LEAN ON ME: SOCIAL SUPPPORT COMPENSATION AND RISK OF DEATH IN OLDER ADULTS WITH TYPE 2 DIABETES

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Type 2 diabetes (T2DM) has an estimated incidence of nearly 11 million US adults aged 65 years and older. Evidence suggests that the quality of the marital relationship is an important factor for diabetes related health outcomes affecting self-management and adherence (Kiecolt-Glaser & Newton, 2001). However, an individual in need may compensate for primary support that is unavailable or not optimal by looking for other sources of support, which may be important for health outcomes (Rini, et al., 2008). The present study examined compensation for poor spousal support through other social relationships. A total of 12,640 participants reported they had diabetes and were married (male = 6,317 and female = 6,323), and of this group 1,084 men and 583 women had died over the course of the study period. Women reported lower spousal support, but significantly more aggregated social support across relationships than men. Few persons reported low spousal support and low support compensation, rendering the cell sizes highly unequal and the associated data uninterpretable. Ancillary analyses were conducted with the idea that some variance in total compensation support may moderate mortality risk finding that higher aggregated social support across non-spousal relationships was associated with lower risk of death accounting for ~3% of the variance in the final model. The current findings demonstrate how an individual can compensate for a poor primary support relationship through a broader support network. These findings should guide future research to focus on how individuals build, maintain, and seek support from social relationships.

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INTRODUCTION

Diabetes is the 7th leading cause of death in the United States and is linked to approximately 200,000 deaths per year (American Diabetes Association [ADA], 2011). Type 2 diabetes (T2DM) is the most prevalent form of the disease, with an estimated incidence of nearly 11 million US adults aged 65 years and older. The disease is associated with serious clinical problems including neuropathy and renal complications and is the leading cause of end-stage renal disease (Cowie et al., 1989; Maser et al., 1989). Moreover, T2DM is a recognized risk factor for hypertension, coronary heart disease, myocardial infarction, stroke, and early mortality (ADA; Merjanian, Budoff, Adfler, Burman, & Mehrotra, 2003). Although modifiable and non-modifiable risk factors for T2DM have been found, a significant amount of risk remains unaccounted for.

Psychosocial factors are increasingly supported as modifiers of diabetic risk (Griffith, Field, & Lustman, 1990; Hagedoorn et al., 2006; van Dam et al., 2004). The salient effect of psychosocial factors (e.g., emotional states, chronic stressors, social ties, social support, and social conflict) is well documented to influence the health and wellbeing of individuals (Berkman & Syme, 1979; Cassel, 1974; Cobb, 1976). Systematic reviews of the literature suggest high levels of social support to be an important determinant in improving diabetic health outcomes and reducing complications (Griffith et al.; House, Landis, & Umberson, 1988; Schlenk & Hart, 1984; van Dam et al.). Several pathways are identified through which social support may reduce diabetic complications such as reducing the experience or impact of life-stressors by buffering the individual, or providing a direct effect on health irrespective of whether an individual is under stress (Cohen & Willis, 1985). Research indicates diabetes self-

management and medical adherence are negatively associated with stress (DiMatteo, 2004). Social support is hypothesized to affect health outcomes by reducing the experience of stress, thereby improving diabetes-relevant health behaviors (Griffith et al.; Schlenk & Hart). The social ties an individual maintains throughout their life have important impacts on health and are primarily manifested through the support one receives.

Individuals with spouses, friends, and family members who provide psychological and material resources are in better health than those with fewer supportive contacts (Kawachi & Berkman, 2001; Uchino, Cacioppo, & Kiecolt-Glaser, 2006). Cobb (1976) defined social support as information that would lead the person to believe they were "cared for and loved, esteemed, and he belongs to a network of communication and mutual obligations" (p. 300). However, different types of social relationships provide various aspects of support or social provisions (Weiss, 1998). Social support is often explored in the context of specific interpersonal relationships. Research indicates that a particular support characteristic can be obtained from more than one relationship type in the form of reliance on other social ties (e.g. adult children, friends, and family). In the absence of suitable support from one group, compensation of other supports may be utilized to contribute to psychological well being (Rook & Schuster, 1996; Rook, Sorkin, & Zettel, 2004). The current study investigates social network compensation in a sample of older men and women with T2DM from a longitudinal perspective and measures social support garnered from spouse, adult children, family, and friends. We hypothesize those individuals with T2DM who use more social network compensation (i.e. compensate for inadequate or unavailable relationships as needed) will have better health and as a result lower incidence of death.

Type 2 Diabetes

Diabetes mellitus is a group of diseases marked by high levels of blood glucose (i.e., hyperglycemia) resulting in deficits of insulin production, insulin action, or both affecting approximately 25 million people in the U.S (ADA, 2011; International Diabetes Federation [IDF], 2009). The chronic hyperglycemiacharacteristic of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, and blood vessels. Many moderators have been identified to combat the deleterious effects of chronic hyperglycemia and help individuals control their diabetes for better outcomes.

Pathophysiology of diabetes. The failure to maintain glucose homeostasis through insulin action results in diabetes. Insulin is a hormone that regulates carbohydrate and fat metabolism in the body. Insulin initiates cells in the liver, muscle, and fat tissue uptake and storage of glucose from the blood for energy. It is produced in the pancreas by β-cells and is released when protein and glucose are detected in the blood. As glucose levels decrease in the blood, insulin release from the β-cells slow or stops (Olefsky & Courtney, 2005). Beta cell dysfunction is responsible for the onset and severity of many systemic disease indicators including, impaired glucose tolerance, hyperglycemia, and insulin resistance in muscle and adipose cells (Thorens, Weir, Leahy, Lodish, & Bonner-Weir, 1990). Poor insulin action by the body leads to insulin resistance, a chronic problem related to poorer health outcomes (Krinsley, 2003).

Insulin resistance is a metabolic hallmark of T2DM, and is characterized by poor insulin regulation by the β-cells of the pancreas, which leads to an inadequate compensatory insulin secretory response (ADA, 2011). Major pathogenic processes are identified in the development

of T2DM. These abnormalities are primarily in carbohydrate, fat, and protein metabolism and result in a deficient action of insulin on target tissues. Research of pancreatic β-cells indicates a combination of molecular events between levels of free fatty acids and the high-fat diet associated with obesity that create a tipping point to effect β-cell glucose sensing and impaired glucose tolerance (Ohtsubo, Chen, Olefsky, & Marth, 2011). Therefore, inadequate insulin secretion and/or diminished tissue responses to insulin occur at one or more points in the hormone response pathway. As a result, this leads to higher levels of circulating glucose in the blood. Individuals with impaired fasting glucose and impaired glucose tolerance are deemed to have prediabetes (ADA).

