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The Role of Religiosity in the Development of Obesity From Adolescence to Adulthood

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Abstract

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MA, Bemidji State University, 1996

BS, Colorado State University, 1992

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Health Psychology

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Abstract

The development of obesity from adolescence to adulthood is not well understood, nor does the research support a multidimensional approach for this understanding. Studies have described primarily cross-sectional bivariate relationships between combinations of obesity, religiosity, depression, and social support, but it is still not known whether there is a relationship between adolescent religiosity, depression, and social support in the development of adult obesity or whether depression and social support mediate the religiosity–obesity relationship. The dynamic, multidimensional, functional model of wellness presented by Hawks was the basis for the spiritual, social, emotional, and physical interactions proposed in this study. The research questions sought to identify the relationship that exists between adolescent religiosity, depression, and social support and adult obesity and considered depression and social support as potential mediators of the religiosity–obesity relationship. This quantitative study employed multiple linear regression while using the prospective nature of the Add Health data set to gain a longitudinal understanding of the religiosity–obesity relationship. Adolescent male religiosity significantly predicted adult obesity, but female religiosity did not. Neither depression nor social support mediated the religiosity–obesity relationship. Social change implications include a rationale for developing sex-based multidimensional approaches, including spiritual approaches, for supporting adolescents in their transition to adulthood. Support for acknowledging the differences between sexes for multiple health interactions is provided and indicated for healthcare providers. Finally, health educators are presented with much-needed support for the concept of the multidimensionality of wellness.

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Dedication

I dedicate this work to my wife, Barbara, and our four children, Tierza, Chase, Janae, and Eliana. The journey has been long and difficult at times, and nobody felt the impact of the dissertation process more than my family. I am forever grateful for the support they each provided. I am particularly thankful and grateful to Barbara for keeping the family intact while I have been "gone" for most of this process. It is my sincere hope that we can regain some momentum as a family now that this chapter in life is complete.

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Chapter 1: Introduction to the Study

Introduction

Health, in American culture, is a hot topic that includes the threats of infectious diseases, such as the yearly flu virus (Centers for Disease Control and Prevention [CDC], 2014b), as well as the reality of chronic diseases/conditions, including elevated blood pressure, cancer, and cardiovascular disease. A noted contributor to increased mortality rates at the worldwide level is obesity (World Health Organization, 2009). Researchers have produced a plethora of cross-sectional studies on correlates of obesity; however, researchers can gain greater understanding of the etiology of obesity using longitudinal designs and incorporating multiple dimensions of wellness (i.e., emotional, social, and spiritual dimensions). This study incorporated such a focus into the study of obesity by using longitudinal data from an archival dataset and by incorporating multiple dimensions. An understanding of the longitudinal development of obesity in light of multiple dimensions of wellness allows for a broader approach to supporting adolescents during their transition to adulthood. Such support may be related to the treatment of overweight and obese individuals or to prevention of overweight and obesity status as individuals mature. Given the physical and social challenges associated with increased levels of obesity, support that helps to prevent the development of obesity can impact American society during the critical transitional period from adolescence to adulthood.

In this chapter, the rationale for the current study is developed. Furthermore, the development of obesity in the United States is outlined and cross-sectional relationships with obesity for religiosity, social support, and depression are discussed. Adolescent

social support and adolescent depression are identified as potential mediators of the relationship between adolescent religiosity and adult obesity. Finally, I identify research questions and hypotheses that relate four dimensions of wellness (the spiritual dimension as represented by religiosity, the emotional dimension as represented by depression, the social dimension as represented by social support, and the physical dimension as represented by obesity). These four dimensions are related directly to the theoretical framework presented by Hawks (2004), in which he proposed that spiritual wellness impacts social and emotional wellness, with further impact on physical and intellectual wellness.

The CDC (2012) defines adult overweight as a body mass index (BMI) range between 25 and 29.9 and adult obesity as a BMI range of 30 or higher. Although BMI is not a direct measure of body fat, it is an accepted estimate for body fat and for disease and health risks related to increases in body fat (CDC, 2012). From 1960 to the present, overweight and obesity levels in the United States rose substantially (National Center for Health Statistics, 2012). Increases in overweight and obesity during this time are evident for children, adolescents, and adults. Adolescent obesity levels increased from 5% in 1960 (Ogden, Flegal, Carroll, & Johnson, 2002) to 16.9% in 2010 (Ogden, Carroll, Kit, & Flegal, 2012a), accounting for the often-stated realization that childhood obesity in the United States has tripled since 1960. Furthermore, obesity rates continue on an upward projection, almost without exception, from childhood into late adulthood for both sexes (Flegal, Carroll, Kit, & Ogden, 2012; Ogden, Carroll, Kit, & Flegal, 2012b). Therefore, within a given section of the American populace, there is an increase in the number of

individuals categorized as overweight or obese as time progresses. A particularly large increase in the rate of obesity is noted in the transition from adolescence to adulthood. Gordon-Larsen, The, and Adair (2010) reported that obesity doubled two separate times (between adolescence and early adulthood and again between early adulthood and the mid-30s) within the cohort they studied.

Increases in obesity rates are related to a number of negative physical, social, and emotional challenges, including heart disease, diabetes, and hypertension (Must et al., 1999); psychological and behavioral problems; ADHD (Halfon, Larson, & Slusser, 2013); and missed days in school (Echeverría, Vélez-Valle, Janevic, & Prystowsky, 2014) and at work (Howard & Potter, 2014). At the national level, initiatives are in place to target the rates of overweight and obesity, and these initiatives are warranted. Despite the overt focus on health in the United States, the American public is not the healthiest population in the world. Japan holds the position atop the health leader-board for women based on life expectancy at birth, and Switzerland holds the top spot for men (U.S. Census Bureau, 2012). Healthy People 2020 is a national initiative with the goal of improving health in the United States across a wide spectrum of health measures. The Department of Health and Human Services (2014) lists *adult obesity* and *childhood and adolescent obesity* as two of 26 leading health indicators associated with Healthy People 2020.

Increased obesity rates are a fairly recent phenomenon, with the first wave of research focusing primarily on cross-sectional correlates to obesity such as depression and stress. Despite the overabundance of current research studies related to obesity, there

is a dearth of research that considers longitudinal measures of obesity as well as multiple dimensions of health in the development of obesity—perhaps the second wave of research will address these concerns. Research consistently demonstrates that overweight and obesity in youth persist into adulthood (Gordon-Larsen, The, & Adair, 2010; Juonala et al., 2011; Rooney, Mathiason, & Schauberger, 2011; Singh, Mulder, Twisk, van Mechelen, & Chipanaw, 2008). Furthermore, for adolescents who are of normal weight, obesity rates increase during the transition to adulthood (Gordon-Larsen et al., 2010; Juonala et al., 2011). The two primary physiological reasons for weight gain are decreases in physical activity and increases in energy consumption. However, research in the tracking of physical activity, which is a measure of consistency for physical activity over time in relation to others, points to the likelihood that physical activity levels from childhood to adulthood track consistently (Telama, 2009; Telama et al., 2005). Regarding the second physiological component of weight gain—increased energy consumption—research provides less definitive results. While Oellingrath, Svendsen, and Brantsæter (2011) found children to maintain stable eating patterns from fourth to seventh grade, Boreham et al. (2004) reported that for Irish participants, dietary tracking was inconsistent from adolescence to adulthood.

Given that obesity increases in adulthood cannot be wholly tied to physical activity level or to energy consumption, researchers have considered other avenues of connection to the transition from adolescence to adulthood. Noted variables of interest in this transition include the role of depression in the development of obesity (Blaine, 2008; Faith et al., 2011), the role of social support in increasing physical activity among

children and adolescents (de la Haye, Robins, Mohr, & Wilson, 2010; Trost & Loprinzi, 2011), and the role of stress in obesity increases (Jääskeläinen et al., 2014; Tomiyama, Puterman, Epel, Rehkopf, & Laraia, 2013). This expanded view of the development of obesity beyond primarily physiological considerations matches current health models.

Health education in the United States consistently promotes a philosophical stance regarding the dimensions of health. Typical dimensions of health (or *wellness*) presented by health educators are physical, emotional, social, spiritual, intellectual, and environmental (some also include occupational, e.g., Hoeger & Hoeger, 2015). Educators stress the principle of interrelatedness of these dimensions indicating that deficiencies in one dimension can often impact individual accomplishment or success in another dimension (e.g., Hoeger & Hoeger, 2015). For instance, an individual who faces physical illness will likely experience impaired functioning in social, emotional, and intellectual interactions throughout the day of his or her illness. Furthermore, there is an assumption that these interactions between health dimensions can be acute as well as longitudinal in nature. Poor physical health may affect one's daily routines; likewise, continuing lack of physical health can have an effect on other dimensions of wellness over longer periods of time.

Researchers in the health fields are increasingly interested in the role of spiritual health/wellness as it relates to other dimensions of health (e.g., Koenig, King, & Carson, 2012). Despite a longstanding taboo against studying the role of spirituality in psychological research, studies of spirituality and religion are common in today's literature. Koenig, King, and Carson (2012) presented an entire volume dedicated to the

association between religion and multiple measures of health; however, much of the research in this area is conflicting (Koenig, King, & Carson, 2012; Musick & Worthen, 2010; Rogers, Krueger, & Hummer, 2010).

What is clear is that obesity is related to biological, environmental, and sociocultural factors, including activity levels, energy consumption, depression, social support for activity and for nutrition, and stress, to name just a few. Research also supports the connection, within the adolescent population, between religiosity and health-enhancing behaviors (Ford & Hill, 2012; Neymotin & Downing-Matibag, 2013; Rew, Arheart, Thompson, & Johnson, 2013) and between religiosity and health-risk behaviors (Salas-Wright, Vaughn, Hodge, & Perron, 2012). What remains unclear is the role of religiosity in the development of obesity in the transition from adolescence to adulthood.

Religiosity and health are connected in some fashion, but the connections to specific forms of health are not consistently identified. Research on the religiosity-obesity link provides such an example of conflicting outcomes. While some researchers have reported a significant relationship between increased religiosity and increased levels of obesity (Dodor, 2012; Feinstein et al., 2010; Yeary et al., 2009), other researchers have concluded there to be no relationship between these two variables (Reeves, Adams, Dubbert, Hickson, & Wyatt, 2012). The rationale for the consideration of a possible relationship between the constructs of religiosity and obesity is based on the role of religion in the management of various health outcomes. Generally, level of religiosity is associated with a longer, healthier life. Obesity, on the other hand, is associated with early mortality and lower health status among the aging. Logic suggests that an increase

in religiosity, which is associated with a healthier life, cannot also be related to increased obesity, which is associated with early mortality and reduced health.

However, conflicting outcomes in the religiosity-obesity research exist. While some of the disparate outcomes may be due to differences in the populations studied or differences in how religiosity and obesity were measured, if dimensions of wellness are truly integral to overall health, a reason for inconsistent conclusions may be a lack of consideration of multiple dimensions working together in the development of obesity. Furthermore, the lack of longitudinal designs in the literature on the development of obesity may have limited the strength of the relationship observed between religiosity and obesity.

The association between religiosity and obesity may be explained by depression and social support. Research conclusions support an inverse relationship between religiosity and depression (Berry & York, 2011; Ji, Perry, & Clarke-Pine, 2011; Koenig, 2009; Pössel et al., 2011; Yonker, Schnabelrauch, & DeHaan, 2012); a relationship between religiosity and social support (Moxey, McEvoy, Bowe, & Attia, 2011; Schnall et al., 2012; Thomas & Washington, 2012); a relationship between depression and obesity (Boutelle, Hannan, Fulkerson, Crow, & Stice, 2010; Faith et al., 2011; Wiltink et al., 2013; Zhong et al., 2010); and a relationship between social support and obesity (Christakis & Fowler, 2007; Leahey, LaRose, Fava, & Wing, 2011). When these relationships were identified in the study, social support and depression were considered as mediators of the religiosity-obesity relationship.

Although the association between health and religiosity is well-established in the literature, the relationship between obesity and religiosity is confounded by weaknesses in the study designs. The vast majority of obesity studies are cross-sectional in nature; summarily, cross-sectional studies only identify that a relationship exists, without insight into the direction of that relationship. Longitudinal studies of obesity are needed to help researchers and health practitioners better prevent the development of obesity and better treat those struggling with obesity. Furthermore, the development of obesity is influenced by multiple biological, environmental, and sociocultural factors. Single connections to obesity (such as gender, race, depression, or socioeconomic status) do not allow for a full understanding of the development of obesity, which is likely impacted by multiple dimensions. Finally, a broader perspective on the relationship of multiple dimensions in the development of obesity will allow for greater responses by the health community in supporting children as they develop socially, emotionally, intellectually, spiritually, and physically into young adults.

Background

Obesity is a complex and deleterious condition associated with early mortality, increases in physical diseases, social segregation, and emotional challenges. In the United States, the obesity rate for children and adolescents was 16.9% in 2010 (Odgen et al., 2012). In essence, obesity rates for children and adolescents have tripled since 1960, when the rate was 5% (Odgen et al., 2002). Among adults, the obesity rate in the United States is 35.3%; however, when overweight and obesity are considered together, the adult rate is 68.5% (National Center for Health Statistics, 2012). Recent trends in obesity in the

United States indicate disproportionate levels for some populations, including higher rates among male children and adolescents compared to female children and adolescents (Ogden et al., 2012a), Mexican American men and women (Rokholm, Baker, & Sorensen, 2010), and non-Hispanic Black adults (Flegal et al., 2012). Furthermore, throughout the lifespan, men and women differ in their levels of obesity based on age, as women have increasing levels of obesity as they increase in age and men hit a plateau in obesity in the 40-59 age range (Flegal et al., 2012). Obesity trends in the United States are discussed more fully in Chapter 2; however, these statistics demonstrate that obesity in the United States is a serious problem that is dependent on age, sex, and race/ethnicity.

Following is a detailed discussion of the multiple correlates that have been identified by researchers to date as related to the variables of interest in the study; each of these relationships is discussed more thoroughly in Chapter 2. The correlates related to religiosity and explored in the next few pages include the following: the relationship between religiosity and general health; the relationship between religiosity and obesity with a focus on various measures of religiosity and confounders to the relationship such as race; and health behaviors leading to obesity rather than specific measures of obesity. Other correlates discussed in the upcoming pages include those between religiosity and depression, depression and obesity, religiosity and social support, and social support and obesity. To start, I discuss the findings related to religiosity and general health.

A factor that impacts obesity and has been less studied than other variables is religiosity; this connection was the primary focus of this study. Religiosity was the chosen measure of spiritual health considered in this study, and obesity was the chosen

measure of physical health considered in this study. Support for the relationship between religion and health is prevalent in scientific studies, with seemingly indelible links between religion and longevity—specifically, the link between religious service attendance and mortality rates (Chida et al., 2009; Musick & Worthen, 2010; Strawbridge, Shema, Cohen, & Kaplan, 2001). Rogers, Krueger, and Hummer (2010) reiterated three hypotheses expressed by Durkheim (1897/2002) to explain the potential role of religion in overall health: (a) the role of social support and social networks through religious affiliation to increase health practices; (b) religious encouragement to abstain from specific lifestyles, such as tobacco use, alcohol use, drug use, and pre- and extramarital sexual intercourse, which are related to the development of cancer, heart disease, and sexually transmitted infections; and (c) the reduction of the stress response through religious affiliation. The potential social and emotional benefits of religiosity hypothesized by Durkheim are most germane to the current study. Might religion provide social support for health behaviors among adolescents that reduce the propensity to develop obesity? Also, might religiosity reduce the incidence of depression among adolescents with subsequent effects on obesity? Although religion has been applied to general health and health practices, there is still inconclusive evidence regarding how or whether religion relates to obesity.

The literature regarding the link between religiosity and obesity is conflicting, as demonstrated by findings in research on denominational affiliation and obesity. Research results indicated higher rates of obesity for states in the United States with higher rates of Baptist affiliation (Ferraro, 1998), among women in the Baptist denomination (Cline &

Ferraro, 2006), and among the African American population for a number of denominations (Dodor, 2012). Conversely, Ferraro (1998) determined that the religiosity and weight relationship identified in his study attenuated when factors of socioeconomic status and race/ethnicity were controlled.

Religiosity can be expressed in a number of ways, including attending religious services or events, prayer, and using religious media. Researchers have concluded consistently that use of religious media is related to increased levels of obesity (Cline & Ferraro, 2006; Musick & Worthen, 2010; Yeary et al., 2009). Dodor (2012) and Feinstein, Liu, Ning, Fitchett, and Lloyd-Jones (2012) supported the connection between increased obesity levels with increased levels of prayer and church attendance. In contrast, for women, specifically, Cline and Ferraro (2006) found church attendance to be negatively associated with obesity levels. Other gendered differences have been noted regarding connections between religion and obesity. Cline and Ferraro determined that men relate to religion differently than do women, and Kim, Sobal, and Wethington (2003) determined that Protestant men were significantly heavier than men without religious affiliation while women demonstrated no relationship between religiosity and body weight. On the other hand, Feinstein et al. (2010) reported there to be no difference between men and women in regard to the religiosity-obesity relationship. The research relating religious attendance, prayer, and religious media to obesity is inconclusive and suffers from combinations of cross-sectional design and study of older adults whose age places them in more consistent developmental patterns. The Cline and Ferraro study is

the one study reporting that church attendance was associated with reduced obesity among women; interestingly, the Cline and Ferraro study used a longitudinal design.

Another area of conflicting conclusions reached by research on the religiosity-obesity connection is race/ethnicity. Dodor (2012) reported that increased prayer and church attendance among the African American population were significantly related to increased obesity; however, Reeves, Adams, Dubbert, Hickson, and Wyatt (2012) found there to be no significant relationship between religiosity and obesity among the African American population. Notably, Dodor (2012) and Reeves et al. (2012) studied different populations within the African American community, and opposing conclusions from the Dodor and Reeves et al. studies may be a reflection of the different African American populations represented by each study, including the differences in the mean age of participants—32 years of age for the Dodor study and 49 years of age for the Reeves et al. study. Furthermore, both studies were cross-sectional in design.

Some researchers have considered the role of religiosity in health behaviors that might lead to obesity rather than having a direct relationship with obesity (Kim & Sobal, 2004; Reeves et al., 2012; Roff et al., 2005). Reeves et al. (2012) reported that increased prayer was associated with lower daily calorie intake. Kim and Sobal (2004) considered fat intake and physical activity in their study of the relationship between religiosity and health. These authors identified that Catholic women consumed less fat in their diet than other groups, and men who prayed more often were more physically active than men who prayed less often (Kim & Sobal, 2004). Roff et al. (2005) concluded that higher levels of church attendance were associated with increased leisure physical activity. Despite the

observed connections between increased religiosity and reduced fat intake and increased physical activity, researchers did not find a connection between religiosity and obesity in the noted studies. Therefore, the link between religiosity and obesity is still not clear.

Studies on race and health behaviors have not provided a clear understanding of the religiosity-to-obesity link. Therefore, other variables—depression and social support—may help to explain the link between religiosity and obesity. Depression and social support are prominent factors in the relationship between religiosity and general health. Powell, Shahabi, and Thoresen (2003) identified depression and social support to be well-established protective factors in the religiosity-physical health relationship. Furthermore, Strawbridge (2001) identified decreases in depression symptoms and increases in social support as specific mechanisms leading to increased longevity for those with higher levels of religiosity. Therefore, there is empirical evidence that social support and depression are likely to interact with religiosity and obesity due to obesity's role in physical health. As with the religiosity-obesity relationship, however, research results for connections to depression and social support are conflicting.

Pratt and Brody (2008) identified depression to be more common for those aged 40-59, for women, for non-Hispanic Blacks, and for those at the poverty level; similarly, Kessler, Chiu, Demler, and Walters (2005) identified levels of major depression to be higher among women, non-Hispanic Whites, and unmarried, low-income, low-education, and rural populations. In the past 10 years, multiple researchers have conducted studies on the relationship between religiosity and depression for a variety of populations (Balbuena, Baetz, & Bowen, 2013; Berry & York, 2011; Meltzer, Dogra, Vostanis, &

Ford, 2011; Pirutinsky et al., 2011; Wenger, 2011). Three general conclusions emerge from these studies.

The first general conclusion from recent studies of the religiosity-depression link is that there is sufficient evidence to indicate that increased religiosity is associated with decreased levels of depression (Berry & York, 2011; Jansen et al., 2010; Koenig, King, & Carson, 2012; Yonker, Schnabelrauch, & DeHaan, 2012). The second conclusion emerging from recent research is that lower depression levels are associated with stronger religiosity levels (Jansen, Motley, & Hovey, 2010; Meltzer et al., 2011) and with higher levels of intrinsic religiosity (Ji, Perry, & Clarke-Pine, 2011; Pössel et al., 2011; Yonker et al., 2012), but that religious affiliation is not related to depression (Jansen et al., 2010; Meltzer et al., 2011). The third general conclusion emanating from recent research is that men and women differ in the religiosity-depression link, with women exhibiting higher levels of depression than men (Eliassen, Taylor, & Lloyd, 2005; Pratt & Brody, 2008) and women exhibiting greater engagement in religion than men (Eliassen et al., 2005; Maselko & Kubzansky, 2006). Eliassen et al. (2005) provided further insight into the gender difference by graphing the religiosity-depression relationship and by noting that the graph for women is U-shaped whereas the graph for men is linear. A connection between depression and religiosity is only one part of the association that needs to be present for mediation to occur. The other half of the mediation analysis requires there to be a measured connection between depression and obesity.

Obesity is a known factor in the development of cardiovascular disease; major depression presents a similar risk for the development of cardiovascular disease as do

smoking and diabetes (Van der Kooy, van Hout, Marwijk, Marten, & Beekman, 2007). Luppino et al. (2010) surmised that the connections of obesity and depression to cardiovascular disease fuel the research linking these two health problems. The literature regarding the depression-obesity link can be considered in three distinct patterns: (a) the possibility that obesity leads to depression; (b) the possibility that depression leads to obesity; and (c) the possibility that obesity and depression share a bidirectional relationship.

Obesity may lead to depression due to the stigma associated with being obese (Faith et al., 2011; Fowler-Brown, Ngo, & Wee, 2012; Goldfield et al., 2010; Needham, Epel, Adler, & Kiefe, 2010; Pan et al., 2012). A second proposal for the way in which obesity leads to depression is through limitations of physical and functional capacities that reduce the individual's quality of life (Faith et al., 2011; Fowler-Brown et al., 2012; Luppino et al., 2010). Finally, obesity and depression promote dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis (Faith et al., 2011; Goldfield et al., 2010; Luppino et al., 2010), and antidepressants and obesity treatments are known to affect the HPA axis (Bornstein, Schuppenies, Wong, & Licinio, 2006), thereby indicating a relationship between obesity and depression. Therefore, stigma, reduced physical functioning, and HPA axis dysregulation are likely causes of the development of depression from obesity.

Connection to the HPA axis is also used to explain the way in which depression might lead to obesity (Goldfield et al., 2010; Luppino et al., 2010; Pan et al., 2012). Another proposed path for depression leading to obesity includes increased consumption

of food and lack of physical activity, which are often symptoms of depression (Faith et al., 2011; Fowler-Brown et al., 2012; Goldfield et al., 2010; Luppino et al., 2010; Needham et al., 2010; Pan et al., 2012). Also, weight gain is a known side effect of antidepressant medication and, therefore, a proposed path for the development of obesity from depression (Faith et al., 2011; Luppino et al., 2010; Pan et al., 2012). Therefore, depression may contribute to obesity development through metabolic disruption, behavior changes associated with depression, or psychopharmological interactions.

Researchers have considered the possibility that the relationship between depression and obesity is bidirectional (Faith et al., 2011; Fowler-Brown, Ngo, & Wee, 2012; Luppino et al., 2010; Needham, Epel, Adler, & Kiefe, 2010; Pan et al., 2012). Results from some research indicate a stronger relationship for depression-to-obesity than for obesity-to-depression (Fowler-Brown et al., 2012; Luppino et al., 2010; Needham et al., 2010; Pan et al., 2012); however, Faith et al. (2011) reported a stronger relationship for the obesity-to-depression link than for the depression-to-obesity link. Once again, research in this area provides conflicting results. However, an interesting connection was drawn from a couple of studies (Luppino et al., 2010; Pine et al., 2001) that reported length of depression, as opposed to just the presence of depression, at baseline to be associated with higher levels of future obesity. Evidence is strong for the bidirectionality of the link between obesity and depression. The potential for depression to lead to obesity was well-supported in the current study.

A second possible mediator between religiosity and obesity is social support. Researchers (Kim & Sobal, 2004; Kim, Sobal, & Wethington, 2003) hypothesized that

religion provides an opportunity for individuals to enhance their social networks and thereby increase social support. Social support may include emotional, instrumental, appraisal, and informational benefits to the individual (Berkman, 2004) that can positively impact health in a variety of ways. Researchers have proposed various relationships between religiosity, social support, and health, including the possibility that religiosity has a direct protective effect on health (Powell, Shahabi, & Thoresen, 2003), that social support mediates the relationship between religiosity and health (Diener, Tay, & Myers, 2011; Koenig & Vaillant, 2009), and that social support provides a direct protective effect on health (Brown, Salsman, Brechting, & Carlson, 2007).

Two recent studies provide evidence for the relationship that exists between religiosity and social support (Moxey, McEvoy, Bowe, & Attia, 2011; Schnall et al., 2012). Moxey et al. (2011) identified increased religious attendance to be related to increased social support among the aging population in Australia, and Schnall et al. (2012) reported more-than-weekly religious attendance to be associated with the emotional/informational construct of social support. Another concept that emerges from the literature and provides a direct connection between religiosity and social support is that of religious social support.

Religious social support has been widely researched in recent studies (Debnam, Holt, Clark, Roth, & Southward, 2012; Holt, Wang, Clark, Williams, & Schulz, 2013; Krause & Hayward, 2013). Debnam et al. (2012) determined that religious social support can benefit the individual beyond the benefit achieved from general social support. In regard to religious social support, Stroope (2012) reported that the religious activity of

church attendance impacted health to a greater degree than did religious belief. There is also evidence that religious social support interacts more directly with emotional health (such as depression) than with physical health (Holt et al., 2013).

Social support is identified in research as a mediator between religiosity and health measures of subjective well-being (Assari, 2013; Diener, Tay, & Myers, 2011) and suicidal ideations and attempts (Robins & Fiske, 2009). However, no known studies have considered the mediational role of social support for the link between religiosity and obesity. The final relationship of interest in the current study is the other half of the mediational relationship–the relationship between social support and obesity.

Christakis and Fowler (2007) provided the most prominent discussion of obesity and social support in recent times. Christakis and Fowler found that obesity levels were high within the social networks of those who were obese and that these increased relationships persisted up to three degrees of separation from the obese individual. Researchers have discovered the same social interaction with obesity levels to exist among younger adults than those studied by Christakis and Fowler (Leahey, LaRose, Fava, & Wing, 2011) and among adolescents (Halliday & Kwak, 2009; Valente, Fujimoto, Chou, & Spruijt-Metz, 2009). Interestingly, the observation of obesity clustering is identified for friends and family of the obese individual but not for neighbors or colleagues (Christakis & Fowler, 2007; Leahey et al., 2011). In all, apart from these few acknowledgements that obese individuals have greater social connections with those who are also overweight or obese, there is a dearth of research connecting social support

to obesity. However, there is substantial research relating social support to physical activity, and physical activity can have an effect on obesity levels.

Engaging in physical activity is an essential part of maintaining healthy weight. Although friends are considered to be important social connections for adolescents, researchers have identified family support as instrumental in promoting physical activity for the adolescent (Baskin et al., 2013; de la Haye, Robins, Mohr, & Wilson, 2010; Trost & Loprinzi, 2011). At the adult level, physical activity may be more influenced by the presence of friends and family who are physically active themselves (Leroux, Moore, Richard, & Gauvin, 2012). For some adult populations, social support for engaging in physical activity is garnered from religious affiliation (Kegler et al., 2012) or from activity companions (Harley et al., 2009). Therefore, while scientific research connecting social support to obesity is scarce, research does support the connection between social support and physical activity, which has potential for influencing obesity level.

I have demonstrated that religiosity is associated with health (primarily measured as longevity), depression, and social support and that obesity is associated with depression and social support. While some of these noted associations are supported longitudinally, many are supported by cross-sectional research. Furthermore, although research consistently supports the connection between religiosity and health, the connection between religiosity and a prominent measure of health—obesity—is less supported. The current study provides insight into the multidimensional interaction that occurs between spiritual, social, and emotional dimensions of wellness in the longitudinal development of obesity from adolescence to adulthood. Such insight can inform

practitioners of holistic tools for treating and preventing the development of obesity among individuals traversing a volatile life transition—the transition from adolescence to adulthood.

Statement of the Problem

There is an obesity problem in the United States, as witnessed by the 16.9% rate of obesity among adolescents (Ogden, Carroll, Kit, & Flegal, 2012) and the 35.3% rate of obesity among U.S. adults (National Center for Health Statistics, 2012). For those who are normal weight during adolescence, the risk for developing obesity as an adult increases with time; Juonala et al. (2011) reported that 14.6% of normal-weight adolescents were obese 23.1 years later, and Gordon-Larsen et al. (2010) reported a doubling in the number of obese individuals between adolescence and young adulthood. Health risks related to obesity include immediate and future physical, social, and psychological challenges that make treating and preventing obesity one of the priorities of the national health initiative Healthy People 2020 (Department of Health and Human Services, 2014).

Religion is also a prominent cultural aspect of life in the United States. Gallup Poll research identified that in 2011, 92% of Americans answered *yes* to the question "Do you believe in God?" (Newport, 2011), and in 2013, 56% of respondents identified religion as *very important* in their life (Newport, 2013). Indeed, there has been a burgeoning response by researchers in the past few decades to identify potential links between religion and various markers of health, much of which has been synthesized by

Koenig, King, and Carson (2012) in their second edition of the *Handbook of Religion and Health*.

Researchers have considered the connections that exist between religious practices and obesity; lifestyle behaviors (i.e., exercise, smoking, and alcohol use), social support, and depression are identified as protective factors in health (Powell, Shahabi, & Thoresen, 2003). Commonly, researchers consider single connections to obesity, such as social support (Leahey, LaRose, Fava, & Wing, 2010), depression (Zhao, Ford, Li, Tsai, Dhingra, & Balluz, 2011), or religious affiliation (Dodor, 2012), and they report on any significant relationships that are observed. However, the results of these studies in aggregate remain inconclusive and often conflicting, primarily due to cross-sectional design or use of individuals in fairly stable states of development.

Furthermore, health is understood to be more than the absence of illness (Hoeger & Hoeger, 2015); health reflects a multidimensional model that includes physical, emotional, social, intellectual, spiritual, environmental, and occupational dimensions (Hoeger & Hoeger, 2015). Despite the presumed connections that exist between dimensions of wellness, researchers rarely view health outcomes in light of multiple dimensions. The problem is that health reasearchers do not have an understanding of the potential role of multiple dimensions of health in the development of a single dimension. Although researchers do know that adolescent religiosity relates to reduced levels of depression and to increased levels of social support for health behaviors such as physical activity and stress management, and researchers know that depression and social support relate to the development of obesity, health reseachers do not know how adolescent

religiosity relates to the development of obesity over time and whether this is mediated by depression and social support.

Purpose of the Study

The purpose of this quantitative study was to explore the longitudinal relationship that exists between adolescent religiosity and adult obesity and to explore such a relationship from a multidimensional health model perspective. This study considered the potential role that social support and depression play as mediators of the adolescent religiosity–adult obesity relationship. Furthermore, this study provides more insight into relationships that have been inconsistently evaluated in previous research, including (a) the religiosity–depression relationship, (b) the religiosity–social support relationship, (c) the depression–obesity relationship, and (d) the social support–obesity relationship. The independent variable in this study was adolescent religiosity, and the dependent variable was adult obesity. The mediational variables were social support and depression. The current study design controlled for gender, race/ethnicity, smoking status, initial depression, factors of socioeconomic status, and obesity status, as these variables have impacted the results of previous studies.

Research Questions and Hypotheses

The following research questions and hypotheses were elicited from the literature surrounding obesity, religiosity, depression, and social support. In Chapter 3, I provide a more detailed discussion of the nature of the study.

Research Question 1. What is the relationship that exists between adult obesity and adolescent religiosity, depression, and social support?

Null Hypothesis 1. There is no statistically significant relationship between adult obesity and adolescent religiosity, depression, and social support. Obesity was measured using body mass index (BMI); religiosity was measured by responses to questions of internal and external religiosity; depression was measured by a variation of the Center for Epidemiologic Studies Depression Scale (CES-D); and social support was measured by responses to eight questions about perceived adult, teacher, parent, and friend care for the individual and family dynamics—all measures were taken from the Add Health data set (see Table 1 for Add Health Wave information).

Alternative Hypothesis 1. There is a statistically significant relationship between adult obesity and adolescent religiosity, depression, and social support. Obesity was measured using BMI; religiosity was measured by responses to questions of internal and external religiosity; depression was measured by the CES-D; and social support was measured by responses to eight questions about perceived adult, teacher, parent, and friend care for the individual and family dynamics—all measures were taken from the Add Health data set.

Research Question 2. To what extent does adolescent depression mediate the relationship between adolescent religiosity and adult obesity?

Null Hypothesis 2. Adolescent depression is not a statistically significant mediator of the relationship between adolescent religiosity and adult obesity. Adolescent depression was measured by the CES-D; adolescent religiosity was measured by internal and external religiosity questions; and adult obesity was measured by BMI—all measures were taken from the Add Health data set.

Alternative Hypothesis 2. Adolescent depression is a statistically significant mediator of the relationship between adolescent religiosity and adult obesity. Adolescent depression was measured by the CES-D; adolescent religiosity was measured by internal and external religiosity; and adult obesity was measured by BMI—all measures were taken from the Add Health data set.

Research Question 3. To what extent does adolescent social support mediate the relationship between adolescent religiosity and adult obesity?

Null Hypothesis 3. Adolescent social support is not a statistically significant mediator of the relationship between adolescent religiosity and adult obesity. Social support was measured by responses to eight questions about perceived adult, teacher, parent, and friend care for the individual and family dynamics; adolescent religiosity was measured by internal and external religiosity questions; and adult obesity was measured by BMI—all measures were taken from the Add Health data set.

Alternative Hypothesis 3. Adolescent social support is a statistically significant mediator of the relationship between adolescent religiosity and adult obesity. Social support was measured by responses to eight questions about perceived adult, teacher, parent, and friend care for the individual and family dynamics; adolescent religiosity was measured by internal and external religiosity questions; and adult obesity was measured by BMI—all measures were taken from the Add Health data set.

Theoretical Foundation

The theoretical framework for this study was the dynamic, multidimensional, functional model for holistic health presented by Hawks (2004). Hawks theorized a

framework whereby spiritual health is the catalyst for the development of social and emotional health that then results in enhanced levels of physical and intellectual health (Figure 1). Hawks (2004) referred to spiritual health as the "linchpin" (p. 14) in holistic health models. As such, Hawks posited that spiritual health provides a basis for meaning and purpose in life that meets a basic life need, allowing the individual to pursue social and emotional wellness. With the basis of spiritual, social, and emotional wellness, the individual is then empowered to engage in wellness-enhancing physical behaviors that support the goal of purpose and meaning in life (Hawks, 2004). This study only considered the paths by which spiritual health impacts social and emotional health with further impact on physical health; the intellectual outcome was not examined.

The model presented by Hawks uses multiple interactions between various dimensions of health and identifies the process of health change as beginning with the spiritual dimension. The spiritual, social, emotional, and physical dimensions of health are multifaceted and therefore impossible to consider in aggregate; as a result, I chose measures to represent each dimension as follows: religiosity was a measure of the spiritual dimension; social support was a measure of the social dimension; depression was a measure of the emotional dimension; and obesity was a measure of the physical dimension. For each of these measures, it should be noted that the construct chosen was merely a subset of the dimensions. Religiosity is a subset of the spiritual dimension in that religious individuals would be considered spiritual, but spiritual individuals might not be religious. Religiosity does, however, align with the concept of a *worldview* and with *commitment* as identified by Hawks in Figure 1. Similarly, looking at depression

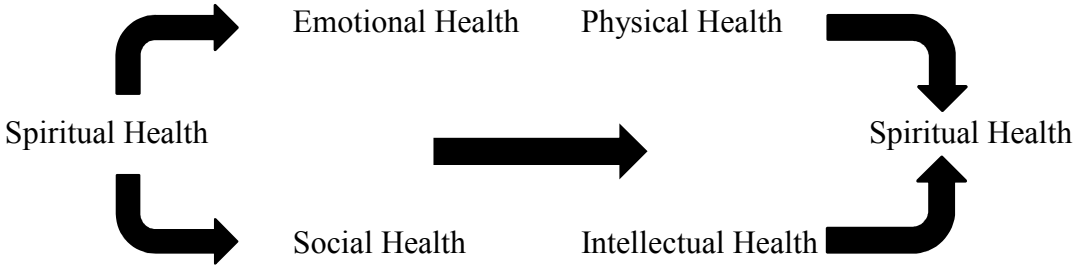
symptoms is one way in which to view emotional wellness, looking at social support is one way in which to view social wellness, and obesity level is one aspect of physical wellness. All of these variables are discussed more fully in Chapter 2, and the measures are detailed in Chapter 3. The multidimensional model proposed by Hawks entwines appropriately with mediational analysis. Hawks proposed a spiritual impact on a physical outcome through the conduit of emotional and social interactions; this study considered the impact of adolescent religiosity (one way to represent spirituality) on adult obesity (one possible measure of physical wellness) as mediated by adolescent social support (one possible measure of social wellness) and adolescent depression (one possible measure of emotional wellness).

