difference between women who reported having no children ($M = 107.693$, $SE = .490$; $M = 111.332$, $SE = .683$) and women who did ($M = 106.723$, $SE = .346$) was not significant. At follow up 2, there was a marginally significant effect of parental status; women who reported having no children ($M = 113.678$, $SE = .802$) had lower SBP than women who reported having children ($M = 114.861$, $SE = .506$) (see figure 18). In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model.

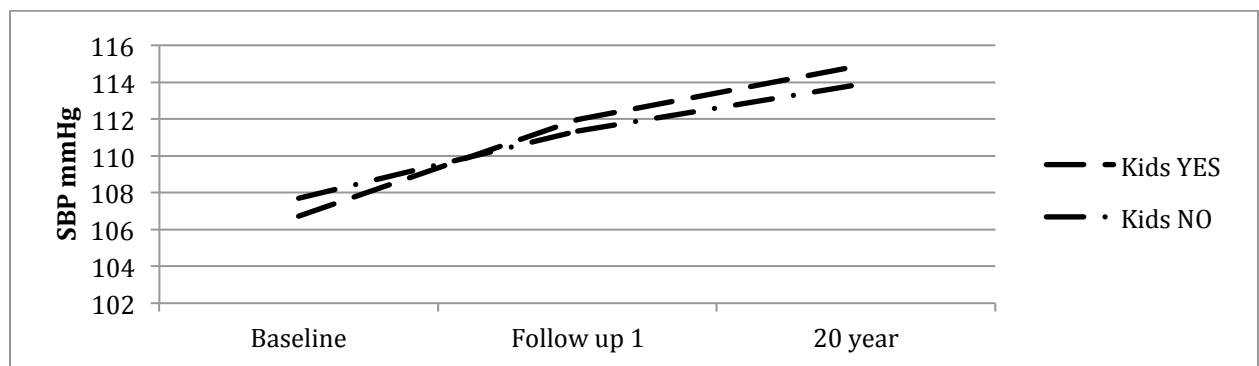


Figure 18. Time by parental status interaction for SBP.

For DBP, the three way interaction and main effects involving parental status were not significant for males. However there was marginally significant parental status by time interaction which remained when the model was tested without the 3 way interaction $F(2, 1721.142) = 2.968$, $p = .05$. Follow up ANCOVAs to investigate this interaction indicated that at baseline, men who reported having no children had higher DBP ($M = 75.414$, $SE = .399$) compared to men who reported having children ($M = 74.337$, $SE = .324$). There was no significant difference between men who reported having no children ($M = 77.196$, $SE = .566$) compared to those who had children ($M = 76.868$, $SE = .445$) at follow up 1 and no difference between those men who reported no

children ($M = 75.198$, $SE = 74.763$) and those who had children ($M = 74.763$, $SE = .465$) at follow up 2 (see figure 19). In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model.

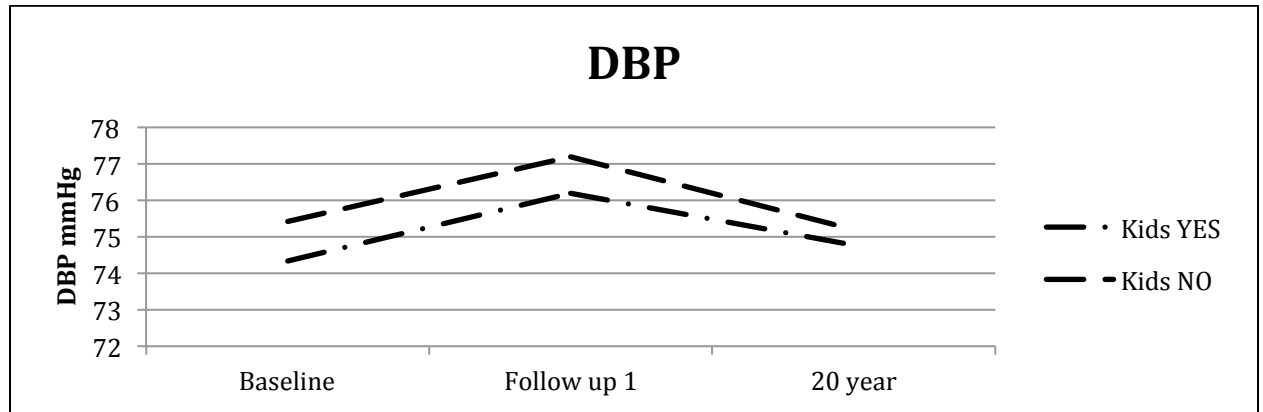


Figure 19. Time by parental status interaction in males

For women, there was a similar pattern of results, in that there was no significant three way interaction among time, parental status, and marital status, or significant main effects of parental status, but there was a significant time by parental status interaction. Follow up ANCOVAs indicate that at baseline, women who reported having no children had higher DBP ($M = 70.952$, $SE = .388$) than women who reported having children ($M = 69.933$, $SE = .274$), while there was no significant difference between women who reported having no children ($M = 72.370$, $SE = .540$) compared to those who had children ($M = 72.585$, $SE = .349$) at follow up 1 and no difference between those women who reported no children ($M = 72.037$, $SE = .574$) and those who had children ($M = 72.616$, $SE = .362$) at follow up 2 (see figure 20). In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model.

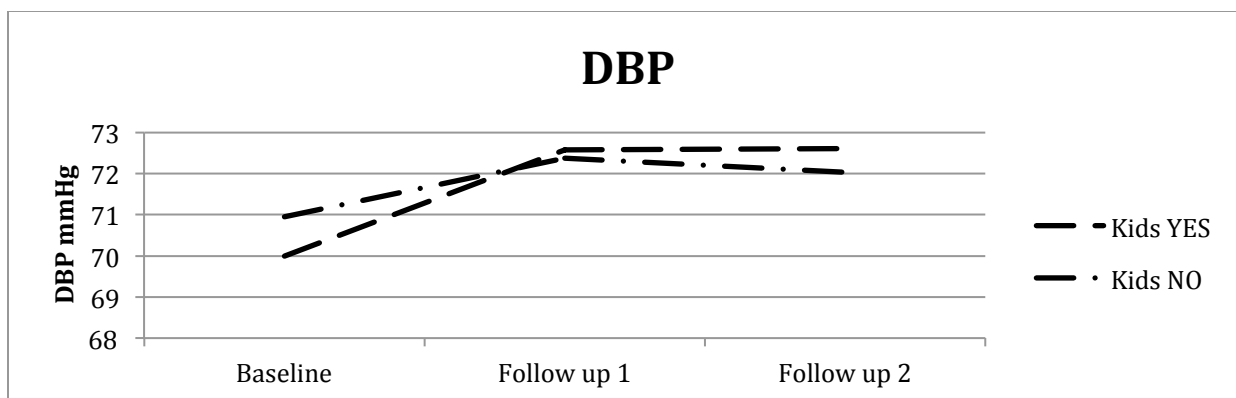


Figure 20. Time by Parental Status interaction on DBP in females

Cholesterol Fractions

For LDL in men, the addition of parental status to the model did not change the overall pattern of results in that there were no interactions or main effect of parental or marital status but there was still a main effect of time as discussed earlier. In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model. For women however, the multilevel model indicates a significant main effect of parental status ($F(1, 5231) = 5.511, p < .05$) and a significant three way interaction among parental status, marital status, and time for LDL with $F(8, 2314.313) = 2.117, p < .05$. Follow up ANCOVAs did not provide any further clarification on the nature of this interaction in that none of the ANCOVAs produced significant results. Specifically, marital status was examined in association with LDL cholesterol separately for women with and without children at each of the three time points to assess where the interaction among these variables was. As stated above, none of these tests were significant, therefore the estimated marginal means for the 3way interaction term were entered into a graph (see figure 21) to further examine the interaction and what may be driving these relationships among marital status, parental status, and time.