Complications of type 2 diabetes. Individuals with prediabetes often encounter issues with glucose regulation in the form of hypoglycemia and hyperglycemia. With time, the pancreas fails to keep up with the insulin demand and may stop producing insulin resulting in chronic problems with glucose regulation in the form of hypoglycemia and hyperglycemia. Hypoglycemia is a state produced by lower than normal blood glucose with principal problems resulting from lowered glucose to the brain (Cryer et al., 2009). In contrast, hyperglycemia is a condition in which an excessive amount of glucose circulates in the blood stream causing frequent urination, excessive thirst, weight loss, and blurred vision. This results in acute, life-threatening consequences of diabetes such as hyperglycemia with ketoacidosis or nonketotic hyperosmolar syndrome (ADA, 2011). Ketoacidosis develops when an individual does not have enough insulin, whereas nonketotic hyperosomolar syndrome occurs due to extremely high blood sugar levels. These conditions cause serious health complications that may lead to dehydration or coma. Additionally, chronic hyperglycemia can produce a wide array of serious

complications including kidney, neurological, and cardiovascular damage, as well as an increased link to mortality (Krinsley, 2003). Impairment of insulin secretion and defects in insulin action frequently coexist and it is often unclear as to the primary cause of hyperglycemia (National Diabetes Data Group [NDDG], 1979).

Individuals with uncontrolled diabetes (i.e., chronic hyperglycemia) are at risk for developing serious health complications including, cardiovascular, renal and retinal diseases. Although the numbers are increasing for adolescents and young adults, it is especially problematic for those individuals aged 65 and older (ADA, 2011; Fagot-Campagna et al., 2000). For example, adults with diabetes have heart disease death rates about 2 to 4 times higher than adults without diabetes (ADA). Individuals with diabetes have a higher rate of hypertension (Benhalima et al., 2011), kidney disease (Adler et al., 2003), and nervous system damage (Davies, Brophy, Williams, & Taylor, 2006), and among those aged 20-79 years half of the deaths attributed to diabetes are estimated to be due to cardiovascular disease (IDF, 2009). Poor health outcomes are ascribed to many mechanisms and are often interrelated, but are best understood in terms of modifiable and non-modifiable moderators of disease progression.

Moderators of Type 2 diabetes.

Non-modifiable influences. Common non-modifiable risk factors for developing T2DM include age, race and ethnicity, gender, and family history (Alberti, Zimmet, & Shaw, 2007; Kao et al., 2005). For example, Kaprio and colleagues (1992) found a higher incidence of T2DM among monozygotic twins than dizygotic twins suggesting a strong heritability factor for the disease. Among individuals with type 2 diabetes, roughly half of their disease risk is attributed

to modifiable factorswith the other half related to non-modifiable factors (Riserus, Willett, & Hu, 2009).

Similar to other chronic conditions, T2DM is unequally distributed across racial/ethnic groups (Centers for Disease Control & Prevention [CDC], 2008). For instance, T2DM is more common in African Americans, Hispanics, Native Americans, and Asian Americans. Compared to non-Hispanic white adults, the risk of diagnosed diabetes is 18% higher among Asian Americans, 66% higher among Hispanics, and 77% higher among non-Hispanic blacks. African Americans, Hispanic/Latino Americans, American Indians, and some Asian Americans are at a higher risk for type 2 diabetes and associated complications (IDF, 2009; Harris, Eastman, Cowie, Flegal, & Eberhardt, 1999). Additionally, Latinos and African Americans with all types of diabetes have poorer glycemic control than their white counterparts as well as have lower medication adherence (Heisler et al., 2007). Thus, racial/ethnic group membership moderates risk for T2DM and associated health complications.

Age, gender, and family history are three factors that are often inter-related in the development of T2DM (Kao et al., 2005; Kaprio et al., 1992). Children with a family history of T2DM are at an increased risk for impaired glucose tolerance especially if exposed to gestational diabetes mellitus (Goran et al., 2004). This may be associated with obesity, an independent risk factor for T2DM, which is a strongly inherited trait (Walley, Blakemore, & Froguel, 2006). Furthermore, these risk factors are influenced by age where men and women have the same cumulative incidence of T2DM until age 60 when the incidence of developing the disease is higher for men (Kaprio et al.). Many non-modifiable risk factors are intertwined

to lead to health outcomes; however, understanding their importance in conjunction with psychosocial factors of health outcomes is especially important for lifestyle modifications.

Modifiable influences. Modifiable risk factors including overweight or obesity, high blood glucose, hypertension, physical inactivity and smoking are implicated in the development of T2DM (Alberti et al., 2007; Turner et al., 1998). For example, several prospective cohort studies suggest that smoking is associated with the development of diabetes. After controlling for other factors, the risk of T2DM among women who were heavy smokers was 1.42 (Rimm, Manson, & Stampfer, 1993) with similar results found for men (Rimm, Chan, Stampfer, Colditz, & Willett, 1995) suggesting an association between smoking and development of T2DM. These risk factors are termed "modifiable" as they can be prevented and/or mitigated through self-management and lifestyle changes, which traditionally include diet and exercise (Eriksson & Lindgarde, 1991; Tuomilehto et al., 2001). Diet is considered a possible cause of T2DM, and studies concentrate on total caloric intake as well as several components of diet including carbohydrates and fats (Lundgren, Bengtsson, Blohme, Isaksson, & Lapidus et al., 1989; Toeller, 1993). In addition, many of the modifiable risk factors are amenable to exercise. Physical activity has been identified as a major determinant of T2DM, and increased activity is shown to improve insulin sensitivity and glycemic control among those with impaired glucose tolerance as well as a reduced incidence of T2DM (Helmrich, Ragland, Leung, & Paffenbarger, 1991; Ivy, Zderic, & Fogt, 1999). For example, Helmrich and colleagues (1991) examined longitudinally the effect of physical activity on incidence of T2DM in a large community sample of middle-aged men and found that total expenditure of energy during leisure time had a protective

effect against the development of T2DM. Negative health outcomes and the development of T2DM are primarily avoided through self-management and education.