Table 1

Add Health Participant Information for Waves I, II, and IV

Wave	Year of collection	Participants
I: In-home	April 1995-December 1995	20,745 U.S. adolescents
II	April 1996-August 1996	14,738 from Wave I with a few exceptions
IV	January 2008- February 2009	15,701 from Wave I

Note. Information taken from http://www.cpc.unc.edu/projects/addhealth/design/designfacts



Worldview	Experience Emotion	Fitness and Strength	Realization of the
Commitment	Express Emotion	Health Behaviors	higher purpose
Higher Power	Love and Acceptance	Logic and Reason	outlined by the
Ethics and Values	Sense of Belonging	Judgment and Sense	spiritual
Purpose and Meaning	Relationships	Wisdom	worldview

Figure 1. A dynamic, functional, multidimensional model of holistic health. From "Spiritual Wellness, Holistic Health, and the Practice of Health Education," by S. Hawks, 2004, *American Journal of Health Education, 35*(1), 14. Used by permission.

Nature of the Study

This study was quantitative in nature and used a prospective design. Creswell (2009) identified that quantitative research is consistent with predictive models in which one or more variables are used to predict a dependent variable. The independent variable in this study was adolescent religiosity, and the dependent variable was adult obesity. Adolescent social support and adolescent depression were mediational variables. This study took advantage of the longitudinal data collected in the Add Health (Harris & Udry, 2014) study, which followed adolescents through to adulthood from 1994 through 2008. Mediation analysis was carried out using multiple regression and based on procedures detailed by Baron and Kenny (1986); the Sobel test (Sobel, 1982), as outlined by Preacher and Hayes (2008), was identified as a method to determine the indirect effect of the mediation.

Definition of Terms

Following are operational definitions of terms used in this manuscript. Study variables are conceptualized from data gathered in the Add Health (Harris & Udry, 2014) study; their construction from the Add Health data is detailed more fully in Chapter 3.

Add Health (Harris & Udry, 2014) is a study that began in 1994 and includes a multitude of survey responses related to physical, social, and emotional factors. The study is representative of adolescents in Grades 7-12 in the United States in 1994 and to date includes four waves of data with the fourth wave culminating in 2008 and a fifth wave scheduled for 2015.

Body mass index (BMI) is a calculation based on one's height and weight that indicates body fatness (Centers for Disease Control and Prevention [CDC], 2014). BMI is calculated in the same way for all individuals but is interpreted differently for adults than for children. Adult classifications of underweight, normal weight, overweight, and obese are based on ranges of BMI; however, children and adolescents are classified based on age- and sex-specific percentiles (CDC, 2014).

The *Center for Epidemiologic Studies Depression Scale (CES-D;* Radloff, 1977) is a 20-item self-report scale that measures symptoms of depression (Masood & Okazaki, 2006). Each item is weighted based on the occurrence of depressed mood, with total scores ranging between 0 and 60; a cut score of 16 is used to identify the need for diagnosis of depression, and higher scores indicate increased symptoms of depression (Masood & Okazaki, 2006).

Depression is a mood disorder characterized by severity and duration and relating to a combination of emotional, cognitive, behavioral, and physical symptoms (Butcher, Mineka, & Hooley, 2010, pp. 220-221).

Extrinsic religiosity is an interest in religion for the gain it can provide in other areas such as status, comfort, or social standing (Allport & Ross, 1967). Allport and Ross (1967) identified extrinsic religious orientation to be expressed by lack of commitment to the tenets of the religion and by self-focus rather than God-focus.

Intrinsic religiosity is a genuine assimilation of religion into one's life whereby one subscribes to the beliefs of the religion and one's motives are outcomes of that belief (Allport & Ross, 1967).

Obesity is an overfat condition carrying with it an increased risk for various comorbidities (McArdle, Katch, & Katch, 2010) including hypertension, dyslipidemia, type 2 diabetes, coronary heart disease, stroke, gallbladder disease, osteoarthritis, sleep apnea, and endometrial, breast, and colon cancer (CDC, 2014).

Religiosity is a measure of "one's religious beliefs/commitment/convictions" (Young, 2011, p. 5) and has been identified in health literature as a self-report of one's closeness to God (Cecero, 2005).

Social support as an early construct was presented through four subtypes: emotional support, appraisal support, belongingness, and instrumental support (Cohen & Wills, 1985). More recently, Berkman (2004) identified the subtypes of social support as emotional support, instrumental support, appraisal support, and informational support and defined each as follows: *emotional support* represents feelings of value, love, and care

received from others; *instrumental support* includes tangible forms of aid available to the individual; *appraisal support* is support that aids the individual in decision making; and *informational support* comes in the form of advice. Berkman acknowledged that emotional, appraisal, and informational support can be difficult to distinguish from each other.

Assumptions

It was assumed that BMI provides an appropriate measure of obesity for adolescents and for adults. The CDC (n.d.) warns that, for children, BMI does not distinguish well between fat and fat-free mass unless the child is obese. While BMI predominates the way in which obesity level is identified in scientific studies, an increased BMI could result when an individual engages in resistance training with a concomitant increase in muscle mass rather than fat mass. Given the thousands of participants who were included in the Add Health study, using height and weight to calculate BMI, versus another more time-consuming method, was the most realistic method for considering a measure of obesity. It was also assumed that self-reported height and weight at Wave I of the Add Health study are significantly reflective of actual height and weight values of the adolescents being surveyed at the time. Regarding self-reporting of height and weight by adolescents in the Wave I data collection, a preferable method would have been for actual heights and weights to be measured by the interviewer. However, the data collection method for Wave I was determined many years ago, and nothing could be done to change that collection for this study. Another assumption of this study was that the CES-D consistently measures the presence of

symptoms of depression among adolescents and young adults. The Add Health study used a slightly modified version of the CES-D. There is evidence that the CES-D is appropriate for use in detecting depression symptoms among children and adolescents as well as among adults.

Scope and Delimitations

In this study, I tested the dynamic, multidimensional, functional model for holistic health presented by Hawks (2004), in which he proposed that spiritual health initiates social and emotional health benefits that result in physical and emotional health outcomes. Because the scope of such variables would have been incredibly expansive, one construct was chosen to represent each variable of interest. In this study, the construct of religiosity represented spiritual health, the construct of social support represented social health, the construct of depression represented emotional health, and the construct of obesity represented physical health. The primary threat to validity in this study was the threat of construct validity based on the appropriateness of each selected construct to represent the stated dimension of health. Religiosity was chosen to represent the spiritual dimension based on the variety of ways in which religiosity can be identified. Common considerations of religiosity are religious denomination, religious practice of prayer or of religious attendance, intrinsic religiosity, and extrinsic religiosity. Following the example of Le, Tov, and Taylor (2007), I used the Add Health questions on religion to identify an internal (intrinsic) religiosity factor and an extrinsic religiosity factor, both of which were combined into one religiosity scale that represented spiritual health.

For emotional health, the construct of depression as measured by 19 questions derived from the CES-D was used. The CES-D can be used to identify the threshold for clinical depression with a cut score of 16 on the 20-question version. Conversely, the score can be used as a continuous score, as done by Le et al. (2007), with higher scores representing greater depression symptomology. Social health was viewed as a measure of social support also derived from the Add Health data. Kort-Butler (2010) used four questions from the Add Health data to elicit a mean score representative of social support. The four questions used in the Add Health study concerned the respondent's feelings of care derived from adults, teachers, parents, and friends. Beaver, Boutwell, and Barnes (2014) included four more questions about family dynamics. The full eight questions were used as a measure of social support. The measure of physical health in this study was obesity level as indicated by BMI. BMI is based on height and weight of the individual, and in the Add Health study, height and weight were self-reported in the Wave I data but were measured by the interviewer in Wave IV. Inconsistencies in self-reports at Wave I may be possible, but self-reports for these measures are generally accepted and supported (Bowring et al., 2012; Fonseca et al., 2010; Yoshitake, Okuda, Sasaki, Kunitsugu, & Hobara, 2012).

This study was delimited to adolescents who participated in Wave I (1994-1995) of the Add Health study, who remained in the study through Wave IV (2008), and who were included in the public-use data provided by the study coordinators. This study was delimited to four out of five dimensions of health proposed by Hawks (2004) in his multidimensional health model. Hawks included an outcome of intellectual health that

was not considered in this study. A final delimitation in this study was the use of one construct for each dimension of health: religiosity for the spiritual dimension; depression for the emotional dimension; social support for the social dimension; and obesity for the physical dimension.

As a result of these delimitations, this study is only generalizable to a specific subset of the American population—those who were adolescents in the United States in the mid-1990s. The Add Health study used a nationally-representative sample of U.S. adolescents in their study design. Results from this study cannot be used to identify relationships for current adolescents moving forward in respect to religiosity, depression, social support, or obesity. This study elucidated the connection that existed, for those growing up in the United States in the mid-1990s, between religiosity and the longitudinal development of obesity. However, care needs to be taken not to graph these relationships onto today's adolescents.

Limitations

This study was reliant on secondary data from the Add Health study. Use of the Add Health data allowed for the longitudinal perspective that is needed for a greater understanding of the development of obesity in the United States; however, the Add Health data were not collected with my study in mind, thereby limiting my ability to provide the most current constructs and measures. The Add Health study design is detailed by its authors on their website. In summary, the study used systematic sampling and implicit stratification to elicit a representative sample of U.S. adolescents from 80 high schools and 52 middle schools; furthermore, this sample is representative of region,

urbanicity, school size and type, and ethnicity among the U.S. adolescent population in 1994-1995 (Harris et al., 2009). The Add Health data included four completed waves of data collection, strengthening the quasi-experimental panel design used for the study over cross-sectional or correlational designs (Frankfort-Nachmias & Nachmias, 2008, p. 123). With a quasi-experimental design, the Add Health data risked a reduction of strength for internal validity; however, such reductions can be accounted for by more rigorous statistical analysis (Frankfort-Nachmias & Nachmias, 2008).

The Add Health study sample protocols account for many of the possible threats to internal validity. The sample size for Wave I was extremely large (interviews of 20,745 adolescents; Harris et al., 2009), accounting for potential dropouts from the study (Creswell, 2009). Add Health researchers negated the potential maturation threat by sampling a large age range (Grades 7-12) and following participants for a considerable amount of time (Creswell, 2009). By randomly choosing study participants, the Add Health researchers also negated the potential selection threat to internal validity, and the testing threat and instrumentation threats were avoided by using years or multiple years between testing and by using the same questions during subsequent waves, respectively (Creswell, 2009).

Despite these strong properties of the Add Health design, this study is limited in its generalizability to current youth, given that the Add Health participants were adolescents approximately 20 years ago. Furthermore, the Add Health data do not allow for a consistent measure of the mediator variables of interest in this study—depression and social support. The time period between Wave I and Wave II of the Add Health data

was approximately one to one-and-a-half years, and the period between Wave II and Wave IV was approximately 11 to 12 years. The disparity in time frame between the various waves of data posed problems with analysis of mediation for the given variables. Finally, depression and social support were not measured over the entirety of the wave; rather, measures for depression and social support reflect a point in time for each of the study participants.

As mentioned earlier, the greatest threats to validity in this study were the threats to construct validity. BMI is the default measure for obesity that was used in the current study due to a lack of other measures to consider from the Add Health data. BMI does not distinguish between weight that is related to fat mass and weight that is related to fat-free mass. Furthermore, while researchers have identified Add Health questions for use as depression, religiosity, and social support measures, only the depression questions, which are derived from the CES-D, were directly related to an identified psychometric scale.

There are a few confounders that were controlled for in this study. In relation to obesity, smoking status is a common confounder by which those who smoke tend to exhibit lower weight. Other known confounders for obesity and for depression are gender and age. Furthermore, race/ethnicity can impact some of the variables that were considered in this study. Socioeconomic status is often controlled for in obesity studies. Finally, weight status and depression status at baseline are known to impact various measures longitudinally, and, therefore, were accounted for in this study.

Significance

Adult obesity is likely to result from multiple contributing factors. This study contributes to a greater understanding of the development of obesity from adolescence through adulthood. The research was unique in that it considered a multidimensional view of the development of obesity from adolescence to adulthood and did so with the support of longitudinal data. There are many studies that correlate obesity rates with one other area of interest, but few studies consider multiple health dimensions in relationship to the development of obesity, and even fewer studies do so from a longitudinal perspective. The current study used both a longitudinal and multidimensional approach to understanding the development of obesity during the time of prominent changes in obesity—the transition from adolescence to adulthood.

In the area of the multidimensionality of health, this study contributes to health education as well. Health education philosophy continues to promote a theoretical connection between multiple dimensions of health, but with little empirical support for such a connection (Hawks, 2004). Religiosity was shown to impact the development of obesity longitudinally and in relation to social support and depression, thereby supporting the interrelational quality of health dimensions

When solid data are analyzed appropriately, and when results from that analysis are shared in a reflective manner consistent with the way in which the data were collected, researchers and practitioners can implement outcomes in meaningful ways. In the United States, 72 million adults are identified as obese (CDC, 2010); therefore, the application of the outcomes of this study is far-reaching within the American population.

This study may influence the way in which health professionals view the development of obesity, which may result in identifying better ways to support those at greatest risk for developing obesity using more holistic approaches to health. This study promotes positive social change by providing a greater understanding of the development of obesity from adolescence to adulthood that will allow for inclusion of more appropriate preventive and supportive strategies for lessening the rate of obesity increases. These supports will benefit both the individual and society as a whole in relation to the physical, emotional, social, and spiritual aspects of wellness. Monetary factors of wellness are also important to the discussion of supporting a healthier population. Both personal health care costs and societal health care costs can be reduced with focused effort toward identifying the causes of obesity in the transition from adolescence into adulthood. Perhaps most importantly, support and prevention during the transition from adolescence to adulthood may help to reduce the debilitating physical and social effects often related to obesity status.

Summary

Religion is a known factor in multiple health outcomes (Koenig, King, & Carson, 2012), and one notable connection is the relationship between increased religiosity and longevity (Rogers, Krueger, & Hummer, 2010). However, there remains a lack of conclusive evidence for the relationship that exists between religiosity and the health marker of obesity. Hawks (2004) proposed a theory of health relationships in which spiritual health initiates emotional and social health benefits that enhance physical and intellectual health outcomes. This study addressed the framework for these interactions

by exploring the relationship between religiosity and obesity. Social support and depression were considered for their role in mediating the religiosity–obesity relationship. In this study, religiosity was the measure chosen to represent the spiritual dimension of health, obesity was the measure chosen to represent the physical dimension of health, social support was the measure chosen to represent the social dimension of health, and depression was the measure chosen to represent the emotional dimension of health.

Evidence regarding the religiosity–obesity link is supported but conflicting and generally identifies an increase in religiosity to associate with an increase in obesity or identifies no relationship to exist (Cline & Ferraro, 2006; Dodor, 2012; Feinstein, Liu, Ning, Fitchett, & Lloyd-Jones, 2010; Kim, Sobal, & Wethington, 2003; Yeary et al., 2009). This study considered depression as a mediator of the religiosity–obesity relationship; therefore, a relationship between religiosity and depression and a relationship between depression and obesity were identified. The evidence for increased religiosity leading to decreases in depression symptoms is well-supported in the literature (Berry & York, 2011; Jansen, Motley, & Hovey, 2010; Koenig, King, & Carson, 2012; Sun et al., 2012; Yonker, Schnabelrauch, & DeHaan, 2012), and this negative relationship is particularly supported for the adolescent population (Ji, Perry, & Clarke-Pine, 2011; Meltzer et al., 2011; Pössel et al., 2011). The literature also supports a significant relationship between depression and obesity (Blaine, 2008; Faith et al., 2011; Goodman & Whitaker, 2002; Stice, Presnell, Shaw, & Rohde, 2005).

The role of social support as a mediator of the religiosity-obesity relationship also requires significant relationships to exist between religiosity and social support and between social support and obesity. Much of the expected support for the religiosity-social support link can be gained from the construct of religious social support. Debnam, Holt, Roth, and Southward (2012) demonstrated that religious social support is an added benefit for those who are receiving general social support. Furthermore, researchers have identified social support to be a mediator between religiosity and subjective well-being (Assari, 2013), between religious attendance and major depression (Ai, Huang, Bjorck, & Appel, 2013), and between religiosity and trait anxiety (Hughes et al., 2004). Finally, Christakis and Fowler (2007) provided support for the relationship between social support and obesity by noting that obesity clusters persist to three degrees of separation. In total, the relationships needed to identify social support and depression as mediators of the religiosity–obesity relationship are supported by research, and, therefore, the proposed multidimensional model of dimensions of health presented by Hawks (2004) was considered.

This study adds to the scientific literature in the area of the development of obesity from adolescence to adulthood. Few studies have considered the religiosity–obesity relationship, and even fewer have done so longitudinally. The literature review that follows in the next chapter is silent regarding studies that consider the longitudinal connection between religiosity and obesity for the adolescent population as it matures into adulthood. Such a perspective, as presented by this study, provides insight into three important aspects of the American lifestyle—religiosity, depression, and obesity—and

allows for better preemptive work to impede the spread of obesity as well as to strengthen the way in which practitioners treat those struggling with obesity. In Chapter 2, statistics related to obesity, religiosity, and depression and research including the multiple relationships between religion, depression, social support, and obesity are discussed in detail.

Chapter 2: Literature Review

Introduction

Obesity develops when calories consumed exceed calories expended; however, humans are multidimensional beings. It is often the case that to understand one outcome (health or otherwise), an appropriate approach is to look beyond what may seem to be the immediate connections to the outcome. The development of obesity in a given individual or population may be related to psychological, social, spiritual, and/or biological factors. Researchers have identified multiple links to obesity, including race (Kirby, Liang, Chen, & Wang, 2012), levels of depression (Wiltink et al., 2013), smoking status (Gümüş et al., 2013), socioeconomic status (Fradkin et al., 2014), social support (Oliveira, Rostila, de Leon, & Lopes, 2013), and activity levels (Brumby et al., 2013), to name just a few. Despite evidence for multiple links to obesity, the nature and strength of these links are not well understood. Much of the recent research in the area of obesity has been more focused on longitudinal connections in order to provide a greater understanding of the efforts of earlier research, which centered primarily on cross-sectional relationships between obesity and other variables. In this literature review, I identify the specific relationships observed in research between obesity and various expressions of other dimensions of health. I present a view of obesity in which multiple dimensions of health are potential precursors to the development of adult obesity.

The theoretical foundation of this dissertation is grounded in a model of health developed by Hawks (2004) in which spiritual health is viewed as a precursor to social and emotional health with further impact on physical and intellectual health. The holistic

model of health presented by Hawks focuses on the role of spiritual health as the necessary ingredient to effect behavioral change; such an approach is at odds with researchers who have been reluctant to incorporate the dimension of spiritual health into health models. However, when asked in a Gallup Poll, "Do you believe in God," 98% of Americans answered *yes* in 1967; more recently, in 2011, Gallup reinstated this polling question and the affirmative response rate was 92% (Newport, 2011). Furthermore, 56% of respondents in a 2013 Gallup Poll indicated that religion was *very important* in their own life; since 1992, between 54% and 61% of Americans stated that religion was *very important* in their own life (Newport, 2013). In 2005, Marks wrote about the role of religion as a significant aspect of health among the U.S. population. Marks (2005) presented a conceptual model of religion and bio-psycho-social health in which he connected religious practices to biological health, religious beliefs to psychological health, and religious community to social health. These four health aspects (spiritual, physical, psychological, and social) are the key constructs being considered in this dissertation.

Social understanding of the dimensions of health, from a health education perspective, has changed significantly over time. Primarily, this change can be identified as a growing acknowledgement of the various ways in which to view health and the avenues through which health can be expressed. Cottrell, Girvan, and McKenzie (2009) explained that while the definition of health is ambiguous, many accept the concept of the multidimensionality of health (p. 85). Many, if not all, health texts include discussion of the multidimensionality of health. As an example, Hoeger and Hoeger (2015) identified

seven dimensions of health (also referred to as *wellness*) as including social, physical, spiritual, mental, emotional, environment, and occupational aspects (p. 7). Whereas Ancient Greek philosophers focused their attention on reconciling the mind and bodily aspects of humanity (Clark, 2009), today's culture typically acknowledges that spiritual, social, and psychological influences also add to the multidimensionality of humans.

Marks (2005) demonstrated an understanding of the significance of the spiritual realm as it pertains to matters of health. However, health educators are reticent about placing the dimensions of health into a hierarchy and often default to talking about physical health above any other dimension of health; reviews of prominent health texts, including the texts by Fahey, Insel, and Roth (2015) and by Hoeger and Hoeger (2015), provide evidence of the primary focus on physical health. Hawks (2004), however, proposed a conceptual model that clearly positions various dimensions of health in a hierarchy.

Hawks's (2004) theoretical model was informed by contemporary components of health education, which portray health as multidimensional (physical, spiritual, social, emotional), dynamic in the the effect of one dimension on another, and functional for reaching higher purposes. Hawks challenged the notion of the multidimensionality of wellness by identifying the overwhelming focus of health promotion to be at the physical level. For Hawks, the inconsistency between health philosophy (multidimensionality) and health education practice (focus on the physical) presents a challenge for current researchers on three levels, as follows: (a) breaking away from the focus of health as being primarily a physical component; (b) identifying widely accepted operational

definitions of measures for the other dimensions of wellness; and (c) breaking through the barrier of social and academic resistance to the role that spirituality can play in health outcomes. As such, Hawks proposed spiritual health to be the catalyst for development of the other dimensions of health.

Specifically, Hawks's model placed spiritual health at the forefront of the development of emotional and social health based on the concept that spiritual aspects (purpose and meaning in life) are grounded in a well-defined worldview. This worldview provides the individual with purpose, meaning, and moral values that support the positive development of emotional and social wellness. Hawks further posited that social, emotional, and spiritual health work synergistically to enhance physical and intellectual health dimensions (which he viewed as supported by the literature) and that behavioral change emmanates from higher levels of physical and intellectual wellness, thus allowing the individual to gain fulfillment in purpose and meaning in life. Therefore, Hawks's holistic health model, as informed by the concepts of the multidimensionality of wellness, is the theoretical framework for this dissertation.

Research findings support the individual relationships identified in the theoretical framework presented by Hawks (2004). For example, religiosity (a measure of spiritual wellness) is associated with depression (a measure of emotional wellness: Balbuena, Baetz, & Bowen, 2013; Berry & York, 2011; Koenig, 2009; Miller et al., 2011; Pirutinsky et al., 2011; Sun et al., 2012; Yonker, Schnabelrauch, & DeHaan, 2012) and with social support (a measure of social wellness: Moxey, McEvoy, Bowe, &Attia, 2011; Schnall et al., 2012). Furthermore, depression (Blaine, 2008; Faith et al., 2011) and social

support (Christakis & Fowler, 2007; Halliday & Kwak, 2009; Leahey, LaRose, Fava, & Wing, 2011; Valente, Fujimoto, Chou, & Spruijt-Metz, 2009) are associated with obesity (a measure of physical wellness). In this archival study, I identify the relationship between a measure of adolescent spiritual health (religiosity) and a measure of adult physical health (obesity) using longitudinal data. Furthermore, I consider the mediator effects of measures of adolescent emotional and social health upon the relationship between religiosity and obesity.

I conducted a literature search primarily using the Academic Search Premier and ProQuest databases, which include a number of peer-reviewed psychological journals. I based my initial searches on combinations of key words such as *obesity* and *religiosity*, *obesity* and *social support*, *obesity* and *depression*, *religiosity* and *depression*, and *religiosity* and *social support*. Search results varied for each combination, with *obesity* and *depression* yielding the most hits at over 4,000 and *religiosity* and *obesity* yielding the fewest hits at 59. I did not initially restrict these searches by date, so studies for inclusion in this review were identified by skimming the titles to confirm relevance and by identifying work done within approximately the previous 5 years. When searches were limited to 2009 and more recent, search results were as follows: *obesity* and *religio** yielded seven studies; *obesity* and *spiritual** yielded 12 articles; *obesity* and *depression* not including *treatment* or *prevention* yielded 507 articles (many of which were still not relevant to the main focus of the present study); *obesity* and *social support* not including *postnatal* or *intervention* yielded 66 articles; *religio** and *social support* yielded 86 articles; *religio** and *depression* not including *treatment* or *prevention* yielded 328

articles; and the combination of *religio** and *depression* and *social support* and *obesity* yielded one article. In general, the further down the list an article was located, the less it was directly related to the present study. Therefore, for the larger searches, I focused on the first 60-80 articles in the search results until the relevance to the present study dropped off dramatically. Some resources consistently pointed to seminal work in a particular field, and I searched for these references individually. Furthermore, I was able to locate a few more resources from reference sections of the articles I had identified through my searches.

In this chapter, I outline the key findings related to obesity research as connected to measures of religiosity, depression, and social support. This review includes cross-sectional and longitudinal findings related to obesity, including the development of obesity across the lifespan and, in particular, during the transition from adolescence to adulthood; conflicting conclusions and research in the areas of interest are also presented. Finally, I present the case for the use of longitudinal research data in providing deeper insight into multifaceted connections to the development of adult obesity.

The Obesity Epidemic: A Worldwide Challenge

Obesity is now considered to be a global trend and challenge. Overweight and obesity account for 5% of worldwide mortality and constitute the fifth-leading risk factor for mortality at the global level (World Health Organization, 2009). High blood pressure, high blood glucose, and physical inactivity were three of the top four risk factors associated with worldwide mortality risk, with rates of 13%, 6%, and 6%, respectively (World Health Organization, 2009); health practitioners recognize the significance of

these three risk factors as related to obesity. Furthermore, elevated BMI is one of eight risk factors associated with 61% of the mortality rate from cardiovascular disease worldwide (World Health Organization, 2009). Stevens et al. (2012) estimated global obesity prevalence to be 6.4% in 1980 and 12.0% in 2008, indicating that global obesity has effectively doubled during this 28-year span. Stevens et al. also indicated that half of the increase in global obesity occurred during the final 8 years of their investigation. Obesity, a contributor to the level of noncommunicable diseases (World Health Organization, 2013), figures prominently in worldwide health and mortality. Within the United States, researchers, as well as the health community at large, demonstrate high levels of interest in the effects and origins of overweight and obesity among their populations.

Obesity in the United States

Overweight and obesity rates in the United States have been of great interest to researchers and to the U.S. public. The 1988-1994 National Health and Nutrition Examination Survey (NHANES) measured adolescent (ages 12-19) obesity and reported an increase in obesity from 5% in the 1960s to 11% at the time of the NHANES testing (Ogden, Flegal, Carroll, & Johnson, 2002). The most recent calculation of obesity rates for children and adolescents recorded by the 2009-2010 NHANES is 16.9% (Ogden, Carroll, Kit, & Flegal, 2012). Furthermore, the total percentage of overweight and obese children in the Unites States in 2009-2010 was 31.8%, among which 12.3% had a body mass index (BMI) that met or exceeded the 97th percentile for their age (Ogden et al., 2012). Ogden, Carroll, Kit, and Flegal (2012) reported 2009-2010 adult obesity rates in

the United States to be 35.7%. From 1960-2010, adult overweight levels increased from 44.8% to 68.5% and obesity levels increased from 13.3% to 35.3% (National Center for Health Statistics, 2012). Clearly, obesity rates in the United States (as measured by BMI) have progressively increased from the 1960s to the present day.

The profound increases in overweight and obesity among all sectors of the U.S. population have been concerning for health professionals. However, some researchers argue that these increases appear to have plateaued. Ogden et al. (2012a) noted that the 16.9% obesity rate for U.S. children and adolescents seen in 2009-2010 had held relatively steady since the 2007-2008 calculations. Rokholm, Baker, and Sorensen, (2010) identified the potential levelling off of obesity rates to be a worldwide phenomenon. In their review of studies from 25 countries, they identified strong evidence that the United States, along with Australia, Denmark, England, and France, had experienced a plataeu for obesity among children and adolescents and that children and adolescents in Japan actually experienced a decrease in obesity. Therefore, it is possible that the trend for increased obesity has plateaued or even reversed in the United States as well as in other countries.

Perhaps obesity rates in the United States have stabilized; however, stabilization does not signify that all demographic sectors have remained constant. Ogden et al. (2012a) considered the demographic changes in obesity more thoroughly and determined that while the obesity rate for U.S. children and adolescents appeared to have settled, male obesity prevalence in ages 2 to 19 significantly increased while female prevalence did not from 1999 to 2010. Similarly, Rokholm et al. (2010) reported that overall U.S.

adult obesity remained stable from 2003 to 2007 despite noted increases among Mexican American men and women. Therefore, despite the possibility that obesity rates in the United States have stabilized, certain populations within U.S. society continue to experience increased levels of obesity.

Further evaluation of recent obesity rates in the Unites States revealed that among U.S. children and adolescents, males exhibited higher rates of obesity than did females; this increase was significant for non-Hispanic White children and adolescents but not for Hispanic or non-Hispanic Black children and adolescents (Ogden et al., 2012a). Other trends presented by Ogden et al. (2012a) included the realization that with increasing childhood and adolescent age (ages 2 to 5, 6 to 11, and 12 to 19), obesity rates increased, and that within the 12- to 19-year-old age range, non-Hispanic White adolescents exhibited the lowest obesity rate (12.2%) followed by Hispanic adolescents (15.8%); non-Hispanic Black adolescents exhibited the highest rate of obesity at 21.4%. Harris, Perreira, and Lee (2009) presented another way to view the changes in obesity from adolescence to adulthood. By longitudinally comparing growth curves, which were based on BMI, Harris et al. (2009) found that girls, Hispanic and Black populations, and generationally established immigrants demonstrated more rapid BMI increases in the transition from adolescence to adulthood than did their counterparts. These studies demonstrate that among adolescents, sex, age, and race/ethnicity impact obesity levels and rate of obesity.

Demographic breakdowns at the adult level have revealed some trends worth noting as well. Flegal, Carroll, Kit, and Ogden (2012b) noted an obesity prevalence rate

among U.S. men of 35.5%, with overall higher rates among non-Hispanic Black men (38.8%). The reported overall obesity prevalence rate for adult women was 35.8% (age-adjusted) with a rate for non-Hispanic White women of 32.2% and a rate of 58.5% for non-Hispanic Black women (Flegal et al., 2012). Obesity prevalence rates for both sexes, based on race/ethnicity, revealed that age-adjusted obesity rates were highest among non-Hispanic Black adults (49.5%), followed by Mexican American adults (40.4%), Hispanic adults (39.1%), and non-Hispanic White adults (34.3%; Flegal et al., 2012). When overweight is considered alongside obesity, age-adjusted prevalence rates in 2009-2010 were 81.2% for Mexican American adults, 78.8% for Hispanic adults, 76.7% for non-Hispanic Black adults, and 66.7% for non-Hispanic White adults. Finally, age adjusted obesity prevalence among men was lowest in the 20- to 39-year-old bracket (33.2%), increased to 37.2% in the 40- to 59-year-old bracket, and dropped down to 36.6% in the 60 and over age bracket; for women, the rates were 31.9% in the 20- to 39-year-old bracket, increasing to 36.0% in the 40- to 59-year-old bracket, and further increasing to 42.3% in the 60 and over bracket (Flegal et al., 2012). As a general trend, increases in obesity at the individual level are a reality. In the next section, I consider the literature that bridges the gap between youth obesity and adult obesity.

Development of Obesity From Adolescence to Adulthood

Individuals who are overweight or obese as youth tend to persist into adulthood categorized as overweight or obese (Gordon-Larsen, The, & Adair, 2010; Juonala et al., 2011; Rooney, Mathiason, & Schauberger, 2011; Singh, Mulder, Twisk, van Mechelen, & Chipanaw, 2008). Singh, Mulder, Twisk, van Mechelen, and Chipanaw (2008)

conducted an analysis of 25 high-quality studies in the area of weight persistence from youth to adulthood and concluded from these studies that those who were overweight or obese in childhood and/or adolescence were at greater risk for the same weight status as adults. Furthermore, the more extreme the weight problem for the child or adolescent, the more persistent was increased weight into adulthood. When considering cumulative results from all studies, Singh et al. concluded that the risk for adult overweight was twice that for overweight children than for normal-weight children. Similarly, Gordon-Larsen, The, and Adair (2010) reported a 90% persistence rate for early adulthood obesity from adolescent obesity. Rooney, Mathiason, and Schauberger (2011) reported that 85% of children and 46% of adolescents who were at or above the 85th percentile for BMI were also obese in young adulthood. Juonala et al. (2011) corroborated these findings in their analysis of four longitudinal studies that included over 6,000 subjects. Juonala et al. reported that 64.6% of overweight or obese children remained obese as adults and that 82.3% of obese children remained obese as adults. While the studies presented here reference youth as a combination of children and adolescents, Reilly et al. (2011) quantified the specific risk of overweight and obesity persistence from childhood to adolescence. Reilly et al. found overweight children to be at 18 to 20 times greater risk for adolescent obesity than normal-weight children. Therefore, research provides substantial evidence that increased weight in childhood predisposes adolescents to increased weight with ensuing risk for increased weight in adulthood.

A consideration must also be made for the reality that those who are normal weight in youth may experience increased rates of obesity in adulthood. Juonala (2011)

reported from their review of four studies of weight persistence and cardiovascular risk factors that 14.6% of normal weight youth were categorized as obese 23.1 years later (the average span at follow-up in the four studies). Gordon-Larsen et al. (2010) reported more extreme increases by noting that within a cohort, the number of obese participants doubled during the transition from adolescence to early adulthood. The number of obese participants doubled again from early adulthood to the mid 30's in the same cohort. Finally, using the same cohort as did Gordon-Larsen et al., The, Suchindran, North, Popkin, and Gordon-Larsen (2010) identified that the number of participants who were severely obese in the cohort increased from 79 during the adolescent wave to 703 merely 13 years later. The evidence from research solidly points to substantial increases in obesity during the transition from adolescence to adulthood as well as persistence of overweight and obesity from youth to adulthood; reasons for the observed increases in weight during this developmental transition are considered next.

Having established the general trend among the American population for increased rates of overweight and obesity from childhood to adolescence, from adolescence to young adulthood, and from young adulthood through mature adulthood, the question remains: What are the risk factors for developing obesity over time? The logical predisposers to increases in weight are increases in calorie intake and decreases in physical activity. However, Bleich, Ku, and Wang (2011) reviewed multiple cross-sectional and longitudinal studies related to these two factors and concluded that the role of each in the development of childhood and adolescent obesity was not clearly identified by research. In light of these proposed factors—physical activity and caloric intake—and

in consideration of other possible factors, researchers have approached the understanding of obesity development by identifying health-risk behaviors and health-promoting behaviors among adolescents. I will discuss first the potential for the role of religiosity in the development of health-promoting behaviors among adolescents.

Religiosity and health promotion. Researchers have linked religiosity to a number of health-enhancing habits among the adolescent population. Ford and Hill (2012) identified personal attitudes towards substance use and the presence of major depression to be mediators of religiosity and substance abuse relationships. Specifically, Ford and Hill attributed religiosity to a disapproving attitude towards excessive alcohol and tobacco use resulting in reduced use of these substances. Similarly, Neymotin and Downing-Matibag (2013) found increased religiosity to reduce drug use among adolescents, but not to reduce sexual activity.