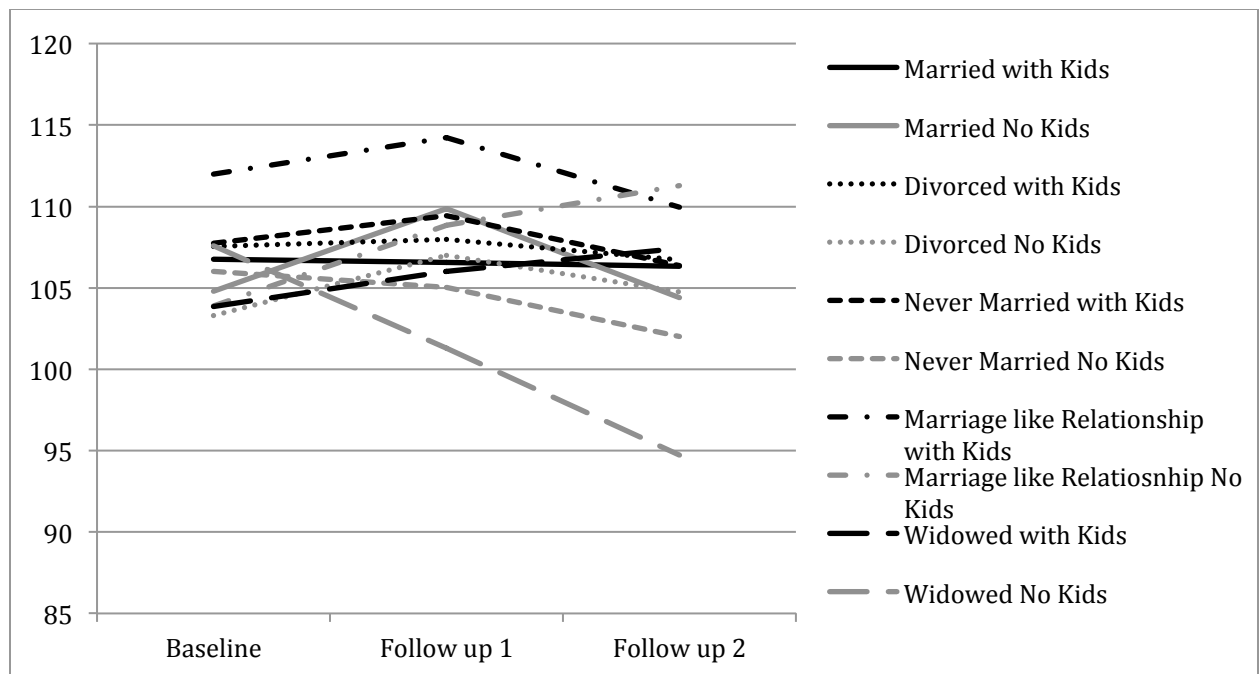


Figure 21. 3-way interaction among parenting status, marital status and time in women on LDL

Examining figure 20 reveals several interesting patterns in LDL scores for women. That is, in general for each marital status LDL levels were higher for those with children than those without. In addition, in general LDL levels went up from baseline to follow up 1 and then down between follow up 1 and follow up 2 for women. However, for both women who were never married with no children (baseline: EMM = 106, SE = 1.452; follow up 1: EMM = 105.01, SE = 1.589; follow up 2: EMM = 102.011, SE = 1.906) and who were widowed with no children (baseline: EMM = 107.575, SE = 4.185; follow up 1: EMM = 101.238, SE = 4.780; follow up 2: EMM = 94.724, SE = 6.036) LDL went consistently down over all three time points. For women who were in a marriage-like relationship with no kids (Baseline: EMM = 103.887, SE = 2.262; follow up 1: EMM = 108.849; SE = 2.594; follow up 2: EMM = 111.278; SE 2.903) or who were widowed with children (Baseline: EMM = 103.854, SE = 2.093; follow up 1: EMM = 106.001, SE = 2.009; follow up 2: EMM = 107.445, SE = 2.338), LDL levels went

consistently up over all three time points. For women who were married with children (baseline: EMM = 106.753, SE = .959; follow up 1: EMM = 106.58, SE = .981; follow up 2: EMM = 106.32, SE = 1.050) LDL levels lowered slightly over all three time points but remained within one point. Although these differences are not necessarily significantly distinct patterns, examining them does help provide a possible explanation for this finding. In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model.

For men, consistent with the previous multilevel model investigating HDL, there were no significant interactions among marital status, parental status and time, neither were there main effects of marital status or parental status. However, the main effect of time discussed earlier was still present. In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model. For women, the three way interaction among marital status, parental status, and time was not significant, but there was a significant main effect of parental status. After removing the three way interaction from the model, the main effect of parental status ($F(1, 5228.315) = 8.608, p < .05$) remained along with the addition of the time by marital status interaction discussed earlier (see figure 22). Pairwise comparisons indicate that women who had children ($M = 55.735, SE = .362$) had significantly lower HDL than women who did not ($M = 57.350, SE = .504$). In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model.

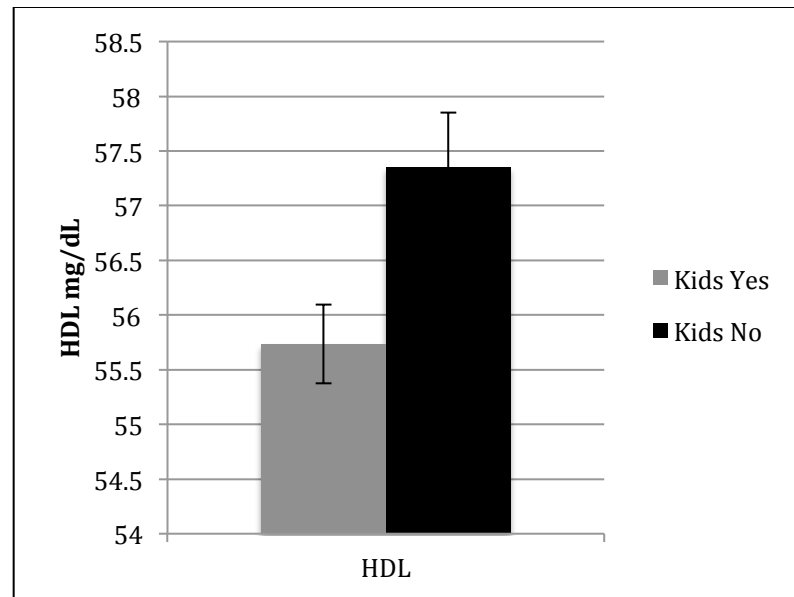


Figure 22. HDL in women with and without children

Depression

For men, there were no significant main effects or two way interactions regarding parental status. There was however, a significant three way interaction among time, marital status and prenatal status with $F(8, 2078.760) = 2.371, p < .05$. Follow up ANCOVAs indicate that the marital status was significantly related to depression scores for men with children ($F(4, 1002) = 2.882, p < .05$) at baseline only. Marital status was not related to depression scores regardless of parental status at any other time point. In order to better understand the overall patterns involving marital and parental status over time, the estimated marginal means provided in the original mixed model for the 3-way interaction are presented in figure 23. In general, it appears that married and widowed men who have children report lower depression levels than their marital status counterparts without children. However, for men who are divorced, and in a marriage-like relationship, it appears in general that men with children report higher depression levels than their childless counterparts. There was no clear pattern for those who were

never married. In addition, it appears that there is a common pattern of depression scores decreasing from baseline to follow up 1 and then increasing again between follow up 1 and follow up 2 (see figure 23). Only those men who were divorced without children, in a marriage-like relationship without kids or widowed with children had a different pattern. Specifically, for divorced men without kids, the scores on the CES-D went up from baseline ($M = 7.534$, $SE = 1.096$) to follow up 1 ($M = 7.762$, $SE = 1.125$) and up again from follow up 1 to follow up 2 ($M = 9.397$, $SE = 1.119$). For men in marriage-like relationships without kids, depression scores went up from baseline ($M = 8.511$, $SE = 1.074$) to follow up 1 ($M = 8.831$, $SE = 1.096$) but then down from follow up 1 to follow up 2 ($M = 7.145$, $SE = 1.102$). Finally, for widowed men with children, depression scores went down from baseline ($M = 11.015$, $SE = 1.091$) to follow up 1 ($M = 10.257$, $SE = 1.117$) and then down again from follow up 1 to follow up 2 ($M = 9.616$, $SE = 1.094$). Although these differences are not necessarily significantly distinct patterns, examining them does help provide a possible explanation for this finding. In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model.