Diabetes self-management is a clustered set of behavior modifications aimed at managing diabetes severity and retarding disease progression. These goals are achieved through improved glycemic control (for review see Skylar, 2004). Glycemic control is traditionally measured through glycated hemoglobin (HbA1c), which is a blood test that provides an average of blood sugar control for the past two to three months, where lower levels indicate better control (IDF, 2009). Diabetes self-management classes focus on improving daily behavioral management through healthy eating, physical activity, blood glucose monitoring, medication adherence, and adaptive coping through an educational format. A main objective of Healthy People 2010 was to increase the proportion of individuals with diabetes who received formal disease training from 40% to 60% (United States Department of Health and Human Services [HHS], 2000) due to significant knowledge and skill deficits in more than half of individuals with diabetes (Clement, 1995). Research demonstrates an inverse relationship between diabetes self-management and glycemic control (Fortmann, Gallo, & Philis-Tsimikas, 2011), providing evidence that education on how to manage diabetes is necessary to achieve better health outcomes.

Success of diabetes management is judged by ability to adhere to the therapeutic regimen and education is a key aspect of this success. Diabetes self-management education (DSME) is the process of facilitating the knowledge, skill, and ability necessary for diabetes self-care (Funnell et al., 2009). Overall objectives of DSME include informed decision-making, self-care behaviors, problem solving, and collaboration with the health care team, which lead to

improved clinical outcomes and quality of life. For example, a meta-analysis by Ellis and colleagues (2004) indicated face-to-face delivery, cognitive reframing, and exercise content were statistically and clinically significant predictors of improved glycemic control in DSME interventions. This education is intended to lead to the ability to safely care for chronic disease on a daily basis. Research suggests DSME is effective for improving metabolic as well as psychosocial outcomes (Norris, Engelgau, & Naranyan, 2001). However, attrition is a common issue that is encountered in these programs (Griffin, 1998). Gucciardi and colleagues (2008) found both personal and contextual factors contributed to attrition in DSME programs. These included availability of support, familiarity of resources, self-efficacy, attitudes, and perceived disease severity. Therefore, reducing attrition behavior requires an integrative approach of support, education, and perceived control.

Psychosocial moderators of type 2 diabetes. In addition to traditional risk factors, psychosocial factors are increasingly recognized as moderators of the course of diabetes (Steed, Cooke, & Newman, 2002). These psychosocial factors can be characterized as qualities or characteristics of the individual including mood as well as more stable factors such as personality and aspects of the social environment including social resources, relationships, and the environment.

Individual level factors. Several studies have examined the impact of general psychological well-being, mood, and coping strategies in the context of psychological stress on intervention adherence and health outcomes. For example, in a study of persons with Type 1 diabetes Tankova and colleagues (2001) found significant improvements on both negative and positive aspects of well-being following a self-management intervention. Additionally, state-

positive affect is associated with lower risk of all-cause mortality in people with diabetes (Moskowitz, Epel, & Acree, 2008). However, mood can also lead to more adverse diabetic health outcomes. For example, mental illness is associated with a higher incidence of impaired glucose metabolism and insulin resistance (Eaton, Aremenian, Gallo, Pratt, & Ford, 1996; Ryan, Collins, & Thakore, 2003). T2DM individuals often exhibit a higher prevalence of clinically significant depressive symptomatology when compared to non-diabetic controls (Garvard, Lustman, & Clouse, 1993). Several studies have shown that higher rates of depression, anxiety, and psychological distress may lead to or coincide with a diagnosis of diabetes (Ali, Stone, Peters, Davies, & Khunti, 2006; Black, Markides, & Ray, 2003; Hamer, Stamatakis, Kivimaki, Kengne, & Batty, 2010; Mezuk, Eaton, Albrecht, & Golden, 2008; Thomas, Jones, Scarinci, & Brantley, 2003), and these may contribute to increased mortality risk (Katon et al., 2005). Although mood can be of help or hindrance in dealing with psychological distress, individuals with diabetes cannot eliminate the stress of living with a chronic disease and coping strategies are of primary importance when confronted with acute stress. Coping styles presumably influence glycemic control by increasing or decreasing stress, which may affect HbA1c levels through adherence to behavioral changes (Peyrot, McMurry, & Kruger, 1999). In a longitudinal study of older women, positive affect and problem focused coping was associated with better control over time and was predictive of lower rates of T2DM (Tsenkova, Love, Singer, &Ryff, 2008). The nature of mood and coping styles in diabetics is very intricate and may lead to, coincide with, or contribute to adverse health outcomes; however other individual and social factors including personality and the social environment are likely contributors to the occurrence of diabetes.

In addition to mood disturbances, personality factors influence the course of diabetes including how the T2DM individual copes with their disease and the subsequent demands of self-management. Individuals identified as opportunistic, alienated, and with explosive temperaments have been found to have poorer glucose control (Lustman, Frank, & McGill, 1991). Moreover, those who are less agreeable/cooperative often perceive a new diagnosis of diabetes as threatening, which can lead to increased stress and poorer adherence (Lawson, Bundy, & Harvey, 2007). In addition to more negative personality factors, those considered as positive can lead to better outcomes. Conscientious individuals are more likely to engage in physical activity, an important modifier of poor health outcomes for those with diabetes (Davies, Mummery, & Steele, 2008). Hence, personality can serve as both a risk or resilience moderator of diabetes.

Social/environmental factors. Aspects of the environment, particularly the social environment, are known to influence the level of risk and course of diabetes. Elements of the built environment including available food choices, exercise opportunities, neighborhood safety, and quality medical care all influence diabetes course and available coping options (Barrera, Toobert, Angell, Glasgow, & MacKinnon, 2006). In addition, a constellation of social risk factors including environmental stress, SES, occupational stress, and social relationships can further influence the disease (Hussain, Claussen, Ramachandran, & Williams, 2007). For example, Kumari, Head, and Marmot (2004) examined social inequalities and the incidence of T2DM longitudinally and found an inverse relationship between social position and incidence of diabetes. More specifically, Kumari and colleagues found that those individuals who were more economically disadvantaged had a higher body mass index and subsequently had a higher

incidence of diabetes. The environment and resources within provide a significant influence on diabetes health outcomes and are often interrelated with other psychosocial factors.