The religious and social environment to which adolescents are exposed can strengthen health-promoting behaviors (Rew, Arheart, Thompson, & Johnson, 2013; Rew, Wong, Torres, & Howell, 2007). Rew, Arheart, Thompson, and Johnson (2013) applied primary socialization theory to the study of adolescent health-promoting behaviors in the areas of nutrition, physical activity, safety, health practices awareness, and stress management. Primary outcomes of the Rew et al. (2013) study included the significance of parental monitoring (an indication of social connectedness) for improving all five health-promoting behaviors and the significance of adolescent's religious commitment for improving all five health-promoting behaviors, but in particular, stress management. Religiosity connected to stress management supported the earlier work by

Rew, Wong, Torres, and Howell (2007) who also found religious commitment to be significantly related to stress management in older adolescents as well as to other health-promoting behaviors such as physical participation, nutrition, social support, identity awareness, and safety. These studies provide support for the role of religiosity in promoting healthy behaviors among adolescents including, most notably, factors of social support and increased ability to manage stress.

Researchers have also considered the role of religiosity for influence on health-risk behaviors (Salas-Wright, Vaughn, Hodge, & Perron, 2012; Stevens-Watkins & Rostosky, 2010). Stevens-Watkins and Rostosky (2010) determined that of three variables—religiosity, family connectedness, and perception of friend's substance use—African American males' practices of binge drinking were connected to their perception of friend's substance use during adolescence. Salas-Wright, Vaughn, Hodge, and Perron (2012) identified the category of *religiously devoted* youth to have decreased levels of substance use as well as less likelihood of delinquent acts. Furthermore, Salas-Write et al. (2012) concluded that the combination of intrinsic and extrinsic religiosity provided the most favorable protection against these health-risk behaviors. Therefore, the influence of religiosity on health-risk behaviors has been shown to be significant in a cross-sectional study but insignificant in a longitudinal study.

The problem remains: We do not have an understanding of the role of religiosity during adolescence in the longitudinal development of obesity. Research indicates that religiosity can positively impact health-promoting behaviors and decrease health-risk behaviors among adolescents, but there is no direct evidence for these effects upon

obesity—one indicator of health—from a longitudinal perspective. A closer look at physical activity and nutritional behaviors associated with obesity can help to illuminate the potential connection between religiosity and obesity.

Physical activity and obesity. As demonstrated previously, adolescent religiosity can enhance physical activity (Rew et al., 2013); furthermore, physical activity is related to obesity levels in children over time (Pate et al., 2013). Pate et al. (2013) reviewed studies completed prior to 2010 and concluded that children and adolescents who engage in higher levels of physical activity have reduced levels of fat, prospectively. However, such findings do not inform the understanding of the development of obesity for those children and adolescents who are not categorized as overweight or obese.

Research in the area of physical activity tracking can help to inform understanding of the potential longitudinal role of physical activity in obesity. For children and adolescents, researchers have studied the tracking of physical activity through the transition to adulthood and the results support the general conclusion that physical activity from childhood to adulthood tracks consistently (Telama, 2009; Telama et al., 2005). Physical activity tracking is a measure of the stability of a person to remain in the same relative position for activity level over time compared to others (Telama, 2009). So, while tracking does not indicate that activity level remains constant, tracking does indicate that in relation to others, there is little variation in physical activity as one transitions from childhood to adulthood. Similarly, Telford et al. (2013) found that children remained consistent in their step count, as measured by pedometers, from ages 8 to 12, but also noted that these same children trended towards reduced levels of physical

activity and increased levels of sedentary time as they aged. Taken together, this research demonstrates that levels of physical activity do not vary greatly throughout childhood and that, relative to others, many people remain consistently active over the lifespan.

Research on the tracking of physical activity provides evidence of notable sex differences. Correlation coefficients related to tracking of physical activity over time were consistently shown to be higher for men than for women (Telama, 2009; Telama et al., 2005). In addition to the sex difference, the Telama (2009) analysis of multiple studies demonstrated that physical activity tracking is not as stable for the childhood to adulthood analysis as it is for the adolescent to adulthood analysis. Therefore, men demonstrate a stronger relationship for physical activity consistency over time than do women, and this relationship is strongest when considering the transition from adolescence to adulthood than is it for the transition from childhood to adulthood.

Regarding the potential relationship between religiosity, physical activity, and obesity from childhood to adulthood, the following conclusions are supported. Adolescent religiosity can positively influence physical activity level; physical activity level is associated with obesity; and physical activity level over time tracks consistently from adolescence to adulthood.

Nutrition and obesity. Another logical connection to increased obesity is increased energy consumption. Similar to physical activity, adolescent religiosity was shown to relate to health-promoting behaviors involving nutrition (Rew et al., 2013). Pate et al. (2013) also reviewed pre-2010 studies of children and adolescents related to nutritional intake and obesity. The most commonly studied nutritional variable was intake

of sugar-sweetened beverages and this variable provided somewhat consistent conclusions. Primarily, among the studies included in the review by Pate et al., increased sugar-sweetened beverages resulted in increased BMI in five out of eight studies. The three remaining studies reported either no association between sugar-sweetened beverages and BMI increases or reported an association only for girls.

Similar to the study of physical activity, researchers study eating patterns over time to determine changes in these patterns for individuals in relationship to others (Boreham et al., 2004; Oellingrath, Svendsen, & Brantsaeter, 2011). Oellingrath, Svendsen, and Brantsaeter (2011) tracked eating patterns from childhood to adolescence among Norwegian children. For the transition from childhood to adolescence, Oellingrath et al. (2011) reported that eating habits from 4th to 7th grade were *reasonably stable*. Interestingly, patterns that included poor eating habits of snacking and junk and convenient processed foods were not linked to an increased risk of becoming overweight for these study participants. There is a dearth of recent research on tracking of diet from adolescence to adulthood, but Boreham et al. (2004) reported that dietary tracking from adolescence to adulthood was inconsistent for Irish participants. Therefore, the relationship of nutrition to BMI as well as the tracking of dietary habits from adolescence to adulthood are inconclusive.

Stress and obesity. Physical activity and dietary intake represent obvious connections to the development of obesity over time. However, another variable should be considered for its consistent contribution and connection to obesity and in light of potential relationships to religiosity. Stress management was identified by Rew et al.

(2007) and Rew et al. (2013) as being influenced by adolescent religiosity. Jääskeläinen et al. (2014) identified a cross-sectional relationship between stress-related eating and obesity among adolescents in Finland. Tomiyama, Puterman, Epel, Rehkopf, and Laraia (2013) identified a longitudinal connection between stress and obesity among U.S. females from ages 10 to 19. Tomiyama et al. (2013) reported that chronic stress was related to increased levels of obesity among Black and White girls although the relationship was stronger for Black girls than for White girls. Therefore, poor stress management may be a pathway by which reduced religiosity can lead to increased levels of stress and ultimately to increased levels of obesity.

In this section, I have discussed the literature related to the primary focal point of the current study—the transition from adolescence to adulthood. The literature points to ever-increasing levels of obesity from childhood through later adulthood; furthermore, there is sufficient evidence for the relationship between religiosity at the adolescent level and the development of obesity over time primarily through relationships associated with physical activity, dietary habits, and stress management. Individuals in the transition from adolescence to adulthood demonstrate low adherence to health-promoting behaviors and increased involvement in health-risk behaviors (Laska, Pasch, Lust, Story, & Ehlinger, 2009), and some of these behaviors are related to the development of obesity. Increased levels of obesity during the transitional period from adolescence to adulthood are associated with a number of health issues; these issues are discussed next.

Obesity-Related Wellness Problems

Percentages of obesity for men and women are significant given the adverse health effects associated with elevated levels of obesity. The World Health Organization (n.d.) listed known links to obesity that included, among others, changes in blood pressure, cholesterol, and triglycerides as well as increases for certain types of cancer (including breast and colon cancer) and for the risk of coronary heart disease and ischemic stroke. Utilizing cross-sectional research, Must et al., (1999) identified comorbidities with overweight and obesity to be irrespective of age or of race/ethnicity; Must et al. observed an increased prevalence ratio among those with higher levels of obesity for type 2 diabetes, gallbladder disease, coronary heart disease, and increases in cholesterol levels and hypertension. Amarya, Singh, and Sabharwal (2014) detailed the health risks associated with the obese elderly population in India and identified these associations to include diabetes, hypertension, arthritis, cardiovascular disease, pulmonary abnormalities, cancer, urinary incontinence, and cataracts. Obesity also affects worker productivity and costs. Howard and Potter (2014) found higher rates of worker absenteeism due to illness among the obese and those with diabetes compared to those of healtheir weight; class III obesity and diabetes were particularly associated with absenteeism. The adverse affects of obesity on health and productivity are not restricted to adults and aging adults. Obese children face significant health risks as well.

Halfon, Larson, and Slusser (2013) conducted a comprehensive review of 19 health risks associated with obesity among children. After controlling for social status variables, Halfon et al. (2013) determined that obese children were at greater risk for

activity restrictions, internalizing problems, externalizing problems, repeating a grade, ADHD, learning disabilities, asthma, allergies, and headaches than were nonobese children. Among children, researchers have considered school absenteeism related to obesity as well. Echeverría, Vélez-Valle, Janevic, and Prystowsky (2014) studied the roles of poverty and obesity on school attendance and determined that for all income categories, increasing levels of BMI were associated with increased absenteeism in school. Similar to obese adults, obese children face significant physical, psychological, and social challenges.

Prevalence rates for adult overweight and obesity are high in the United States (age-adjusted estimate of 69.4% in 2008) although not the highest in the world (WHO, 2010). High obesity rates are a cause of great concern for those interested in U.S. public health and safety as evidenced by the declaration of the Department of Health and Human Services (2014) that *adult obesity* and *childhood and adolescent obesity* are two of 26 leading health indicators in America. The fact that a country, such as the United States, posts such a high rate of obesity seems incongruous with the fact that the same country can post a high rate of religiosity as well given that high religiosity is associated with health and longevity while obesity is associated with decreased health and longevity. In the next section I discuss significant research regarding the spiritual dimension of health as reflected by religiosity.

Religiosity: A Measure of the Spiritual Dimension of Health

It can be a challenge for the scientific community to be open to the role that spirituality can play in the development of human health. In fact, as Miller and Thoresen

(2003) and Marks (2005) pointed out, prominent psychologists of the 20th century, such as Freud, were quite convinced that reliance on religion was tantamount to expressing psychopathological disorders. This belief as well as the belief that a consideration of religion reduces scientific thought to unscientific levels (Miller & Thoresen, 2003) have served to keep overt research on religion out of the mainstream for a considerable period of time.

Despite long-standing challenges to the role of religion and spirituality as an area of study related to psychology and health, consideration for the interrelatedness of health and religion are prominent in todays literature. Marks (2005) cited the rise of the empirical study of religion as beginning in the 1970s with significant works of research (i.e., the four complete volumes of *The Faith Factor,* which provided annotated bibliographies of nearly 400 studies on religion-health) in circulation by the 1990s. Abdel-Khalek (2007) identified that the study of religion had spread to multiple disciplines including "psychology, psychiatry, medicine, epidemiology, gerontology, and geriatrics" (p. 571). As the study of religion related to these various disciplines expanded, researchers identified problems with standardizing the conceptualization of religion and spirituality.

Researchers have identified a number of challenges related to the study of religion and spirituality. A primary challenge has been in determining responsible and accurate ways in which to define spirituality, religion, and religiosity in order to effectively measure these constructs. Miller and Thoresen (2003) defined spirituality as an individual-level construct dealing with the transcendent or divine and defined religiosity

(or religion) as a social construct with prescribed beliefs. The definitions for spirituality and religion provided by Miller and Thoresen are consistent with modern-day definitions of these terms. Throughout this dissertation, I will be using the term *religiosity* as a reflection of a specific measurement of spiritual health.

Young (2011) identified the measurement of the construct of religiosity as a particular challenge in religiosity research. Young observed that too often single measures of religiosity are used in research and that primarily, when this is done, religious affiliation becomes the default measure of religiosity. Attendance at religious events is also frequently used to measure religiosity. Young elaborated on other measures of religiosity including experiential, ritualistic, ideological, intellectual, and consequential dimensions as well as an intrinsic-extrinsic model. Finally, Young detailed a generic measure for the construct of religiosity; the exemplar that Young provided is to use multiple measures of religiosity when conducting such research. Following is a review of research in the area of religiosity and other dimensions of health as related to this dissertation.

Religiosity and Health

Strawbridge, Shema, Cohen, and Kaplan (2001) asserted that the search for the connection between religion and death rates is more than a century old. Powell, Shahabi, and Thoresen (2003) indicated that while religion and health are related, the evidence for the construct of this connection is unclear. Chida, Steptoe, and Powell (2009) concluded that evidence existed for the relationship between higher levels of religiosity/spirituality as a protective effect on health particularly among healthy populations. Furthermore,

Chida et al. (2009) identified religious attendance (attending church or religious services) to be the primary measure of religiosity leading to lower mortality rates. Musick and Worthen (2010) supported the salience of religious attendance in connection with health and referred to religious attendance as "…the strongest and most consistent association with health" (p. 259). Powell et al. (2003) considered multiple studies through 2003 that reported a relationship between religiosity and/or spirituality with physical health. I consider more fully some of the general hypotheses related to religiosity and health that were discussed by Powell et al.

Protective effect of religious attendance against death. Results of the Powell et al. (2003) analysis demonstrated support for a research hypothesis in which religious attendance protected against death. This protective factor was evaluated by Strawbridge, Shema, Cohen, and Kaplan (2001) who observed that mortality rates dropped for those who attended religious services weekly over those who attended less frequently. Improving negative health behaviors, decreasing levels of depression, and evidence of greater social support were the primary mechanisms responsible for this protective effect (Strawbridge et al., 2001). Interestingly, Strawbridge et al. (2001) determined a greater effect related to improving poor health behaviors than for maintenance of already established good behaviors. Strawbridge et al. also observed improvements in health behaviors and decreases in depression to be stronger for women than for men; ultimately, there was a 25% difference between weekly attenders and less than weekly attenders, making this a particularly notable study connecting religiosity with physical health (Powell et al., 2003).

Protective effects of religiosity against cardiovascular disease (CVD). Powell et al. (2003) expressed belief that religious attendance is likely to promote a healthier lifestyle thus translating to improved cardiovascular status. Chida et al. (2009) found there to be a 28% reduction in hazard ratios for cardiovascular mortality in their meta-analysis of quality studies of healthy participants. Masters and Hooker (2012) also concluded that a connection existed between religiosity/spirituality and CVD mortality and noted that most measures of religiosity/spirituality were based on attendance at religious services. Powell et al. (2003) attributed the religiosity/spirituality connection to reduced CVD death to protective effects of a healthy lifestyle. Conversely, Feinstein, Liu, Ning, Fitchett, and Lloyd-Jones (2010) concluded, in their study of religiosity and CVD risk factors, that there was no cross-sectional connection for religious participation, prayer, or spirituality to CVD risk factors or to subclincial CVD. Furthermore, four-year longitudinal analysis did not reveal signficiant relationships for these measures to CVD events.

Protective effect of religiosity/spirituality against cancer. Powell et al. (2003) found only two studies that related religiosity/spirituality to cancer mortality and concluded that these two studies offered a level of connection between religiosity/spirituality and reductions in all forms of death except for cancer. Powell et al. proposed that the populations that contracted cancer were more likely to seek out religious support after diagnosis of cancer than before. Masters and Hooker (2012) also found no conclusive evidence for a reduction in cancer mortality due to religiosity/sprituality connections. However, their meta-analysis revealed that women

who attended church/services more frequently demonstrated fewer risk factors for breast cancer. Masters and Hooker pointed out that research in this area often considered positive and negative religiosity/spirituality coping; results demonstrate that positive coping is associated with higher levels of well-being among cancer patients and negative coping is associated with lower levels of well-being.

Powell, Shahabi, and Thoresen (2003) supported the general connection that exists between religion and health, but stated that such a connection "may be more limited and more complex than has been suggested by others" (p. 50). I presented examples in this section of the review that support the general conclusions of Powell et al. (2003). Religious attendance and longevity are inextricably linked. However, the connection between religiosity and CVD (the number one cause of death among adults in the United States) is unclear and the connection between religiosity and cancer (the number two cause of death in the Unites States) is even less supported.

In summary, research in the area of religion and health supports the religion-health relationship primarily as evidenced by the relationship between increased religious attendance and longevity. Connections between religion and the top two causes of death in the United States, CDV and cancer, are less supported. I now turn to a discussion of the role of religiosity in the development of a specific measure of physical health – obesity.

Religiosity and Obesity

Empirical evidence for the connection between religiosity and obesity is conflicting. General hypotheses for studies relating these two factors include an

acknowledgement that greater degrees of religiosity are associated with better health and that higher levels of obesity (or weight) are associated with decreased health levels; therefore, the conjecture is that higher levels of religiosity should be related to lower levels of obesity (e.g., Kim, Sobal, & Wethington, 2003). As stated, however, the connection between religiosity and obesity is not so easily identified. I begin with a consideration of the research relating obesity and denominational affiliation as this area of study identifies the way in which the religiosity-obesity relationship was first researched.

Obesity and Denominational Affiliation

Early research interests in the relationship between religion and obesity included the role of denominational affiliation in the development of weight. Ferraro (1998) completed a state-by-state analysis of ecological data and determined that states in the United States with higher Baptist affiliation were also higher in obesity levels. A couple of studies supported the prevalence of overweight and obesity within the Baptist denomination (Cline & Ferraro, 2006; Dodor, 2012). Cline and Ferraro (2006) observed a significant relationship of increased obesity among women in the Baptist denomination. Dodor (2012) studied a specific section of the African American population, African Americans with at least one parent with a college education, and reported that obesity was high among Baptists (32.2%) in this population.

Dodor (2012) found the African American Baptist population to have a high rate of obesity (32.2%); however, Dodor reported reformation-era Protestants to have a higher rate of obesity (38.0%) and nondenominational Protestants to have the third

highest rate of obesity at 31.9%. The rate of protestant obesity was supported by Kim, Sobal, and Wethington (2003) in their study of religious affiliation and obesity. Among adults 25-74 years of age, Kim et al. (2003) concluded that men who were Conservative Protestants measured approximately five pounds heavier than men with no particular religious affiliation. It should be noted that while Kim et al. (2003) identified four measures of religiosity that were significantly related to obesity, the significant relationships observed became insignificant when controlling for smoking status.

Conclusions from some studies (Dodor, 2012; Ferraro, 1998; Kim et al., 2003; Yeary et al. 2009) counter the connection between religious affiliation and increased obesity. As stated earlier, Ferraro (1998) identified a relationship between the Baptist denomination and increased levels of obesity; conversely, in another part of the study, Ferraro found there to be no relationship between religion and body weight when accounting for socio-economic status (SES) and race/ethnicity. Yeary et al. (2009) found no relationship for increased religiosity among African Americans versus Whites for obesity levels. Kim et al. (2003) presented conflicting findings in which male religious affiliation related to obesity but female religious affiliation did not relate to obesity. Dodor identified obesity rates to be elevated for African Americans in the reformation-era Protestant, Baptist, and nondenominational Protestant groups; however obesity rates for African Americans with no religious affiliation were at 36.4%, which placed the nonreligious group between the obesity rates for reformation-era Protestants and Baptists. Furthermore, when considering the rate of extreme obesity, African Americans with no religious affiliation recorded a 17.4% rate, which exceeded the rates of all religious

denominations for extreme obesity. Taken as a whole, the research relating obesity to denominational groups did not provide much insight into the obesity-religiosity relationship. The reasons for lack of clarity in this research are: (a) cross-sectional relationships make it impossible to identify causation, (b) the mainstream U.S. religions are Christian, so there has been little to compare them to, and (c) many of the studies have focused on heterogeneous sections of the population.

Obesity and Various Religious Expressions

Some of the most compelling information regarding religion and obesity is the connection that exists between different expressions of religious activity and obesity level. Studies of the use of religious media indicate an increase in obesity among those who use more religious media (Cline & Ferraro, 2006; Yeary et al., 2009). Cline and Ferraro (2006) considered religious media practice (defined by use of religious books, television, and radio) as a factor in obesity and identified a relationship among women for increased obesity with increased religious media practice. Yeary et al. (2009) also considered the factor of religious media and found an increase, among the White population, for BMI with increased consumption of religious media. Although not concluded in these studies, level of obesity may be the antecedent to use of religious media. The studies did not control for injury or debilitations that may have predisposed these individuals to seek religious interaction in ways other than active participation.

Research results related to church attendance and obesity are equivocal. Increased levels of prayer and religious attendance were related to increases in obesity for various populations (Dodor, 2012; Feinstein, Liu, Ning, Fitchett, & Lloyd-Jones, 2012). Dodor

(2012) identified obesity to be related to prayer and church attendance among the African American population; obesity levels were significantly increased among those engaging in higher levels of prayer and of church attendance. Feinstein, Liu, Ning, Fitchett, and Lloyd-Jones, (2010) considered the role of religious participation, prayer, and spirituality in the expression of obesity and found increases in each measure to be significantly related to increased obesity. The participants in the Feinstein et al. (2012) study represented diverse ethnic groups and both genders, but the average age of the participants was 63, which could indicate substantial life changes due to retirement. Regardless, the religiosity-obesity connection in the Feinstein et al. study was cross-sectional; thus limiting an understanding of causality for this relationship. In opposition to the findings of Dodor and of Feinstein et al., Cline and Ferraro (2006) identified increased church attendance among women to be associated with decreased levels of obesity. The conflicting findings related to church attendance and obesity may reflect research design and life-stage development. The studies that found increased church attendance to relate to increased obesity were both cross-sectional in design. What sets the Cline and Ferraro study apart from the other studies is that the Cline and Ferraro study, as it relates to religious attendance, considered women from a somewhat younger age bracket and used a longitudinal approach for determining this religiosity-obesity relationship. As demonstrated by the Cline and Ferraro study, there are gender differences that exist in the religion-obesity connection. Gender-specific findings are considered more fully next.

Obesity, Religion, and Gender

A subtle, but notable, theme that is present in this review of the religiosity and obesity connection is that the connection exists at different levels based on gender. One study indicated there to be no significant difference between genders for the religiosity-weight relationship (Feinstein et al., 2010); however, two studies identified gender differences related to the interaction of religiosity and obesity (Cline & Ferraro, 2006; Kim, Sobal, & Wethington, 2003). Cline and Ferraro (2006) noted specifically the differences in gender related to the religiosity-obesity connection. Cline and Ferraro concluded that women demonstrated higher susceptibility to obesity the more they engaged in religious media practices while men engaged with religion for comfort and support, which helped to reduce obesity levels. Kim, Sobal, and Wethington (2003) identified Conservative Protestant men to be heavier than men with no religious affiliation but noted that women did not demonstrate a relationship for body weight to religiosity. Therefore, as is true in multiple areas of research, men and women demonstrate different relationships for the religiosity and obesity association. Another common consideration among those studying obesity is the role of race/ethnicity in the development of obesity. Race/ethnicity as a factor in the religiosity-obesity connection is considered next.

Obesity, Religion, and Race/Ethnicity

The main area of research related to obesity, religion, and race is related to these constructs among the African American population. Much of the interest in this area relates to the noted role of religion as a significant influence in African American culture

(Dodor, 2012; Reeves, Adams, Dubbert, Hickson, & Wyatt, 2012). Dodor (2012) reported that higher levels of prayer and of church attendance among the African American population was significantly related to higher levels of obesity. Reeves, Adams, Dubbert, Hickson, and Wyatt (2012) provided a cross-sectional view of highly religious African Americans in the southeastern United States and considered the possible role of mediation played by social support, depression, demographic variables, and multiple health behaviors in the relationship between multiple measures of religiosity and obesity. However, Reeves et al. (2012) found no statistically significant relationship between religiosity and obesity to exist. A possible reason for the opposing conclusions reached in the Dodor and Reeves et al. studies is that Dodor studied members of the African American population who had at least one parent who had earned a college degree while Reeves et al. studied members of the African American population from the southeastern United States with deeply held religious views. Reeves et al. (2012) were unable to test for mediation in their study because there was no relationship between religiosity and obesity; however, Reeves et al. identified religiosity effects on health behaviors, social support, and depression that supports the basis for the current study. Dodor and Reeves et al. also reported conflicting evidence related to the effect of religiosity on behaviors; Dodor concluded that prayer and church attendance were related to increased obesity levels among African Americans, but Reeves et al. concluded there to be no relationship between these variables; however, both studies employed cross-sectional analysis, which limits identification of causation. Furthermore, the specific African American populations studied by both studies were dissimilar. Reeves et al. did,

however, further the discussion of the role of religiosity in behaviors associated with obesity. Such behaviors are considered next.

Religion and Obesity-Related Behaviors

Three studies considered the role of religiosity in the engagement of obesity-related behaviors (Kim & Sobal, 2004; Reeves et al., 2012; Roff et al., 2005). Reeves et al. (2012) identified a significant association between the religious practice of prayer and lower caloric intake per day. Prayer, along with church attendance and other spiritual measures, was associated with reduced alcohol use, and smoking was less prominent among those who attended church more often (Reeves et al., 2012). Notably, psychosocial aspects of social support and depression (variables of consideration in the present study) were related to prayer and church attendance. Kim and Sobal (2004) considered the role of religion in specific behaviors presumed to be associated with weight management—fat intake and physical activity. Kim and Sobal (2004) found women in the Catholic denomination significantly less likely to consume fat in their diet than Conservative Protestant women and women of *Other* faith when controlling for age, race, and other factors. Kim and Sobal reported that among men in the various denominations there was no relationship between religion and fat intake. In regard to physical activity, men who were more engaged in prayer were more physically active than men not engaged in prayer; women who demonstrated greater commitment to religion (measured, in part, by financial support for the denomination) were more physically active than women not commited to religion (Kim & Sobal, 2004). Because the Kim and Sobal (2004) study did not specifically relate obesity levels to fat intake and

physical activity, the study does not provide a greater understanding of the relationship between religiosity and obesity *per se*. The study does, however, provide possible affiliational and gender considerations for the development of obesity based on lifestyle behaviors.

Roff et al. (2005) considered exercise in the relationship between religion and obesity, and accounted for smoking status in their study of adults 65 years of age and older. Roff et al. found lifetime smoking to be less likely for those more likely to attend church or religious functions and for those more engaged in private expressions of religion, such as prayer. Increased church attendance was also significantly related to an increase in time spent in leisure physical activity; despite these relationships, none of the measures of religiosity were significantly related to obesity in this population.

In summary, the religiosity-obesity findings are varied and seemingly contradictory. However, a few determinations can be made. Primarily, there have been multiple ways in which religiosity has been measured including denominational affiliation, attendance at church or religious activities, engagement in private forms of religiosity, and the salience of one's religion in their daily functioning and approach to life; Young (2011) recommended that, when possible, researchers use multiple measures of religiosity. Secondly, some health behaviors increase with increased religiosity and some health behaviors decrease; regardless, even when increased religiosity is associated with healthier behaviors, this does not necessarily translate to decreased levels of obesity. Increased religiosity has been shown to be related to decreased levels of smoking and increased levels of physical activity (Roff et al., 2005) and prayer has been linked to

lower daily caloric intake (Reeves et al., 2012); men and women demonstrate differing connections to relgiosity that engage greater levels of physical activity among the genders (Kim & Sobal, 2004). However, despite noted relationships to healthy behaviors, religiosity in these studies was not significantly related to obesity.

A third determination is that race/ethnicity are not consistently evaluated in this literature. Although Dodor (2012) found prayer and church attendance to be significantly related to obesity rates among the African American population, Reeves et al. (2012) and Yeary et al. (2009) found no relationship to exist between religiosity and obesity among the African American population. Finally, cross-sectional research characterizes the majority of the work concerning the relationship between religiosity and obesity. The lack of longitudinal designs for studying the relationship between religiosity and obesity during more volatile transitional periods has left a void of understanding of the potential relationship that may exist.

Although the mechanisms by which religion can lead to longevity are not known, Rogers, Krueger, and Hummer (2010) reemphasized three prominent hypotheses ascribed to Durkheim (1897/2002) for the relationship between religious attendance and reduced mortality; they are (a) social support and social ties gained through religious involvement allow for social integration with those who can provide financial, emotional, and instrumental forms of support, which may reduce negative health effects; (b) beliefs and practices encouraged by the specific religious denomination provide health-enhancement at the individual level (e.g., reduced levels of smoking tobacco and use of alcohol and illegal drugs); and (c) religious attendance reduces stress levels by providing

greater meaning in life, peace, a sense of belonging, and coping behaviors for the tough times in life.

Potential Mediators of the Religiosity–Obesity Relationship

Throughout the literature, there is a common theme of hypothesized relationships for two variables in connection to religiosity and to obesity—they are depression and social support (Powell, Shahabi, & Thoresen, 2003; Reeves et al., 2012; Strawbridge et al., 2001). Strawbridge et al. (2001) concluded that longevity was enhanced for those who more frequently practiced religious attendance and that this enhancement was due to improving health behaviors including decreases in depression symptoms and increases in monthly engagement with social supports such as family and friends. At baseline, the age range of participants in the Alameda County Study, the data set used by Strawbridge et al., was 17 to 95 years of age. In the current study, the focus will be on the development of obesity during the transition from adolescence into adulthood. Strawbridge et al. did not look at the effect of depression and social support specific to the development of obesity.

Similarly, Powell, Shahabi, and Thoresen (2003) identified preemptively that social support, depression, and health lifestyles were established protective factors in the link between religiosity and physical health but did not link the connection specifically to obesity. Powell et al. (2003) used the control of these variables as indication of the quality of the research for classifying studies in their levels-of-evidence design. If a study did not control for social support, depression, and healthy lifestyle, it was not included in their review. Likewise, if a study was cross-sectional in design, it was not included in

their review. Powell et al. (2003) accepted the role of social support and depression in the religiosity-health relationship, but did not study these specific relationships. For those studies that did control for social support and depression and were longitudinal in nature, the most consistent connection between religiosity and health, identified by Powell et al., was the protection of church/service attendance against death. Powell et al. did not report any conclusions related to religiosity and obesity.

In a more recent study, Reeves et al. (2012) considered depression and social support for moderation and mediation effects for the relationship between religiosity and obesity. While the Reeves et al. study included a robust level of participants (more than 2,000), the study design was cross-sectional and considered only the African American population. Furthermore, the average age of the participants in this study was 53.6 years of age with a standard deviation of 12.39 years. Reeves et al. reported there to be no cross-sectional association between various religiosity measures and BMI or waist circumference among this population. The studies by Strawbridge et al. (2001), Powell et al. (2003), and Reeves et al. demonstrate the progression of interest in and evidence for religion affecting social support and depression with concommitant affects on physical health but either did not consider the connection to obesity or did so for a restricted population and in a cross-sectional manner. Furthermore, these studies did not consider the longitudinal interaction of these variables prior to adulthood. Following is a closer look at one aspect of possible mediation between religiosity and obesity—depression.

Depression Demographics in the United States

Rates of depression in the United States are dependent on age, gender, race/ethnicity, and income level (Kessler, Chiu, Demler, & Walters, 2005; Pratt & Brody, 2008) and on marital status, educational level, and rural versus urban living (Kessler et al., 2005). Pratt and Brody (2008) presented information from the National Health and Nutrition Examination Survey, 2005-2006, which identified 2-week prevalence rates for depression in the United States of 5.4% for those 12 years of age and over. The rates of depression were related to specific demographics–notably, depression was more common for those in the 40-59 year age bracket, for women, for non-Hispanic blacks, and for those living below the poverty level. Kessler, Chiu, Demler, and Walters (2005) reported somewhat different values for 12-month depression prevalence rates with a total rate for major depressive disorder of 6.7% for those 18-years of age or older in the United States. Kessler et al. reported that major depression correlated more stongly with women, non-Hispanic White, unmarried, low educational level, lower income, and those in nonrural locations. While the Kessler et al. findings match somewhat with the Pratt and Brody findings, the differences in race and ethinicity may be due to the fact that Kessler et al. correlated in relation to major depression rather than to depression as a general measure.

Depression and Religiosity

Individuals holding to religious ideations have long been maligned by some psychologists; Freud is perhaps the most notable psychologist to express disregard for religion in scientific study. Freud (1927) stated that religious ideas were not “precipitates of experience or end-results of thinking: they are illusions, fulfillments of the oldest,

strongest and most urgent wishes of mankind" (p. 30). Freud went on to state that these illusions "come near to psychiatric delusions" (p. 31; also cited in Simmonds, 2006). Unfortunately, acquiescence to the thought that religious ideation was tantamount to psychiatric delusion resulted in the practice of ignoring the role of religion and spirituality in psychological constructs. Despite somewhat heavy opposition to the consideration of religion as a salutary contributor to mental health, researchers have developed significant work that considers the role that religion and spirituality may play in a variety of health outcomes including that of mental health. In fact, within the past 10 years, research regarding the religiosity-depression link included the following populations: adolescents in Great Britain (Meltzer, Dogra, Vostanis, & Ford, 2011), adolescents in Austria (Wenger, 2011), Canadian populations (Balbuena, Baetz, & Bowen, 2013; Rasic, Asbridge, Kisely, & Langille, 2013) adolescents in the United States (Ji, Perry, & Clarke-Pine, 2011; Possel et al., 2010), Muslim adolescents (Abdel-Khalek, 2007), Asian Americans (Ai, Huang, Bjorck, & Appel, 2013), African Americans (Colbert, Jefferson, Gallo, & Ronnie, 2009), Jews (Pirutinsky et al., 2011) Mormons (Bartz, Richards, Smith, & Fischer, 2010); college students (Berry & Adams-Thompson, 2008; Berry & York, 2011; Jansen, Motley, & Hovey, 2010; Lester, 2012), and older adults (Roff et al., 2004; Sun et al., 2012). Despite early and ardent opposition to the inclusion of religion as a variable of consideration in scientific endeavors, these recent examples of studies on religiosity and depression are indicators that religion is now a viable area of research. General conclusions can be made from the recent research

in the area of religiosity and depression; the first supported conclusion is that religiosity and depression share an inverse relationship.

The inverse relationship between religiosity and depression. Evidence for the relationship between religiosity and depression is mixed; however, the preponderance of the evidence indicates an inverse relationship between religiosity and depression (Koenig, 2009). Koenig, McCullough, and Larson (as cited in Koenig, 2009) outlined the general findings that two-thirds of cross-sectional studies identified higher degrees of religiosity to be associated with lower rates of depression or of depressive symptoms; furthermore, of the 34 studies opposing this finding, a statistically significant relationship of increased religiosity to increased depression was found in only four studies. Koenig (2009) also referenced 22 longitudinal studies that tested the religiosity-depression relationship and reported that 15 of these studies also support the inverse relationship between religiosity and depression over time. While study results do not all point in the same direction regarding the relationship between religiosity and depression, acceptance for an inverse relationship between these two variables is widely supported by research (Koenig, 2009).

Recently, researchers have identified the negative relationship between religiosity and depression to exist for a variety of populations including the elderly (Roff et al., 2004; Sun et al., 2012), Orthodox Jews (Pirutinsky et al., 2011), a sample of Canadians (Balbuena, Baetz & Bowen, 2013), and adults at high risk for depression (Miller et al., 2012). Roff et al. (2004) reported a significant cross-sectional relationship between high religiosity and decreased symptoms of depression for the elderly population.

Furthermore, Sun et al. (2012) identified a significant relationship between increased religious service attendance and decreased symptomology of depression among southern, community-dwelling older adults. In their study of Orthodox Jews, Pirutinsky et al. (2011) found higher intrinsic relgiosity to be associated with lower depression symptomology. Balbuena, Baetz, and Bowen (2013) reported that even monthly attendance at religious functions reduced the risk of depression by 22% over those who were categorized as nonattenders among a large sample of Canadians 16 years of age and older. In a longitudinal study, Miller et al. (2012) considered the relationship between religiosity and depression among adults who were at high risk for depression by virtue of being offspring of parents with depression. In general, those in the study, who reported high importance for religion or spirituality at the 10-year wave, exhibited one-fourth the risk for major depression in the 10 years that followed; furthermore, for those who were the offspring of adults with depression, high religiosity at the 10-year wave corresponded with one-tenth the risk of recurrence of major depression during the following 10 years as compared to offspring of adults with depression who were not highly religious. The referenced studies support the assertion of an inverse relationship between religiosity and depression for a variety of populations from both cross-sectional and longitudinal studies. One population that has been of particular interest to researchers is the population of college students.