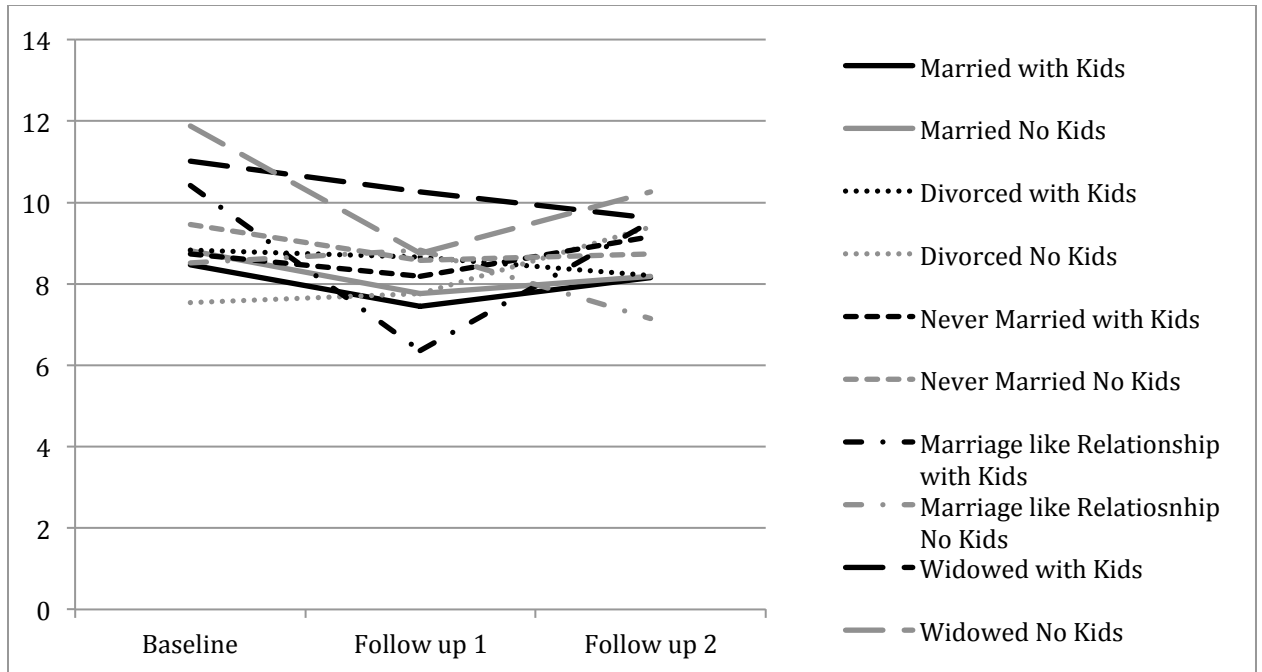


Figure 23. 3-way Interaction among parental status, marital status, and time for men and depression scores

For women, there was no change in the overall pattern of significance with the addition of parental status to the model. That is, there were no significant interactions or main effects of marital status or parental status, however the significant main effect of time discussed earlier remained. In order to summarize the overall results for each hypothesis, a summary table is provided below (see Table 4). In addition, in terms of covariates, the pattern of results regarding race did not change with the addition of parent status to the model.

Table 4. Summary of Results

<i>Summary of Results</i>			
<i>Hypothesis 1a</i> Widowed individuals will show greater risk than divorced individuals who in turn will show greater risk than single individuals who in turn will show higher risk than married and cohabiting individuals. These relationships will vary by sex.	<i>Risk factor:</i>	<i>Men</i>	<i>Women</i>
	SBP	Interaction, Main effect Marital status and Time significant <i>Hypothesis partially supported</i>	Main effect Marital status and Time Significant <i>Hypothesis partially supported</i>
	DBP	Main effect Time significant <i>Hypothesis not supported</i>	Main effect Time significant <i>Hypothesis not supported</i>
	LDL	Main effect Time significant <i>Hypothesis not supported</i>	Main effect Marital status and Time significant <i>Hypothesis partially supported</i>
	HDL	Main effect Time significant <i>Hypothesis not supported</i>	Interaction, Main effect Marital status and Time significant <i>Hypothesis partially supported</i>
	Depression	Main effect Marital status and Time significant <i>Hypothesis partially supported</i>	Main effect Time significant <i>Hypothesis not supported</i>
<i>Hypothesis 1b</i> Divorced individuals will show higher risk compared to individuals who are divorced and cohabiting. These relationships will vary by sex.	<i>Risk factor:</i>	<i>Men</i>	<i>Women</i>
	SBP	Significant <i>Hypothesis supported</i>	Significant <i>Not supported</i>
	DBP	Significant <i>Hypothesis supported</i>	NS
	LDL	NS	NS
	HDL	NS	NS
	Depression	NS	NS
<i>Hypothesis 1c</i> Individuals who are widowed or separated will show greater risk compared to those who are widowed or separated and cohabiting. These relationships will vary by sex.	<i>Risk factor:</i>	<i>Men</i>	<i>Women</i>
	SBP	NS	NS
	DBP	NS	NS
	LDL	NS	Significant <i>Hypothesis supported</i>
	HDL	NS	NS
	Depression	NS	NS

<i>Hypothesis 2</i> For those reporting divorce status, biological and psychosocial risk factors will initially increase and then decrease by years since divorce. These relationships will vary by sex	<i>Risk factor:</i>	<i>Men</i>	<i>Women</i>
	DBP	NS	NS
	SBP	NS	NS
	LDL	NS	NS
	HDL	NS	NS
	Depression	NS	NS
<i>Hypothesis 3</i> Having children will increase risk in all marital status categories compared to those who report having no children.	<i>Risk factor:</i>	<i>Men</i>	<i>Women</i>
	SBP	NS <i>Hypothesis not supported</i>	Parental status X time interaction significant <i>Hypothesis not supported</i>
	DBP	Parental status X Time interaction significant <i>Hypothesis not supported</i>	Parental status X Time interaction significant <i>Hypothesis not supported</i>
	LDL	NS <i>Hypothesis not supported</i>	3-way Interaction Main effect Parental Status significant <i>Hypothesis partially supported</i>
	HDL	NS <i>Hypothesis not supported</i>	Main effect Parental status significant <i>Hypothesis not supported</i>
	Depression	3-way Interaction significant <i>Hypothesis partially supported</i>	NS <i>Hypothesis not supported</i>

EXPLORATORY ANALYSES

A series of ANCOVAs was run looking at the psychosocial variables of job strain, social support and social network to assess if there were any relation among these variables and the marital statuses examined in the above research hypotheses. Bonferroni correction was applied to all follow up comparisons. Unfortunately because these variables were not available at all three time points, they could not be added into the mixed models as covariates to determine if the pattern of results would change. All analyses controlled for race, age, BMI, household income, and smoking status and run separately for men and women.

Job Strain

Job strain consisted of psychological demands and control indices. For men, neither decisional latitude (control) or psychological demands were associated with marital status. For women, while decisional latitude was not associated with marital status, psychological demands were ($F(4,2071) = 3.347, p < .01$, partial eta squared = .006) in that women who were divorced at baseline reported higher psychological demands ($M = 32.89, SE = .383$) than women who were married ($M = 31.387, SE = .240$) while women who were single-never married ($M = 32.406, SE = .318$), living in a marriage-like relationship ($M=31.328, SE = .511$) or widowed ($M = 32.040, SE = .563$) did not differ significantly from any other marital status in terms of their psychological demands scores (see figure 24).