Functional aspects of our social networks, particularly the support we receive from others (Cohen, 2004), and the quality of our social interactions (Ruiz, Hutchinson, & Terrill, 2008) are important to health outcomes. For example, the social environment and the resources it provides are important aspects to diabetes self-care regimen. Barrera and colleagues (2006) found that broader social-ecological resources (e.g., restaurants that serve healthy foods and recreation areas) found in neighborhoods, organizations, and media effect changes in fat consumption, physical activity, and HbA1c over a 6-month intervention period. In a follow-up study, Barrera, Stryker, MacKinnon, and Toobert (2008) found diet specific and activity specific social-ecological resources act as mediators of behavioral intervention effects on diet and physical activity at 2-year follow-up. Importantly, they determined that lifestyle changes often occur in the social context of family and friends to influence adherence to a diabetes self-care regimen. The social network of T2DM management often resides in the family and is identified as an important component of influence on diabetes management. The social environment represents the most powerful influential web of intimate personal relationships while providing many supportive or deleterious effects on individual behavior, health, and well-being (Fisher et al., 1998). Furthermore, influence is often received in the form of support from others, which has been extensively studied due to the impact support has on health.

Social Support

Social support refers to the psychological and material resources provided by the social

network that gives an individual the ability to cope with stress. Social support is a primary psychosocial moderator of health outcomes with higher social support being associated with a range of health behaviors and outcomes including lower all-cause mortality (Holt-Lunstad, Smith, & Layton, 2010; Ruiz, Prather, & Kauffman, in press). Lower social support is associated with faster development and progression of cardiovascular disease (Ertel, Glymour, & Berkman, 2009), and impaired immune function (Robles & Kiecolt-Glaser, 2003). Adequate social integration is as strong a predictor of mortality as traditional risk factors such as cholesterol level, tobacco use, and hypertension in a patient population when compared to healthy others (Mookadam & Arthur, 2004). In addition, social integration is associated with fewer inflammation markers associated with heart disease and less cognitive decline with age (Ertel, Glymour, & Berkman, 2008; Loucks, Berkman, Gruenewald, & Seeman, 2006). Individuals with T2DM who participate in a social network are subject to influences on normative health behaviors. Social networks may influence self-management behaviors such as engagement in exercise, healthy diet, and close supervision of blood glucose levels. For example, T2DM women with larger support networks have lower HbA1c levels and lower triglycerides (Kaplan & Hartwell, 1987). In addition to integration and network size, research has linked social support to better glycemic control in all T2DM individuals (Okura, Heisler, &Langa, 2009), which may lead to slower disease progression. Moreover, social support is related to diet and medication adherence in a diabetic population (Garay-Sevilla, Nava, Malarca, Huerta, & Diaz de Leon et al., 1995). Diabetic health behaviors influence disease progression and clinical events. Diabetes is the most common cause of end-stage renal disease. Adequate social support is associated with lower rates of death in T2DM patients suffering from end stage renal disease (McClennan,

Stanwyck, & Anson, 1993).

Social support pathways to health. Social factors promote health through two mechanisms but through different processes of support and integration. Two primary psychosocial models: the main-effect model and the buffering hypothesis are theorized to link social support and the specific biological and psychosocial processes affected (Cohen, 1988). The main-effect model of social support proposes that social resources have a beneficial effect by providing positive experiences and stability in life situations. The stress-buffering model hypothesizes that support "buffers" an individual from the pathogenic effects of stressful events. Prospective research on stress buffering indicates those with high stress who perceived high levels of emotional support were at a substantially lower mortality risk (Rosengren, Orth-Gomer, Wedel, & Wilhelmsen, 1993). This may be especially important for T2DM individuals who must manage their disease while undergoing many life stressors. For example, Griffith and colleagues (1990) found for individuals with T2DM under high stress, more social support was related to better control while low stressed individuals with high and low levels of social support did not differ in their glucose control. This suggests an interaction between stress and social support with protection from adverse health outcomes. In contrast, the main-effect model states that social connectedness is beneficial whether or not one is under stress. Interacting with others is thought to aid in emotional regulation increasing positive affect (Cohen, 1988). Positive health behaviors and emotions are thought to be beneficial because they reduce psychological despair and enhance immune function particularly for older adults (Pressman & Cohen, 2005; Uchino et al., 1996). In regards to diabetes health outcomes, research indicates positive effects associated with social support broadly defined, including

HbA1c levels (Fortmann et al., 2011) and mortality (Zhang, Norris, Gregg, & Beckless, 2007) for all individuals.

Sources of social support. Supports can take the form of any person or group, real or imagined, present of inferred, which provides comfort (Barrera, 1986; Smith, Ruiz, & Uchino, 2004; Uchino, 2004). Marriage, parenthood, divorce, and widowhood are important transition periods in adulthood that may affect social networks and integration. Social ties shift during romantic relationships, the transition to parenthood, as well as during retirement and widowhood. For older individuals, the health benefit of social support is primarily manifested through the marital relationship and may be particularly important for health outcomes in the T2DM population. For example, as marital stress increases, depression and diabetes-related stress increases (Trief et al., 2006). The quality of the marital relationship is an important factor that may affect diabetes related health outcomes through self-management. Systematic reviews suggest marital quality (whether supportive or stressful) influences health related behaviors and subsequent physiological outcomes (Burman & Margolin, 1992; Kiecolt-Glaser & Newton, 2001) including greater life satisfaction and greater blood pressure dipping (Holt-Lunstad, Birmingham, & Jones, 2008). Conversely, marital strain has deleterious effects on cardiovascular, endocrine, and immune functions and is considered a chronic social stressor (for a review see Robles & Kiecolt-Glaser, 2003). Unhappily married people are worse off in terms of physical health (Wickrama, Lorenz, Conger, & Elder, 1997) and psychological well-being (Gove, Hughes, & Style, 1983). Research suggests that past negativity in relationships predicts greater mortality (Friedman et al., 1995). The marital relationship provides important influences on health and is a primary form of social support in the older T2DM population.

However, what happens if the primary form of support is unavailable or inadequate? When faced with these obstacles, compensation of social support may occur to prolong the health benefits of social support.