An inverse relationship between depression and religiosity among college students. There is a considerable amount of recent research in the area of religiosity-depression in which college students are the population of consideration (Berry & York,

2011; Jansen, Motley, & Hovey, 2010; Yonker, Schnabelrauch, & DeHaan, 2012). This research supports further the inverse relationship between religiosity and depression. Berry and York (2011) studied the religiosity measurement of *religious coping* and reported a significant, negative correlation for this measure with depression. Jansen, Motley, and Hovey (2010) also studied college students for the relationship between religiosity and depression but conducted their study cross-sectionally. Jansen et al. (2010) measured religiosity as self-reported religiosity and as self-reported religious influence on their lives and each measure was found to have a significant, negative relationship with depression among college students. Likewise,church attendance among college students correlated negatively with depression (Jansen et al., 2010). Yonker, Schnabelrauch, and DeHaan (2012) conducted a meta-analysis of multiple psychological outcomes related to spirituality and religiosity among adolescents and emerging adults. Primary findings included evidence that higher levels of religiosity (and spirituality) were associated with reduced symptoms of depression and that this relationship was more salient for adolescents than for emerging adults. Recent research focussed on the religiosity-depression link among adolescents consistently identified the relationship to be negative and there is evidence that this relationship is stronger for adolescents than for emerging adults. Such a finding is important as it relates to this dissertation in that adolescent depression is being considered as a mediational variable between adolescent religiosity and adult obesity. Researchers have also considered extensively the relationship between religiosity and depression among the adolescent population.

An inverse relationship between depression and religiosity in the adolescent population. Recently, researchers have attempted to quantify the relationship between religiosity and depression among adolescents. Similar to the findings within other populations, adolescents in the majority of recent studies demonstrated a negative relationship between some measure of religiosity and level of depression and these findings applied to various adolescent populations (Abdel-Khalek, 2007; Ji, Perry, & Clarke-Pine, 2011; Meltzer et al., 2011; Pössel et al., 2011; Rasic, Asbridge, Kisely, & Langille, 2013). Meltzer et al. (2011) studied the association between religiosity and mental health among adolescents in Great Britain and concluded that adolescents with weak association to religion have a greater risk of emotional disorders (including depression) than do adolescents with no stated religious affiliation. Abdel-Khalek (2007) studied adolescents in the Muslin faith and reported a significant negative cross-sectional relationship between religiosity and depression.

The results of two studies identified intrinsic religiosity to be related to lower depression levels, and both studies reported extrinsic religiosity to interact differently with depression level (Ji, Perry, & Clarke-Pine, 2011; Pössel et al., 2011). Ji, Perry, and Clarke-Pine (2011) identified a relationship between higher levels of intrinsic religiosity and lower levels of depression among church-going adolescents but reported contradictory results for extrinsic religiosity; these results are discussed later. Pössel et al. (2011) considered the role of intrinsic and extrinsic religiosity among adolescents on level of depressive symptoms, but did so longitudinally. Similar to the findings of Ji et al. (2011), Pössel et al. determined there to be a significant relationship between increased

intrinsic religiosity and decreased levels of depressive symptomology; extrinsic religiosity was not related to depression scores in this longitudinal study.

One study of adolescents detailed the differences between boys and girls in the religiosity-depression relationship (Rasic, Asbridge, Kisely, & Langille, 2013). Rasic, Asbridge, Kisely, and Langille (2013) conducted a longitudinal study of adolescents related to religiosity and depression and found that among adolescents in Nova Scotia, beginning level of depression was a significant indicator of the religiosity/depression interaction. Primarily, Rasic et al. (2013) determined that girls with high religious attendance who were also low on depression symptoms at baseline were less likely to have elevated depression symptoms at the 2-year follow-up. Boys in this study, who were depressed at baseline (actually a dichotomization based on depressive symptoms) but were attending church, were less likely to remain depressed at the 2-year follow-up.

The majority of recent research supports the negative relationship between religiosity and depression among the adolescent population; however one recent study found no significant correlation to exist for this relationship (Wenger, 2011). Wenger (2011) studied a population of adolescents from Austria but reported that, unlike the adolescent populations mentioned previously in this review, there was no signficant correlation between religiosity and depression. Wenger also determined that the Austrian adolescent population did not "seem to be very religious" (p. 524) compared to other adolescent populations. An example of this disparity is seen by the percentage of Austrian adolescents who attended church (9%) compared to church attendance for U.S. adolescents (38%; Smith et al. as cited in Wenger, 2011). The lack of religiosness among

the adolescent population in the Wenger study separates this population from the majority of the other adolescent populations included in this review, and may account for the disparity in the findings. Results from other studies also counter the expectation that religiosity is negatively associated with depression.

Studies demonstrating divergent relationships between depression and religiosity. Not all studies support the relationship of increased religiosity to decreased depression. Colbert, Jefferson, Gallo, and Davis (2009) studied aspects of the relationship between religiosity and depression among African American adults and reported that increased religiosity did not relate statistically to decreased depression levels. Eliassen, Taylor, and Lloyd (2005) reported that no relationship existed between degree of religiosity and depressive symptoms for young males (primarily ages 19-21). Similarly, Ji et al. (2011) found increased levels of extrinsic religiosity to be related to increased levels of depression among church-going adolescents. Lester (2012) reported that religiosity was associated with increased levels of depression in a cross-sectional study of college psychology students; however, the study is also limited by sample size and generalizability. Therefore, while there is sufficient support for the negative relationship between religiosity and depression, there remains some evidence that for some populations, the relationship is not signficant or is positive. The potential reason for the divergent findings of these studies is the cross-sectional nature of the studies as well as the specific focus on religious groups as targeted participants in two of the studies. Colbert et al. (2009) studied members of a specific religious African American group and

Ji et al. (2011) studied adolescents in schools with a specific church affiliation. Neither population was representative of the general U.S. population.

Strength of religiosity and intrinsic religiosity. The second general finding of the religiosity-depression link is that the strength of one's religious beliefs affects depression level, and that intrinsic religiosity was more closely related to reduced depression than was extrinsic religiosity. Among a variety of populations, the strength of religiosity indicated a greater relationship to depression level than did religious affiliation (Jansen et al., 2010; Meltzer et al., 2011). Meltzer et al. (2011) concluded that while adolescents in Great Britain were not highly religious, regardless of religious affiliation, adolescents with weakly held beliefs or those who did not believe religious practice to be very important were at greater risk for depression than adolescents who indicated no religious affiliation. Likewise, Jansen et al. (2010) concluded that college students did not receive protection from depression based on religious affiliation, but rather based on level of religiosity. The Meltzer et al. and Jansen et al. studies focussed on the strength of religiosity as a measure that indicates greater connection to depression. Another way to distinguish between *types* of religiosity, which are more strongly connected to depression, is to consider the differences between intrinsic and extrinsic religiosity.

The findings of intrinsic and extrinsic religiosity in relation to depression are divergent. Lester (2012) defined intrinsic religiosity as a measurement of relgiosity "which captures whether religion provides a source of meaining in one's life" (p. 247); Pirutinsky et al. (2011) further explained instrinsic religiosity as the "sincere and intentional integration of religion into one's life" (p. 490). Extrinsic religiosity, however,

is comprised of factors that meet the needs of the individual (Lester, 2012) or that have an external benefit to the individual, such as social prominence (Pirutinsky et al., 2011). Research findings support the relationship of intrinsic religiosity over extrinsic religiosity in connection to reduced levels of depression.

There are a number of studies that have considered the specific role of intrinsic versus extrinsic religiosity as related to depression and that support the relationship between high intrinsic religiosity and lower rates of depression (Ji, Perry, & Clarke-Pine, 2011; Pössel et al., 2011; Roff et al., 2004; Sun et al., 2012; Yonker, Schnabelrauch, & Dehaan, 2012). Roff et al. (2004) identified a positive relationship between high religiosity, which included intrinsic religiosity as a factor, and lower depression symptoms. Sun et al. (2012) studied depression and religiosity among older adults and found that higher levels of intrinsic religiosity at baseline among older adults was related to decreased depression symptomology over four years while older adults with lower intrinsic religiosity at baseline experienced an overall increase in depression symptoms over four years. Ji, Perry, and Clarke-Pine (2011) studied the role of religiosity in the development of depression and subsequent suicidal ideation among adolescents and concluded that increased intrinsic religiosity among adolescents was associated with decreased depression and suicial ideation while increased extrinsic religiosity was significantly associated with increased level of depression and suicidal ideation. Pössel et al. (2011) also considered the role of intrinsic and extrinsic religiosity among adolescents and concluded that intrinsic religiosity significantly predicted depression four months later such that higher intrinsic religiosity was associated with lower depression among

adolescents. However, measures of extrinsic religiosity were unrelated to depression among this same sample of adolescents. Yonker, Schnabelrauch, and Dehaan (2012) provided a fitting summary to the factor of intrinsic and extrinsic religiosity in relation to depression. Yonker et al., (2012) concluded in their meta-analysis of adolescent studies that intrinsic religiosity was a key factor in the decreased level of depression; the studies mentioned here support such a conclusion.

There are a handful of studies that do not support the existence of a negative relationship between intrinsic religiosity and depression. Wenger (2011) studied Austrian adolescents and found there to be no relationship between intrinsic religiosity and depression as measured by the Depression-Happiness Scale. Pirutinsky et al. (2010) presented an informative study that included both strength of religiosity and type of religiosity (intrinsic versus extrinsic) in their study of the effects of religiosity on physical health and depression in the Jewish population. For Jews high in intrinsic religiosity, Pirutinsky et al. (2010) reported no relationship between physical health and depression; however among Jews with low intrinsic religiosity, reduced physical health was related to higher levels of depression. Once again, the research in the area of the relationship between instrinsic religiosity and depression presents opposing results; however, the subjects of these studies are not representative of the general U.S. population. The majority of the research presented here indicates that intrinsic religiosity is related moreso to decreased depression levels than is extrinsic religiosity.

Gender differences for the religiosity–depression relationship. The third general observation relating depression and religiosity is that males and females exhibit

different responses for this relationship. Two general concepts to note when studying the role of gender in the religiosity-depression link are (a) women in the United States have a higher rate of depression than men (Balbuena et al., 2013; Eliassen, Taylor, & Lloyd, 2005; Pratt & Brody, 2008), and (b) women in the United States are more religiously engaged than are men (Balbuena et al., 2013; Eliason et al., 2005; Maselko & Kubzansky, 2006). Pratt and Brody (2008) reported the 2005-2006 rate of depression among women (6.7%) to be significantly higher than for men (4.0%), and Maselko and Kubzansky (2006) determined that women scored signficantly higher than men for three religiosity measures of weekly public activity, daily private activity, and daily spiritual experience. Eliassen, Taylor, and Lloyd (2005) combined these two findings when concluding from their study of young adults that women were more religious and had more symptoms of depression than men. More recently, Balbuena et al. (2013) supported each of these concepts in their study in which a smaller percentage of men attended religious events monthly or more often compared to women, and a greater percentage of men never attended religious events compared to women. Women in the Balbuena et al. study were at siginificantly greater risk for diagnosis of major depressive episode than were men. Wenger (2011) reported that even among fairly nonreligious adolescents in Austria, girls were significantly more engaged in meditation, prayer, and interest in religious questions and discussions than were boys. These studies support the general conclusion that females engage to a higher degree in religious practices than do males and that females exhibit a higher level of depression than do males.

Through this review of the depression-religiosity link, I have identified strong evidence for an inverse relationship between religiosity and depression. To assorted degrees, research demonstrates an inverse relationship between religiosity and depression for various populations including college students, diverse adolescent groups, the elderly, and multiple religious and ethnic groups. Furthermore, I considered the role of the type of religiosity most closely associated with depression—instrinsic religiosity. Finally, I discussed varying conclusions related to gender differences in the religiosity-depression link. Now, I consider another aspect of the mediation equation—the depression-obesity link.

Depression and Obesity

Depression and obesity are significant public health problems that can inhibit daily functioning, are associated with social stigma, and tend to associate with decreased levels of health. Luppino et al. (2010) suggested that the cross-sectional study of depression and obesity is so extensive due to their intrinsic connection to cardiovascular disease—the obesity connection to cardiovascular disease being well-documented already and the depression connection to cardiovascular disease as evidenced by Penninx et al. (2001) and more recently by Van der Kooy, van Hout, Marwijk, Marten, and Beekman (2007) who concluded that major depression held as strong a risk for the development of cardiovascular disease as did smoking and diabetes. I have identified statistics for obesity and for depression in the United States previously. I now consider the multiple ways in which depression and obesity may be associated.

The obesity-to-depression link. There are a number of theories explaining why obesity may lead to increased depression levels. Socially, stigma associated with being obese has been proposed as a possible path to susceptibility to depression (Blaine, 2008; Faith et al., 2011; Fowler-Brown, Ngo, & Wee, 2012; Goldfield et al., 2010; Goodman & Whitaker, 2002; Needham, Epel, Adler, & Kiefe, 2010; Pan et al., 2012). Many of the social stigmatization issues are extensively reviewed and addressed by Puhl and Heuer (2009). Primarily, stigmatization and teasing about weight have been shown to be related to depression among obese adults, among those who experienced teasing as children, and among those seeking weight-related surgery (Puhl & Heuer, 2009). Chen (as cited in Puhl & Heuer, 2009) identified weight stigma to account for 32.6% of depression scores thereby supporting stigmatization as a path to susceptibility for depression. Therefore, research supports the role of weight stigma as a precussor to the development of depression.

Weight stigma accounts for approxiamtely one-third of the development of depression; another contributor to the development of depression among those with increased weight levels is the physical and functional tolls associated with carrying extra weight. A second proposed path by which obesity can lead to depression is through reduced quality of life related to physical/functional limitations (Faith et al., 2011; Fowler-Brown et al., 2012; Luppino et al., 2010). Janke, Collins, and Kozak (2007) found in their review of obesity and pain literature that being overweight/obese early in life was related prospectively to pain and to reduced quality of life. Heo, Pietrobelli, Wang, Heymsfield, and Faith (2010) demonstrated multiple areas for which extreme obesity

(class III obesity) was shown to impact physical and functional abilities including reduced yearly wage earnings, lower activity levels, increased joint pain, increased levels of hypertension, and increased mental health problems. Therefore, obesity can reduce quality of life ratings with concommitant increases in depression.

Obesity may also impact the biological response of the body to stress. A third way in which obesity is proposed as a predictor of depression is through the physiological stress response associated with the hypothalamic-pituitary-adrenal (HPA) axis (Faith et al., 2011; Goldfield et al., 2010; Goodman & Whitaker, 2002; Luppino et al., 2010). Bornstein, Schuppenies, Wong, and Licinio (2006) point out that both depression and obesity share the biological structure of the HPA axis with disregulation of this system as a common outcome. Futher evidence of this shared biology are seen by the fact that antidepressant and antiobesity treatments affect the HPA axis (Bornstein et al., 2006). While any of these three paths to depression seem plausible, there is evidence that the reverse process (depression leading to obesity) can also occur.

The depression-to-obesity link. There is burgeoning literature indicating that depression can lead to obesity. A number of researchers point to factors of increased eating and decreased levels of activity as correlates of depression that could contribute to weight gain (Blaine, 2008; Faith et al., 2011; Fowler-Brown et al., 2012; Goldfield et al., 2010; Goodman & Whitaker, 2002; Luppino et al., 2010; Needham et al., 2010; Pan et al., 2012; Stice, Presnell, Shaw, & Rohde, 2005; Vogelzangs et al., 2008). Farmer et al. (1988) are often cited for their cross-sectional and prospective study of 1,900 adults in which low physical activity and increased depression were reported cross-sectionally and

in which physical activity was identified as a predictive factor in depression. Therefore, depression resulting in reduction in physical activity can lead to weight gain.

Another popular explanation for the way in which depression can lead to obesity is through side-effects of antidepressant medications (Faith et al., 2011; Luppino et al., 2010; Pan et al., 2012; Vogelzangs et al., 2008). Zimmerman, Kraus, Himmerich, Schuld, and Pollmacher (2003) provided an extensive review of the myriad of pharmacologic treatments available for psychiatric patients. Zimmerman et al. (2003) concluded that all classes of psychotropics (antidepressants, antipsychotics, and mood stabilizers) can lead to weight gain for the user of such medication. The third possible mechanism by which depression may lead to obesity is through disregulation of the HPA axis and serotonin-related changes (Golfield et al., 2010; Goodman & Whitaker, 2002; Luppino et al., 2010; Pan et al., 2012; Stice et al., 2005; Vogelzangs et al., 2008). Notably, HPA axis disregulation is also promoted as evidence for the obesity-to-depression link discussed earlier.

Does depression lead to obesity or does obesity lead to depression? The cross-sectional approach to studying these questions is helpful in elucidating an understanding of the connection that may exist between depression and obesity. Goldfield et al. (2010) identified a relationship between depression symptoms and obesity among adolescents in Canada for specific subscales of depression (anhedonia, and negative self-esteem) and for overall depression scores. Among adults, Wiltink et al. (2013) reported a variety of obesity measures to be associated cross-sectionally with depression in those between 35 and 74 years of age. Obesity measures in the Wiltink et al. (2013) study included BMI,

waist circumference, waist-to-height ratio, and waist-to-hip ratio; all of these measures demonstrated strong relationships to the somatic-affective symptoms of depression in men, and all but the waist-to-hip ratio was strongly related to the somatic-affective symptoms of depression in women. Similarly, Zhong et al. (2010) identified a cross-sectional relationship between depression and obesity among 21-84 year old participants and found the depression domain of somatic complaints to be influenced most by obesity. These studies support the cross-sectional relationship between obesity and depression; however there are a few cross-sectional studies that do not demonstrate a relationship between obesity and depression.

While cross-sectional research supports the depression-obesity relationship, a few studies demonstrate a lack of relationship for these variables (Benson, Williams, & Novick, 2013; Pine, Goldstein, Wolk, & Weissman, 2001). Benson, Williams, and Novick (2013) found no relationship to exist between depression scores—measured as continuous values—and BMI among obese children and adolescents in Pennsylvania. Similarly, Pine, Goldstein, Wolk, and Weissman (2001) reported there to be no significant relationship between depression and obesity in childhood or in adulthood. Cross-sectional studies in the area of depression and obesity are inconsistent in their findings, and do not allow researchers to determine causality. In order to identify causality, researchers must employ longitudinal studies, and such studies are considered next.

Longitudinal research considering the obesity-to-depression link. While cross-sectional research can identify some helpful connections, longitudinal research

allows for determinations of causality. Faith et al. (2011) conducted a meta-analysis of prospective studies, on the obesity-to-depression relationship, which were conducted between 2000 and 2009. Faith et al. noted that methodology for these studies varied immensely in terms of sample size (approximately 400 to 74,000) and longitudinal period (1 year to 31 years). No single study involved in this review met both criteria of measured values for height and weight (as opposed to self-reported values) and of assessment of depression via interview (as opposed to a questionnaire: Faith et al., 2011). Ultimately, Faith et al. concluded that there was "good evidence" (p. e449) for the obesity-to-depression connection based on the nature of the longitudinal research in the 10 studies associated with their review. Eight of the 10 studies found significant prospective relationships between obesity and depression. In fact, the two studies that did not yield significant findings in this area were studies of the adolescent population and were conducted over only a one year period (Faith et al., 2011). Faith et al. concluded there to be solid support for the obesity-to-depression connection based on the results of their meta-analysis; however, some researchers have reported varied findings depending on the way in which they chose to measure obesity or depression and depending on the population they chose to study.

Longitudinal findings of the obesity-to-depression relationship are sometimes impacted by the way in which depression is measured or by the population being studied. Boutelle, Hannan, Fulkerson, Crow, and Stice (2010) considered the role of obesity in later development of depression among adolescent females. Boutelle et al. (2010) concluded that when considering the obesity-to-depression link using a dichotomous

evaluation of major depression (either the individual met criterion for major depression, or did not), adolescent females exhibited no significant relationship. However, when the measure of depression was a continuous variable, Boutelle et al. found increased adolescent female obesity level was significantly related to depression symptomology. This study by Boutelle et al. demonstrates that an observed relationship between obesity and depression may be dependent on the way in which depression is measured.

Disparities in the obesity-to-depression link are also demonstrated by two studies that measured depression in ways unique to prior research (Chang & Yen, 2012; Gariepy, Wang, Lesage, & Schmitz, 2010). Gariepy, Wang, Lesage, and Schmitz (2010) chose to consider the obesity-to-depression link by measuring the incidence of depression, rather than prevalence of depression, in relation to weight status. In doing so, Gariepy et al. (2010) reported that for middle-aged, Canadian participants who were followed for 12 years, increased obesity was significantly related to a decrease in major depressive episode in men but not in women. Chang and Yen (2012) also reported lower depression levels for overweight and obese elderly Taiwanese participants than for normal weight women and underweight men, and they cited ethnic differences (the Chinese culture) as the reason for their findings. Therefore, the difference in significant findings in the obesity-to-depression link may be impacted by the way in which depression is measured or by cultural influences.

There are multiple ways to measure obesity; and the choice for how to measure obesity may impact the statistical evaluation of a relationship. Hamer, Batty, and Kivimaki (2012) considered the obesity-to-depression link and accounted for obesity in

relation to metabolic health among an aging population in England. Participants who had two or more metabolic risk factors (from among hypertension, increased HbA1c readings, high C-reactive protein levels, high cholesterol, or high triglycerides) were identified as *metabolically unhealthy* and then were also categorized based on obesity status. Hamer et al. (2012) determined metabolically unhealthy obese participants to be at greater risk for depression symptoms at the two-year follow-up than were metabolically healthy obese participants. In this case, the obesity-to-depression relationship was supported by a varied approach to measuring obesity. As with cross-sectional research, longitudinal research in the depression-obesity realm also considered the alternate path of development—the depression-to-obesity link.

Longitudinal research considering the depression-to-obesity link. Evidence exists to support the possibility that depression can lead to obesity and much of this evidence is summarized in meta-analytical studies presented by Blaine (2008) and Faith et al. (2011). Blaine (2008) conducted a meta-analysis of research regarding the depression-to-obesity connection and concluded that among adolescents, depression increased the odds of weight gain by 2.5 times over adolescents who were not demonstrating depression symptoms. Blaine did not account for the role of antidepressants in weight gain, and the conclusion was only signficant for girls as studies of boys were scarce. Faith et al. (2011) incorporated parameters other than those utilized by Blaine (2008) in their review of 15 studies, between 1992 and 2009, in which depression was viewed as a predictor of obesity. Faith et al. described these studies as demonstrating wide variation in methodology, including large differences in sample size

(nearly 200 to over 9,000), variable longitudinal time-frames (1 year to 21 years), and different combinations of two key measurement strategies – self-reported versus measured height and weight, and interviews to assess depression versus questionnaire assessment. Faith et al. reported that eight of the 15 studies identified a significant relationship between depression and some measure of obesity (categorically, or in terms of BMI or weight gain). Interestingly, three of the eight studies reporting relationships found the relationship between depression and obesity to be negative (Faith et al., 2011). Similar to Blaine, Faith et al. concluded the depression-to-obesity link to be most prominent for females, and concluded that baseline weight in conjunction with baseline depression appears to be a factor in the depression-to-obesity relationship. Therefore, evidence from meta-analyses of longitudinal studies support the depression-to-obesity relationship.

There are two earlier studies that were considered in the previously discussed meta-analyses by Blaine (2008) and by Faith et al. (2011); these studies are considered more fully here as they relate directly to the parameters to be used in the current study—the maturational process from adolescence to adulthood. Franko, Striegel-Moore, Thompson, Schrieber, and Daniels (2005) presented a longitudinal study of Black and White women and followed them from adolescence into adulthood. Franko et al. (2005) evaluated the depression-to-obesity link for girls from age 16 to age 21 and from age 18 to age 21. Even in this short period of time, depression symptoms at age 16 and at age 18 were shown to be significantly related to obesity and BMI at age 21. Pine, Goldstein, Wolk, and Weissman (2001) also considered children and adolescents, ages 6-17, in their

10-15 year longitudinal study. Individuals who were treated for major depression at baseline were at greater risk of obesity as adults than those who were not identified with depression at baseline. Further investigation by Pine et al. (2001) revealed that the longer a child or adolescent remained in depression, the greater the chance of adult obesity for that individual. These studies by Franko et al. and Pine et al. provide longitudinal support for the depression-to-obesity link as adolescents progress into adulthood; other research considered the stated link for the adolescence phase alone.

Some research in the area of depression-leading-to-obesity has focused exclusively on the range of childhood and adolescence, but this research is less conclusive than the research in this area for other age groups. Incledon, Wake, and Margaret (2011) conducted a meta-analysis of research in the area of depression-to-obesity but limited their inclusion of studies to those that were conducted among children and adolescents. Primary findings from the Incledon et al. (2011) study were that support for the depression-to-obesity link in children was lacking, but that two studies of the adolescent depression-to-obesity link provided more compelling evidence of the link at one year and at four years from baseline.These studies were also included in the reviews by Blane (2008) and Faith et al. (2011) and are considered here.

The meta-analytic reviews of studies concerning the depression-to-obesity relationship highlighted two significant studies in this area. Incledon et al., (2011), Blaine (2008), and Faith et al. (2011) referenced earlier work by Stice, Presnell, Shaw, and Rohde (2005) in their reviews of the depression-to-obesity link. Stice et al. (2005) tracked the effects of behavioral and psychological risk factors, for the development of

obesity among girls, over four yearly measurements beginning in adolescence. Odds ratios supported the significant relationship between depression symptoms and obesity at the 4-year follow-up with each additional symptom of depression increasing the risk of obesity onset by four times (Stice et al., 2005). The second of two adolescent studies referenced by Incledon et al. (2011), Blaine (2008), and Faith et al. (2011) was the study completed by Goodman and Whitaker (2002). In this study, Goodman and Whitaker (2002) identified a relationship between depression symptoms at baseline among adolescents, who were not obese, and the development of obesity one year later; furthermore, among adolescents who were obese and were exhibiting depressed mood at baseline, adjusted BMI scores were also increased after one year. The cut points used for identifying *depressed mood* in this study were higher than the typical score of 16 referenced for the Center for Epidemiologic Studies Depression Scale (CES-D). Together, the Stice et al. and the Goodman and Whitaker studies provide a significant consideration for the depression-to-obesity link among the adolescent population.

Despite the support presented so far for the connection between depression and developing obesity, the way in which obesity is defined may yield varied results. For example, Tanofsky-Kraff et al. (2006) considered the effect of depression in childhood as it relates to obesity in adulthood and measured obesity using the dual-energy x-ray absorptiometry (DEXA) technique. The longitudinal timeframe involved in the Tanofsky-Kraff et al. (2006) study varied from as little at 0.10 years up to 7.9 years and used a population of children considered at risk for developing adult obesity. Among this specific population, depression in childhood was not significantly related to increases in

adult body fat mass as measured by DEXA. On the other hand, Vogelzangs et al. (2008) studied 70- to 79-year old Black and White individuals in the United States and found that depression in this age group was related to an increase in abdominal obesity (as measured by CT scans) in the 5-year follow-up but not to overall obesity (as measured by BMI and percent body fat). These studies by Tanofsky-Kraff et al. and Vogelzangs et al. demonstrate that longitudinal relationships between depression and obesity may be subject to the way in which obesity is measured.

The evidence in this review suggests that obesity can lead to depression and that depression can lead to obesity; thus, indicating that the relationship may be bidirectional. Specific research on the bidirectionality of obesity and depression is considered next.

Longitudinal research considering a bidirectional relationship. Some researchers have considered a possible bidirectional relationship between depression and obesity (Fowler-Brown, Ngo, & Wee, 2012; Luppino et al., 2010; Needham, Epel, Adler, & Kiefe, 2010; Pan et al., 2012); while there is wide support from this research for depression leading to obesity, conclusions from these studies are varied. Luppino et al. (2010) conducted a meta-analysis of longitudinal studies and identified 15 studies that fit their inclusion criteria and that considered odds ratios for the mentioned links. Based on odds ratios, Luppino et al. determined the depression-to-obesity link to be stronger than the obesity-to-depression link, thus, providing the primary generalization from research in this area.

As follow-up to the meta-analysis by Luppino et al. (2010), three more researchers have supported the general conclusion of the strength of the depression-to-

obesity relationship. Using data from the National Longitudinal Survey of Youth, which began in 1979, Fowler-Brown, Ngo, and Wee (2012) examined the bidirectional relationship for depression and obesity from 1992-1994. Fowler-Brown et al. (2012) found the obesity-to-depression relationship to lack significance but noted significant relationships with depression for overweight/obese Hispanic women and for obese women in the highest income bracket. Needham, Epel, Adler, and Kiefe (2010) evaluated the potential bidirectional relationship between depression and obesity over the span of 20 years and for Blacks and Whites between 18- and 30-years of age in their review of data from the Coronary Artery Risk Development in Young Adults study. Needham et al. (2010) did note a significant relationship for the depression-to-obesity link in that among Whites, depressive symptoms in early adulthood were associated with increased BMI in later adulthood and with waist circumference in later adulthood for both Whites and Blacks in the study. Pan et al. (2012) conducted a study of the bidirectional nature of the obesity/depression relationship by comparing measurements over 10 years for middle-aged and older female, registered nurses. Women in the Pan et al. (2012) study, who were categorized with depression at baseline, were significantly more likely to become obese at follow-up. These studies demonstrate that depression can lead to obesity for certain populations given a time period as little at 2 years and as great at 20 years.

While there is strong support for the depression-to-obesity relationship as evidenced over time, there are some specifics to these findings that should be identified. Luppino et al. (2010) reported that a significant relationship existed between baseline depression and future obesity, but not future overweight. On the other hand, Fowler-

Brown et al. (2012) reported that depression played a role in the development of both overweight and obesity among Hispanic women. Therefore, while the role of depression in the development of obesity is fairly solid, the role of depression in the development of overweight status is inconsistently characterized in the literature.

Another observation from longitudinal research in this area, which bears mention, is the role that the length of depression may play on future obesity levels. Luppino et al. (2010) concluded that a greater length of depression at baseline increased the predicted risk of obesity; this finding supports the earlier finding by Pine et al. (2001) in this area. A final observation, from this area of longitudinal research, is that depression leading to obesity may be different for men than for women. While Luppino et al. (2010) and Needham et al. (2010) found the depression-to-obesity relationship to be indicated in both sexes, Fowler-Brown et al. (2012) and Pan et al. (2012) reported a significant depression-to-obesity link only for a subset of women (Hispanic and high-income women as in Fowler-Brown et al., and female registered nurses as in Pan et al.). However, it should be noted that the Pan et al. study only included women.

Thus far in this review, I have discussed the key relationship of the current study as being the relationship between religiosity and obesity. I have further discussed the potential role of mediation that depression may play in this relationship. This review includes evidence that religiosity is primarily inversely related to depression and that increased depression is associated with increased obesity in both cross-sectional and longitudinal studies with strong evidence for this relationship coming from longitudinal

research. I now consider one final potential mediator of the proposed religiosity-obesity relationship—that of social support.

Social Support: A Potential Mediator Between Religiosity and Obesity

In this review, I have identified two potential mediators for the link between religiosity and obesity; they are depression and social support. Having just reviewed the role of depression as a mediator, I now consider the role of social support as a mediator of the religiosity-obesity relationship. Kim, Sobal, and Wethington (2003) considered a possible link between weight loss and religious social support, and Kim and Sobal (2004) considered the role of religion in the development of social networks with concomitant increases in social support. These stated studies represent the general hypothesis that religious engagement of various kinds afford an individual an opportunity to increase their social contacts and thereby gain support that may lead to a number of emotional and physical advantages that can lead to improved health or to reports of increased health status.

Social support is one avenue by which the larger construct of social networks is believed to impact health (other pathways being social influence, social engagement, and access to material resources; Berkman, 2004). There are four generally recognized types of social support—emotional, instrumental, appraisal, and informational (Berkman, 2004). Schafer (2013) offered a definition for social support as "the provision of resources between people" (p. 35). Schafer used this definition in studying the relationship between religiosity, social support, and health with a focus on intercessory prayer as a symbol of social support—prayer being a provision shared between

individuals. With the aforementioned definitions and constructs in mind, I now turn to a discussion of the connection that may exist between social support and religiosity.

Social Support and Religiosity

Religiosity and social support are often logically linked in research. While some theorists concluded there to be a direct protective effect of religiosity on health (Powell, Shahabi, & Thoresen, 2003), other theorists proposed that social support factors mediate the relationship between religiosity and health (Diener, Tay, & Myers, 2011; Koenig & Vaillant, 2009), and yet others concluded social support to provide a direct protective effect on health (see Brown, Salsman, Brechting, & Carlson, 2007, for a description of the protective effect of social support on alcohol consumption).

Researchers identify social support in a variety of ways; Edgell, Mather, and Tranby (2013) offered their own perspective on social support. Edgell et al. (2013) identified four profiles using latent class probability procedures to connect religion and social support in their study. Edgell et al. (2013) identified the four profiles for anticipated social support (a specific type of social support) as: (a) religious support, (b) broad support, (c) secular support, and (d) limited support. Religious support includes support from church leadership and members of one's church; secular support elicits support from decidedly nonchurch entities; broad support indicates the use of a wide variety of forms of support; and limited support indicates use of only one's spouse or significant other for support. Spousal and family support were common to all profiles presented by Edgell et al. Another research method used by researchers to identify the social support-religiosity connection is qualitative research.

Qualitative researchers have considered the relationship between religiosity and social support in efforts to identify more clearly the interaction that takes place. Shoemaker (2012) used qualitative research to provide insight into the connection between religiosity and social support through his interviews with war veterans. First, Shoemaker identified that churches in the United States can function to provide a level of social support for those serving in the military. Shoemaker further identified that symbols of support from the church (or individuals) at home (i.e., church bulletins, a Bible) were constant reminders of the support they were receiving from God and from those at home. The results of such religious social supports were realized through a sense of community and through a sense of comfort despite living in the reality of intensely dangerous situations and circumstances. Shoemaker recognized these supports as coping mechanisms for war veterans in active duty. Shoemaker also concluded that the social network engaged in during these deployment scenarios included a sense, by the deployed individual, of mutual responsibility between them and the church at home. In other words, while the church was supporting the deployed individual, the deployed individual felt a sense of responsibility to protect the church through his/her actions abroad. Shoemaker concluded his research with a discussion about religious social support and identified that religious social support consists of "spiritual coping (prayer), spiritual support (perceived comfort from God), congregational coping (rituals), and congregational support (support from fellow congregants)" (p. 18). This example of qualitative research links two variables of interest in this study—religiosity and social support. I now consider the general concept of religious social support more fully.

Religious social support. Religious social support can benefit the recipient of such support even beyond that derived from general social support (Burkman, 2004; Debnam, Holt, Clark, Roth, & Southward, 2012). An example of this added benefit is seen by the results of the Debnam, Holt, Clark, Roth, and Southward (2012) study in which a health behavior (fruit and vegetable consumption) was increased among African Americans with higher levels of general social support and was further increased (although not significantly) for those identified with higher levels of emotional religious support (for fruit consumption only). Further support for this view of religious social support was the significant increase in physical activity associated with anticipated religious social support. Additionally, religious social support resulted in a significant decrease in alcohol consumption beyond the already reduced levels associated with general social support. Therefore, while social support can benefit an individual who receives such support, religious social support may be an added benefit to the same individual.

Researchers have broadened the scope of studies related to religious social support to include considerations of denominational affiliation (Stroope, 2012), the African American population (Holt, Wang, Clark, Williams, & Schulz, 2013), and the Mexican American population (Krause & Hayward, 2013). Stroope (2012) connected the concept of social networks (as defined by embeddedness) with religiosity while considering a number of faith traditions. Stroope observed that higher levels of social embeddedness was associated with greater engagement in both religious activities and religious belief; however, embeddedness had a stronger connection to religious activity

than to religious belief. Such a finding supports the general understanding of the connection between church attendance and health in that the religious activity of church attendance is believed to result in greater social support (and therefore greater embeddedness) than does religious belief (Stroope, 2012). Stroope determined that those in the Catholic faith were less reflective of the embeddedness-religious activity interaction than were Protestants. Therefore, denominational affiliation may play a role in the expression of religious social support.

Two studies included consideration of racial minorities in the United States and the conclusions offer further insight into the role of religious social support among these groups. Holt, Wang, Clark, Williams, and Schulz (2013) considered the role of religious social support among an African American population representing a wide range of ages. Holt et al. (2013) found religious emotional support to mediate the association between religious involvement and emotional functioning as well as the association between religious involvement and depression. However, religious support was not associated with physical functioning in this group. Physical functioning included physical limits, due to health, for activities such as bowling or moving a table. Krause and Hayward (2013) considered the Mexican American population and reported no significant difference in the amount of support exchanged with other church members by Mexican American women as opposed to Mexican American men. Krause and Hayward also determined that both genders experienced a greater sense of belonging the more frequently they offered social support in the church setting. Finally, Krause and Hayward determined that Mexican American men secured greater benefit from the social

environment at church than did Mexican American women. These two studies demonstrate that among different races, religious social support relates to emotional functioning but not to physical functioning and that gender and racial status may affect the role of religious social support.