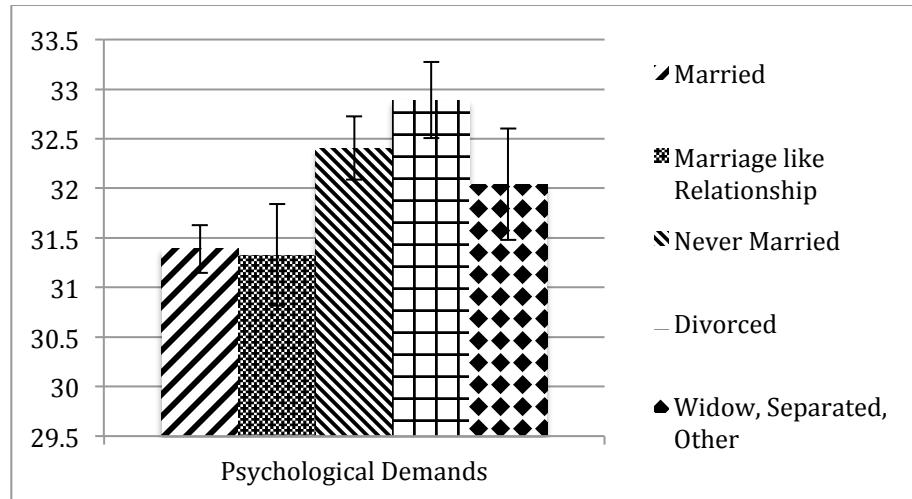


Figure 24. Psychological demands and marital status for women at baseline

Social Support

Emotional social support at follow up 1 was significantly related to marital status for both men ($F(4, 1459) = 6.19, p < .01$, partial eta square = .017) (see figure 25) and women ($F(4, 1813) = 3.269, p < .05$) (see figure 25). Specifically, men who were married ($M = 13.905, SE = .108$) had higher levels of emotional support from the close friends and family than those who were single-never married ($M = 13.157, SE = .127$). Men who were divorced ($M = 13.401, SE = .190$), in a marriage-like relationships ($M = 13.543, SE = .233$) or widowed ($M = 13.897, SE = .307$) did not differ from any other marital status. While for women, those who were married ($M = 14.014, SE = .102$) had higher emotional support from friends and family than those who were in a marriage-like relationship ($M = 13.349, SE = .200$). Women who were divorced ($M = 13.643, SE = .138$), never married ($M = 13.763, SE = .121$) or widowed ($M = 13.972, SE = .217$) did not differ significantly from any other marital status (see figure 26).

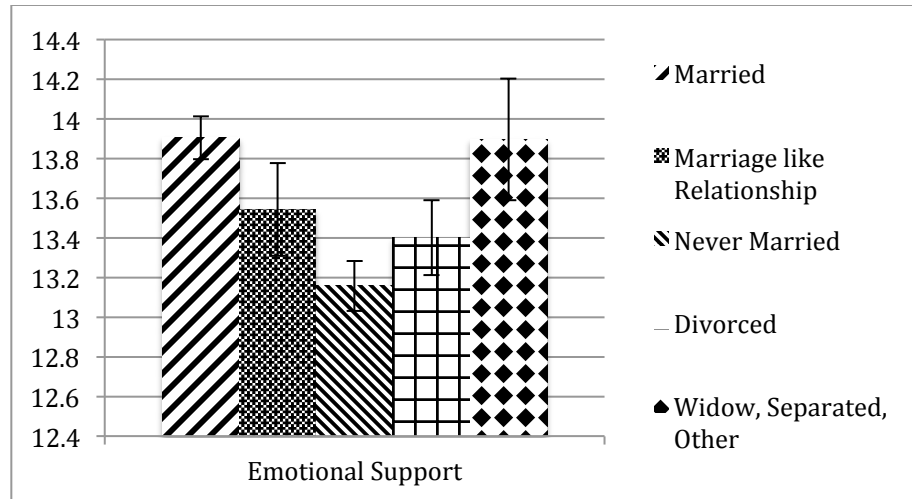


Figure 25. Emotional support and men's marital status at follow up 1

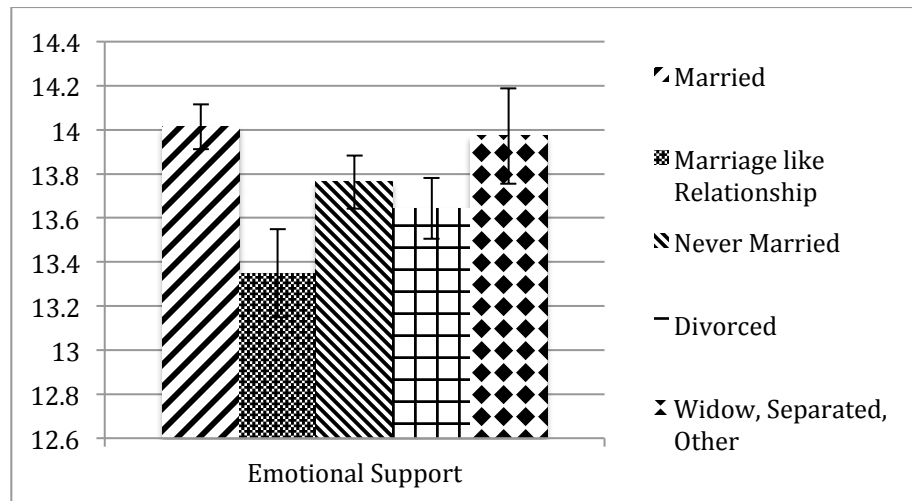


Figure 26. Emotional support and women's marital status at follow up 1

Emotional support at follow up 2 was significant only for women ($F(4, 1704) = 13.074, p < .01$, partial eta squared = .01) not men (see figure 27). Pairwise comparisons indicated that women who had never been married ($M = 12.274, SE = .108$) had lower emotional support from their close friends and family than women who were either married ($M = 12.803, SE = .084$) or divorced ($M = 12.692, SE = .107$). Women who were in a marriage-like relationship ($M = 12.581, SE = .160$) or widowed ($M = 12.569, SE =$

.179) differed from no other marital status. Demandingness was not related to marital status for men or women at follow 1 or 2.

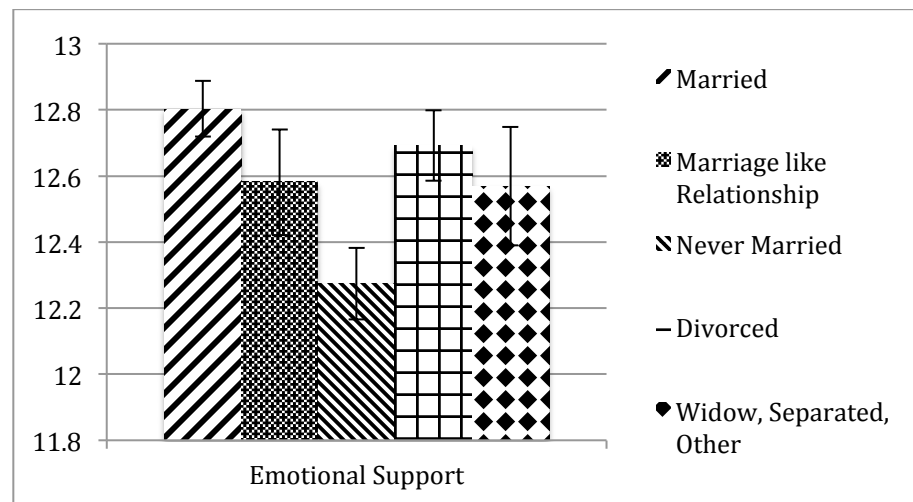


Figure 27. Emotional support and women's marital status at follow up 2

Social Network Index

For both men and women social network as reported by the number of friends and family members seen in the past month was not associated with marital status at either follow up 1 or at follow up 2.