Compensation Theory

Compensation implies an adjustment mechanism by which the functional impact of loss is minimized by focusing on alternative resources. For example, an individual with an inattentive spouse may compensate for their poor relationship by seeking out friends by joining an organization (e.g. church, sports leagues, or junior league), or through fostering relationships with their extended family. In the context of social support, compensation may be the best option for some individuals who have lower social support in some social relationships or have suffered a loss in their social network. One strategy for minimizing the functional impact of declines as a result of network loss and/or diabetes related health is compensation, or relying on alternative resources. Compensation is conceptualized as making up for a loss, a deficiency, or a decline and may be especially important for diabetic health outcomes for those with higher social support.

Few studies have focused on social network compensation or have undertaken formal tests of the extent to which alternative social ties provide compensatory benefits on health outcomes (Rook & Schuster, 1996). In one study, Rice (1989) found that older, childless widows who had a confidant to provide emotional support formerly provided by a spouse reported higher life satisfaction than did the childless widows who lacked a confidant. Similar findings were found in mothers with critically ill children. Those women who received more supplemental support from family and friends when spousal support was inadequate had more

stable trajectories of physical and mental health over a one-year time period (Rini et al., 2008). However, evidence on social support compensation is mixed. Zettel and Rook (2004) examined social network compensation effects on psychological well-being. They found compensation often occurs for widows but is not associated with enhanced well-being, rather widows with more rekindled relationships had higher levels of depressive symptoms and loneliness. Social support compensation is studied in mothers and widows but is rarely examined for other individuals where social support may be an important influence on health outcomes.

Although social support compensation is a useful tool for health outcomes, there are few studies that examine it directly. For example, Block, Kremer, and Gaylor (1980) studied compensation indirectly through subjective pain reporting in patients with attentive and non-attentive spouses. The patients believed they were being observed by their spouse or a neutral observer and were asked to rate their current pain as well as pain over the past week. Block and colleagues found that patients with relatively non-attentive spouses reported less pain when observed by their spouse than when observed by a neutral observer. For those patients with a more attentive spouse, more pain was reported when the spouse was observing than in the presence of the neutral observer. Their findings suggest an attempt to elicit support from a neutral other compensated for inadequate social support. In this way, social support does not necessarily have to be visible to provide relative health benefits if primary social support is inadequate or unavailable.

Social support may come in many forms and alternative forms of social support including relationships with friends and family, and even strangers, may influence health through decreasing reactivity to and appraisal of stress. Support from a friend is more strongly

associated with attenuated reactivity to stress than support provided from a confederate (Edens, Larkin, & Abel, 1992). Further evidence suggests a friend-support group is associated with lower stress reactivity compared to a confederate support group (Christenfeld et al., 1997). Reduced reactivity has also been observed when subjects have a partner present during a task (Phillips, Carroll, Hunt, & Der, 2006), or receive a supportive note from a friend in a separate room (Uno, Uchino, & Smith, 2002). However, evidence also suggests confederates can be a helpful form of support if other forms of support are unavailable. Bolger and Amarel (2007) found invisible supportive confederates helped to reduce emotional reactivity when compared to no support or visible confederate support, which suggests knowing help is available, is just as beneficial as actual help and better than no support at all. Therefore, alternative sources of support are cultivated and relied upon in the face of enduring strains and a lack of support (Syrotuik & D'Arcy, 1984).

For individuals with T2DM, inadequate social support may lead to declines in healthover time as a result of poor management of their disease (Zhang et al., 2007). Evidence suggests that individuals will first look to a spouse for support, but if this support is inadequate or unavailable they begin to look for alternative sources of support (Block et al., 1980). Understanding how individuals with T2DM compensate for inadequate or unavailable social support may have important implications for understanding psychosocial aspects of disease management and downstream clinical events including mortality. Although research primarily focuses on compensation of widows, compensation may occur in all relationships regardless of significant loss and is rarely addressed in the literature. However, there are relatively few cross-sectional and no published longitudinal studies examining these issues.

Current Study

The current study is a longitudinal design with a nationally representative sample of older, married adults to better understand how social support compensation may provide a lower risk of mortality. It is unknown whether a lack of social support, and subsequent compensation for that support, has a significant effect on the risk of death in a married, T2DM sample. The study was guided by the general question, "Do older, married individuals with T2DM use a compensation model of social support for continued health benefits when primary support is unavailable or less than optimal?" More specifically, we tested the hypothesis that individuals with T2DM who use more social network compensation, when spousal support is less than optimal, have a lower likelihood of death.

METHOD

Participants

The Health and Retirement Study (HRS), is a nationally representative, longitudinal study of persons born in 1947 or earlier. The study was initiated in 1992 by researchers at the University of Michigan for the purpose of providing information about the U.S. population over the age of 50. Data has been continuously collected every two years since 1992. For the purpose of this study, data was analyzed within the 2004 wave of the study due to inconsistent measurement of social support throughout the waves. Additionally, 2004 was the first year that psychosocial measures were added to the study. At each interview detailed information is collected about the respondent's health behaviors, disease and disability, medical care usage, and psychosocial characteristics. Data was taken for each individual on his or her death status from the 2010 wave of the study. This information is collected each wave by using the National Death Index (NDI) from the CDC. Details of the methodology are reported elsewhere (Juster & Suzman, 1995; Heeringa & Connor, 1995).

The HRS is composed of four cohorts who entered the study in different calendar years. Eligibility for each cohort was based on birth year, although spouses were interviewed regardless of their age. Once participants have entered the study, they are interviewed every two years. The sample for each cohort was generated using a multi-stage, clustered, area probability sample of households based on the national sample frame in which Blacks, Hispanics, and Floridians were oversampled to provide a realistic sample of the national population of individuals over the age of 50. The sample includes approximately 17,000

respondents. Re-interview rates for all cohorts at each wave have been between 92% and 95% (Health and Retirement Study, 2011).

Measures

Outcome measures. Incident events were dichotomized into either deceased (documented date of death) or living (answering questions in the next wave or no death date documented). For deceased participants or those unavailable for a direct interview, proxy informants, predominantly spouses or children provided information of the event leading to mortality and completed an exit interview providing final information about the participant. Incident events were coded with either a 0 or 1.

Social support measure. Social support was assessed with a 34-item questionnaire (Walen & Lachman, 2000), which provided measures of structural (i.e., network size) and functional (i.e., the perceived emotional support provided) aspects of support. Items assessed social integration (number of social ties) and the quality of the interaction with those ties across four types of relationships: partner/spouse, adult children, extended family, and friends. Network size was assessed by asking whether participants live with their spouse and how many close relationships they have with children, other family, and friends. Quality of spousal relationship was measured by determining the closeness of the relationship and ranged from very close to not at all close.