In this review of religious social support I have shared evidence that religious social support can provide protection beyond that provided by general social support. Using the terms shared by Edgell et al. (2013), *religious support* may offer added benefit over *broad support*. Furthermore, engaging in religious activity may be more salubrious than engaging in religious belief. I have also revealed evidence that religious support may present a stronger connection to emotional health (depression) than to physical functioning, and I have identified that all of these interactions may be different for women and men based on the race of the individual. Religious social support demonstrates the inherent relationship between religion and social support. However, researchers have studied the relationship between religiosity, social support, and health as separate entities. I now consider some research from this broader context.

Religiosity, social support, and health. Researchers have demonstrated interest in the associations between religiosity, social support, and health. As would be expected, there is evidence that increased religiosity is associated with increased social support (Moxey, McEvoy, Bowe, &Attia, 2011; Schnall et al., 2012). Moxey, McEvoy, Bowe, and Attia (2011) found some level of religious attendance, among the aging population in Australia, was associated with higher levels of social support compared to those who did not attend religious events at all. Moxey et al. (2011) also noted that aging Australian

men experienced less social support the less frequently they attended religious events. Schnall et al. (2012) considered multiple measures of social support in their study of varying levels of religious attendance among menopausal women participating in the Women's Health Initiative Observational Study. Positive social support, emotional/informational support, affection support, and positive social interaction were each positively related to all levels of religious attendance for those who reported at least one religious attendance in the previous month; overall, attendance was positively associated with optimism and negatively associated with depression. Schnall et al. reported that more-than-weekly religious attendance related most readily with the emotional/informational construct of social support while tangible support was least associated. These two studies demonstrate the relationship of religious attendance to increased social support for two populations—the aging Australian population, and menopausal women.

The general hypothesis of religiosity, social support, and health studies is that increased religiosity increases social support that leads to increased health status of the individual. However, a few researchers have reported that increased religiosity is related to lower level of physical health (Moxey et al., 2011; Thomas & Washington, 2012). Thomas and Washington (2012) considered the roles of religiosity and social support in the health-related quality of life of a population of African Americans who were undergoing hemodialysis treatment. Thomas and Washington observed that social support related significantly to emotional and physical health-related quality of life for these patients. Further analysis revealed that lower religiosity associated with greater

physical health-related quality of life among this population. Similarly, Moxey et al. (2011) noted that for the aging population in Australia, physical health was lower for those who demonstrated higher religiosity and social support. Moxey et al. concluded that the elderly with greater physical ailments used social support received through religious involvement to cope more successfully with their physical health. Although a reasonable hypothesis is that increased religiosity increases social support and further increases health status, those with significant health problems may be most likely to use religious engagement as a means of social support to help them through their ailment. In line with the premise of this study, in which social support is considered as a possible mediator between religiosity and obesity, I will consider more fully, here, the evidence related to social support as a mediator between various religion measures and various health measures.

Social support as mediator. Researchers have tested social support for its mediational role between religiosity and various health outcomes; studies identify social support as a mediator between religiosity and subjective well-being (Assari, 2013; Diener, Tay, & Myers, 2011) and religiosity and components of psychological or emotional function (Ai, Huang, Bjorck, & Appel, 2013; Hughes et al., 2004; Robins & Fiske, 2009). Assari (2013) determined that religious social support mediated the relationship between religiosity and subjective well-being among African Americans. This relationship was specific to ethnicity, with Afro-Carribeans and non-Hispanic Whites not demonstrating the same significant interaction. Diener, Tay, and Myers (2011) attempted to decipher a much broader view of the role of religiosity, social

support, and subjective well-being by considering a world-wide population of 455,104 individuals representing 154 countries. Despite great variation for religiosity, economic stability, and resource availability among countries, social support mediated the religiosity and subjective well-being relationship at the world-wide level. Furthermore, Diener et al. (2011) considered four major religions around the world for which they had ample data. For Buddhists, Christians, Hindus, and Muslims, religiosity and subjective well-being were associated. Diener et al. identified gender differences in which women in Buddhist and Christian societies demonstrated significantly higher levels of religiosity than did men, and men dominated in religiosity among the Muslim societies. For a wide range of nationalities and even for targeted populations within the United States, social support mediates the religiosity-subjective well-being relationship.

Social support is a recognized mediator in the relationship between religiosity and aspects of psychological (or emotional) wellness—namely, depression (Ai, Huang, Bjorck, & Appel, 2013; Holt et al., 2013), trait anxiety (Hughes et al., 2004), and suicidal ideation and attempt (Robins & Fiske, 2009). Ai, Huang, Bjorck, and Appel (2013) considered the mediational role of social support in the relationship of religious attendance and major depression among Asian Americans and concluded social support to be a mediator of this relationship. Holt et al. (2013) also considered the mediational role of social support in its relationship to religiosity and depression and reported religious social support to mediate the relationship between religious involvement and emotional functioning as well as the relationship between religious involvement and depression among African Americans. Hughes et al. (2004) also determined social

support to be a mediator in their study of 228 catheterization patients from a wide range of ages; social support mediated the relationship between religiosity and trait anxiety in these patients. Finally, Robins and Fiske (2009) determined that among undergraduate psychology students, higher levels of extrinsic religiosity were associated with fewer suicidal ideations and attempts. Social support was identified by Robins and Fiske as a mediator in this relationship. Contrary to these studies, Dulin (2005) reported that social support did not mediate a religiosity-major depression relationship, but, rather, moderated the relationship in community dwelling older adults. Dulin reported that those with low social support and higher levels of religious participation demonstrated less psychological distress. While a number of studies corroborate mediation by social support of various religiosity-psychological/emotional relationships, the population being studied may direct conclusions to moderation rather than mediation, as in this example, by Dulin, of community dwelling older adults.

Evidence is strong for the mediational role of social support in the relationship between religiosity and the constructs of subjective well-being and of aspects of emotional wellness; however, evidence for the mediational role of social support in the relationship between religiosity and physical wellness is less consistent (Chen & Contrada, 2007; Holt et al., 2013). Holt et al. (2013) identified a mediational role of religious social support for the relationship between religious involvement and the dependent variables of emotional functioning and of depression among the African American population; however, religious social support was not a mediator of the relationship between religious involvement and physical functioning. Chen and Contrada

(2007) studied the role of perceived social support in the relationship between religious involvement and cardiovascular health in studying the cardiovascular reactivity of 108 college students who were subjected to laboratory stressors. In this case, Chen and Contrada determined that perceived social support was not a mediator between religiousness (multiple components being considered) and cardiovascular reactivity; rather, perceived social support acted as a moderator between religiousness and cardiovascular reactivity such that high religiousness and high perceived social support resulted in lower systolic blood pressure under stress among these college students. The role of social support in the relationship between religiosity and some physical measures of health are not supported in the literature. Similarly, some research determined social support to play no mediational role but, rather, a role of moderation.

Studies mentioned previously (Chen & Contrada, 2007; Dulin, 2005) demonstrated that social support is a moderator rather than a mediator of relationships between religiosity and various health measures. Two other studies also refuted the specific role of social support as a mediator in these types of relationships; Son and Wilson (2011) considered the role of multiple psychological resources (emotional well-being, psychological well-being, and social well-being) as mediators in the religiosity-health link. Son and Wilson concluded that high religiosity in the home when growing up was related to adult health of varied age in 1995 that further impacted health 10 years later. While Son and Wilson identified emotional and psychological well-being as mediators between religiosity and health, social well-being, as measured by social integration, was not a mediator of the stated relationship. Edlund et al. (2010) also

determined that social support was not a mediator of the religiosity-substance use disorder relationship in their study of over 36,000 U.S. adults. Although a number of studies demonstrate a mediational role of social support in relationships of religiosity and various health measures, the mediational role is not evident for all measures of psychological or physical health.

I demonstrate, in this section, the varied role of social support as a mediator between religiosity and multiple health outcomes. In some cases, social support is a mediator; in other cases, social support is a moderator, and in other cases social support plays no role in the relationship between religiosity and the dependent variable being considered. Therefore, the evidence regarding mediational effects of social support is varied.

In this discussion of the link between religiosity and social support, I have demonstrated that religiosity can provide a level of social support that is connected to a number of health outcomes. The ways in which this connection occurs are varied based on how religiosity and social support are measured and are varied by gender and race. Primarily, I considered the role of social support as a mediator between religiosity and health. I now discuss the other aspect of this mediation—the relationship between social support and obesity.

Social Support and Obesity

Recent research linking social support and obesity is primarily focused on the role of social networks in the spread of obesity. Much of this focus centers on the work of Christakis and Fowler (2007) who reported that increased obesity of the ego (a term in

social network research referring to the participant in the study) and within the ego's social network are associated, and that this obesity clustering persisted to three degrees of separation from the obese individual. In other words, the friends and family of an obese individual were more likely to be obese than were individuals not affiliated with the ego; furthermore, friends and family of friends and family were more likely to be obese and represent the second degree of separation. Christakis and Fowler analyzed 32 years of obesity data from the Framingham Heart Study and concluded that one degree of separation from an obese individual in a social network represented a 45% higher chance of obesity for the alter (the social network term for a friend or family member of the ego) than for a member of a social network constructed at random. At two degrees of separation, the chances of obesity for alters were 20% higher, and at three degrees of separation the chances of obesity for alters were 10% higher. Christakis and Fowler identified a social phenomenon in which obesity tracks significantly among alters of an obese individual.

While research of social networks provided the basis for an understanding of the spread of obesity, the nature of social interactions provided even further insight. Christakis and Fowler (2007) found that, among mutual friends, obesity increased 171% when one became obese. Among nonmutual relationships—when the ego identified an individual as an alter, but the alter did not identify the ego as such—the likelihood of obesity by the alter still increased 57% subsequent to the ego's growing obesity. Christakis and Fowler observed obesity clusters for same-sex friends but not for opposite-sex friends, and determined that same-sex siblings demonstrated obesity clustering more

prominently than did opposite-sex siblings. Finally, Christakis and Fowler determined that there was a 37% increased chance of becoming obese among married couples if one spouse became obese, but that obesity clustering did not exist with immediate neighbors.

Other researchers confirmed the concept of obesity clustering for the young adult population (Leahey, LaRose, Fava, & Wing, 2011) and for adolescents (Halliday & Kwak, 2009; Valente, Fujimoto, Chou, & Spruijt-Metz, 2009). Christakis and Fowler's (2007) study included participants with a mean age of 38 years. Leahey, LaRose, Fava, and Wing (2011) considered younger adults (ages 18-25) in their study of the role of social ties in BMI and intention to lose weight. Leahey et al. (2011) concluded that the young adult population exhibited obesity clustering and reported a positive correlation between BMI for young adult egos and the number of overweight alters. Likewise, Valente, Fujimoto, Chou, and Spruijt-Metz (2009) identified obesity clustering for adolescents ages 11-15 with overweight adolescents exhibiting a two times greater likelihood of having overweight friends compared to normal weight adolescents. Halliday and Kwak (2009) also reported the clustering of adolescents based on BMI and reported the clustering to be stronger for girls than for boys. Halliday and Kwak also found clustering for height among adolescents, especially for boys, and concluded that such clustering is likely due to choosing to engage with those similar to oneself (homophily) rather than to peer effect. Similar to the Christakis and Fowler results, Leahey et al. noted that obesity clustering existed with close friends and family but was insignificant when considering colleagues and neighbors.

Researchers do not agree on the path by which obesity is transmitted through social networks. Christakis and Fowler (2007) concluded that social norms may be the controlling factor in the spread of obesity across social ties. This is to say that because obesity is associated with social ties, rather than geographic ties, egos accepted personal weight increases after identifying weight gain in alters with whom they connected. However, neighbors and colleagues who were geographically positioned near the ego did not generate the same effect. Leahey et al. (2011) countered the concept that social norms contributed to the development of obesity among egos and their alters through their determination that social norms were not a statistical mediating factor for obesity clustering among young adults. Hruschka, Brewis, Wutich, and Morin (2011) also discounted the hypothesis that social norms provided the path to obesity clustering. Hruschka et al. (2011) confirmed in their study of women and social norms that as obesity increased among egos, obesity also increased among alters. The only social norm pathway that elicited any type of significant interaction with obesity clustering was the antifat stigma pathway; however, the authors summarily discounted this pathway due to weak analytic support. The evidence regarding social norms as the pathway by which obesity is transmitted through social networks is inconclusive.

Although research presents a strong case for the spread of obesity through social networks, there is a dearth of research that relates social support directly to obesity. Therefore, I consider a plausible connection to another factor that will help to identify a more clear potential for the role of social support in expressed levels of obesity. Physical

activity is related to obesity levels, and recent research of the role of social support in enhancing health behaviors is substantial.

Social support for physical activity. Physical activity is a key factor in the development of obesity. Support, from an individual's social network, can influence the choices an individual makes for engagement in physical activity (de la Haye, Robins, Mohr, & Wilson, 2010; Trost & Loprinzi, 2011). Furthermore, childhood and adolescence is considered to be an important time for the forming of habits including lifestyle health habits (Gesell et al. 2008) that may be associated with obesity. Williams, Holmbeck, and Greenley (2002) identified parents, peers, school, and work as areas of interaction that influence the formation of healthy habits among children and adolescents.

Physical activity during childhood is an important factor in maintaining healthy weight during childhood and adolescence. Parental social support (Trost & Loprinzi, 2011) and social support from friend networks (de la Haye, Robins, Mohr, & Wilson, 2010) are two social influences for which there is evidence of the impact of social support on physical activity levels of children and adolescents. Trost and Loprinzi (2011) reviewed 103 studies and considered a number of factors in relation to physical activity levels of children including parental physical activity and parental support for physical activity. Trost and Loprinzi reported that parental support for physical activity significantly increased activity levels of children in 69% of the 71 studies reviewed, but that parental physical activity did not significantly influence the activity level of their children. De la Haye, Robins, Mohr, and Wilson (2010) studied Australian adolescents to determine the role of friendship networks on high-calorie food consumption, physical

activity, and screen time—all three are considered factors of obesity. De la Haye et al. (2010) reported that adolescent friends in two of the three male networks and one female network were significantly similar for engagement in organized physical activities such as school sports. However, for all six networks studied by de la Haye et al., adolescents and their friends were statistically dissimilar for unorganized physical activities such as active play outside of school and viewing of television. Researchers have identified that parental social support can influence the physical activity levels of children and that friend networks provide a significant level of interaction for organize physical activity but not for unorganized physical activity.

At the adolescent level, some researchers have considered the differences that may exist for physical activity among differing racial and ethnic minorities. In particular, recent research in this area includes considerations of African American adolescents (Baskin et al., 2013) and Latino adolescents (Gao, 2012; Gesell et al., 2008). Baskin et al. (2013) considered a number of factors related to moderate-to-vigorous physical activity (MVPA) among African American adolescents. Social support, from family members, for physical activity was one variable for which a significant relationship was identified. Specifically, MVPA increased by 5.46 minutes/day for each unit increase in family social support and this was a statistically significant increase. Therefore, among African American adolescents, familial social support for physical activity is associated with significantly increased levels of physical activity.

Social support also influences the level of physical activity among Latino youth (Gao, 2012; Gesell et al., 2008). Gesell et al. (2008) found that social support received

from friends and family of overweight, preadolescent, Latino youth, related significantly to the amount of activity in which these youth were engaged. Gesell et al. measured social support as friends and family who valued physical activity and those who encouraged or participated in physical activity with the study participant. Similarly, Gao (2012) considered the role of social support (which in this case included parental encouragement and three conduits of support—parental, peer, and teacher) for increasing physical activity levels of Latino children. Gao found that social support was significantly and positively correlated with increased physical activity among Latino children, and that social support predicts physical activity among Latino children. Therefore, when family, friends, and teachers of Latino youth encourage and support physical activity, Latino youth demonstrate an increase in physical activity.

Among adults, social support is a factor in engagement in physical activity (Harley et al., 2009; Leroux, Moore, Richard, & Gauvin, 2012) and in obesity (Leroux et al., 2012), but the relationship is not clearly defined (Siceloff, Coulon, & Wilson, 2013). Leroux, Moore, Richard, and Gauvin (2012) studied urban-dwelling adults and made comparisons between younger adults (ages 25-54) and older adults (those 55 and over). Primarily, among older adult egos, physical activity provided the strongest association to obesity with further influence from the number of alters perceived to be exercisers. Furthermore, exercising alters who lived in the same neighborhood demonstrated a greater influence on activity and obesity levels of egos than did exercising alters not in the neighborhood. Harley et al. (2009), while using qualitative research, identified that physical activity companions provided emotional, instrumental, informational, and

appraisal forms of support for physical activity among African American women. Leroux et al. (2012) and Harley et al. emphasized the role of the social environment on physical activity among different adult populations; whereas, Siceloff, Coulon, and Wilson (2013) considered the role of the physical environment on physical activity and BMI of underserved African American adults. Siceloff et al. (2013) identified physical activity as a mediator between infrastructure for walking and BMI among underserved African American adults; however peer social support for physical activity did not relate to BMI for this population. Therefore, social support affected physical activity levels among older adults and African American women, but not for underserved African American adults.

Previously, I discussed the relationship between religiosity and social support as well as the concept of religious social support. While the study of religious social support related to obesity is lacking, Kegler et al. (2012) conducted a qualitative study to compare African American and White adult perceptions of the rural church's role in providing social and environmental support related to healthy eating and physical activity. Adults in the Kegler et al. study were diverse in their feelings about health messages coming from church leaders or from the pulpit; however these same adults indicated that they talked with friends from church about eating healthy, losing weight, and exercising. Kegler et al. concluded that the social support received at church is an important venue for developing healthy habits and should be part of health intervention programs for rural African American and White populations.

The research relating social support directly to obesity is lacking, but some researchers have studied the role of social support in weight loss efforts (Kiernan et al., 2012; Kumanyika et al., 2009) and have reported divergent findings. Kiernan et al. (2012) considered the role of social support in weight loss among overweight/obese women engaging in a behavioral program. Women in this program reported *never* or *rarely* experiencing support from friends or family for healthy eating or for physical activity; percentages for these experiences ranged from 77.2% to 90.3%. Furthermore, sabotage for healthy eating and for exercise from friends and family was also high among this population of weight-loss seekers, though not as powerful as the support factors. Findings from research by Kumanyika et al. (2009) advocate the role of social support in weight loss. Kumanyika et al. found that among African American adults, partnering an individual with a family member, friend, or a random member of the weight loss group enhanced the effectiveness of the weight-loss program as long as the partner was engaged in the program and was losing weight as well.

To this point, I have not elucidated findings from research that directly connect social support to obesity. There is at least one study that considered the role of social support in the development of obesity. Ball and Crawford (2006) considered biological, psychological, social, and environmental domains for correlation to BMI among 790 young women. In addition to finding significant correlations between BMI and biological, psychological, and environmental domains, Ball and Crawford identified significant social correlations to BMI. However, results from the Ball and Crawford study are counterintuitive; women who perceived greater family support for healthy eating and

women who experienced less sabotage for healthy eating and for physical activity recorded higher BMI scores than women from other social support and sabotage categories. At least one study has approached the question of social support in relation to obesity, and the relationship demonstrated in this study indicates that increased positive social support relates to increased BMI and increased negative social support relates to decreased BMI.

In this section of this literature review, I have detailed the potential association between social support and obesity. There is strong evidence that social networks provide a path for the transmission of obesity, but this pathway is not clear to researchers. Social support is one component of social networks, and while few studies directly linked social support to obesity, a number of studies included connections between social support and physical activity (a known factor in obesity levels). As with many of the other relationships discussed in this review, the majority of studies were conducted using cross-sectional research approaches. Cross-sectional research does not inform the reader of causation, and the use of cross-sectional design likely contributes to the divergent results of research in this area; for this reason, there is a need for substantive longitudinal research in the relationships presented in the current study.

The Need for Longitudinal Research

Cross-sectional research design is a commonly used design in the social sciences (Frankfort-Nachmias & Nachmias, 2008). However, a limitation of cross-sectional research is that results do not indicate causality; rather, they indicate relationship (Frankfort-Nachmias & Nachmias, 2008). The overwhelming majority of studies that I

have presented in this review are cross-sectional in nature. As a result, although research indicates there is a relationship between higher levels of religious attendance and reduced depression symptoms among community-dwelling older adults (Sun et al., 2012), the cross-sectional nature of the research does not identify whether religious attendance leads to reduced depression or whether reduced depression leads to religious attendance.

For each of the relationships of interest in this study, researchers who conducted cross-sectional studies suggested the use of longitudinal designs to ellucidate the direction of the noted relationships. Authors of cross-sectional research in the area of religiosity and obesity advocate for the use of prospective designs to futher understand the role of religiosity and obesity among the African American population (Dodor, 2012; Reeves et al., 2012). Researchers of the religiosity-depression link have also identified the limitations of cross-sectional research (Ai et al., 2013; Gupta et al., 2011; Meltzer et al., 2011; Pirutinsky et al., 2011; Wenger, 2011). Similarly, researchers of the religiosity-social support link have identified the limitations of cross-sectional research to understanding of the link (Moxey et al., 2011; Schnall et al., 2012; Thomas & Washington, 2012). The one relationship that has ample support from longitudinal research is the relationship between depression and obesity (Blain, 2008; Faith et al., 2011; Franko et al., 2005; Goodman & Wittaker, 2002; Incledon, 2011; Pine et al., 2001; Stice et al., 2005). The interaction of variables for which there is the least empirical support is the interaction of social support and obesity; therefore, researchers have not identified the specific need for longitudinal research in this area.

In this review of the literature, I have identified plausible connections that exist between religiosity and obesity, religiosity and depression, religiosity and social support, depression and obesity, and, to a lesser extent, social support and obesity. Much of the empirical evidence relating these variables is in the form of cross-sectional designs, which do not allow for interpretation of causation. Furthermore, the strength of the relationships are dependent on the ways in which obesity and religiosity are defined and measured as well as the measures for determining types of social support or depression. Race, ethnicity, and gender are often important factors in the interactions of the stated variables, as is smoking status for the development of obesity. The problem remains—there is not a clear understanding of the way in which obesity develops from the adolescent years through adulthood. Furthermore, researchers have seldom considered the role of multiple dimensions of health in the development of obesity over time. I conducted a prospective study of the development of obesity from adolescence through adulthood and in light of the factors of religiosity, social support, and depression while considering the potential role of social support and depression as mediators of the religiosity-obesity relationship.

I will be using logistic regression to identify the relationships that exist between adolescent religiosity, adolescent social support, adolescent depression, and adult obesity. Furthermore, I will be using logistic regression to identify the extent to which social support and depression may mediate the religiosity-obesity relationship in the transition from adolescence to adulthood. In chapter 3, I detail the use of logistic regression for these purposes and in relation to the identified research questions of this study.

Chapter 3: Research Method

Introduction

The purpose of this archival, quantitative study was to explore the relationship that exists between adolescent religiosity and adult obesity while accounting for potential mediational roles of social support and depression for this relationship. The transition from adolescence to adulthood is a period of time during which increased obesity level occurs for many in the United States. To date, this transition has not been studied longitudinally from a multidimensional health perspective. While simple correlates between obesity and other variables have been identified for individuals in this transitional stage, I found no study that incorporated the associations of religiosity, social support, depression, and obesity from a longitudinal perspective for this population.

In this chapter, I discuss the chosen research design, the analytical approach for the study, the data collection methods as they relate to secondary data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris & Udry, 2014), and ethical considerations for this study.

Research Design and Rationale

In this archival study, using longitudinal data, of the relationship between adolescent religiosity and adult obesity, the independent variable (IV) was adolescent religiosity and the dependent variable (DV) was adult obesity. The variables of social support and depression were considered as mediators of the religiosity–obesity relationship when such a relationship was present. The research questions I have presented in this study concern an understanding of the relationships that may exist

between adolescent religiosity and adult obesity, adolescent religiosity and adolescent social support, adolescent religiosity and adolescent depression, adolescent social support and adult obesity, and adolescent depression and adult obesity.

My research questions also included the possibility that social support and depression were mediators of the religiosity–obesity relationship. As related to this study, the primary relationship of consideration was that of adolescent religiosity and adult obesity. Where adolescent religiosity was shown to be significantly associated with adult obesity, I considered the roles of adolescent social support and of adolescent depression as mediators of this relationship.

Generally, a prospective study of this nature would be beyond the scope of a dissertation due to the need for repetitive measures over time. However, the Add Health (Harris & Udry, 2014) data provided religiosity, social support, depression, and BMI measures for multiple waves of data collection, which began in 1994 and continued through 2008-2009. Use of the Add Health data allowed me to complete the longitudinal aspect of this study without the time delay and provided further insight into the longitudinal development of obesity.

Methodology

The Add Health (Harris & Udry, 2014) study began in 1994 and consisted of four waves of data collection through 2008-2009, with a fifth wave scheduled for 2015. The current study was based on the data collected in the Add Health study and allowed for a longitudinal perspective on the development of obesity. Methodology for all four waves of the Add Health study are presented in detail online at

www.cpc.unc.edu/projects/addhealth/design/designfacts; in this section, I identify the methodology that formed the basis for the Add Health study as related to the current study.

Population

The Add Health study design yielded a representative sample of U.S. adolescents in Grades 7-12 using an unequal probability method that included systematic sampling and implicit stratification (Harris et al., 2009). Wave I in-home data of the Add Health study were collected in April through December 1995 and were garnered from 12,105 core adolescent participants (Add Health, 2014); however, the dataset used by this study was the public-use dataset. The Add Health public-use data included responses from half of the original core sample and half of the African American oversample from Wave I of the Add Health data for a total of 6,504 participants; the public-use participants were randomly chosen from the original data set and are considered to be a nationally representative sample of U.S. adolescents in 1994-1995 (Harris et al., 2009). Wave II of the Add Health public-use data was collected in April through August 1996 and included responses from 4,834 of the 6,504 Wave I respondents; Wave III data were not used to answer the primary research questions of the current study but were used in the latent growth curve model analysis; and Wave IV data were collected in January 2008 through February 2009 and included responses from 5,114 of the Wave I respondents (Harris et al., 2009). There were 3,924 respondents in common to Waves I, II, and IV of the public-use dataset.

Sampling and Sampling Procedures of the Add Health Study

The sampling procedure used in the Add Health (Harris & Udry, 2014) study was a systematic and implicit stratification design (Harris et al., 2009) in which 80 high schools were chosen from a sampling frame of 26,666 high schools as representative of U.S. schools based on size, type, region, urbanization, and ethnicity. Feeder schools for the core high schools were identified for participation in the study, and for schools that chose not to participate in the study, replacement schools were identified randomly but based on predetermination as a specific category of school (Add Health, 2014). Over 100 schools were in the final core study, from which more than 90,000 student questionnaires were collected (Add Health, 2014).

Add Health researchers identified 12 strata based on sex and grade and set targets for equal sample sizes in each stratum. The sampling frame for in-home interviews of adolescents was based on completed in-school questionnaires and student rosters of 145 middle, junior high, and high schools (Add Health, 2014). From these core schools, 12,105 adolescents were chosen for in-home interviews; furthermore, specific groups were targeted for oversampling, including Black adolescents with college-educated parents, Cuban and Puerto Rican adolescents; Chinese adolescents; and physically disabled adolescents (Add Health, 2014). Ultimately, Wave I in-home interviews were conducted on 27,000 adolescents (Harris & Udry, 2014).

The in-home interviews were conducted using a variety of methods including audio computer-assisted self-interviews, computer-assisted self-interviews, computer-assisted personal interviews, paper-and-pencil interviews, telephone interviews

(including computer-assisted telephone interviews), face-to-face interviews, onsite observations and questionnaires, record abstracts, and cognitive assessment tests (Harris & Udry, 2014). Data from the Add Health study are maintained by the Inter-University Consortium for Political and Social Research (ICPSR). While the restricted dataset must be garnered through official steps with the ICPSR, the public-use datasets for Waves I, II, III, and IV of the Add Health study are available for direct download from the ICPSR website (Harris & Udry, 2014).

Instrumentation and Operationalization of Constructs

I measured or constructed four variables from the Add Health datasets. The independent variable was adolescent religiosity, and the dependent variable was adult obesity. Potential mediating variables were adolescent social support and adolescent depression. Adolescent religiosity was chosen to represent the spiritual dimension with the understanding that many spiritual individuals would not identify as religious yet an individual high in religiosity would also be viewed as high in spirituality. Adolescent religiosity was constructed from Add Health (Harris & Udry, 2014) Wave I data, which were collected between April and December 1995. Adolescent depression was measured using data from Wave I and Wave II of the Add Health data. Wave II data were collected between April and August 1996. Wave I data were used to control for high symptomology for depression during adolescence, while Wave II was used to identify the development of depression symptoms. Social support was constructed from Wave II data. Obesity was constructed from Wave I and Wave IV data, with Wave I data used to

control for obesity level during adolescence and Wave IV data used to identify obesity in adulthood. Wave IV data were collected between January 2008 and February 2009.

Depression. Depression, in the Add Health (Harris & Udry, 2014) study, was measured using a variation of the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The full CES-D is a 20-item scale that can yield scores between 0 and 60 and that represents depression symptomology among the general population (Radloff, 1977). Responses to each item are based on feelings experienced during the previous week and are coded on a scale between 0 (*rarely or none of the time*) and 3 (*most or all of the time*). Items in the scale include such statements as *I felt lonely* and *I enjoyed life* (Radloff, 1977). The cut score for the CES-D is 16, meaning that individuals with a total score below 16 on the CES-D do not exhibit significant clinical symptomology of depression (The Center for Epidemiologic Studies Depression Scale Revised, 2008).

Wave I and Wave II data of the Add Health study include 19 of the 20 items. In accordance with another study that used the Add Health data for studying adolescent depression (Needham, 2009), I summed the scores from the 19 items and used the continuous score in my analyses. Responses ranged between 0 and 3 for each item for a possible total depression score of 57, with a higher score representing greater depression symptomology. Wave I depression scores were used to control for depression by including Depression at Wave I as an independent variable in the regression analysis. Wave II depression scores were used as continuous measures and were included as a primary independent variable and as a mediating variable. Reliability of the Add Health

version of the CES-D in identifying adolescent depression symptomology is strong (Cronbach's alpha = .86, Katcher, 2014; Cronbach's alpha = .84-.88 for Wave I and .85-.89 for Wave II, Le, Tov, & Taylor, 2007; and Cronbach's alpha = .87 for Wave I, Needham, 2009).

Religiosity. While the Add Health study did not use a formalized scale for identifying religiosity, researchers have identified ways to operationalize the construct of religiosity from the Add Health data (Le, Tov, & Taylor, 2007). Researchers often dichotomize religiosity into intrinsic and extrinsic constructs (Le, Tov, & Taylor, 2007; Nkansah-Amankra et al., 2012). This dichotomization is used to help differentiate between different foci for religious expression. Individuals with high intrinsic religiosity demonstrate commitment to personalizing their religion while those with high extrinsic religiosity demonstrate a commitment to outward forms of religious commitment such as attending church or religious services.

Le et al. (2007) identified four questions from the Add Health survey that related to religiosity. Internal religiosity was identified by the questions "How important is religion to you?" and "How often do you pray?" The first question used a response code of 1 (*very important*) to 4 (*not important at all*), and the second question used a response code of 1 (*at least once a day*) to 5 (*never*). Two questions were used to represent external religiosity. For the question "In the last 12 months, how often did you attend religious service," the response code was 1 (*once a week or more*) to 4 (*never*). The final question related to attending religious youth activities and was coded on a 1 (*once a week or more*) to 4 (*never*) scale. Le et al. reverse coded all four measures and added them

together to create one continuous score for religiosity (α = .86 to .91) in which a higher score represented a higher level of religiosity.

In the current study, I used a continuous score determined by reverse coding responses for the four noted questions related to religiosity. A higher religiosity score represented increased levels of religiosity. Furthermore, researchers have identified intrinsic and extrinsic religiosity to associate differently with studied variables. However, Le et al. (2007) found that intrinsic and extrinsic religiosity for adolescents in Wave I of the Add Health study were highly correlated. For this reason, I considered only the composite score of religiosity in my study.

Social support. The social support measure was derived from eight questions in the Add Health study. Adolescents were asked in the Wave II survey, "How much do you feel that [adults, teachers, parents, friends] care about you?" The remaining four questions were about family support, including the extent to which the participant felt understood by the family, wanted to leave home, had fun with the family, and was paid attention by the family. Each question was coded on a 1 (*not at all*) to 5 (*very much*) scale. Beaver, Boutwell, and Barnes (2014) used the sum of the responses to these eight questions to create a social support scale (Cronbach's α = .76, Wave II).

Obesity. The final variable of consideration in the current study was adult obesity, which was calculated by determination of BMI from measured height and weight values taken during the Wave IV in-home surveys. BMI is calculated by dividing the weight of the individual (kg) by the squared height (m) of the individual (CDC, 2014). The resulting BMI value is used to categorize adults for weight status as follows: A BMI

below 18.5 is considered underweight; a BMI between 18.5 and 24.9 is considered normal; a BMI between 25.0 and 29.9 is considered overweight; and a BMI of 30.0 and above is considered obese (CDC, 2014). I used BMI as a continuous measure in the primary statistical analyses. Further discussion of changes in obesity included the categorization of adult individuals as nonobese for a BMI under 30.0 and obese for a BMI at or above 30.0. To control for obesity level during adolescence, I evaluated males and females separately and used the continuous measure of BMI. Further evaluation of changes in obesity was accomplished by identifying BMI classifications for adolescents in Wave I based on age. Categorization of obesity through BMI for children and adolescents is different than for adults. Ogden and Flegal (2010) described the most recent classification standards for children and adolescents being used by the National Center for Health Statistics; BMI for children and adolescents is dependent on age and sex, for which growth charts from the CDC are used. Children with a BMI-for-age that places them in the 85th percentile and up to the 95th percentile are considered to be overweight, while those at the 95th percentile and above are categorized as obese. When categorization of obesity levels for adolescents was used, the current percentile values based on the CDC were used.

Control variables. I have already mentioned the control for high depression symptomology and for obesity for the Wave I measures of adolescents. Other variables being controlled in this study included sex, smoking status, race, age, and income. Smoking status was identified using the following question from Wave I: "During the past 30 days, on how many days did you smoke cigarettes?" Respondents indicating no

cigarette use in the previous 30 days were identified as *nonsmokers*, while respondents indicating that they had smoked a cigarette in the previous 30 days were identified as *smokers*. The same question from Wave IV was used to identify smokers versus nonsmokers at Wave IV. Race was identified by the following questions in Wave I: "What is your Hispanic or Latino background?" followed by six possible choices; and "What is your race?" followed by choices of *White, Black or African American, American Indian or Native American, Asian or Pacific Islander,* and *Other*. Those identifying a Hispanic/Latino background were categorized as Hispanic, and all others were designated for race based on their response to the race question. Sex was controlled using Wave I determination of biological sex. Age at Wave I was also determined from relevant demographic information. Family income was included as a socioeconomic control at Wave I and was determined using imported data from the parental questionnaire of the Add Health study. Parents were asked at Wave I to report their total household income. Income at Wave IV was determined as a range for total household income as reported by the respondent. The factors of race, age, smoking status, and household income were controlled for by including them as independent variables in the regression analysis.

Table 2

Variables Associated With Various Waves of the Add Health Data

Variable	Wave I	Wave II	Wave III	Wave IV
BMI (DV)	X[a]	X[b]		X
Religiosity (IV)	X			
Depression (MV)	X[c]	X		
Social support (MV)		X		

Note. BMI = body mass index as a measure of obesity; MV = mediating variable. [a]Used to control for adolescent BMI. [b]Used in structural equation model. [c]Used to control for adolescent depression.

Data Analysis

In this study, I employed two forms of analysis to answer my research questions related to the relationship that exists between obesity and measures of religiosity, social support, and depression. First, I determined the extent to which factors of adolescent religiosity, adolescent social support, and adolescent depression predicted an outcome of adult obesity. Where there was a noted relationship between adolescent religiosity-adult obesity, I determined the potential role of adolescent social support and of adolescent depression as mediators of this relationship. Regression analysis is one method for predicting an outcome based on one or more variables (Field, 2009, p. 198) and is the method I used to answer my research questions. For mediation analysis, I followed the steps presented by Baron and Kenny (1986).