Race and Ethnicity Exploratory Analyses

Exploratory analyses were conducted to assess whether the pattern of results were similar across races. This sample consists of both African American and Caucasian participants. To explore differences among African Americans and Caucasian participants, the sample was split by race and separate mixed model analyses were run to see if there were any variations in the patterns of results for findings regarding marital status and parental status across participants' racial identification. For the hypothesis that widowed individuals will show greater risk than divorced individuals who in turn will show greater risk than single individuals who in turn will show higher risk than married

and cohabiting individuals, for African American men, results followed a similar pattern as when all male participants were combined in one analyses. The only differences were in regard to SBP and depression. Specifically, the interaction of marital status and time in regard to SBP was no longer significant, although it was marginal at $p = .08$, and only a main effect of time remained ($p < .001$). For results regarding depression scores, there was a main effect of time only ($p < .01$) and no main effect of marital status as in the original analyses.

Several differences were observed for Caucasian men when examined separately as well regarding SBP, DBP, HDL, and depression. Specifically, similar to African American men, there was no longer a significant interaction regarding SBP, and only a main effect of time ($p < .001$). For DBP on the other hand, not only was there the original main effect of time ($p < .001$), but also a main effect of marital status emerged ($p < .05$) indicating never married men having lower DBP than married men who had lower DBP than widowed men, divorced men, and finally, men in a marriage like relationship. Whereas for African American men, the results regarding HDL remained the same as the original analyses, for Caucasian men, there was a significant marital status by time interaction for HDL when they were examined on their own ($p < .05$) and main effect of marital status ($p < .01$) indicating married men had the lowest HDL, followed by divorced, then widowed men, men in a marriage like relationship and finally never married men. Only married and never married men were significantly different from each other. Lastly, the original analyses reveal both main effects of time and marital status regarding depression, however when analyzed separately African American men showed only the main effect of time and Caucasian men showed only the main effect of marital

status ($p < .001$) with marital status categories in the same order of level of depression as the original analyses.

Overall there were several small deviations from the original pattern of results regarding men's risk pattern when African American and Caucasian men were examined separately. That is, when examined separately marital status appeared to play no part in African American men's risk for heart disease. Time was related to risk for all variables however. For Caucasian men on the other hand, when examined separately, marital status category was involved in the risk profile for all but SBP and LDL risk factors.

To examine the impact of race for hypotheses 1b and 1c, that divorced and widowed individuals will show higher risk compared to individuals who are divorced and cohabiting or widowed and cohabitating, the sample was again split by race. In the original analyses the two main findings were relationships between SBP and marital status and DBP and marital status. When examined separately, it appears that these relationships were still significant for divorced African American men only. For divorced Caucasian men there was no significant relationship between cohabiting and heart health risk. For men who were widowed, the original analyses revealed no significant findings, however, when African American and Caucasian men were examined separately, it appears that for Caucasian men, cohabiting after widowhood was associated with DBP in that those who were not cohabiting had increased DBP and those who were cohabiting had lower DBP ($p < .05$). This was the only significant finding for men regarding cohabiting after widowhood.

To examine the impact of race on parental status, the mixed models were run again for African American and Caucasian men separately. In the original analyses,

parental status was significantly related to risk for men in regards to depression only. For African American men, this pattern changed when they were examined separately in that parental status was no longer related to any risk factor. For Caucasian men on the other hand, there was no longer an interaction of parental status, marital status, and time in regards to depression, there was a main effect of marital status ($p < .05$) and a marginal main effect of time ($p = .054$). Additionally, there was a significant parental status by time interaction ($p < .05$) and a main effect of time ($p < .001$) for SBP. Lastly, for Caucasian men there was a 3way interaction of parental status, marital status and time ($p < .01$) and a 2-way interaction of parental status and time ($p < .05$), but no main effect of parental status in regards to HDL.

For women, as well as men, there were several deviations from the original pattern of results regarding marital status when African American and Caucasian women were examined separately. For African American women, the overall pattern of results was similar except for the results regarding LDL and HDL. Specifically, whereas the original analyses revealed an interaction of time and marital status for HDL, there were only main effects of marital status ($p < .01$) and time ($p < .001$) for African American women when examined on their own. Results indicate that married women had lower HDL than both never married and women in a marriage like relationship. Additionally, for LDL, the original main effect of marital status dropped off leaving only the main effect of time ($p < .01$) when African American women were examined separately. For Caucasian women there were several variations in the pattern of results when they were examined on their own. Specifically, marital status was no longer significantly related to SBP or LDL. Time was no longer significantly related to LDL. Lastly, the interaction

between marital status and time was no longer significant as was seen for African American women regarding HDL, and there were both significant main effects of time ($p < .001$) and marital status ($p < .01$) in relation to HDL. Results indicate that married women had higher HDL than both divorced and never married women.

To examine the impact of race for hypotheses 1b, regarding divorce, the sample was again split by race. In the original analyses the main finding was a relationship between SBP and cohabitation. When examined separately, this result was marginally significant ($p = .066$) for African American women only and not apparent in the results for Caucasian women. However, unlike the original analyses, for Caucasian women only, those who were divorced and cohabiting had lower HDL cholesterol than those who were divorced and not cohabiting ($p < .05$). For women who were widowed, the original analyses revealed only a significant relationship between cohabiting and LDL cholesterol for women. However when African American and Caucasian women were examined separately, it appears that this relationship was driven by African American women because it was no longer significant for Caucasian women but remained significant for African American women ($p < .05$). In addition, for African American cohabiting after widowhood was significantly related to depression in that those who were cohabiting had higher depression scores than those who were not ($p < .05$). This was not found for Caucasian women. Lastly, when examined on their own, Caucasian women who were cohabiting reported higher SBP than those who were not ($p < .05$). This was not found for African American women.

To examine the impact of race on parental status, the mixed models for hypothesis 3 were repeated for African American and Caucasian women separately. There were

several variations in the original pattern of results. The original parental status by time interaction for SBP was only significant for Caucasian women ($p < .01$) and not for African American women. Neither was there a main effect of parental status for African American women in regards to SBP. The parental status by time interaction regarding DBP was marginal for African American women ($p = .08$) however no longer significant for Caucasian women. Neither was there a main effect of parental status for either African American or Caucasian women regarding DBP. The 3 way interaction regarding LDL was marginally significant for Caucasian women ($p = .069$), but not significant for African American women. There were no main effects of parental status regarding LDL. The main effect of parental status regarding HDL remained for African American women, and was marginal ($p = .067$) for Caucasian women. Lastly, there remained no findings regarding parental status and depression scores for women when African American and Caucasian women were examined separately.

There was no change in the pattern of results for either men or women for hypothesis 2, that for those reporting divorce status, biological and psychosocial risk factors will initially increase and then decrease by years since divorce when regression analyses were run separately for African American and Caucasian participants.

CHAPTER 4: Discussion

This study investigated the relationship of marital status to various psychosocial and physiologic cardiac risk factors. Further, these relationships were examined over a 15 year time period. The obtained relationships in the present study were not evident for all risk factors and differed for males and females. Since the literature indicates that there are differences in the patterns of risk for males and females and therefore, this discussion will first consider results for males and then separately for females.

EFFECTS IN MEN

It was hypothesized that widowed individuals would show greater risk than divorced individuals, who in turn would show greater risk than single individuals, who in turn would show higher risk than married and cohabiting individuals. In addition, it was hypothesized that for individuals who had been either divorced or widowed, those who were cohabitating would show less risk compared to those who were not cohabitating. Overall, these hypotheses were partially confirmed for men.

Overall, it appears that married and cohabitating men had lower SBP and depression compared to divorced, never married, or widowed men. In accordance with the finding that married men have reduced risk, it appears that cohabitating after divorce is also associated with lower risk for men. The current results support the assertion that for men, cohabitating following divorce is associated with decreased risk in some instances (e.g., lower blood pressure), however not for all risk factors examined (e.g., cholesterol). Mastekaasa (40) discussed evidence for the effect of cohabitation after divorce being associated with reduced well-being. Several authors (e.g., 17, 32) have found different patterns of relationships among risk factors and unhealthy behaviors (e.g.

smoking, eating habits) and cohabitation following divorce as described earlier. The results of the current study provides additional risk factors which show a similar pattern regarding cohabiting after divorce.