For each type of perceived social support, questions are divided into positive (e.g., How much can you rely on them if you have a serious problem?) and negative (e.g., How much do they criticize you?) worded items. Participants answered these questions based on a Likert-type scale (1 = *a lot* to 4 = *not at all*) with six questions for each relationship type (e.g., spousal,

child, other family, friends). The questions were divided into negative and positive aspects of perceived support and were also pooled together to get a total perceived support score by reverse scoring both the negative and positive items andaveraging the scores within each relationship type where higher numbers equal more support. The social support measure was added to the core questionnaire in 2004. Reliability coefficients were calculated for total forms of social support and the positive and negative scales of social support across the three waves of 2004, 2006, and 2008. All four types of relationships had Cronbach's alpha ranging from .68 to .83 as shown in Table 1 across the three waves of the study. The survey was given to a selected portion of the participants in each wave; therefore for the purpose of the analysis, only data from the 2004 wave was used. As indicated by Table 1, spousal social support had the highest reliability (α = .80) with child social support (α = .79), family social support (α = .78), and friend social support (α = .70) with moderately reliable scales.

Bivariate correlations were conducted to examine simple associations between the social support variables of interest (spousal quality, spousal support, child support, family support, friend support, and total support) and are reported in Table 2. Quality of spousal relationship was used to identify those individuals in good and poor relationships to form two groups. Participants who responded they were very close and quite close with their spouse were categorized into a good relationship while those who responded as not very close or not at all close were categorized as in a poor spousal relationship. Total social support was calculated for all types of social support by reverse scoring both the negative and positive items and averaging the scores within each relationship type where higher numbers equal more support to obtain total scores.

Additionally, it was hypothesized that individuals who use compensatory social support would have a lower risk of death. Participants with low spousal support and low support in all other relationship types were hypothesized to have a higher risk of death than those with low spousal support and high social support in at least one other type of support (i.e., family, friends, children). Married participants with T2DM were categorized into two groups: those who compensated for low spousal support (i.e., 1 SD below the mean) by reporting high support (i.e., 1 SD above the mean) in at least one other relationship (1), and those who did not by reporting low support in at least one other relationship (0). A logistic regression analysis was used to calculate risk of death and corresponding odds ratios. Individuals who compensated for poor spousal support were expected to have a lower risk of death than those who did not.

Analytic Approach

The objectives of the study were to compare individuals with T2DM who were married on the basis of (1) cross-sectional associations with diabetes, age, race and ethnicity, other comorbid conditions, and different types of social support; and (2) incidence of death between those who compensated for poor spousal social support and those who did not.

Multivariable logistic regression was performed to assess the effect of social support compensation on mortality after adjusting for the effect of four covariates found to be predictive of death via comparative analysis: gender, Hispanic/Latino, race (White, Black, or Other), and age. The sample size was evaluated for missing data and to determine how the groups were split. For the purpose of the study, only married individuals with T2DM were used in the analysis and totaled 12,640 participants. Although individuals who are separated are technically still married, it is unlikely that someone who separates from their spouse still views

them as a significant source of support. Furthermore, 79% of couples that separate ultimately end their relationship in divorce (Tumin & Qian, 2012) and with this information; individuals who were separated were ultimately kept from further data analysis. Analyses were further restricted to subjects who completed the social support questionnaire and totaled 1,535.

It was hypothesized that individuals with higher marital quality also had higher spousal support. Individuals with higher marital quality were hypothesized to have a lower mortality risk across the study. Initial logistic regression models compared probability of death when spousal quality was high or low for participants with T2DM and it was expected that individuals in a higher quality relationship would have lower death incidence.

RESULTS

Sample Characteristics

A total of 12,640 participants reported they had diabetes and were married (male = 6,317 and female = 6,323), and of this group 1,084 men and 583 women had died over the course of the study period. The marital status make-up of those participants with diabetes is reported in Table 3. The racial and ethnic makeup of the sample consisted of approximately 78% who were non-Hispanic White, 10% non-Hispanic Black, 8% Hispanic White, and less than 1% Hispanic Black and is reported in Table 4.

The mean age of the participants was 67.81 (*SD* = 9.92) with men (*M* = 69.21, *SD* = 9.68) significantly older than the women (*M* = 66.50, *SD* = 9.97). The mean age of deceased participants was76 years old whereas the mean age of participants who were still alive was 67 years old, and these differences were significant $t(10720) = 1.48$, $p <= .01$. T2DM males had a mean estimated body mass index (BMI) of 29.07 (*SD* = 8.61) with T2DM women being significantly larger with a mean BMI of 32.65 (*SD* = 26.88). The average BMI for participants who were still alive at the end of the study was 30.92 (*SD* = 20.37) with participants who had died having a slightly lower BMI of 29.26 (*SD* = 6.77) although these differences were not significant $t(2359) = .69$, $p = .49$.

Descriptive statistics were computed and *t* tests were performed for all baseline control variables for married, T2DM participants, comparing individuals who compensated for social support to those who did not as well as to individuals with self-reported good and poor quality of spousal support. Men reported significantly more spousal support (*M* = 16.57, *SD* = 1.78) than women (*M* = 16.40, *SD* = 1.98), whereas women reported significantly more child, family,

and friend social support than men. Women also reported significantly more aggregated social support (M = 61.00, SD = 6.26) than men (M = 59.20, SD = 6.77). Table 5 gives an overview of these relationships and associated t-tests. Individuals who compensated for social support reported significantly more spousal support (M = 13.28, SD = 1.17) than those who did not compensate (M = 12.15, SD = 2.27), $t(216) = -3.14$, $p <. 01$, and were also significantly different when comparing their perceived social support from all other relationships as shown in Table 6.

In general, the sample of married, T2DM participants was evenly divided between men and women and was primarily non-Hispanic White. Men were older but had lower average BMI and more spousal support than women. However, women reported more aggregated support and had more support across other relationships than did men.

Specific Hypothesis Testing

Hypothesis 1. Individuals with higher marital quality will have higher spousal support. Pearson's bi-variate correlations were conducted and found that better marital quality was related to higher spousal support (r = .102, $p < .001$). Quality of the marital relationship was dichotomized into high and low quality by conducting a median split. Participants who reported higher marital quality had significantly higher spousal support (M = 16.53, SD = 2.38) than those with low marital quality (M = 15.72, SD = 1.82), $t(2113) = -5.16$, $p < .01$. These findings were consistent with the hypothesis that higher marital quality is associated with higher spousal support.