Second, to more fully describe the development of obesity from adolescence to adulthood in relationship to key factors of religiosity, social support, and depression, I analyzed the change in obesity over time through structural equation modeling. Singer and Willett (2003) identified three requirements for the study of change over time as follows: (a) multiple waves of data available for analysis, (b) an outcome exhibiting systematic change over time, and (c) a cogent time scale. The Add Health data provides four waves of data from which to compare the changes in BMI. Furthermore, Gordon-Larson, The, and Adair (2010) used the Add Health data to demonstrate the systematic change in obesity from Wave II to Wave IV, and noted that the prevalence of obesity doubled two times—obesity among normal weight adolescents doubled by Wave III and then again by Wave IV. Finally, the Add Health data represents an appropriate time scale in which to observe changes in obesity with approximately 5 years between waves II and III and 7 years between Waves III and IV. In the Gordon-Larson et al. (2010) study, the obesity rate for adolescents in Wave II was 13.3%, and increased to 36.1% in Wave IV. Similarly, Juonala et al. (2011) found that in a study of international cohorts, the percentage of normal weight adolescents who became obese in adulthood was 14.6% over the average span of 23.1 years. Therefore, in the United States as well as internationally, increased levels of obesity is a trend during the transition from adolescence to adulthood and this trend has been established using repeated measures of obesity within the Add Health data previously. My research questions were as follows:

Research Question 1. What is the relationship that exists between adult obesity and adolescent religiosity, depression, and social support?

Null Hypothesis 1. There is no statistically significant relationship between adult obesity and adolescent religiosity, depression, and social support.

Alternative Hypothesis 1. There is a statistically significant relationship between adult obesity and adolescent religiosity, depression, and social support.

Research Question 2. To what extent does adolescent depression mediate the relationship between adolescent religiosity and adult obesity?

Null Hypothesis 2. Adolescent depression is not a statistically significant mediator of the relationship between adolescent religiosity and adult obesity.

Alternative Hypothesis 2. Adolescent depression is a statistically significant mediator of the relationship between adolescent religiosity and adult obesity.

Research Question 3. To what extent does adolescent social support mediate the relationship between adolescent religiosity and adult obesity?

Null Hypothesis 3. Adolescent social support is not a statistically significant mediator of the relationship between adolescent religiosity and adult obesity.

Alternative Hypothesis 3. Adolescent social support is a statistically significant mediator of the relationship between adolescent religiosity and adult obesity.

Public-Use data for Wave I, II, and IV of the Add Health (Harris & Udry, 2014) study was downloaded from the ICPSR website. Statistical analysis was conducted using Stata 14 Data Analysis and Statistical Software, and weights developed by the Add Health researchers were used to account for unequal sampling of the Add Health data. Within Stata, data from the Add Health study was manipulated to create the scores related to religiosity, depression, social support, BMI and the various control variables.

The primary analytical procedure used was multiple linear regression, which considered three independent variables (adolescent religiosity, social support, and depression), a continuous dependent variable (adult BMI), and five control variables (sex, age, smoking status, race/ethnicity, and income). The Public-Use data of the Add Health study was determined to be of sufficient size to meet the needs of logistic regression analysis. Using G*Power 3.1.9.2 (Faul, Erdfelder, Buchner, & Lang, 2009) and accounting for an alpha level of .05, a power level of .80, and an odds ratio of 1.4, the a priori sample size for logistic regression is 360. The identified alpha level of .05 and power level of .80 are supported as standard levels in quantitative research (Burkholder, n.d.). Due to a lack of longitudinal studies that yield odds ratios for the religiosity-obesity relationship, I determined the odds ratio using the depression-obesity relationship. The odds ratio is derived from the meta-analysis conducted by Luppino et al. (2010) in which the adjusted odds ratio for depression leading to obesity among adults was 1.4.

In order to improve the variance associated with my analysis, I chose to work with my dependent variable (adult obesity) as a continuous measure rather than as a dichotomous variable. This resulted in the change to using linear regression rather than logistic regression as my main analysis. G*Power 3.1.9.2 analysis for linear multiple regression, alpha level of .05, power level of .80, effect size of .1, and accounting for 11 predictors yielded a sample size requirement of 81. The Public-Use data was of sufficient size for the study.

Multiple linear regression was used to identify if adolescent religiosity, adolescent social support, and adolescent depression (IVs) could significantly predict the dependent

variable of adult obesity as measured by BMI (Research Question 1). The analysis related to Research Question 1 (RQ1) controlled for sex by analyzing males and females in separate regression analyses; race, age, initial BMI, initial depression, family income at waves I and IV, and smoking status at waves I and IV were control variables and were included as independent variables in the regression analyses. All variables were utilized as continuous scores except for race, income at Wave I and at Wave IV, and smoking status at Wave I and at Wave IV. Regression coefficients and confidence intervals were reported separately for males and females.

Research Questions 2 and 3 related to the possibility that adolescent depression and adolescent social support, respectively, mediated the relationship between adolescent religiosity and adult obesity. In order to test for mediation, the relationship between adolescent religiosity (the independent variable) and adult obesity (the dependent variable) had first to be established. I used regression analysis to determine if a significant correlation existed between adolescent religiosity and adult obesity. All analyses related to this portion of the mediation relationship controlled for smoking status, race, age, depression symptomology, adolescent obesity status, and income in the same way as outlined for RQ1. Adult obesity, as measured by BMI, was regressed on adolescent religiosity. The next step of meditational analysis was to regress the mediator on the independent variable, which, in this case, was a regression of adolescent social support and adolescent depression on adolescent religiosity. The final step of mediation was to regress the dependent variable on the mediators, which, in this case was a regression of adult obesity on adolescent depression and adolescent social support. Had

all of these relationships been significant and had the direct relationship from adolescent religiosity to adult obesity been reduced to insignificance by the mediating relationship, then the Sobel test (Sobel, 1982) would have been utilized to account for an indirect effect (Preacher & Hayes, 2008). However, the relationships needed to account for mediation were not present and further analysis was unnecessary.

Repeated measures ANOVA was proposed for further analysis of the changes seen in obesity over time and in relation to religiosity in adolescence. Limitations of the analysis that could be used to analyze complex survey data using Stata 14 resulted in a change in analysis from repeated measures ANOVA to growth curve modeling as a function of structural equation modeling (SEM). Paths for adolescent religiosity, depression, and social support were associated with the BMI scores for which data was collected. Latent variables representing mean intercept, linear slope, and quadratic slope were used to identify the change in obesity over time and age categories were used to identify the growth in BMI over time. Obesity, as measured by BMI, was taken from Wave I, Wave II (Wave I and II BMI were from self-reported measures), Wave III, and Wave IV (Wave III and IV BMI were from measured values). Figure 2 demonstrates the primary longitudinal interactions observed in this study in relation to the Add Health wave information.

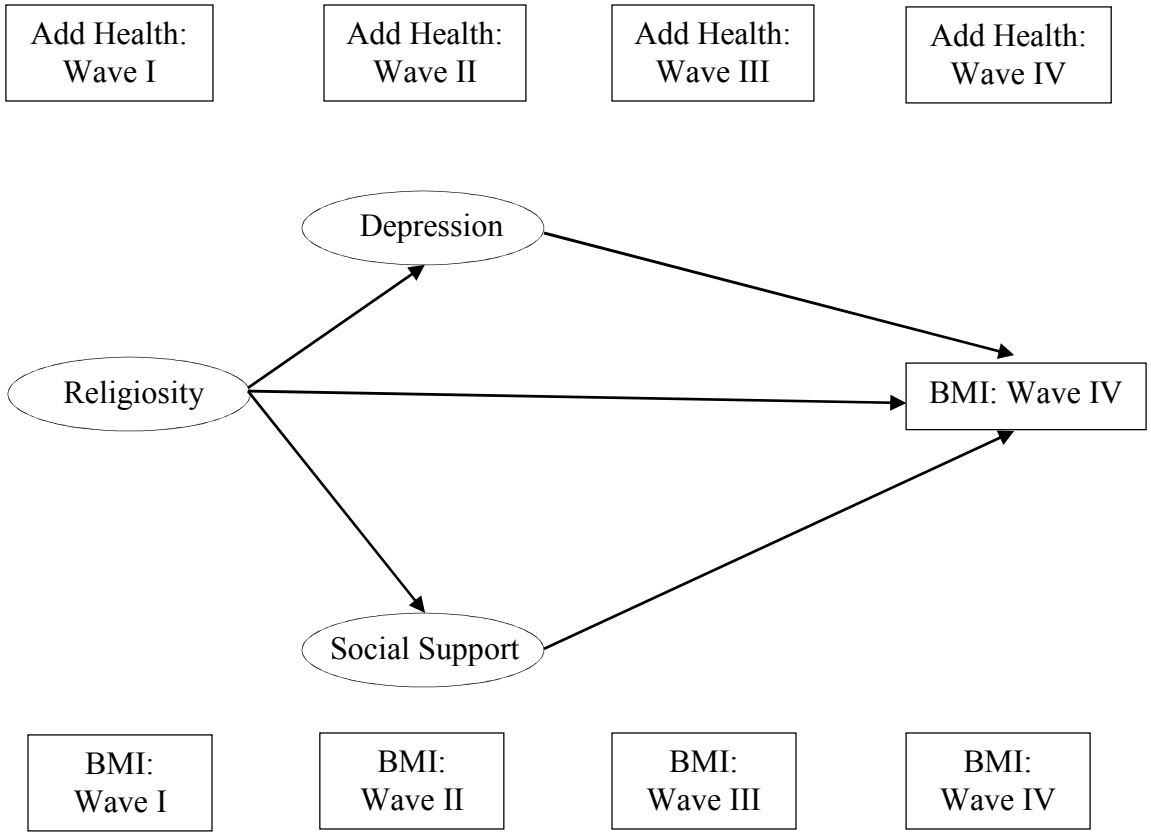


Figure 2. Study interactions demonstrating the longitudinal format for the two potential mediational relationships as well as the BMI data to be used in the SEM analysis.

Threats to Validity

Generally, threats to internal validity and to external validity are considered so that researchers can account for ways in which to strengthen their research design in order to identify causation and generalizability (Frankfort-Nachmias & Nachmias, 2008). However, the data used in this study had been previously collected by the Add Health (Harris & Udry, 2014) researchers. Therefore, in this section, I discuss, first, the ways in which the Add Health study accounted for the primary threats to validity in their data collection efforts, and then I discuss the potential threats to validity for this study.

Threats to internal validity. The primary threat to internal validity for the Add Health data is the threat posed from selection (Creswell, 2009). Chantala and Tabor (2010) identified that the Add Health data is an unequal sampling of adolescents in Grades 7 through 12 in the United States with Wave I data being collected in 1994 and Wave II data being collected in 1996. In order to produce results that are nationally representative from this data, researchers must apply weights to the data to account for the unequal samplings of the Add Health data. In particular, the unequal sampling in the Add Health data included oversampling of some ethnic groups, sampling of students with disabilities, and sampling of genetic relations such as twins and various sibling combinations (Chantala & Tabor, 2010). The Add Health data also accounts for two specific threats to internal validity—maturation and mortality, as described by Creswell (2009). Maturation is accounted for by the Add Health data due to the selection of participants from the same age group of adolescents (Grades 7-12). Mortality is accounted for by virtue of the large sampling of the study. The nationally representative sample included 18,924 Wave I in-home participants and 13,570 Wave II in-home participants (Chantala & Tabor, 2010). Finally, the Add Health researchers accounted for the testing and instrumentation threats to internal validity by spreading out the in-home surveys and by using some of the same questions in various waves of the study. Wave I surveys were conducted in 1994 and 1995, Wave II in 1996, Wave III in 2001 and 2002, and Wave IV in 2008 and 2009 (Harris et al., 2009). In some cases, questions were identical between various waves, and in some cases, questions for particular constructs changed.

Threats to external validity. Generalization of outcomes from the Add Health data are limited due to the fact that the Add Health data is representative of U.S. adolescents in 1994 and 1995. Care must be taken not to generalize the findings of Add Health data to other or all generations of U.S. adolescents or to adolescents from other countries or time periods.

Threats to validity in the current study. Although most of the internal threats to validity have been accounted for by the Add Health study, there were a couple of threats that I needed to account for in the current study. The threat to internal validity caused by extreme scores in the regression analysis was accounted for by the robust nature of survey data analysis using Stata. Secondly, I accounted for the selection threat identified previously by appropriately applying weights and cluster information provided by the Add Health researchers.

The primary threats to validity in the current study were construct validity threats. Previously, religiosity has been measured in relation to attendance at religious services, engaging in prayer, and feelings about the importance of religion in one's life; furthermore, religiosity has been viewed as separate constructs of intrinsic religiosity and extrinsic religiosity. Researchers have used questions from the Add Health data to consistently identify the construct of religiosity. Nonnemaker, McNeely, and Blum (2003) used the terms public religiosity and private religiosity in their study based on the Add Health data. Private religiosity was derived from questions in the Wave I data about prayer and the importance of religion to the individual, while public religiosity was associated with questions about attending religious services and youth church functions.

These same questions and constructs were used by Le et al. (2007) and Nkansah-Amankra et al. (2012) in their respective studies that used the Add Health data. However, Le et al. (2007) specifically found that private and public measures of religiosity were highly correlated in the Wave I data. Le et al. chose to consider a total measure of religiosity in their study rather than sublevels of the construct; I also used the total measure in this study.

Another potential threat to construct validity was the use of BMI to represent obesity. The CDC (2014) identifies three populational examples for which BMI does not correlate well with body fatness. The three examples identify that body fatness can vary, given the same BMI score, for differences in sex, age, and status as a trained athlete. A recommendation for determining obesity-related health risks is to measure waist circumference as this accounts for increased amounts of abdominal fat, which is a known contributor to obesity-related diseases (CDC, 2014). Wave IV surveys from the Add Health study included measurements for waist circumference; however, because similar measurements were not taken in previous waves of the study, there was no baseline for comparison using waist circumference measurements.

I controlled for obesity status in adolescence, thus, presenting another concern for construct validity. The BMI components of weight and height were self-reported in the Wave I surveys. Research consistently indicates that boys and girls tend to lack precision in their estimates and reports of height and weight, and they underestimate their weight; however, the inconsistencies do not significantly affect the determination of BMI (Bowring et al., 2012; Fonseca et al., 2010; Yoshitake, Okuda, Sasaki, Kunitsugu, &

Hobara, 2012). Fonseca et al. (2010) concluded that use of adolescent self-reported height and weight is most warranted when used as a continuous variable. I used self-reported values for height and weight from the adolescents in Wave I and II of the Add Health study, thereby reducing this concern for construct validity.

Ethical Considerations

The North Carolina School of Public Health IRB oversaw the collection and storage of all facets of the Add Health study and written informed consent was obtained from all participants in the study (Harris et al., 2009). While the Add Health data contain ID numbers, the files do not contain identifiers of respondents. This is important due to the sensitive nature of many of the Add Health survey questions. Furthermore, in-home surveys were completed by interviewers on computers to alleviate the potential of a paper trail; for sensitive questions, participants heard the question while wearing earphones and recorded their own score. The Add Health researchers demonstrate the bulk of the responsibility for the ethical treatment of the study participants and for the storage and availability of the data associated with the study. My responsibility with the Add Health data was limited due to the unrestricted availability of the Public-Use dataset from the ICPSR website. Individuals who download the data are required to follow formalized steps with ICPSR, which include registering an email address with ICPSR and identifying organizational affiliation prior to using the data. I received approval from the Walden IRB (approval number 07-06-15-0169976) for use of the Add Health dataset. I downloaded the data to my office computer, which was password protected and was located in an individual, locked office. Furthermore, by agreeing to terms of the ICPSR, I

resolved not to attempt to identify participants of the Add Health study. In my presentation of the results, I did not include information that might allow for the identification of any of the study participants.

Summary

This study was a prospective study of the general relationship that exists between adolescent religiosity, adolescent social support, adolescent depression, and adult obesity (RQ1). A significant relationship between adolescent religiosity and adult obesity was followed up for consideration of the role of adolescent social support and depression as mediators of the religiosity-obesity relationship (RQs 2 and 3). All three research questions were addressed using multiple linear regression. Adolescent religiosity was the independent variable, adult obesity was the dependent variable, and adolescent social support and depression were potential mediating variables. All analyses controlled for age, sex, race/ethnicity, smoking status, and income as these factors have elicited significant differences in previous studies utilizing one or more of the study variables. Identification and strength of interaction for multiple dimensions of wellness are reported in Chapter 4. Elucidation of the multiple relationships that may exist between spiritual, emotional, social, and physical dimensions of wellness will aid in the scientific understanding of the development of obesity. Such an understanding of the development of obesity can help to prevent the spread of obesity and to better inform practitioners on potential therapies for treating those already struggling with the realities of obesity.

Chapter 4: Results

Introduction

The purpose of this archival study was to quantitatively explore the relationship that may exist between adolescent religiosity and adult obesity while accounting for the possibility of mediation of this relationship by depression and social support. The following research questions and hypotheses were used to guide the statistical analyses in this study:

Research Question 1. What is the relationship that exists between adult obesity and adolescent religiosity, depression, and social support?

Null Hypothesis 1. There is no statistically significant relationship between adult obesity and adolescent religiosity, depression, and social support.

Alternative Hypothesis 1. There is a statistically significant relationship between adult obesity and adolescent religiosity, depression, and social support.

Research Question 2. To what extent does adolescent depression mediate the relationship between adolescent religiosity and adult obesity?

Null Hypothesis 2. Adolescent depression is not a statistically significant mediator of the relationship between adolescent religiosity and adult obesity.

Alternative Hypothesis 2. Adolescent depression is a statistically significant mediator of the relationship between adolescent religiosity and adult obesity.

Research Question 3. To what extent does adolescent social support mediate the relationship between adolescent religiosity and adult obesity?

Null Hypothesis 3. Adolescent social support is not a statistically significant mediator of the relationship between adolescent religiosity and adult obesity.

Alternative Hypothesis 3. Adolescent social support is a statistically significant mediator of the relationship between adolescent religiosity and adult obesity.

In this chapter, I describe the Add Health (Harris &Udry, 2014) dataset in relation to the variables used in this study; I present descriptive characteristics of the sample population; I report the results of regression analyses related specifically to each research question; and I discuss the supporting statistical procedures used in the study.

Data Collection

The Add Health study (Harris &Udry, 2014) is a longitudinal study that includes four periods of data collection. The study involves a nationally representative sample of U.S. students in Grades 7-12 in 1994-1995 (Harris, Halpern, et al., 2009). Wave I data were collected in 1994-1995, and the public-use data, which were used in the present study, consisted of 6,504 respondents who were randomly selected from the full contractual dataset; Wave II data were collected in 1996 and included 4,834 of the Wave I public-use respondents; Wave III data were collected in 2001-2002 and included 4,882 of the Wave I public-use respondents; and Wave IV data were collected in 2008 and included 5,114 of the Wave I public-use respondents. See Tables 3 and 4 for information on the consolidation of the various waves to create working datasets for the current study. For analyses involving Waves I, II, and IV of the Add Health data, there were 3,923 respondents in common to all three datasets. It should be noted that because the statistical analyses needed to account for clustering and for unequal probability of selection in the

Add Health study (Chen & Chantala, 2014), weights were identified using Wave IV respondents, and all 5,114 were included in the dataset, allowing Stata to take care of respondents who had missing data. In total, the 5,114 Wave IV respondents represent 22,014,038 individuals who were in Grades 7-12 in the United States during the 1994-1995 school year according to statistical results in Stata. Survey descriptives also reveal that this combined dataset, based on Wave IV data, included one stratum with 132 sampling units consisting of a minimum of five observations per unit, a maximum of 106 observations per unit, and an average of 38.7 observations per sampling unit.

Table 3

Consolidation of the Add Health Public Use Wave I, II, and IV Datasets

Public-use dataset	Respondents	Respondents in common
Wave I	6,504	
Wave II	4,834	4,833
Wave IV	5,114	3,923
Total possible study respondents (Waves I, II & IV)		3,923

Table 4

Consolidation of the Add Health Public Use Wave I, II, III, and IV Datasets

Public-use dataset	Respondents	Respondents in common
Wave I	6,504	
Wave II	4,834	4,833
Wave III	4,882	3,843
Wave IV	5,114	3,341
Total possible study respondents (Waves I-IV)		3,341

There were a number of demographic variables that were accounted for by using them as independent variables in statistical analysis in the current study. They included biological sex, race/ethnicity, age, family income, and smoking status. Table 5 lists the

representative cell proportions for each of the categorical variables and includes the number of observations contributing to the proportion as well as the resulting representative population of students in Grades 7-12 in the United States during the 1994-1995 school year. There was one participant who did not have a biological sex listed from the Wave I or Wave II data. I was able to determine this individual's sex from Wave IV data. One individual was coded as a male for Waves I, II, and IV but as a female for Wave III. I assumed Waves I, II, and IV to be the correct evaluation. One individual was coded as female for Waves I and II but as male for Waves III and IV. Another individual was coded as male in Wave I but as female for Waves III and IV. A third individual was coded as male in Wave I and as female in Wave IV. For these three individuals, because it could not be determined whether the codings were mistakes or if the individuals had identified with the opposite gender at some point in their development, the biological sex for these individuals was changed to *missing*, thereby nullifying any data analysis that was related to the sex of the individual. For the dataset based on Wave IV responses, of the 5,114 possible cases, 5,111 were used for analyses involving the individual's sex.

Regarding age, data were missing for three individuals (due to refusal to answer); however, Wave IV included birthdates, and these were used to determine the missing values at Wave I. Furthermore, I changed ages 11, 19, 20, and 21 at Wave I to missing data due to small cell counts of 3, 77, 10, and 3, respectively. I also accounted for age at Wave II in order to identify appropriate responses to a specific question about social support. In the Wave II dataset, 97 participants responded *does not apply* to the question "How much do you feel that you want to leave home?" This prompted me to focus Wave

II scales for depression and social support as representative of adolescents likely to be living at home with their parents. I chose 18 as my cut point for age at Wave II and removed depression and social support scores for those over age 18 by changing their depression and social support scores to missing (the effect of this decision on my sample is discussed later). This decision also supported my general interest in these scales as representative of the stage of adolescence. The average weighted age of the full sample (5,114 observations) was 15.44 years of age, while the average weighted age of the adjusted sample (5,021 observations) was 15.36 years of age.

Table 5

Descriptive Statistics of Covariates for the Sample Population Including Weighted Values

Variable	Subcategory	Cell proportion (mean)	Observation (5,114 possible)	Representative population
Biological sex			5,111	22,006,447
	Female	49.46%	2,759	
	Male	50.54%	2,352	
Identified race/ethnicity			5,098	21,944,291
	White	69.96%	3,207	
	Hispanic	11.09%	532	
	African American	15.44%	1,160	
	Other	3.51%	199	
Age (Wave I)			5,021	21,573,210
	12	3.12%	157	
	13	15.09%	670	
	14	16.77%	816	
	15	17.11%	888	
	16	17.17%	923	
	17	16.74%	889	
	18	14.00%	678	
Smoking status			5,083	21,896,330
	Nonsmoker	73.03%	3,787	
	Smoker	26.97%	1,296	
BMI (Wave I)	Female	(22.28)[a]	2,638	10,409,693
	Male	(22.67)[a]	2,315	10,963,471
Family income (mean)	Overall	($46,689)	3,940	17,177,330
	White	($51,333)[b]	2,604	12,530,338
	Hispanic	($33,517)[b]	379	1,751,293
	African American	($31,671)[b]	802	2,292,470
	Other	($45,169)[b]	145	551,924
Depression (Wave I)	Female	(11.69)[c]	2,749	10,842,441
	Male	(9.99)[c]	2,336	11,036,113

Note. Percentages represent cell proportions that contribute to the total representative population when sample weights are applied.
[a]Obesity at Wave I was significantly lower for females than for males, $t(131) = -2.85$, $p = .005$. [b]Family income between White and Hispanic students was significantly different, $t(131) = 6.29$, $p < .001$; family income between White and African American students was significantly different, $t(131) = 6.87$, $p < .001$; family income between Other and Hispanic students was significantly different, $t(131) = 2.52$, $p = .013$; family income between Other and African American students is significantly different, $t(131) = 3.10$, $p = .002$. [c]Depression at Wave I is significantly higher for females than for males, $t(131) = 7.85$, $p < .001$.

The primary independent variable in this study was adolescent religiosity, which was measured using Wave I data. The dependent variable was adult obesity, which was measured using Wave IV data. Secondary independent variables (being considered for mediational properties) included adolescent depression and adolescent social support, both of which were measured using Wave II data. Baseline descriptive statistics for each of these variables are provided in Table 6.

Religiosity Scores

Wave I Religiosity scores were derived from four questions in the Wave I survey. The Religiosity score reflects church/worship attendance, prayer habits, the importance of religion to the individual, and attendance at religious youth activities. Responses to these questions were reverse coded so that a higher score indicates a higher level of religiosity. Missing responses for any of the four questions resulted in a missing Religiosity score. Very few participants responded to these questions with *refused* or *don't know*; however, 879 respondents were coded as *legitimate skip* due to their answer on a previous question about religious affiliation of *no religion*.

Researchers using the Add Health data have handled these legitimate skips differently. Harden (2009) assigned the lowest possible religiosity score to those who were legitimate skips due to no religion. As applied to the current study, legitimate skips would have received scores of 4 because the lowest possible score for Religiosity is 4. Nooney (2005), on the other hand, concluded that legitimate skips for no religious affiliation needed to be treated as missing data. Nooney pointed out that giving such an individual a reverse score of 1 to indicate that this individual never prays may involve an

incorrect assumption. An individual who does not have a specific religious affiliation may still engage in prayer. Following the example of Nooney, I decided to identify legitimate skips for religiosity questions as missing data so that these individuals were not included in the statistical analyses. Of the 6,504 possible religiosity scores derived from Wave I data, 909 were coded as missing data. The resulting number of participants in the combined datasets (Wave I, II, and IV) with Religiosity scores was 4,430; 684 participants in the combined dataset were missing a Religiosity score. Cronbach's alpha for this scale was .75, which exceeded the acceptable level of .70 as identified by Frankfort-Nachmias and Nachmias (2008). Adolescent girls demonstrated a higher mean Religiosity score than did adolescent boys, and the African American adolescents scored higher than all other groups for Religiosity; Other races were second, followed by the Hispanic population and finally the White population. Significant differences between means of various groups for the primary variables in this study are detailed in Table 7 and were determined using *t test* analysis through the *lincom* command in Stata.

Depression Scores

As mentioned earlier, this study considered Depression and Social Support scores for adolescents at Wave II. However, the Social Support scale was interrupted by responses of *does not apply* in relation to feelings of wanting to leave home. For this reason, I decided to focus more intently on participants in Wave II most likely to be considered adolescents; I did this by recording missing values for Depression and Social Support for respondents over 18 years of age at Wave II. Table 8 demonstrates the impact of this decision, as well as the choice to mark small cell sizes for age at Wave I as

missing, on the resulting sample size. Final sample sizes while controlling for factors of age at Wave I and Wave II were still quite impressive at n = 3,699 for Depression and n = 3,555 for Social Support. Cronbach's alpha for the Wave I Depression scale was high at .86; equally high was the alpha for Wave II Depression at .87. Overall, female Depression scores at Wave II were higher than were male Depression scores (Table 6); Hispanic adolescents exhibited higher depression symptoms than did the other racial/ethnic groups. Statistical significance of these differences can be seen in Table 7.

Social Support Scores

The sample sizes for social support are included in Table 8. For the same reasons discussed in relation to the Depression scores, the sample that included Social Support scores was reduced to n = 3,555. Cronbach's alpha for Wave II Social Support was found to be .77. On average, females reported greater levels of Social Support at Wave II than did males, and the African American adolescent population reported the greatest level of Social Support at Wave II. Statistical significance of these differences can be seen in Table 7.

BMI Scores

BMI scores were calculated using either height and weight, as reported by the respondent, or by measured height and weight when this information was available. For statistical analysis, BMI data for women who were determined to be pregnant at the time of data collection were changed to *missing* for that wave. For the combined dataset of all four waves and retaining all 5,114 of the Wave IV respondents, six women were identified as pregnant at Wave I, 39 women were identified as pregnant at Wave II, 349

women were identified as pregnant at Wave III, and 189 women were identified as pregnant at Wave IV.

Women at Wave IV recorded a higher average for BMI than did men at Wave IV (Table 6). Based on race and ethnicity, African Americans reported a higher average BMI than all other groups, followed by the Hispanic population, the White population, and then the Other Race group. Interestingly, when the Other Race group was parceled out, the American Indian group exhibited the highest average BMI (32.28) and the Asian group exhibited the lowest average BMI (26.63). Statistical significance of the differences in Wave IV BMI by group can be seen in Table 7.

The measurement of BMI is naturally a continuous measure as it is the result of taking one's weight in kilograms and dividing by the square of one's height in centimeters. However, the continuous BMI scores are also categorized into levels of obesity as follows: underweight, normal weight, overweight, and obese (with a variety of levels for obesity). For individuals between the ages of 2 and 20, BMI classifications for obesity are based on age and sex; for adults, BMI classifications are based entirely on the calculated BMI regardless of age or sex. BMI categories at Wave I were calculated using the 2000 CDC Growth Charts found at www.cdc.gov/growthcharts/clinical_charts.htm. Another way in which to view obesity is related to changes in obesity category over time. For males, 2,262 participants at Wave I representing 10,687,088 U.S. students in Grades 7-12 in 1994-1995 were classified by BMI into obesity categories based on their age. At Wave I, 65.5% of students were categorized as normal weight, with overweight and obese levels reported at 17.8% and 13.6%, respectively. At Wave IV, the classifications

for obesity were based on 2,316 observations representing 10,959,662 males, and only 27.5% were considered to be normal weight, with categories of overweight and of obesity reaching 35.7% each (see Table 9).

Categorization of obesity for women trended similarly to men in that at Wave I, 73% of females were categorized as normal weight while 15% and 9.8% were categorized as overweight and obese, respectively. These percentages were based on U.S. girls in Grades 7-12 in 1994-1995 originating from 2,602 observations representing 10,264,917 adolescents. Wave IV percentages were based on 2,536 women representing 9,996,636 individuals and demonstrate that 34.8% of women were normal weight, 24.6% were overweight, and 38.6% were categorized as obese (see Table 9).

The changes in obesity over time demonstrate notable movement between categories of obesity between adolescence and early adulthood. In order to gain a better sense for what this movement looks like, I categorized individuals as either dropping by two BMI categories, dropping by one BMI category, remaining constant for BMI category, increasing to the next highest BMI category, moving up two BMI categories, or moving up three BMI categories between adolescence and early adulthood. For males, 40.8% remained constant in their BMI category over time while 41.9% increased by one BMI category and 13.3% increased by two BMI categories. Among women, 44.8% remained in their BMI category from adolescence through early adulthood while 33.2% increased by one BMI category and 17.8% increased by two BMI categories. Table 10 reflects percentages for all categories of changes in BMI between adolescence and adulthood.

Table 6

Baseline Descriptive Statistics for the Primary Independent and Dependent Variables

Variable	Subcategory	Means [95% CI]	Observations	Representative population
Religiosity	Biological sex		4,427	18,845,030
Wave I	Male	12.09 [11.84, 12.33]		
	Female	12.80 [12.53, 13.07]		
	Race/ethnicity		4,420	18,803,847
	White	12.12 [11.86, 12.38]		
	Hispanic	12.49 [12.13, 12.86]		
	African Am	13.74 [13.45, 14.03]		
	Other	12.78 [12.17, 13.38]		
Depression	Biological sex		3,701	15,940,178
Wave II	Male	9.48 [9.05, 9.91]		
	Female	11.81 [11.36, 12.26]		
	Race/ethnicity		3,690	15,888,740
	White	10.00 [9.61, 10.39]		
	Hispanic	12.71 [11.70, 13.73]		
	African Am	12.08 [11.31, 12.85]		
	Other	11.72 [10.21, 13.24]		
Social	Biological sex		3,557	15,295,046
support	Male	31.86 [31.55, 32.17]		
Wave II	Female	32.29 [31.96, 32.61]		
	Race/ethnicity		3,546	15,243,607
	White	32.13 [31.85, 32.42]		
	Hispanic	32.15 [31.25, 33.05]		
	African Am	31.78 [31.29, 32.27]		
	Other	31.96 [30.80, 33.11]		
BMI	Biological sex		5,042	21,703,410
Wave IV	Male	28.96 [28.65, 29.28]		
	Female	29.20 [28.71, 29.69]		
	Race/ethnicity		5,026	21,633,663
	White	28.62 [28.24, 29.00]		
	Hispanic	29.98 [29.34, 30.63]		
	African Am	30.79 [30.17, 31.41]		
	Other	27.81 [26.67, 28.95]		

Table 7

Statistical Comparison of Means for the Sex and Race Components Within the Primary Independent and Dependent Study Variables

Variable	Comparison means (mean difference)	SE of the difference	*t*-value	*p*-stat
Religiosity (Wave I)				
Male-Female	12.09 – 12.80 (-.7082)	.1230	-5.76***	.000
White-Hispanic	12.12 – 12.49 (-.3757)	.2047	-1.84	.069
White-Afr. Am.	12.12 – 13.74 (-1.622)	.1754	-9.25***	.000
White-Other	12.12 – 12.78 (-.6568)	.3242	-2.03*	.045
Hispanic-Afr. Am.	12.49 – 13.74 (-1.246)	.2327	-5.36***	.000
Hispanic-Other	12.49 – 12.78 (-.2811)	.3513	-0.80	.425
Afr. Am-Other	13.74 – 12.78 (.9654)	.3258	2.96**	.004
Depression (Wave II)				
Male-Female	9.48 – 11.81 (-2.330)	.2598	-8.97***	.000
White-Hispanic	10.00 – 12.71 (-2.712)	.5222	-5.19***	.000
White-Afr. Am.	10.00 – 12.08 (-2.079)	.4403	-4.72***	.000
White-Other	10.00 – 11.72 (-1.725)	.7959	-2.17*	.032
Hispanic-Afr. Am.	12.71 – 12.08 (.6326)	.6502	0.97	.332
Hispanic-Other	12.71 – 11.72 (.9873)	.9138	1.08	.282
Afr. Am-Other	12.08 – 11.72 (.3548)	.9179	0.39	.700
Social Support (Wave II)				
Male-Female	31.86 – 32.29 (-.4340)	.1884	-2.30*	.023
White-Hispanic	32.13 – 32.15 (-.0141)	.4571	-0.03	.976
White-Afr.Am.	32.13 – 31.78 (.3545)	.2800	1.27	.208
White-Other	32.13 – 31.96 (.1772)	.5991	0.30	.768
Hispanic-Afr.Am.	32.15 – 31.78 (.3685)	.4920	0.75	.455
Hispanic-Other	32.15 – 31.96 (.1912)	.7272	0.26	.793
Afr. Am-Other	31.78 – 31.96 (-.1773)	.6420	-0.28	.783
BMI (Wave IV)				
Male-Female	28.96 – 29.20 (-.2392)	.2709	-0.88	.379
White-Hispanic	28.63 – 30.04 (-1.407)	.3600	-3.91***	.000
White-Afr. Am.	28.63 – 30.69 (-2.055)	.4020	-5.11***	.000
White-Other	28.63 – 27.81 (.8210)	.6100	1.35	.181
Hispanic-Afr. Am.	30.04 – 30.69 (-.6474)	.5154	-1.26	.211
Hispanic-Other	30.04 – 27.81 (2.228)	.6142	3.63***	.000
Afr. Am-Other	30.69 – 27.81 (2.876)	.6841	4.20***	.000

*$p < .05$. **$p < .01$. ***$p < .001$.

Table 8

Adjustment of the Sample Size Related to Depression and Social Support Scales to Control for Adolescent Age at Wave I & II

	Sample size (5,114 possible)
Depression	
All respondents with score at Wave II	3,907
Control for age at Wave I	3,870
Control for age at Wave I & II	3,699
Social Support	
All respondents with score at Wave II	3,719
Control for age at Wave I	3,695
Control for age at Wave I & II	3,555

Table 9

Categorization of Obesity Based on BMI at Wave I and Wave IV for Those With BMI Data for Both Waves

BMI classification	Wave I		Wave IV	
	Observations	Percentage	Observations	Percentage
Females				
Underweight	49	2.2	51	2.2
Normal weight	1,720	72.7	840	35.6
Overweight	363	15.3	599	25.0
Obese	254	9.9	896	37.3
Males				
Underweight	62	3.1	25	1.1
Normal weight	1,490	65.9	635	27.6
Overweight	377	17.8	776	35.6
Obese	298	13.3	791	35.6

Note. Female statistics were based on 2,386 respondents with BMI data for both waves and represented 9,410,591 female students. Male statistics were based on 2,227 respondents with BMI data at both waves and represented 10,528,638 male students.