Interpreted in terms of the stress buffering hypothesis, men's decreased risk as described above may be due to protective factors of increased social support against stress (18). Measures of social support in the current study were related to marital status at follow up one only for men. Because there were no measures of stress in the CARDIA dataset at these time points, it was not possible to test the role of social support in buffering stress. However, we were able to assess for the direct effects hypothesis regarding social support. The direct effects hypothesis asserts that social support directly benefits individuals regardless of stress levels (11, 10). In accordance with the main effect hypothesis, married men reported the highest emotional social support (feeling that they can be open with and find support from friends and family) and lower risk compared to other marital status categories. However, men who were single and never married reported low social support and low risk as well, which is contradictory to the main effects hypothesis. These results are similar to Dupre et al.'s (16) findings that relationships among marital status and health risk depend on men's age; in the current study the relationships among marital status categories and risk were stronger earlier on in current the study at baseline when men were aged 28-45 and dissipated over time.

Regarding hypothesis 2, that risk would decrease over time since divorce, there was no relationship among time since divorce and any of the risk factors examined at baseline. It may be that the variation in risk factors occur over a shorter time period than was captured by the current data. For instance, it may be that the fluctuations occur for

the most part over the course of the first year since divorce and then average out over time. If this is the case, the time since divorce as measured in years in the current study would not be sensitive enough to detect differences between study participants and represents a limitation to the study.

Adding parental status to the model for men changed the pattern of results very little. For men, parental status was related to DBP but not SBP in that men who had children at baseline appeared to have higher DBP. However, this interaction was only marginally significant and the difference between being a parent or not on DBP dissipated over time (at follow up 1 and 2). In addition, for men, there was no change in the models for either LDL or HDL cholesterol with the addition of parental status. There was an effect of marital status and parenting on depression scores, however. It appears that married and widowed men who have children, report lower depression levels than their marital status counterparts without children. However, for men who are divorced and in a marriage-like relationship, it appears in general that men with children report higher depression levels than their childless counterparts.

Although it makes intuitive sense that having children may reduce depression scores regardless of marital status, perhaps by providing perhaps a sense of meaning, or even simply increased social support, this was not a consistent finding in this study. It may be that the added stress and added complications of having children during and following a divorce may serve to increase certain risk factors. However since men in a marriage-like relationship who had children reported increased depression as well men who were divorced, results are not consistent with this explanation either. Another potentially important issue not assessed in the current study was whether the children of

participants lived with the parent full-time, or were children produced within the current relationship. There are several further inconsistencies in results for effects of marital status and parenting on depression. Specifically, the results were not consistent across physiological risk factors, even those such as blood pressure which consistently appeared related to marital status in the previous analyses. In addition, although the initial 3-way interaction among marital status, parental status and time was significant in the mixed model in regard to depression, the univariate follow-up analyses revealed only one significant relationship, which increases the likelihood that the initial relationship may have been due simply to chance rather than to any strong association among parental status, marital status and time. Finally, it should be noted that the actual differences in the scores on the CES-D among participants was rather small and limited in terms of their clinical significance. In sum, all relationships among marital status, parental status and risk factors varied across risk factors. However, obtained results are complex and difficult to interpret.

Effects in Men African-Americans and Caucasians

Overall, examining race as a covariate in the main analyses indicated that risk in the current sample did in fact vary according to race. The exploratory analyses regarding race revealed that for African American men, marital status and parental status is not associated with heart health risk outcomes. In contrast, for Caucasian men, several risk factors are related to both marital status and parental status including relationships found in the original analyses as well as relationships that were not evident when African American and Caucasian men were examined together. In regards to cohabiting after divorce, it appears the findings of the original analyses were driven by African American

men in that there was no longer a significant relationship for Caucasian men when examined separately. The opposite was true for cohabiting following widowhood where findings were significant only for Caucasian men.

EFFECTS IN WOMEN

It was hypothesized that widowed individuals would show greater risk than divorced individuals, who in turn would show greater risk than single individuals, who would show higher risk than married and cohabiting individuals. In addition, it was hypothesized that for individuals who had been either divorced or widowed, those who were cohabiting would show less risk compared to those who were not cohabiting. Overall, these hypotheses were partially confirmed for women. The findings in the current study for women are contrary to the hypothesized relationships as well as previous research which places married individuals at decreased risk compared to other marital status categories (e.g., 43).

In the current study, the patterns of social support reported by women in varying marital status categories did not consistently correspond with expectations regarding risk in terms of the main effects or stress buffering hypothesis. Rather than decreasing stress, as proposed by the stress buffering hypothesis, it may be that women's stress increases in certain marital status roles and thereby increases risk. Unfortunately, this could not be examined in this study. However, it may also be that women are more likely to modify healthy eating and physical activity habits due to relationship factors (e.g., more likely to eat out) which may also influence the results found here. In a similar manner, the decreased risk evidenced in women who were not cohabiting following divorce may be a result of similar factors. However, given the small number of significant results for

effect of cohabiting after divorce or widowhood, it is possible that the single finding for each are due to type one error, and should be interpreted cautiously. Time since divorce was also not related to risk for women and this may be due to similar reasons as described above for men.

Overall, women's risk increased when they had children. However the relationships of having children to risk factors did not differ across specific marital status categories as hypothesized. The exception is LDL cholesterol, which demonstrated a significant marital status by parental status by time interaction. The general patterns for this finding were in accordance with the hypothesis that having children will increase risk (LDL cholesterol) across marital statuses. The findings in the current study echo prior literature finding that cholesterol, and LDL specifically increases with pregnancy (38). However, the literature also indicates that LDL levels return to pre-pregnancy levels within one year of giving birth. The current study did not control for or examine time since pregnancy to be able to make a comparison to prior research in this regard. However, as mentioned above, the univariate follow up analyses revealed nonsignificant results, casting doubt on the reliability of the initial finding.

Adding parental status to the model modified several other findings for women but, in general, did not support the hypothesis that parental status would increase risk across all the marital status categories. For blood pressure, there were time by parental status interactions for both SBP and DBP. Specifically, women's blood pressure increased over time and for women who had children, increased more over time compared to women who did not have children. For cholesterol, women who had children had lower HDL compared to women who did not which is in accordance with

prior literature documenting decreasing HDL levels with each successive pregnancy (38). For LDL the interaction among marital status, parental status and time yielded results which should be interpreted with caution as described above. For depression, marital status and parental status were not related to depression scores in women. Although the results of the current study indicated that having children was associated with increased heart health risk factors for women, the hypothesis that parenting would interact with marital status was not supported for women.

Effects in Women for African-Americans and Caucasians

Overall, examining race as a covariate in the main analyses indicated that risk in the current sample did in fact vary according to race. The exploratory analyses regarding race revealed that for women, marital status and parental status were often associated with cholesterol fractions for African American women whereas marital status and parental status were more often associated with blood pressure for Caucasian women. Previous research has asserted that African American women may be at increased risk compared to Caucasian women other individuals ethnic groups due to more substantial weight gain following marriage (29). The exploratory analyses in the current study did not directly compare African American and Caucasian women. However, when these groups were examined separately, it appeared that their patterns of risk depended on the risk factor examined. Specifically, African American women have increased cholesterol, and Caucasian women in this sample appear to have increased blood pressure.

CLINICAL IMPLICATIONS

Cardiovascular diseases such as coronary artery disease are progressive and deadly diseases. There are many risk factors that influence the development and

progression of these disease and understanding the correlates involved can help inform risk categorization and subsequent intervention. Prior research has attempted to create a risk stratification model in order to assess for follow up treatment and reduce re-hospitalizations in patients with heart diseases, understanding social support networks such as family structures may prove useful to these models (22, 59). In addition to risk stratification, understanding how family structure and marital status are related to risk may be a first step in targeting psychosocial interventions. While it may be helpful to implement more in depth interventions for a variety of family structure's, it may also be useful in continuing to investigate what exactly it is about these family structure categories that is responsible for increased or decreased risk and then those mechanisms could be targeted for intervention. For instance, psychosocial interventions targeting married women's healthy eating, stress, or adjustment to relationship changes may be beneficial for their heart health while interventions targeting these in men who are divorced or widowed and not cohabiting may be beneficial for their heart health. Interventions such as these will necessitate moving to a more person centered care model with multiple components to treatment team and plan. Although the current project may be a first step towards contributing to these clinical goals, there are several limitations to the current work which should be addressed in future studies to work on understanding what about these marital status and family structure categories confer increased risk.