Hypothesis 2. Individuals with higher marital quality will have a lower mortality risk across the study. A multivariable logistic regression was conducted after controlling for gender, ethnicity, race, and age to determine the likelihood of death based on spousal quality. The final

model accounted for 15% of the variance in mortality, Nagelkerke R^2 = .151, Model χ^2 (5) = 96.31, p < .001. Older age, male gender, and being Hispanic were all significant predictors (see Table 7). Contrary to expectation, martial quality was not a significant predictor in the model, Walden value = .117, p = .73.

Hypothesis 3. Individuals who compensate for poor spousal support will have a lower risk of death than those who do not compensate for poor spousal support. Individuals with low spousal support and low support in their other relationships are hypothesized to have a higher risk of death than those with low spousal support and high social support in at least one other relationship (i.e., family, friends, and children). Compensation of social support was categorized by comparing married, T2DM individuals with low spousal support (i.e., 1 standard deviation below the mean) into 2 groups 1) compensators (i.e., 1 SD above the mean in at least one other relationship, and 2) non-compensators (no other major source of support). The non-compensators group was evenly divided between men (n = 7) and women (n = 6) whereas more women (n = 134) than men (n = 71) compensated for poor spousal support. Initial analyses of the groups found of the 218 individuals 13 did not compensate for poor spousal support. Given these cell size discrepancies, the associated analyses were not conducted (see Table 8).

Ancillary Analyses

Ancillary analyses were conducted with the idea that some variance in total compensation support may moderate mortality risk. Specifically, we examined whether individuals in low supportive marriages who had high total support (1 SD above the mean) were at lower mortality risk compared to those with low total support (1 SD below the mean).

A multivariable logistic regression was conducted to determine the likelihood of death based on total support in all relationships after controlling for age, gender, race, and ethnicity. The final model accounted for 12% of the variance in mortality, Nagelkerke R^2 =.12, Model χ^2 (5) = 19.05, $p < .01$. Older age, male gender, and Hispanic status were significant predictors of mortality risk. Higher total social support across relationshipswas associated with decreased mortalityrisk, accounting for ~3% of the variance (see Table 9). The odds of mortality for individuals who did not have high total social support was .35 indicating those in a low supportive marriage who did not compensate were at higher risk of mortality than individuals who compensated for poor spousal support.

DISCUSSION

The goal of the current study was to determine if social support compensation led to a lower risk of death among married persons with diabetes when social support was inadequate in a national cohort sample. Overall, men reported more spousal support than women. However, women reported significantly more aggregated social support across all identified relationships relative to men. As expected, individuals with higher reported marital quality had higher spousal support. However, higher spousal support was not associated with a lower risk of death, despite expectations to the contrary.

Most germane to the current study, was the issue of whether greater social support compensation for low spousal support was associated with mortality risk amongst married persons with diabetes. Few persons reported low spousal support and low support compensation, rendering the cell sizes highly unequal and the associated data uninterpretable. Therefore, a set of ancillary analyses were conducted to examine whether the degree of variance in total compensation support moderated mortality risk. Consistent with this idea, higher aggregated social support across non-spousalrelationships was associated with lower mortality risk. These results support the idea that strong social relationships moderate the risk of mortality, when compensating for poor spousal support. Such findings strengthen the concept of social support compensation as an important psychosocial health buffer and suggest it may be a mechanism linking network size with beneficial health outcomes.

These findings add to the sporadic data supporting social support compensation and better health outcomes. Past research has examined compensation in regards to health outcomes, life satisfaction, and pain behaviors. For example, Rini and colleagues (2008) found

that mothers who compensated for poor spousal support with other forms of social support had better overall health outcomes than those with poor support. Additionally, higher life satisfaction was found among childless widows who had a close confidant (Rice, 1989). It should be noted that few studies have examined specific compensation models of social support in relation to objective health outcomes. A study by Block and colleagues (1980) found that subjective pain decreased when individuals sought a secondary form of social support when their primary social support (spouse) was inadequate. Although this study took place in the context of an objective physical condition (i.e., chronic pain) it also relied on subjective reporting and behavior. Hence, the current findings broaden the literature on social support compensation as amongst the first to examine a non-subjective outcome.

More broadly, the current studyunderscores the importance of large social networks and the associated physical health benefits, particularly in the context of diabetes. Consistent with past research (Kaplan & Hartwell, 1987; Okura, Heisler, & Langa, 2009), having a broad social network and seeking those relationships in a time of need was associated with a reduced risk of death.This study provides evidence of a caveat that flexibility in drawing support from the network - ability to pull from secondary sources when primary sources are not optimal – may have important implications for health.

Limitations and Future Directions

Although thefindings of this study provide valuable insights into social support compensation, several important limitations should be noted. First, the study used an ad hoc measure of support not previously validated. Although we drew upon conceptually related items, the relation between this measure and better-validated measures of support is less well

known. Caution is therefore warranted in interpreting the associated findings. Second, and related, the measure used here was not intended to describe a compensation model, which further limits generalizability. Third, like all such studies, self-report has inherent error bias, which could affect differences between reported, perceived, and actual support. Fourth, although this study drew from a national cohort, the sample was largely homogenous older and predominately non-Hispanic Caucasian adults. Spousal support and other supportive relationships may vary in persons of different races, ethnicities, age, and overall health status, and a greater diversity of participants could yield different results. Finally, analyses for the main hypothesis were not conducted due to the unequal group sizes of individuals who engaged in social support compensation compared to those who did not. Finding an adequate way to measure and conceptualize social support compensation would be beneficial to understanding how this aspect of social support contributes to long-term health and mortality.