Table 10

Level Change in BMI From Wave I to Wave IV

	2 level decrease	1 level decrease	No change	1 level increase	2 level increase	3 level increase
Female						
Percentage	3.6	3.8	44.8	33.2	17.8	0.1
Count	8	92	1,077	793	415	1
Male						
Percentage	0.6	3.2	40.8	41.9	13.3	0.1
Count	15	65	939	901	305	2

Regression Analysis

Research Question 1 (RQ1) for Male Participants

The first research question presented in this study was the question of the relationship that exists between adult obesity (DV) and adolescent religiosity (IV), adolescent depression (IV), and adolescent social support (IV). Adult obesity was measured using BMI, which can be expressed as either a continuous or as a categorical value. In order to reduce the loss of variance associated with making BMI dichotomous (not obese/obese), I decided to use continuous BMI scores in all analyses. As a result, the anticipated use of logistic regression for this analysis was changed to linear regression.

For all linear regression analyses, the *survey data analysis* option in Stata 14 was used and grand sample weights were accounted for by setting the CLUSTER2 variable as the primary sampling unit and GSWGT4_2 as the sampling weight; this was done in accordance with recommendations set by Chen and Chantala (2014). The Public-Use data sets for Add Health do not contain a strata variable; however as Chen and Chantala (2014) point out, there is a minimal effect on standard errors by not including this variable in the survey data analysis.

The finalized variables discussed in the previous section were used, when possible, in the linear regression models to answer RQ1. Therefore, the Religiosity score at Wave I did not include those who had legitimate skips for the religiosity questions, and the Depression and Social Support scores from Wave II did not include respondents who were older than 18 years of age at the Wave II interview. The choice to limit the age range being referenced at the Wave II data collection to those under 19 years of age was based on a data collection issue identified at Wave II. As mentioned earlier, a number of respondents at Wave II were coded as *does not apply* in relation to a question about wanting to leave home. This indicates that the individual was already out of the home and inclusion of these responses would have impacted the social support scale. Given this data collection circumstance in Wave II, I determined to identify the terminology of *adolescence* in relation to American Psychological Association (APA) standards. Although there is no standard age-range that is universally accepted as representative of adolescence, the APA (2002), in their professional reference document, established adolescence to be individuals between the ages of 10 and 18 (p. 5). Limiting responses to those under 19 years of age at Wave II allowed me to more clearly identify the variables of adolescent depression and adolescent social support for this current study.

One of the control variables in this analysis was obesity level at Wave I. Obesity was identified in relation to BMI, and BMI for children and adolescents carries a unique quality in that for individuals between the ages of 2 and 20, BMI categorization of obesity is based on percentiles for sex and age (CDC, 2015). Based on percentiles, individuals who are below the 5^{th} percentile for their age and sex are considered to be

underweight; those between the 5th and less than the 85th percentile for their age and sex are considered to be normal weight; those between the 85th and less than the 95th percentile for their age and sex are considered to be overweight; and those at the 95th percentile and above for their age and sex are considered to be obese (CDC, 2015). As a result, a 16-year-old male with a BMI of 28.2 is considered to be obese yet a female of the same age with a BMI of 28.2 is considered to be normal weight. Given the different scales by which children and adolescents are categorized according to BMI, I decided to run separate analyses for males and females to answer this first research question.

The role of adolescent religiosity, depression, and social support in the development of adult obesity among males. The resulting sample size for the primary variables of interest for RQ1 was n = 1,386 males and represented 6,471,975 boys in Grades 7-12 in 1994-1995.The relationship of the primary variables of interest (adolescent religiosity, adolescent depression, adolescent social support, and adult BMI) was just beyond the level of significance, $F(3, 129) = 2.59$, $p = .056$; the $R^2 = .0080$ for this relationship was extremely low. Adolescent religiosity, $t(131) = 2.66$, $p = .009$ was a significant predictor in this overall statistically insignificant relationship. Adolescent depression, $t(131) = 1.32$, $p = .189$, and adolescent social support, $t(131) = .13$, $p = .898$, did not contribute significantly to this prediction (see Model 1 in Table 11).

There were a number of control variables identified from research in the area of obesity that were applied to this analysis. Sex was accounted for by using separate analyses for males and for females. The other control variables included race/ethnicity, baseline age, baseline BMI, baseline depression, baseline family income, baseline

smoking status, smoking status at Wave IV, and income at Wave IV. Model 2 demonstrates the prediction of adult BMI based on these control variables (see Table 11). The resulting sample size involving these control variables was n = 1,600 males and represented 7,582,985 male students in Grades 7-12 in 1994-1995. The prediction of adult obesity from these demographic variables was statistically significant, $F(13, 119) = 73.50$, $p < .001$. The R^2 for this relationship was 0.533. In this model, neither initial depression symptoms nor smoking status at Wave I were significantly predictive of adult obesity; family income, as reported in the parent survey, was just beyond the .05 level of significance, $t(131) = -1.97$, $p = .051$. All other variables were significantly predictive of adult obesity. In relation to the White population, being of Hispanic origins significantly predicted adult obesity at $t(131) = 3.70$, $p < .001$, but being of African American and Other origins did not significantly predict adult obesity; $t(131) = 0.65$, $p = .520$ for African American populations and $t(131) = 0.37$, $p = .714$ for the Other race population. Age at Wave I, $t(131) = -5.60$, $p < .001$, significantly contributed to the prediction of adult obesity; raw BMI scores at Wave I, $t(131) = 29.18$, $p < .001$, significantly contributed to the prediction of adult obesity; smoking status at Wave IV, $t(131) = -5.77$, $p < .001$, significantly contributed to the prediction of adult obesity; and all levels of income quintiles, except for the second quintile, contributed significantly to the prediction of adult obesity, $t(131) = 3.35$, $p = .001$ for the third quintile, $t(131) = 2.59$, $p = .011$ for the fourth quintile, and $t(131) = 2.42$, $p = .017$ for the fifth quintile (see Model 2, Table 11).

The final combined model was determined by considering the theoretical rationale for including control and predictor variables as well as observing the contribution of the control and predictor variables in the model. Model 3 (Table 11), is the final model of interest for answering RQ1. All control variables were retained on a theoretical basis as well as based on an observation that the model was strongest when all variables were retained. The resulting model included data for 1,021 respondents, representing 4,812,148 male students in Grades 7-12 in 1994-1995 and was a statistically significant predictor of adult obesity, $F(16, 116) = 33.52$, $p < .001$. The R^2 for this model was .561, which indicated that while demographic data was a strong predictor of adult obesity, the prediction was enhanced with data on measures of religiosity, depression, and social support during adolescence. The change in R^2 between Model 2 and Model 3 was .028. In this complete model, adolescent religiosity was a significant predictor of adult obesity, $t(131) = 3.31$, $p = .001$. Neither adolescent depression nor adolescent social support at Wave II was a significant predictor of adult obesity. The Hispanic male population remained a significant predictor of adult obesity in the full model, $t(131) = 3.27$, $p = .001$, but the African American population was not significantly different from the White population for predicting adult obesity. Initial age remained a significant predictor, $t(131) = -6.08$, $p < .001$; and initial BMI remained a significant predictor, $t(131) = 22.03$, $p < .001$. Wave IV smoking status was a significant predictor of adult obesity in the full model, $t(131) = -5.46$, $p < .001$.Wave IV income categorization was also a significant predictor with all four upper income quintiles identified as significant predictors of adult obesity in relation to the lowest income quintile; values were $t(131) = 2.52$, $p = .013$ for

the second quintile, $t(131) = 4.00$, $p < .001$ for the third quintile, $t(131) = 2.71$, $p = .008$ for the fourth quintile, and $t(131) = 2.79$, $p = .006$ for the fifth quintile. Although Wave II depression and social support were not significant contributors to the prediction of adult obesity, removing Wave II depression, Wave II social support, or both from the model did not increase the overall power of the relationship. When Wave II depression was removed, the R^2 for the model was .5595; when Wave II social support was removed, the R^2 was .5547; and when both Wave II depression and social support were removed, the R^2 was .544. In summary, the null hypothesis for RQ1 is rejected as adolescent religiosity, adolescent depression, and adolescent social support contribute to the significant prediction of adult male obesity; however, only adolescent religiosity is a significant predictor of adult obesity.

Table 11

Coefficients, Standard Errors, 95% CIs, and Significance Levels of the Various Regression Models in the Prediction of Adult Male Obesity

Model	β	SE	*t*	*p*-value	95% CI
Model 1: Primary variables					
Religiosity (W1)	.1467	.0552	2.66	.009**	[0.04, 0.26]
Depression (W2)	.0534	.0404	1.32	.189	[-0.03, 0.13]
Social support (W2)	.0058	.0453	0.13	.898	[-0.08, 0.10]
Constant	26.4400	1.7019	15.54	.000***	[23.07, 29.81]
Model 2: Demographic predictors					
Race/ethnicity					
Hispanic					
African American	1.4750	.4045	3.70	.000***	[.69, 2.30]
Other	.2600	.4031	0.65	.520	[-.54, 1.06]
Age (WI)	.2835	.7716	0.37	.714	[-1.24, 1.81]
Baseline BMI (WI)	-.5275	.0942	-5.60	.000***	[-.71, -.34]
Smoking status (WI)	1.0708	.0367	29.18	.000***	[.99, 1.14]
Smoker					
Baseline depression (WI)	-.1325	.2889	-0.46	.647	[-.70, .44]
Family income (WI)	.0167	.0218	0.77	.444	[-.03, .06]
Smoking status (WIV)	-.0037	.0019	-1.97	.051	[-.01, .00]
Smoker					
Income quintile (WIV)	-1.5520	.2691	-5.77	.000***	[-2.08, -1.02]
Second quintile					
Third quintile	.9066	.5339	1.70	.092	[-.15, 1.96]
Fourth quintile	1.8307	.5462	3.35	.001**	[.75, 2.91]
Fifth quintile	1.4040	.5413	2.59	.011*	[.33, 2.47]
Constant	1.2384	.5118	2.42	.017*	[.23, 2.25]
	12.0827	1.4700	8.22	.000***	[9.17, 14.99]
Model 3: Combined primary and demographic variables					
Religiosity	.1439	.0435	3.31	.001**	[.06, .23]
Depression (WII)	.0238	.0275	0.87	.388	[-.03, .08]
Social support (WII)	-.0275	.0351	-0.78	.435	[-.10, .04]
Race/ethnicity					
Hispanic	1.6780	.5133	3.27	.001**	[.66, 2.69]
African American	.2656	.4670	0.57	.570	[-.66, 1.19]
Other	.5243	1.0243	0.51	.610	[-1.50, 2.55]
Age (WI)	-.7090	.1166	-6.08	.000***	[-.94, -.48]
Initial BMI (WI)	1.0960	.0498	22.03	.000***	[1.00, 1.19]
Initial depression (WI)	-.0039	.0291	-0.13	.894	[-.06, .05]
Smoker (WI)	.1652	.3894	0.42	.672	[-.61, .04]
Family income (WI)	-.0024	.0026	-0.92	.362	[-.01, .00]
Smoker (WIV)	-1.6450	.3014	-5.46	.000***	[-2.24, -1.05]
Income quintile (WIV)					
Second quintile	1.6001	.6356	2.52	.013*	[.34, 2.86]
Third quintile	2.6685	.6665	4.00	.000***	[1.35, 3.99]
Fourth quintile	1.8922	.6981	2.71	.008**	[.51, 3.27]
Fifth quintile	1.814	.6497	2.79	.006**	[.53, 3.10]
Constant	12.4565	2.0753	6.00	.000***	[8.35, 16.56]

*$p < .05$. **$p < .01$. ***$p < .001$.

Table 12

Statistical Comparison of Means for Factor Variables in the Prediction of Obesity Among Males in the Full Model (Model 3)

Group comparison	Coef	Std. Error	*t*	*p*-value	95% CI
White—Hispanic	-1.6780	.5133	-3.27**	.001	[-2.69, -.66]
White—African Am.	-.2656	.4670	-0.57	.570	[-1.19, .66]
White—Other	-.5243	1.0243	-0.51	.610	[-2.55, 1.50]
Hispanic—African Am.	1.4124	.6659	2.12*	.036	[.10, 2.73]
Hispanic—Other	1.1537	1.1607	0.99	.322	[-1.14, 3.45]
African Am.—Other	-.2587	.9905	-0.26	.794	[-2.22, 1.70]
IncQuin1—IncQuin2	-1.6001	.6356	-2.52*	.013	[-2.86, -.34]
IncQuin1—IncQuin3	-2.6685	.6665	-4.00***	.000	[-3.99, -1.35]
IncQuin1—IncQuin4	-1.8923	.6981	-2.71**	.008	[-3.27, -.51]
IncQuin1—IncQuin5	-1.8141	.6497	-2.79**	.006	[-3.10, -.53]
IncQuin2—IncQuin3	-1.0683	.4419	-2.42*	.017	[-1.94, -.19]
IncQuin2—IncQuin4	-.2921	.4907	-0.60	.553	[-1.26, .68]
IncQuin2—IncQuin5	-.2140	.4391	-0.49	.627	[-1.08, .65]
IncQuin3—IncQuin4	.7762	.5526	1.40	.163	[-.32, 1.87]
IncQuin3—IncQuin5	.8543	.4386	1.95	.054	[-.01, 1.72]
IncQuin4—IncQuin5	.0781	.5227	0.15	.881	[-.96, 1.11]

*$p < .05$. **$p < .01$. ***$p < .001$.

Assumptions of linear regression analysis for male data. Researchers, who utilize linear regression techniques, must address a number of assumptions associated with the analysis (Field, 2009, p. 220). These assumptions are addressed here; however, due to the complex survey design of the dataset and the way in which Stata accounts for that design, not all assumptions can be addressed definitively. I followed the web book example provided by Chen, Ender, Mitchell, and Wells (2003) in which they discuss the use of Stata commands for linear regression diagnostics. When possible, I applied the analyses to the weighted data, however not all of the diagnostic tools in Stata are available when working with survey data. While the reality of loss of diagnostic

capability for survey data is undeniable, many of the assumption tests that could not be applied here are potentially accounted for by the robust nature of the regression protocol for analyzing survey data with Stata.

Field (2009) identified that an assumption of normally distributed data can be attended to by use of robust tests. The use of the *svy* command in Stata results in analyses that produce standard errors that are "robust to heteroscedasticity" (J. Wang, StataCorp Statistician, personal communication, September 10, 2015). The specific test that produces these robust standard errors is identified by StataCorp (2015) as the Huber/White/sandwich estimator (p. 6 of the Stata Survey Data Reference Manual). Furthermore, Chen et al. (2015) corroborated the use of robust tests to account for potentially problematic data in their statement:

> Such robust standard errors can deal with a collection of minor concerns about failure to meet assumptions, such as a minor problems about normality, heteroscedasticity, or some observations that exhibit large residuals, leverage or influence. For such minor problems, the robust option may effectively deal with these concerns. (Section 4.1.1 of the Stata Web Book).

Outliers and influence. I was unable to run the specific commands in Stata that would produce evaluations of outliers, leverage, and influential data points such as Cook's D, and DFITS due to the nature of my survey data. I was also unable to determine statistics for DFBETA on my weighted data. Chen et al. (2015) identify these two methods as typical ways for identifying outliers and influential data. In relation to weighted data, I was able to produce visual relationships between Wave IV BMI and the

three independent variables (adolescent religiosity, depression, and social support). These representations are provided in Figure 3 and they demonstrate that there are a couple of observations that are potential outliers. For all three comparisons, the same observation was found to be a visual outlier and there is one other clear observation that is a potential outlier demonstrated in the BMI-Depression matrix. A handful of observations show separation from the group in the BMI-Religiosity matrix as well. Because the specific Stata statistical commands were not available to evaluate these potential outliers, I am acknowledging that my data was comprised of a few potential outliers. However, I was unable to determine the specific influence these potential outliers have on the dataset.

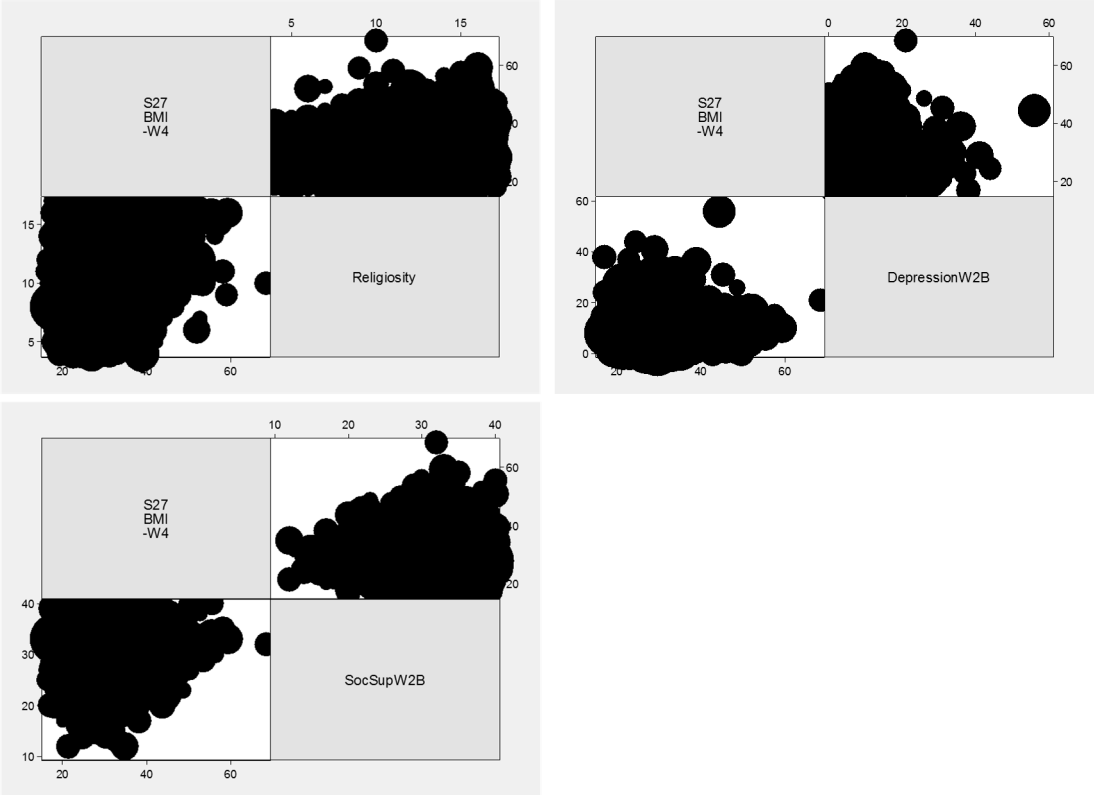


Figure 3. Scatterplots representing each predictor variable against the outcome variable, adult obesity. The scatterplots represent weighted data as seen by the mass they occupy, as opposed to the representation of a single point, and indicate potential outliers.

Normality of residuals. As described by Field (2009) and Chen et al. (2015), the residuals of a regression model are assumed to be normally distributed. Checking this assumption was particularly challenging due to the use of survey data; in fact, many of the specific checks for normality could not be performed while accounting for the survey design. After determining the residuals for the 1,021 males included in the full model, I was able to produce a kernel density plot using the *kdensity* command, a standardized normal probability plot using the *pnorm* command, a plot of quantiles using the *qnorm* command, an inter-quartile range determination using the *iqr* command, and the Shapiro-Wilk W test for normality using the *swilk* command (UCLA, 2015, Chapter 2.2). The kernel density plot demonstrates leptokurtosis and positive skewness (Figure 4). The standardized normal probability plot is used to identify shifts from normality for the mid-range data (Chen et al., 2015); Figure 5 shows departure from the plot at mid-range. The quantile plot is used to identify shifts from normality at the ends (Chen et al., 2015); Figure 5 demonstrates a slight problem at the bottom end with more defined departure from normality at the top. The inter-quartile range analysis identified 7 mild outliers on the lower end and 19 mild outliers on the upper end; three of the upper end outliers were identified as severe outliers in this analysis. Finally, a significant *p*-value for the Shapiro-Wilk W test indicated that the distribution of the residuals lacks normality. The results of the Shapiro-Wilk W test for the unweighted data revealed that the residuals were not normally distributed, $z = 8.022$, $p < .001$.However, it should be noted that all of the commands in this section on normality of residuals were applied to unweighted data. Due to the fact that this analysis is not based on weighted data, there is no definitive measure

for the role of the outliers on the weighted analysis. In this case, the robust test used to complete survey analysis is believed to account for these potential outliers as some large residuals can be offset by robust tests (Chen et al., 2015).

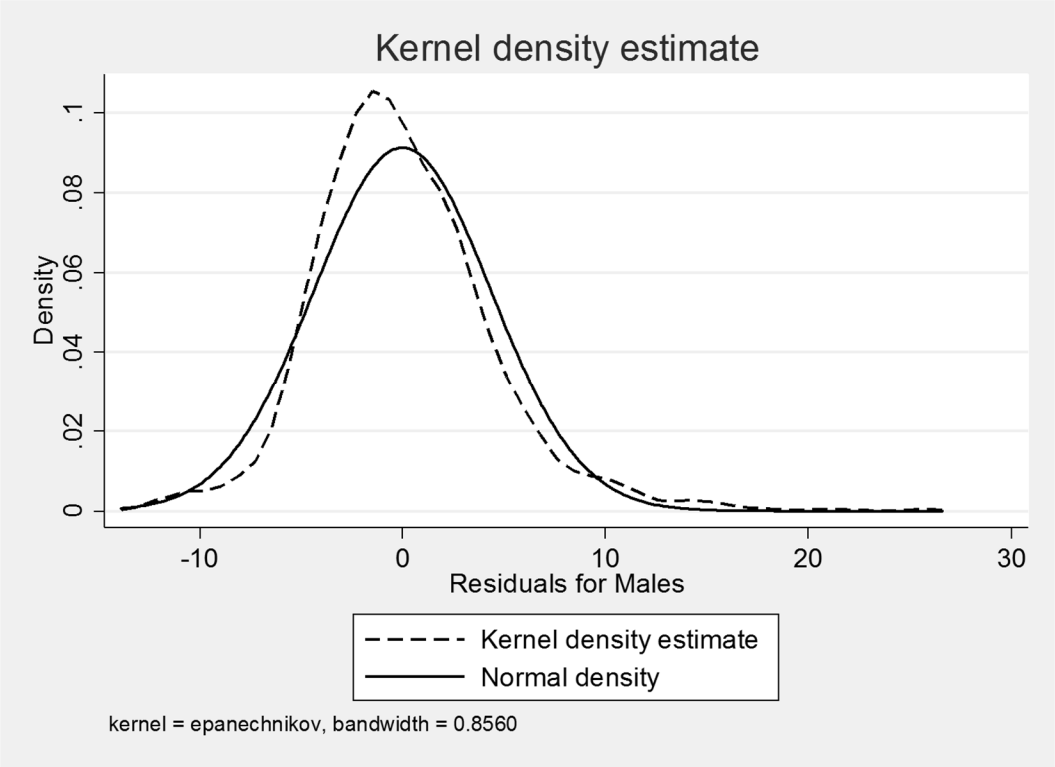


Figure 4. Kernel density plot of residuals for male participants.

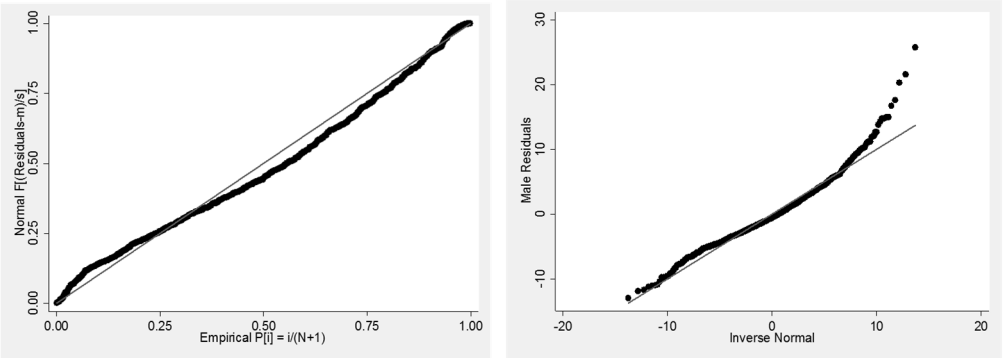


Figure 5. Standardized normal probability plot and quantile plot for male participant data.

Homoscedasticity. Homoscedasticity is an analysis of the residuals in relation to the predictor variables; the expectation is that for each level of the variable, the variance should be the same (Field, 2009). The use of survey data in this study limited the statistical tests that could be used for post estimation analysis. The primary test to check for homoscedasticity in regression post estimations is the *estathettest* command; however, this command does not work with survey data (StataCorp, 2015, p. 2126). I was, therefore, left with a visual analysis for homoscedasticity. Field (2009, p. 229) described a plot of standardized residuals against standardized predicted values as a visual check for meeting the assumptions of homoscedasticity. Using Stata 14, Chen et al. (2015) outlined steps to produce a related chart. I was able to generate predicted values for Wave IV BMI using the *predict xb* command. I was also able to generate residuals for the Wave IV BMI using the *predict, residuals* command. I graphed the residuals against the predictions and was able to apply weights to this graph (see Figure 6), which resulted in masses rather than points associated with each data point.

It appears that the weighted data demonstrated a level of heteroscedasticity due to the fact that the data did not plot randomly (Figure 6). Because survey analysis in Stata is "robust to heteroscedasticity" (J. Wang, StataCorp Statistician, personal communication, September 10, 2015), the possibility of some heteroscedasticity in the current male dataset is not of great concern.

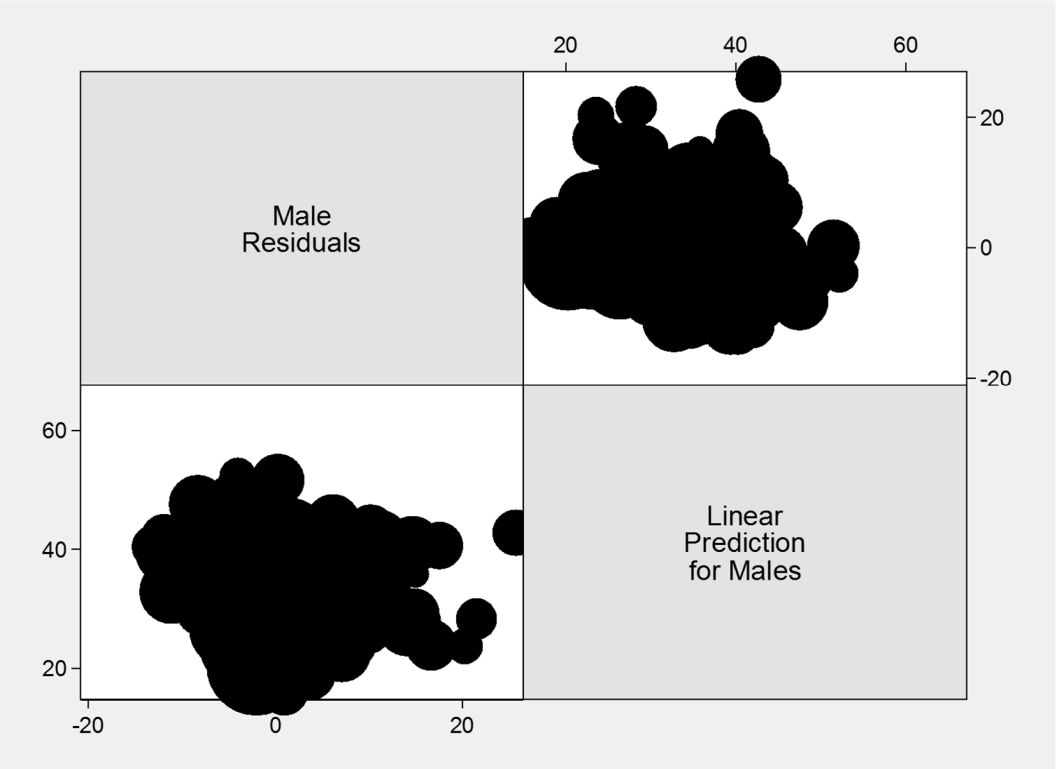


Figure 6. Wave IV BMI residuals plotted against the predicted values of Wave IV BMI based on the full regression model and on residuals and predicted values with weights applied to the graph.

No perfect multicollinearity. Perfect multicolinearity can be identified by predictor variables that are perfectly correlated (r= 1.0; Field, 2009). In Table 13, I record the correlations for all predictor variables. These correlations were derived from properly weighted survey data and they demonstrated that for all continuous predictor variables there was no perfect correlation between any two variables. Despite the seemingly low correlations between predictor variables, many of the correlations were significant at the $p < .05$ level or below. Religiosity was positively, significantly related to Wave II Social Support, but not to the other continuous predictors. Wave II Depression shared significant negative relationships with Wave II Social Support and with Wave I Family Income and a significant positive relationship with Age and Depression at Wave I. Wave II Social

Support, in addition to its positive relationship with Wave I Religiosity, also demonstrated a significant negative relationship with Age and with Depression at Wave I. There existed a positive significant relationship between BMI at Wave I and Depression at Wave I. Wave I Family Income shared significant negative relationships with Depression at Wave II, BMI at Wave I, and Depression at Wave I.

Another check for multicollinearity is to consider variance inflation factors (VIF) for the predictor variables (Field, 2009). These values are listed in Table 14 and were determined while accounting for survey data characteristics using linear regression. The analysis included 1,038 observations representing 4,889,455 males. All VIFs were between one and two and were less than the value of concern of 10, which Myers (as cited by Field, 2009) indicates is a concern for multicollinearity. Variables with multiple factors (Race and Income Quintile) could not be parceled out when analyzed as survey data. However, I was able to apply sample weights to the regular *regress* command and these results are reported with an asterisk in Table 14; VIFs from both types of analysis yielded nearly identical results for all of the other variables. The tolerance statistics (the reciprocal of VIF) for the predictor variables were all above the .2 level, which is noted by Field as the cutoff for cause for concern. Field points out that an average VIF "substantially greater than 1" (p. 242) can be an indication of multicolinearity; the average of the VIFs for the full model was 1.22 and therefore not considered to be of concern. Having considered the statistical significance of correlations for the predictors in the dataset and having identified reasonable VIFs for predictor variables in the dataset, I conclude that there was no outstanding demonstration of multicollinearity in this dataset.

Table 13

Correlation Coefficients and Significance Levels for Predictor Variables (Factor Variables Not Included) for the Male Model

	Religiosity (Wave I)	Depression (Wave II)	Social support (Wave II)	Age (Wave I)	BMI (Wave I)	Depression (Wave I)	Family income (Wave I)
Religiosity (Wave I)	1.000						
Depression (Wave II)	-0.068 (.077)	1.000					
Social support (Wave II)	0.113 (.003**)	-0.451 (.000***)	1.000				
Age (Wave I)	-0.060 (.089)	0.085 (.007**)	-0.158 (.000***)	1.000			
BMI (Wave I)	-0.001 (.970)	0.077 (.051)	-0.020 (.495)	0.195 (.000***)	1.000		
Depression (Wave I)	-0.049 (.191)	0.553 (.000***)	-0.344 (.000***)	0.078 (.016*)	0.048 (.117)	1.000	
Family income (Wave I)	-0.049 (.069)	-0.109 (.000***)	0.016 (.586)	0.056 (.121)	-0.075 (.005**)	-0.128 (.000***)	1.000

Note. Weighted correlations for these variables are based on 1,128 observations representing 5,350,661 male high school students in the United States in 1994-1995. Significance levels are in parentheses.
*$p < .05$, **$p < .01$. ***$p < .001$.

Table 14

Variance Inflation Factors (VIF) for Independent Variables in the Regression Analysis

Variable	VIF	Tolerance statistic (1/VIF)
Religiosity	1.08	0.93
Depression (Wave II)	1.72	0.58
Social support (Wave II)	1.40	0.72
Age (Wave I)	1.12	0.90
Race	1.09	0.91
Hispanic	*(1.07)	*(0.94)
African American	*(1.12)	*(0.89)
Other	*(1.04)	*(0.96)
BMI (Wave I)	1.07	0.94
Depression (Wave I)	1.52	0.66
Smoking status (Wave I)		
Smoker	1.20	0.83
Family income (Wave I)	1.10	0.91
Smoking status (Wave IV)		
Smoker	1.12	0.89
Income quintile (Wave IV)	1.10	0.91
Quintile 2	*(2.59)	*(0.39)
Quintile 3	*(3.33)	*(0.30)
Quintile 4	*(2.57)	*(0.39)
Quintile 5	*(2.51)	*(0.40)
Mean VIF	1.22	
	*(1.60)	

* VIFs for individual factor variables while applying sample weights in *regress* without accounting for the cluster variables.

Linearity. The assumption of linearity as related to regression analysis is that the outcome variable is linearly related to each predictor variable (Chen et al., 2015). In multiple regression, one way to check for linearity using Stata is to use the *acprplot* command, which plots the augmented component-plus-residual plot against the predictor value. However, as with some of the previous assumption tools, the *acprplot* command could not be applied to weighted data. The only analysis I could apply to weighted data was a plot of the residuals against the ordinal- and ratio-level predictors with the hope

that breaks from linearity might be clearly visible. Once again, because the weighted plots are represented by masses as opposed to a single plot, the graphs can be difficult to decipher. I could make no determination of clear violation of linearity, such as u- or s-shaped graphs, based on the weighted plots, which can be seen in Figure 7.

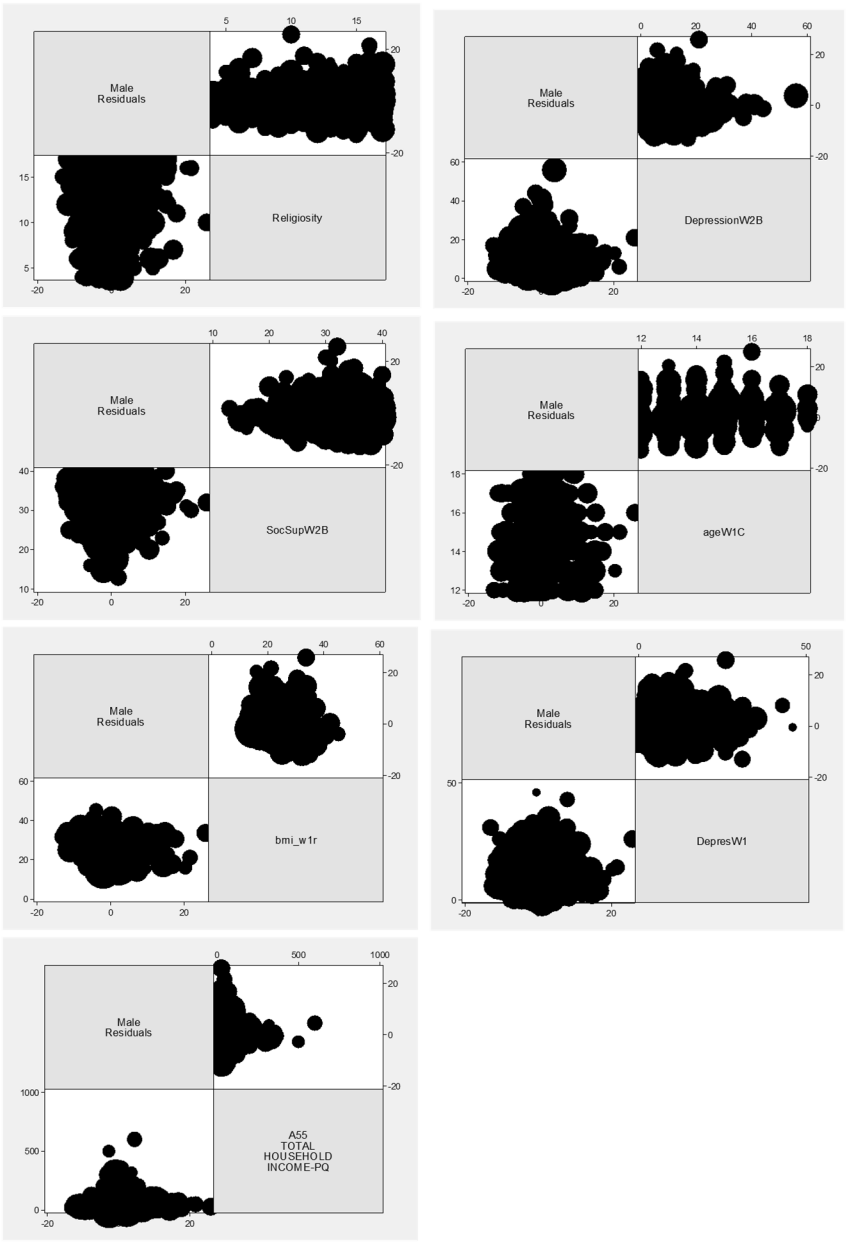


Figure 7. Residual plots against each of the ordinal- and ratio-level predictor variables.