LIMITATIONS

The current study has several limitations which should be noted. As a general limitation, this study used self report measures for psychosocial variables which are less reliable than other types of measurement (14). This is an issue which is common to

research of this type. A more pressing limitation is that many of the psychosocial variables (e.g., as social support and job strain) which could have been used to explain the present results were measured only at one or two time points and therefore could not be included in the data analyses. Although the analyses presented in this study do reveal several interesting patterns, it is unclear by what mechanism or mechanisms these patterns occur because measures of the possible mechanisms were not present at all time points. In addition, the model utilized in this study did not allow for considering whether it was current marital status or the pattern of transitions over time which influences risk, and could not infer causality. Another limitation is that this study used many statistical comparisons, which presents the risk of findings being due to type I error. Specifically there were 5 dependent variables used in 2 main models resulting in a total of 10 models. However, in each of these models Bonferroni correction was used reducing the risk of type I error. In addition, the fact that multiple findings in each analyses were significant (e.g., main effects as well as interactions) reduces concern for type I error for some, but not all of the variables examined. Lastly, the a priori power analysis indicates that the study had sufficient power for the analyses used.

The timeline of the current study in which data was collected at 5 year intervals may also represent a significant limitation. Specifically, as mentioned in the discussion, it may be that a smaller time frame (e.g., months instead of years) may be needed to discern patterns in regard to marital status transitions and heart health risk patterns. Including multiple follow ups at regular intervals and increased frequency may yield more substantial findings in regard to time since divorce and risk patterns over time. Overall, time is important in considering the progression of risk for heart diseases. However, the

inclusion of time as a factor in the mixed model analyses without specific mention of time in many of the hypotheses may have affected and complicated the results. In addition, sex was not included in the models used to test the study hypotheses. However, as described earlier, analyses indicated that there would be interactions with sex and these interactions may have significantly decreased the interpretability of results. Additionally, given the concerns about type I error, adding another independent variable into the models instead of splitting the sample by that variable (e.g., sex) may have increased the possibility of type I error by increasing the comparisons. Overall, it appears that splitting the sample by sex rather than including sex as a variable in the models was the most appropriate method for analyzing the data. Lastly, this study included only African American and Caucasian participants and excluded other racial profiles during the recruitment process. This limits the generalizability of results. However it also allows for examination of African American men and women in comparison to Caucasian men and women. Several of the limitations above present important directions for future research to follow in order to further understand correlates of heart health risk.

CONCLUSION AND FUTURE DIRECTIONS

Heart diseases are one of the leading causes of death in the United States and the progressive nature of heart diseases adds to medical costs. There are several physiologic and psychosocial risk factors associated with the development and progression of these heart health diseases and understanding their correlates in young and middle aged adults may improve the care provided these individuals as well as prevent the onset of the disease. The current study examined several possible correlates of heart health risk factors including marital status, parental status and cohabitation following divorce or

widowhood. The results of this study indicate that structural aspects of individuals social support network, and specifically, the family structure, are in fact related to the level of risk factors such as blood pressure, cholesterol, and depression. This current study identified several relationships among these variables. However, further research which addresses the limitations cited above as well as attempts to create a deeper understanding of the mechanisms guiding those relationships is warranted. Such research may prove useful in aiding health care providers at assessing risk profiles for individuals, implementing preventative measures to address risk reduction, and also design and implement interventions to reduce continued development of risk and disease.

REFERENCES

1. American Heart Association 2011. Heart disease and stroke statistics-2011 update: A report from the American heart association. *J. of the Am. Heart Asso.*
2. American Heart Association 2012. Heart disease and stroke statistics-2012 update: A report from the American heart association. *J. of the Am. Heart Asso.*
3. American Psychiatric Association 2013. The Diagnostic and Statistical Manual of Mental Disorders 5. American Psychiatric Association Press
4. Bennett SJ, Huster GA, Baker SL, Milgrom LB, Kirchgassner A, et al. 1998. Characterization of the precipitants of hospitalization for heart failure decompensation. *Am. J. of Crit. Care* 7: 168-74
5. Berkman LF & Syme SL. 1979. Social networks, host resistance, and mortality: A nine-year follow-up study of Alameda county residents. *Am. J. of Epid.* 109: 186-204
6. Brummett BH, Babyak MA, Mark DB, Clapp-Channing NE, Siegler IC, & Barefoot JC. 2004. Prospective study of perceived stress in cardiac patients. *An. of Beh. Med.: a Pub. of the Soc. of Beh. Med.* 27: 22-30
7. Brummett BH, Barefoot JC, Siegler IC, Clapp-Channing NE, Lytle BL et al. 2001. Characteristics of socially isolated patients with coronary artery disease who are at elevated risk for mortality. *Psychos. Med.* 63: 267-72
8. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, et al. 2003. Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. *Hyper.* 42: 1206-52.
9. Cohen S, Schwartz JE, Epel E, Kirschbaum C, Sidney S, et al. 2006 Sociodemographic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Psycho. Med.* 68: 41-50
10. Cohen S, Gottlieb BH, & Underwood LG. 2000. Social relationships and health. In *Social Support Measurement and Intervention: A guide for health and social scientists*, ed. S Cohen, LG Underwood, BH Gottlieb, pp. 3 – 25. NY: Oxford University Press
11. Cohen S. & Wills TA. 1985. Stress, social support, and the buffering hypothesis. *Psych. Bul. Am. Psych. Asso.* 98: 310 -57
12. Czajkowski SM, Arteaga SS. 2012. Social Support and Coronary Heart Disease. In *Heart and Mind*, ed. R Allan, & J Fisher, pp.17-53. Washington, DC: APA

13. DeKlyen M, Brooks-Gunn J, McLanahan S, Knab J. 2006. The mental health of married, cohabitating, and non-coresident parents with infants. *Am. J. Pub. He.* 96: 1836-1841
14. Dawes RM, Faust D, & Meehl PE. 1989. Clinical versus Actuarial Judgment. *Sc.* 243, 1668-1673
15. Dunbar SB, Clark PC, Quinn C, Gary RA, & Kaslow NJ. 2008. Family influences in heart failure self-care and outcomes. *J. of Card. Nur.* 23: 258-65
16. Dupre ME, Beck AN, & Meadows SO. 2009. Marital trajectories and mortality among US adults. *Am. J. of Epid.* 170: 546-55
17. Eng PM, Kawachi I, Fitzmaurice G, & Rimm EB. 2005. Effects of marital transitions on changes in dietary and other health behaviors in US male health professionals. *J. of Epid. Com. Heal.* 59: 56-62
18. Feldman P, & Cohen S. 2000. Social support. In *Encyclopedia of Psychology*, ed. AE Kazdin. New York: Oxford University Press
19. Fisher L, & Weihs KL 2000. Can addressing family relationships improve outcomes in chronic disease? *The J. of Fam. Prac.* 49
20. Friedmann E, Thomas SA, Liu F, Morton PG, Chapa D, et al. 2006. Sudden Cardiac Death in Heart Failure Trial Investigators. Relationship of depression, anxiety, and social isolation to chronic heart failure outpatient mortality. *Am. Hear. J.* 152: 940.e1-940.e8
21. Gallo LC, Troxel WM, Matthews KA, & Kuller LH. 2003. Marital status and quality in middle-aged women: Associations with levels and trajectories of cardiovascular risk factors. *Heal. Psych.* 22: 453-63
22. Giamouzis G, Kalogeropoulos A, Georgiopoulou V, Laskar S, Smith AL, et al. 2011. Hospitalization epidemic in patients with heart failure: Risk factors, risk prediction, knowledge gaps, and future directions. *J. of Card. Fail.* 171: 54-75
23. Gunderson EP, Chiang V, Lewis CE, Catow J, Quesenberry CP, et al. 2008. Long-term blood pressure changes measured from before to after pregnancy relative to nonporous women. *The Am. Col. of Obst. & Gyn.* 112: 1294-1302
24. Hewitt B, Baxter J, & Western M. 2006. Family, work and health: The impact of marriage, parenthood, and employment on self-reported health of Australian men and women. *J. of Soc.* 42
25. Holt-Lunstad J, Birmingham W, Howard A, & Thoman D. 2009. Married with children: The influence of parental status and gender on ambulatory blood pressure. *Ann. of Beh. Med.* 38: 170-79