Despite these limitations, the present study broadens understanding of social support compensation as a social construct. Future studies should focus on this concept of compensation by conducting studies with a validated measure of social support. In addition, a strategy for measuring support compensation more accurately should be a top research priority. Adequate measurement is necessary for understanding how individuals compensate for inadequate support and may further enlighten the importance of support received from many different social categories. In addition to addressing the aforementioned limitations, future research should examine how specific relationships buffer an individual from mortality. For example, older adults usually gain primary support from their spouse, but do some relationships matter more than others? In the present study, having more support was

associated with lower death risk but due to measurement issues we were unable to address whether certain aspects of the social network were more important than others. Special attention should also be given to perceived compared to actual support given from important social network members. Each group may influence the individual's self-perception of the type and availability of social support. Personality influences also deserve more direct attention. For example, do individuals higher in hostility perceive and react to social support in a way that directly influences availability of supportive others? Additional research on personality influences may facilitate a more complete understanding of psychosocial influences in the risk of mortality for individuals with a chronic illness and guide the development of appropriate interventions. Finally, understanding how social support influences chronic diseases such as T2DM, including management and health outcomes, is useful for health professionals and caregivers. The current study was plagued with ambiguous and uninterpretablefindings; however, this may have been the result of a failure to account for social support coming from many different external sources.

Conclusions

Social networks play an important role in health. The current findings demonstrate how an individual can compensate for a poor primary support relationship through a broader support network. These findings should guide future research to focus on how individuals build, maintain, and seek support from social relationships. Understanding the many facets of these relationshipscan be a useful guide for individuals with a chronic illness, and may be especially important for individuals with T2DM who face a myriad of health issues in addition to the natural aging process. The current study informs the literature by emphasizing the

importance of a large social network and turning to many supportive aspects of that network for continued health benefits.

Table 1

Scale and Subscale Reliability by Year

Types of Social Support	2004 □	2006 □	2008 □
Total Spouse	0.797	0.812	0.818
Positive Spouse	0.796	0.807	0.815
Negative Spouse	0.688	0.732	0.735
Total Child	0.787	0.796	0.795
Positive Child	0.817	0.829	0.82
Negative Child	0.71	0.732	0.731
Total Family	0.785	0.763	0.763
Positive Family	0.855	0.856	0.859
Negative Family	0.733	0.757	0.769
Total Friends	0.679	0.702	0.688
Positive Friend	0.835	0.839	0.833
Negative Friend	0.679	0.714	0.72

Table 2

Perceived Social Support Correlation Matrix

	Age	Spousal Quality	Spouse Support	Child Support	Family Support	Friend Support
Spousal Quality	.042					
Spousal Support	.006	.111**				
Child Support	-.005	.009	.343**			
Family Support	-.077**	.010	.233**	.425**		
Friend Support	-.082**	.004	.217**	.342**	.451**	
Total Support	-.067**	.029	.576**	.722**	.824**	.780**

*p<.05; **p<.01

Table 3

Marital Status by Sex

Marital Status	Total	Male	Female
Married	12640	6317	6323
Separated	264	139	225
Divorced	2189	778	1411
Widowed	4049	746	3303
Never Married	720	307	413
Other	43	17	26

*p<.05; **p<.01, **p<.001

Table 4

Summary of Race and Ethnicity of the Sample

	White	Black	Other
Hispanic Unknown Type	2	0	0
	.01%	0%	0%
Mexican American	670	1	117
	5.3%	.01%	.9%
Other Hispanic	341	18	73
	2.7%	.1%	.6%
Not Hispanic	9855	1309	252
	78.0%	10.4%	2.0%

Table 5

Demographics by Sex

	Overall	Male	Female	*t*
Age	67.81	69.21	66.50	14.30***
	(9.92)	(9.68)	(9.97)	
BMI	30.87	29.07	32.65	-4.35***
	(20.10)	(8.61)	(26.88)	
Spousal Support	16.48	16.57	16.40	2.07*
	(1.88)	(1.78)	(1.98)	
Child Support	15.33	15.09	15.55	-4.73***
	(2.16)	(2.26)	(2.05)	
Family Support	14.30	13.89	14.66	-6.63***
	(2.63)	(2.70)	(2.52)	
Friend Support	14.07	13.65	14.45	-6.98***
	(2.56)	(2.54)	(2.53)	
Total Support	60.16	59.20	61.00	-5.65***
	(6.57)	(6.77)	(6.26)	

*p<.05; **p<.01; ***p<.001

Table 6

Age & Social Support Relationships by Social Support Compensation

	Overall	Compensation	No Compensation	*t*
Age	67.81	68.35	65.42	-1.03
	(9.92)	(9.50)	(10.30)	
Spousal Support	16.48	13.28	12.15	-3.14**
	(1.88)	(1.17)	(2.27)	
Child Support	15.33	14.40	11.00	-5.81***
	(2.16)	(2.04)	(2.16)	
Family Support	14.30	13.39	8.92	-6.13**
	(2.63)	(2.56)	(2.14)	
Friend Support	14.07	13.33	9.38	-5.34**
	(2.56)	(2.62)	(1.85)	
Total Support	60.16	54.52	41.46	-8.97**
	(6.57)	(4.94)	(6.31)	

*p<.05; **p<.01; ***p<.001

Table 7

Logistic Regression of Spousal Quality

Variable	B	SE	Wald	Exp(B)	Exp(B) 95% CI
Age	.112	.01	72.66***	1.12	1.09-1.15
Gender	-.392	.23	2.99*	.68	.43-1.06
Hispanic/Latino	-.314	.09	11.01***	.73	.61-.88
Race	-.036	.12	.087	.97	.76-1.22
Spousal Quality	-.165	.48	.117	.85	.33-2.18
Constant	-8.98	1.11	65.49***	.00	

-2 Log Likelihood = 652.54, χ^2 = 96.31, p< .001
*p<.05; **p<.01; ***p<.001

Table 8

Mortality by Social Support Compensation Crosstabs

	No Compensation	Compensation	Total
Alive	11	183	194
Dead	2	22	74
Total	13	205	218

Table 9

Logistic Regression of Total Social Support

Variable	B	SE	Wald	Exp(B)	Exp(B) 95% CI
Mortality					
Block 1					
Age	.056	.02	5.48*	1.06	1.01-1.11
Gender	-.074	.47	2.57	.47	.19-1.18
Hispanic/Latino	-.39	.15	6.80**3	.69	.50-.91
Race	-.047	.16	.09	.95	.70-1.30
Mortality					
Block 2					
Total Support	-1.06	.46	5.35*	.35	.14-.85
Constant	-3.51	1.95	3.24	.03	

[a]Overall R^2 = .084; -2 Log Likelihood = 177.507, χ^2 = 13.48, p< .01
[b]Overall R^2 = .12; -2 Log Likelihood = 171.939, χ^2 = 19.05, p< .01
*p<.05; **p<.01; ***p<.001

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