26. Heo S, Lennie TA, Moser DK, & Okoli C. 2009. Heart failure patients' perceptions on nutrition and dietary adherence. *Eur. J. of Card. Nurs.* 8: 323-28
27. Karasek R, Baker D, Marxer F, Ahlbom A, & Theorell T. 1981. Job decision latitude, job demands, and cardiovascular disease: A prospective study of Swedish men. *Am. J. of Pub. Heal.* 71: 694-705
28. Klatsky AL. 2010. Alcohol and cardiovascular health. *Phys. and Beh.* 100: 76-81
29. Koball HL, Moiduddin E, Henderson J, Goesling B, & Besculides M. 2010. What do we know about the link between marriage and health? *J. of Fam. Iss.* 31: 1019-40
30. Kop WJ, & Plumhoff JE. 2012. Depression and Coronary Heart Disease: Diagnosis, Predictive Value, Biobehavioral Mechanisms, and Intervention. In *Heart and Mind*, ed. R Allan, J Fisher, pp. 17-53. Washington, DC: APA
31. Krantz DS, Whittaker KS, & Sheps DS. 2012. Psychosocial Risk Factors for Coronary Heart Disease: Pathophysiologic Mechanisms. In *Heart and Mind*, ed. R Allan, J Fisher, pp. 17-53. Washington, DC: APA
32. Lee S, Cho E, Grodstein F, Kawachi I, Hu FB, & Colditz GA. 2004. Effects of marital transitions on changes in dietary and other health behaviors on US women. *Int. J. of Epid.* 34: 69-78
33. Lett HS, Blumenthal JA, Babyak MA, Strauman TJ, Robins C, & Sherwood A. 2005. Social support and coronary heart disease: epidemiologic evidence and implications for treatment. *Psych. Med.* 67: 869-78
34. Lehman BJ, Taylor SE, Kiefe CI, & Seeman TE. 2009. Relationship of early life stress and psychological functioning to blood pressure in the CARDIA study. *Hea. Psych.* 28: 338 – 46
35. Levy D, Larson MG, Vasan RS, Kannel WB, & Ho KK. 1996. The progression from hypertension to congestive heart failure. *JAMA*, 275: 1557 -62
36. Luke A, Dugas LR, Durazo-Arvizu RA, Cao G, & Cooper RS. 2011. Assessing physical activity and its relationship to cardiovascular risk factors: NHANES 2003-2006. *BMC Publ. Heal.* 11
37. Lund R, Hulvej N, & Christensen U. 2010. Are negative aspects of social relations predictive of angina pectoris? a 6-year follow-up study of middle-aged Danish women and men. *J. of Epid. and Com. Heal.*
38. Mankuta D, Elami-Suzin M, Elhayani A, & Vinker S. 2010. Lipid profile in consecutive pregnancies. *Lip. in Heal. and Dis.* 9: 9-58.

39. Maselko J, Bates LM, Avendano M, & Glymour M. 2009. The intersection of sex, marital status, and cardiovascular risk factors in shaping stroke incidence: Results from the health and retirement study. *Am. Ger. Soc.* 57: 2293-9.
40. Mastekaasa A. 1994. The subjective well-being of the previously married: The importance of unmarried cohabitation and time since widowhood or divorce. *Soc. For.* 73: 665-92
41. Meadows SO. 2009. Family structure and fathers' well-being: Trajectories of mental health and self-rated health. *J. of Heal. and Soc. Beh.* 50: 115-131
42. Meadows SO, McLanahan SS, Brooks-Gunn J. 2008. Stability and change in family structure and maternal health trajectories. *Am. Soc. Rev.* 73: 314-34
43. Molloy JG, Stamatakis E, Randall G, & Hamer M. 2009. Marital status, gender and cardiovascular mortality: behavioural, psychological distress and metabolic explanations. *Soc. Sc. & Med.* 69: 223-228
44. Muhammad A, & Gagnon A. 2010. Why should men and women marry and have children? parenthood, marital status and self-perceived stress among Canadians. *J. of Heal. Psych.* 15: 315-25
45. Musselman DL, Evans DL, & Nemeroff CB. 1998. The relationship of depression to cardiovascular disease: Epidemiology, biology, and treatment. *Arch. of Gen. Psych.* 55: 580-92
46. Packard RR, & Libby P. 2008. Inflammation in atherosclerosis: from vascular biology to biomarker discovery and risk prediction. *Clin. Chem.* 54: 24-38
47. Parker ED, Schmitz KH, Jacobs DR, Dengal DR, Schreiner PJ. 2007. Physical activity in young adults and incident hypertension over 15 years of follow-up: The CARDIA study. *Am. J. of Pub. Heal.* 97: 703-9
48. Radloff LS. 1977. The CES-D scale: A self-report depression scales for research in the general population. *App. Psych. Meas.* 1: 385-401
49. Rauchhaus M, Clar AL, Doehner W, Davos C, Bolger, et al. 2003. The relationship between cholesterol and survival in patients with chronic heart failure. *J. of the Am. Coll. of Card.* 42: 1933-40
50. Scherbaum CA, & Ferreter, JM. 2009. Estimating statistical power and required sample sizes for organizational research using multilevel modeling. *Org. Res. Meth.* 12: 347-67
51. Simons LA, Simons J, Friedlander Y, & McCallum J. 2012. Childbearing history and late-life mortality: the Dubbo study of Australian elderly. *Ag. and Ag.* 1-6

52. Smith TW, & Traupman EK. 2012. Anger hostility and aggressiveness in coronary heart disease: Clinical applications of an interpersonal perspective. In *Heart and Mind*, ed. R Allan, J Fisher, pp. 17-53. Washington, DC: APA
53. Smith TW, Uchino BN, Florsheim P, Berg CA, Butner J, et al. 2011. Affiliation and control during marital disagreement, history of divorce, and asymptomatic coronary artery calcification in older couples. *Psych. Med.* 73: 350-7
54. Stewart JC, Fitzgerald GJ, & Kamarck TW. 2010. Hostility now, depression later? longitudinal associations among emotional risk factors for coronary artery disease. *Ann. of Beh. Med.: a pub. of the Soc. of Beh. Med.* 39: 258-66
55. Vedhara K, Shanks N, Anderson S, & Lightman S. 2000. The role of stressors and psychosocial variables in the stress process: a study of chronic caregiver stress. *Psych. Med.* 62: 374-85
56. Venters M, Jacobs DR, Pirie P, Luepker RV, Folsom AR, et al. 1986. Marital status and cardiovascular risk: The Minnesota Heart Survey and the Minnesota Heart Health Program. *Prev. Med.* 15: 591-605
57. Wang HXX, Leineweber C, Kirkeeide R, Svane R, Schenck-Gustafsson K, et al. 2007. Psychosocial stress and atherosclerosis: family and work stress accelerate progression of coronary disease in women. the Stockholm female coronary angiography study. *J. of Int. Med.* 261
58. Wilson PWF, D'Agostino RB, Levy D, Belanger AM, Silbershatz H. 1998. Prediction of coronary heart disease using risk factor categories. *J. of the Am. Hear. Ass.* 97: 1837- 47
59. Zaya M, Phan A, & Schwarc ER. 2012. Predictors of re-hospitalization in patients with chronic heart failure. *Wor. J. of Card.*, 4: 23-30