Model specification. An assumption of multiple regression is that the model does not omit relevant variables (Chen et al., 2015). To test for this assumption in Stata, there are two commands—*linktest* and *ovtest*. The *linktest* command creates two variables, a prediction variable and a squared prediction variable (Chen et al., 2015). Upon rerunning the regression with these two variables as predictors, the expectation is that the prediction variable will be significant, but that the squared prediction variable is not a significant predictor. Running the *linktest* command on the weighted data yielded a significant relationship for the predictor variable, $t(131) = 4.15$, $p < .001$, and an insignificant relationship for the squared prediction variable, $t(131) = 0.12$, $p = .905$. The *ovtest* command also checks for omitted variables and should yield a nonsignificant relationship in order to indicate no errors with the model (Chen et al., 2015). The *ovtest* as applied to the weighted data yielded a nonsignificant relationship, $F(3, 2104) = 0.86$, $p = .461$ and, therefore, also confirmed that the model has no omitted variables and is specified accurately.

Independence. The assumption of independence has to do with the correlation of errors between observations (Chen et al., 2015). A common test for this assumption is the Durbin-Watson test statistic, which can be run using Stata. Because the regression used in this analysis includes a lagged variable of BMI at Wave I on the right hand side of the regression, the Durbin-Watson test is invalid, but Durbin's alternative test can be used in these cases (StataCorp, 2015). This test could not, however, be applied fully to survey data. Alternatively, I used the general *regress* command and applied sample weights but not the *cluster* variable. This analysis was applied to 1,021 males and the weighted sum

of respondents was 4,812,100. The results of the Durbin's alternative test for autocorrelation are $X^2(1) = 1.361$, $p = .243$; therefore, the null hypothesis of no serial correlation is accepted for this regression analysis.

Variable types. This assumption is a general assumption discussed by Field (2009). The outcome variable for the regression analysis was adult BMI (collected at Wave IV) and is a continuous variable. Rather than the outcome variable being an interval measurement, however, BMI scores were ratio measures. By nature, BMI is also unbounded in that there are no scientifically identified top or bottom scores for which one's BMI must fit. The predictor variables in this analysis were either quantitative or categorical as specified by Field (2009). The quantitative (or interval measures) included Religiosity (Wave I), Depression (Wave II), Social Support (Wave II), Age at Wave I, BMI at Wave I (a ratio score), Depression at Wave I, and Family Income at Wave I. The predictor variables that were categorical in nature included Race, Smoking Status at Wave I and at Wave IV, and Income Quintile at Wave IV.

Conclusion about meeting assumptions for the regression analysis for males. In conclusion, the use of survey data presented a number of problems with postestimation related to the regression analysis for males. Many of the typical checks for assumptions were not available in the *survey* commands of Stata 14. In particular, I was not able to generate specified approaches to determining the assumptions related to identification of outliers, normality of residuals, homoscedasticity, or linearity. To address these potential problems, I note that robust tests in Stata using the *survey* command are recognized as attending to problems with data including normality, heteroscedasticity, and large

residuals (Chen et al., 2015). Stata was useful in allowing me to address the assumptions of multicollinearity and model specification while accounting for survey data and these assumptions were met.

Research Question 1 (RQ1) for Female Participants

Research question 1 for females was the same as that for males: what is the relationship that exists between adult obesity (DV) and adolescent religiosity (IV), adolescent depression (IV), and adolescent social support (IV)? For all linear regression analyses, the *survey data analysis* option in Stata 14 was used and grand sample weights were accounted for by setting the CLUSTER2 variable as the primary sampling unit and GSWGT4_2 as the sampling weight. As was discussed with the male dataset, the Religiosity scores for females at Wave I did not include those who had legitimate skips for the religiosity questions, and the Depression and Social Support scores from Wave II did not include respondents who were older than 18 years of age at the Wave II interview.

The role of adolescent religiosity, depression, and social support in the development of adult obesity among females. The resulting sample size for the primary variables of interest for RQ1 was n = 1,563 females and represented 6,120,344 girls in Grades 7-12 in 1994-1995. The relationship between the primary variables of interest (adolescent religiosity, adolescent depression, adolescent social support, and adult BMI) was not significant, $F(3, 129) = 1.60$, $p = .194$. Adolescent Religiosity, $t(131) = 0.30$, $p = .76$, and adolescent Social Support, $t(131) = 0.80$, $p = .427$, were not significant predictors of adult Obesity among females. Although the overall model was not

statistically significant, adolescent Depression was a significant predictor of adult Obesity, $t(131) = 2.20$, $p = .030$ (see Model 1 in Table 15).

Control variables included in the initial analysis for males were also used in this analysis for females. The control variables were: race/ethnicity, baseline age, baseline BMI, baseline depression, baseline smoking status, family income at baseline, smoking status at Wave IV, and income at Wave IV. Model 2, which is represented in Table 15, demonstrates the prediction of adult female BMI based on all of these variables. The resulting sample size involving these control variables was $n = 1{,}691$females and represented 6,774,423 female students in Grades 7-12 in 1994-1995. The prediction of adult obesity from these demographic variables was statistically significant, $F(13, 119) = 83.92$, $p < .001$. The R^2 for this relationship was 0.503. In this model, Age at Wave I, Depression at Wave I, and Smoking Status at Wave I were nonsignificant predictors of adult obesity. All other variables were significantly predictive of adult obesity. In relation to the White population, being of Other race or ethnicity significantly predicted adult obesity at $t(131) = -2.66$, $p = .009$, but being of Hispanic and African American origins did not significantly predict adult obesity; $t(131) = -1.14$, $p = .258$ for the Hispanic population and $t(131) = 0.41$, $p = .681$ for the African American population. BMI at Wave I, $t(131) = 28.96$, $p < .001$, significantly contributed to the prediction of adult obesity; family income at Wave I, $t(131) = -2.79$, $p = .006$, significantly contributed to the prediction of adult obesity; smoking status at Wave IV, $t(131) = -2.45$, $p = .016$, significantly contributed to the prediction of adult obesity; and the fourth and fifth levels of Income Quintiles (in relation to the first quintile) contributed significantly to the

prediction of adult obesity, $t(131) = -2.07$, $p = .041$ for the fourth quintile, and $t(131) = -3.16$, $p = .002$ for the fifth quintile (see Model 2, Table 15).

The final combined model for women was determined by considering the theoretical rationale for including control and predictor variables as well as observing the contribution of the control and predictor variables in the model. Model 3 (Table 15), is the final model of interest for answering RQ1. In this model, smoking status at Wave I was dropped from the regression due to its low statistical contribution to the R^2 for the model. When considering all available independent variables and control variables, smoking status at Wave I in this model lacked statistical significance at $t(131) = 0.46$, $p = .644$. When dropping smoking status at Wave I from the model, the R^2 for the model remained unchanged at .5419. The resulting model (Model 3, Table 15) included data for 1,106 respondents, representing 4,440,158 female students in Grades 7-12 in 1994-1995 and was a statistically significant predictor of adult obesity, $F(15, 117) = 70.92$, $p < .001$. The R^2 for this model was .542, which indicated that while demographic data was a strong predictor of adult obesity, the prediction could be enhanced with data on measures of religiosity, depression, and social support during adolescence. The change in R^2 between Model 2 and Model 3 was .039. In this complete model (see Table 15), neither adolescent Religiosity, $t(131) = -0.40$, $p = .688$, adolescent Depression, $t(131) = -0.29$, $p = .769$, nor adolescent Social Support, $t(131) = 1.91$, $p = .058$ were significant predictors of adult Obesity among females, although Social Support was just beyond the .05 level of significance. BMI at Wave I, $t(131) = 25.70$, $p < .001$, remained a significant predictor of adult Obesity in the full model. Family Income at Wave I remained a significant predictor

of adult Obesity, $t(131) = -2.04$, $p = .043$. Wave IV Smoking Status remained a significant predictor of adult Obesity in the full model, $t(131) = -2.21$, $p = .029$. All Wave IV income categorizations, except for the third quintile, were significant predictors of adult Obesity in relation to the lowest income quintile; values are $t(131) = -2.19$, $p = .030$ for the second quintile, $t(131) = -2.34$, $p = .021$ for the fourth quintile, and $t(131) = -2.86$, $p = .005$ for the fifth quintile. Although Wave II Depression and Social Support were not significant contributors to the prediction of adult Obesity, removing Wave II Depression, Wave II Social Support, or both from the model did not increase the overall power of the relationship. When Wave II Depression was removed, the R^2 for the model was .5395; when Wave II social support was removed, the R^2 was .532; and when both Wave II Depression and Social Support were removed, the R^2 was .520. In summary, the null hypothesis for RQ1 was rejected as adolescent religiosity, adolescent depression, and adolescent social support contributed to the significant prediction of adult female obesity despite the fact that none of these variables individually contributed statistically significantly to the model.

Table 15

Coefficients, Standard Errors, 95% CIs, and Significance Levels of the Various Regression Models in the Prediction of Adult Obesity for Females

Model	β	SE	*t*	*p*-value	95% CI
Model 1: Primary variables					
Religiosity (W1)	.0228	.0754	0.30	.763	[-.13, 0.17]
Depression (W2)	.0665	.0302	2.20	.030*	[0.01, 0.13]
Social support (W2)	.0466	.0584	0.80	.427	[-0.07, 0.16]
Constant	26.3932	2.3048	11.45	.000***	[21.83, 30.95]
Model 2: Demographic predictors					
Race/ethnicity					
Hispanic					
African American	-.5242	.4618	-1.14	.258	[-1.44, .39]
Other	.2053	.4990	0.41	.681	[-.78, 1.19]
Age (WI)	-1.6706	.6286	-2.66	.009**	[-2.91, -.43]
Baseline BMI (WI)	-.1769	.1522	-1.16	.247	[-.48, .12]
Smoking status (WI)	1.2675	.0438	28.96	.000***	[1.18, 1.35]
Smoker					
Baseline depression (WI)	.0588	.4005	0.15	.884	[-.73, .85]
Family income (WI)	.0081	.0248	0.33	.745	[-.04, .06]
Smoking status (WIV)	-.0092	.0033	-2.79	.006**	[-.12, -.00]
Smoker					
Income quintile (WIV)	-1.0708	.4375	-2.45	.016*	[-1.94, -.21]
Second quintile					
Third quintile	-1.0284	.6403	-1.61	.111	[-2.30, .24]
Fourth quintile	-.6582	.7113	-0.93	.357	[-2.07, .75]
Fifth quintile	-1.5241	.7366	-2.07	.041*	[-2.98, -.07]
Constant	-2.0947	.6629	-3.16	.002**	[-3.41, -.78]
	5.2244	1.8992	2.75	.007**	[1.47, 8.98]
Model 3: Combined primary and demographic variables					
Religiosity	-.0249	.0620	-0.40	.688	[-.15, .10]
Depression (WII)	-.0098	.0333	-0.29	.769	[-.08, .06]
Social support (WII)	.0905	.0473	1.91	.058	[-.00, .18]
Race/ethnicity					
Hispanic	.0745	.6432	0.12	.908	[-1.20, 1.35]
African American	.2775	.5614	0.49	.622	[-.83, 1.39]
Other	-1.8703	1.0973	-1.70	.091	[-4.04, .30]
Age (WI)	-.1640	.1315	-1.25	.214	[-.42, .10]
Initial BMI (WI)	1.2983	.0505	25.70	.000***	[1.20, 1.40]
Depression (WI)	.0275	.0258	1.07	.289	[-.02, .08]
Family income (WI)	-.0071	.0035	-2.04	.043*	[-.01, -.00]
Smoking status (WIV)					
Smoker	-.8742	.3958	-2.21	.029*	[-1.66, -.09]
Income quintile (WIV)					
Second quintile	-1.6005	.7302	-2.19	.030*	[-3.04, -.16]
Third quintile	-.9202	.6575	-1.40	.164	[-2.22, .38]
Fourth quintile	-1.8532	.7923	-2.34	.021*	[-3.42, -.29]
Fifth quintile	-2.2608	.7910	-2.86	.005**	[-3.83, -.70]
Constant	1.5904	3.0432	0.52	.602	[-4.43, 7.61]

*$p < .05$. **$p < .01$. ***$p < .001$.

Table 16

Statistical Comparison of Means for Factor Variables in the Prediction of Obesity Among Females in the Full Model (Model 3)

Group Comparison	Coef	Std Error	*t*-stat	*p*-value	95% CI
White—Hispanic	-.0745	.6432	-0.12	.908	[-1.35, 1.20]
White—African Am.	-.2775	.5614	-0.49	.622	[-1.39, .83]
White—Other	1.8703	1.0973	1.70	0.91	[-.30, 4.04]
Hispanic—African Am.	-.2029	.8356	-0.24	.808	[-1.86, 1.45]
Hispanic—Other	1.9448	1.1294	1.72	.087	[-.29, 4.18]
African Am—Other	2.1478	1.0782	1.99*	.048	[.01, 4.28]
IncQuin1—IncQuin2	1.6005	.7302	2.19*	.030	[.16, 3.04]
IncQuin1—IncQuin3	.9203	.6575	1.40	.164	[-.38, 2.22]
IncQuin1—IncQuin4	1.8532	.7923	2.34*	.021	[.29, 3.42]
IncQuin1—IncQuin5	2.2608	.7910	2.86**	.005	[.70, 3.83]
IncQuin2—IncQuin3	-.6802	.5582	-1.22	.225	[-1.78, .42]
IncQuin2—IncQuin4	.2527	.6964	0.36	.717	[-1.13, 1.63]
IncQuin2—IncQuin5	.6604	.6136	1.08	.284	[-.55, 1.87]
IncQuin3—IncQuin4	.9329	.5800	1.61	.110	[-.21, 2.08]
IncQuin3—IncQuin5	1.3406	.5388	2.49*	.014	[.27, 2.41]
IncQuin4—IncQuin5	.4077	.6915	0.59	.557	[-.96, 1.78]

*$p < .05$. **$p < .01$. ***$p < .001$.

Assumptions of linear regression analysis for female data. The same procedures to check for adherence to assumptions of liner regression that were applied to male data were applied to the data for females. The same issues regarding the use of survey data were encountered with the female data as was discussed with the male data. When possible, assumptions were addressed in relation to the *survey* mode of Stata 14. The survey mode accounts for weights and for the identification of clusters in the data. In some cases, assumptions were addressed while only accounting for sample weights because the particular command was invalid for use with clusters. I identify if the

assumption was addressed for survey data or if it was addressed using only sample weights for each of the assumptions listed below.

Outliers and influence. I was unable to run the specific commands in Stata that would produce evaluations of outliers, leverage, and influential data points such as Cook's D, and DFITS due to the nature of my survey data. I was also unable to determine statistics for DFBETA on my weighted data. Using weighted data, I was able to produce visual relationships between Wave IV BMI and the three independent variables (adolescent religiosity, depression, and social support). These representations are provided in Figure 8 and they demonstrate that there was one clear data point that demonstrated separation from the pack in the Religiosity graph. Depression and Social Support graphs were more cohesive with just a few points that were distinguishable from the group. Because the specific Stata statistical commands were not available to evaluate these potential outliers, I am acknowledging that my data was comprised of a handful of potential outliers. However, I was unable to determine the specific influence these potential outliers had on the dataset.

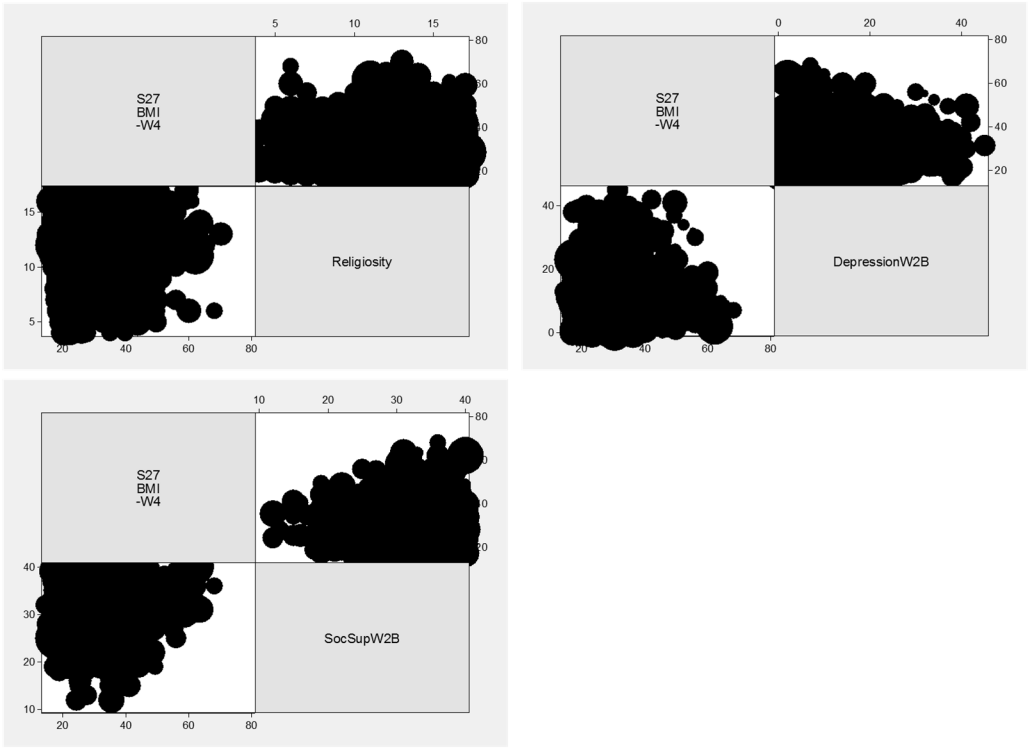


*Figure 8.*Scatterplots representing each predictor variable against the outcome variable, adult obesity, for females in the study. The scatterplots represent weighted data as seen by the mass they occupy, as opposed to the representation of a single point, and indicate potential outliers.

Normality of residuals. Many of the specific checks for normality could not be performed while accounting for the survey design. After determining the residuals for the 1,106 females included in the full model, I was able to produce the following: a kernel density plot using the *kdensity* command; a standardized normal probability plot using the *pnorm* command; a plot of quantiles using the *qnorm* command; an inter-quartile range determination using the *iqr* command; and the Shapiro-Wilk W test for normality using the *swilk* command (UCLA, 2015, Chapter 2.2). The kernel density plot demonstrated leptokurtosis and slight positive skewness (Figure 9). The standardized normal probability plot is used to identify shifts from normality for the mid-range data (Chen et

al., 2015); Figure 10 shows slight departure from the plot at mid-range. The quantile plot is used to identify shifts from normality at the ends (Chen et al., 2015); Figure 10 demonstrates departure from normality at both the top and bottom end of the graph. The inter-quartile range analysis identified seven mild outliers on the lower end and 27 mild outliers on the upper end; one of the upper end outliers was identified as a severe outlier in this analysis. Finally, a significant *p*-value for the Shapiro-Wilk W test indicated that the distribution of the residuals lacked normality. The results of the Shapiro-Wilk W test for the unweighted data revealed that the residuals were not normally distributed, $z = 6.975$, $p < .001$. As discussed in the analysis of the male data, it should be noted that all of the commands in this section on normality of residuals were applied to unweighted data. Due to the fact that this analysis was not based on weighted data, there was no definitive measure for the role of the outliers on the weighted analysis. In this case, the robust test used to complete survey analysis was believed to account for these potential outliers as some large residuals can be offset by robust tests (Chen et al., 2015).

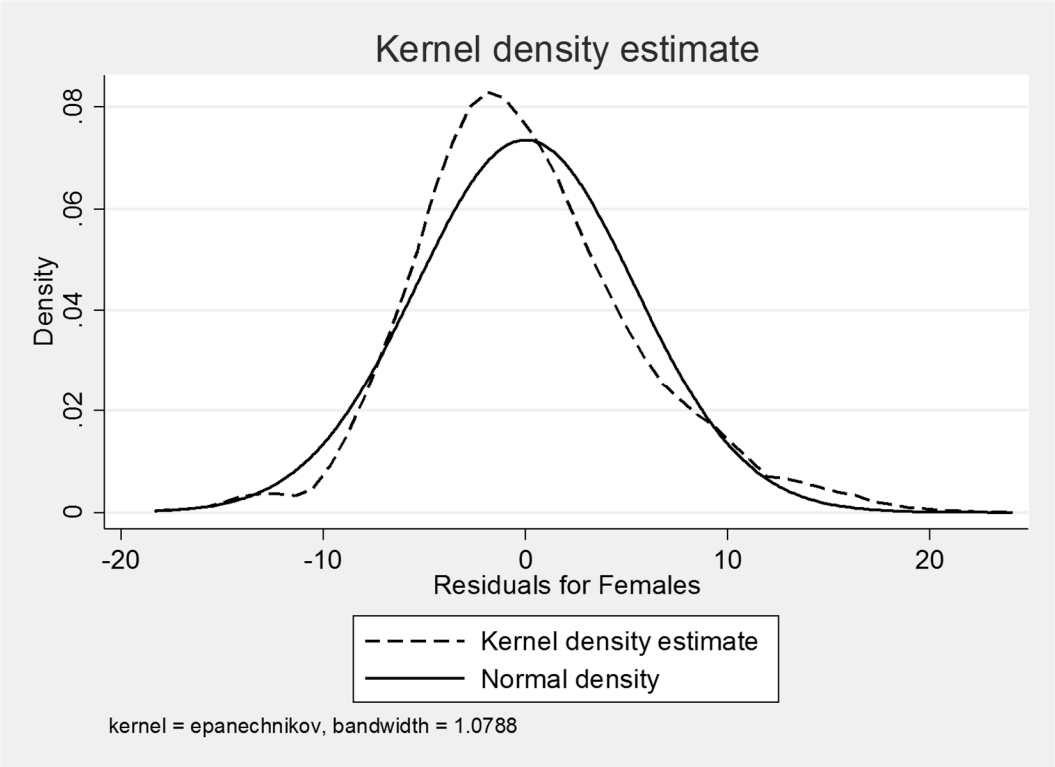


Figure 9. Kernel density plot of residuals for female participant data.

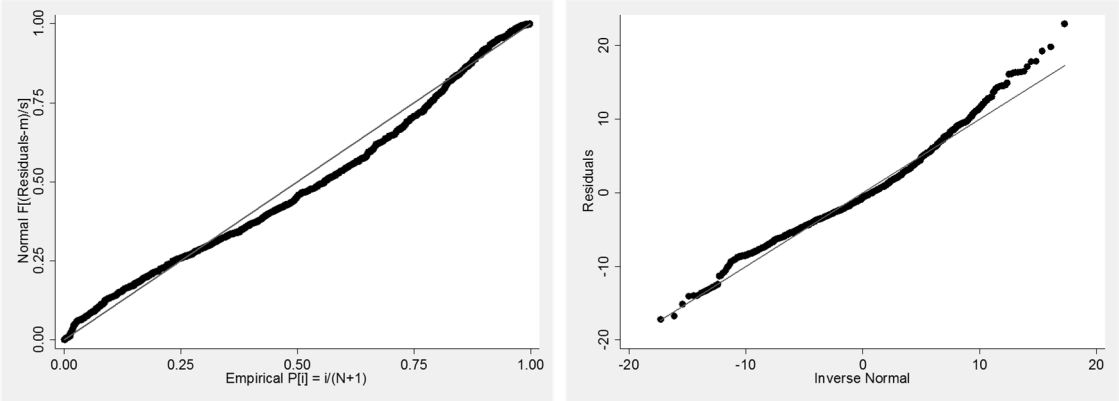


Figure 10. Standardized normal probability plot and quantile plot for female participant data.

Homoscedasticity. Homoscedasticity is an analysis of the residuals in relation to the predictor variables (Field, 2009). The use of survey data in this study limited the statistical tests that could be used for postestimation analysis. I was only able to evaluate for homoscedasticity with a visual representation. Field (2009, p. 229) described a plot of standardized residuals against standardized predicted values as a visual check for meeting the assumptions of homoscedasticity. Using Stata 14, Chen et al. (2015) outlined steps to produce a related chart. I was able to generate predicted values for Wave IV BMI using the *predict xb* command. I was also able to generate residuals for the Wave IV BMI using the *predict, residuals* command. I graphed the residuals against the predictions and was able to apply sample weights to this graph (see Figure 11), which resulted in masses rather than points associated with each data point.

It appears that the weighted data demonstrated a level of heteroscedasticity due to the fact that the data did not plot as random plots but as clear clusters with a few breaks in continuity (Figure 11). However, because survey analysis in Stata is "robust to heteroscedasticity" (J. Wang, StataCorp Statistician, personal communication, September 10, 2015), the possibility of some heteroscedasticity in the current female dataset was not of great concern.

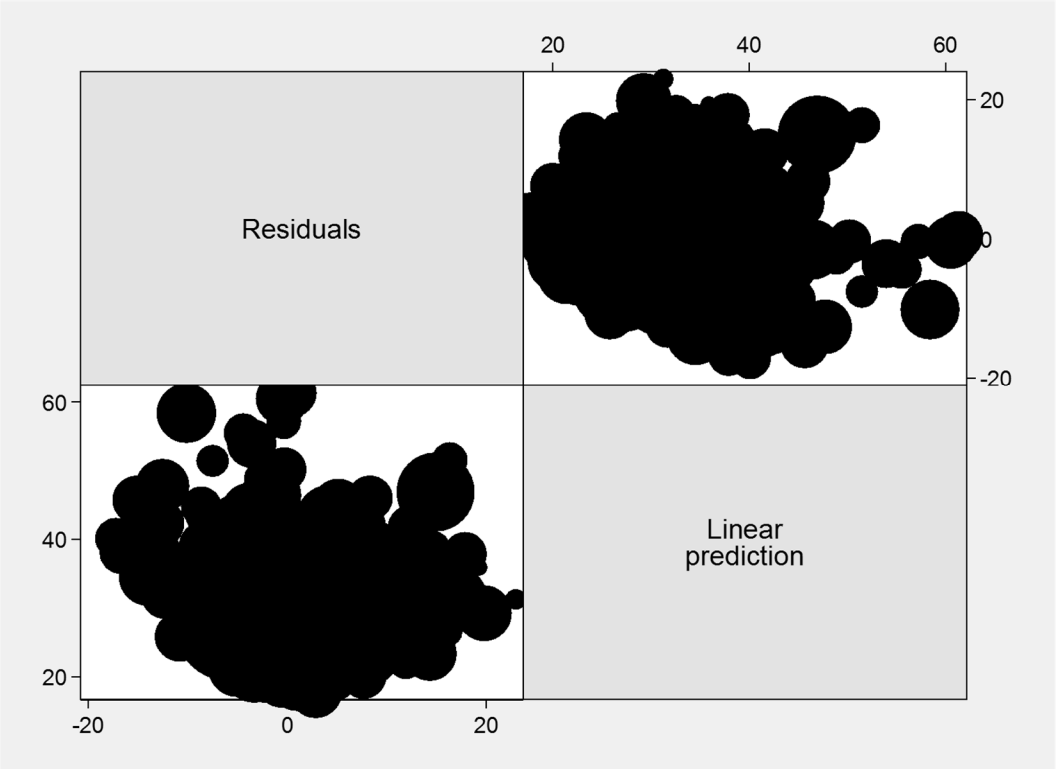


Figure 11. Wave IV BMI residuals for females plotted against the predicted values of Wave IV BMI based on the full regression model and on residuals and predicted values with weights applied to the graph.

No perfect multicollinearity. Perfect multicolinearity can be identified by predictor variables that are perfectly correlated (r= 1.0; Field, 2009). In Table 17, I recorded the correlations for all nonfactor predictor variables. These correlations are representative of weighted data and demonstrated that there was no perfect correlation between any two predictor variables. Despite the seemingly low correlations between predictor variables, many of the correlations were significant at the $p < .05$ level or below. Religiosity at Wave I was not significantly correlated with BMI at Wave I or with Family Income at Wave I. Social Support at Wave II was significantly correlated with every other predictor variable except for BMI at Wave I and Family Income at Wave I.

Age at Wave I was significantly correlated with each predictor variable except for Family Income at Wave I.

Another check for multicollinearity is to consider variance inflation factors (VIF) for the predictor variables (Field, 2009). These values are listed in Table 18 and were determined from the survey weighted data. The individual factor VIFs for Race and Income Quintile are listed in parenthesis below and were not available in the survey data; they were determined using the regular *regress* command and by applying sample weights but not the *cluster* variable. Both the survey analysis and the regular *regress* analysis yielded similar results for VIF. All VIFs from the survey data were between one and two and were less than the value of concern for multicollinearity of 10 (Field, 2009). Furthermore, the tolerance statistics (the reciprocal of VIF) for the predictor variables were all above the .2 level, which is noted by Field as the cutoff for cause for concern. The average of the VIFs for the full survey weighted model was 1.23 and not of concern as it was not substantially higher than 1.

Table 17

Correlation Coefficients and Significance Levels (in Parenthesis) for Predictor Variables in the Female Dataset

	Religiosity (Wave I)	Depression (Wave II)	Social support (Wave II)	Age (Wave I)	BMI (Wave I)	Depression (Wave I)	Family income (Wave I)
Religiosity (Wave I)	1.000						
Depression (Wave II)	-0.132 (.000***)	1.000					
Social support (Wave II)	0.210 (.000***)	-0.400 (.000***)	1.000				
Age (Wave I)	-0.081 (.033*)	0.064 (.037*)	-0.132 (.000***)	1.000			
BMI (Wave I)	-0.042 (.213)	0.071 (.012*)	-0.040 (.249)	0.142 (.000***)	1.000		
Depression (Wave I)	-0.144 (.000***)	0.601 (.000***)	-0.320 (.000***)	0.092 (.007**)	0.100 (.000***)	1.000	
Family income (Wave I)	0.039 (.219)	-0.115 (.000***)	0.005 (.883)	0.040 (.196)	-0.080 (.025*)	-0.073 (.004**)	1.000

Note. Weighted correlations for these variables are based on 1,296 observations representing 5,183,911 female high school students in the U.S. in 1994-1995.

Table 18

Female Dataset Variance Inflation Factors (VIF) for Independent Variables in the Regression Analysis

Variable	VIF	Tolerance statistic (1/VIF)
Religiosity	1.10	0.91
Depression (Wave II)	1.79	0.56
Social support (Wave II)	1.27	0.79
Race/ethnicity	1.13	0.89
Hispanic	*(1.09)	*(0.92)
African American	*(1.18)	*(0.84)
Other	*(1.03)	*(0.97)
Age (Wave I)	1.08	0.93
BMI (Wave I)	1.09	0.92
Depression (Wave I)	1.63	0.61
Family income (Wave I)	1.06	0.94
Smoking status (Wave IV)		
Smoker	1.07	0.94
Income quintile (Wave IV)	1.12	0.89
Quintile 2	*(2.11)	*(0.47)
Quintile 3	*(2.45)	*(0.41)
Quintile 4	*(1.87)	*(0.54)
Quintile 5	*(1.87)	*(0.54)
Mean VIF	1.23	
	*(1.44)	

* VIFs for individual factor variables while applying sample weights in *regress* without accounting for the cluster variables.

Linearity. The assumption of linearity as related to regression analysis is that the outcome variable is linearly related to each predictor variable (Chen et al., 2015). The only analysis that could be applied to weighted data to check for this assumption was a plot of the residuals against the ordinal- and ratio-level predictors with the hope that breaks from linearity might be clearly visible. Once again, because the weighted plots are represented by masses as opposed to a single plot, the graphs can be difficult to decipher. I could make no determination of clear violation of linearity based on the weighted plots, which can be seen in Figure 12.

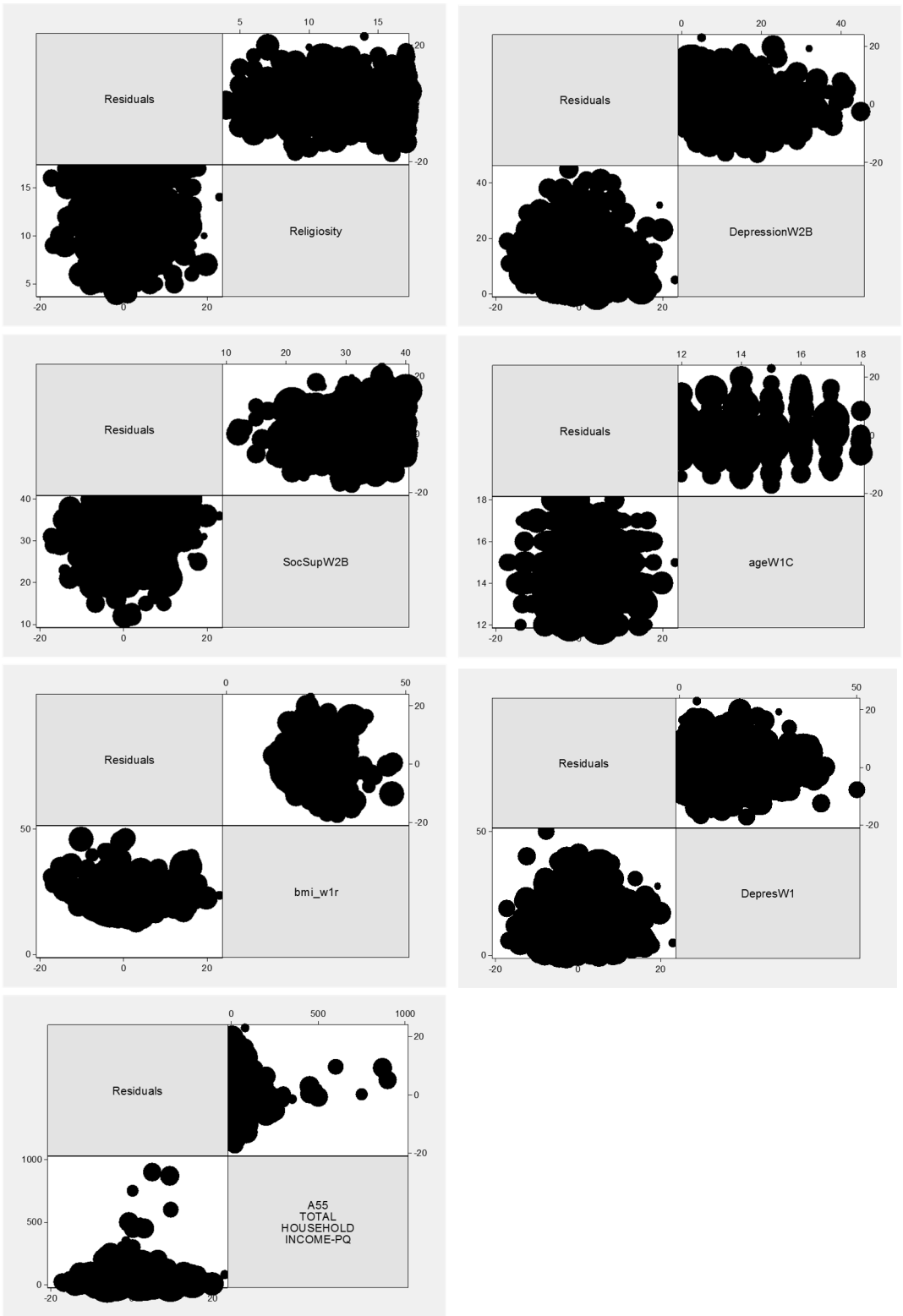


Figure 12. Residual plots against each of the ordinal- and ratio-level predictor variables.

Model specification. An assumption of multiple regression is that the model does not omit relevant variables (Chen et al., 2015). The command used to test for this assumption in Stata is *linktest*. The *linktest* command creates two variables, a prediction variable and a squared prediction variable (Chen et al., 2015). Upon rerunning the regression with these two variables as predictors, the expectation was that the prediction variable would be significant, but that the squared prediction variable would not be a significant predictor. Running the *linktest* command on the survey weighted data yielded a significant relationship for the predictor variable, $t(131) = 6.63$, $p < .001$, and an insignificant relationship for the squared prediction variable, $t(131) = -1.64$, $p = .103$. The *ovtest* command also checks for omitted variables and should yield a nonsignificant relationship in order to indicate no errors with the model (Chen et al., 2015). The *ovtest* as applied to the weighted data yielded a nonsignificant relationship, $F(3, 2112) = 1.43$, $p = .2320$ and, therefore, also confirms that the model has no omitted variables and is specified accurately. Based on the results of these two commands, I concluded that this model was specified correctly.

Independence. The assumption of independence has to do with the correlation of errors between observations (Chen et al., 2015). A common test for this assumption is the Durbin-Watson test statistic, which can be run using Stata. Because the regression analysis used a lagged variable on the right hand side (Obesity at Wave I as a predictor of Obesity at Wave IV), I was not able to use the typical Durbin-Watson test for this analysis. For regressions in which lagged variables are present, Durbin's alternative test

can be used and in Stata it can be used with regression using the *vce(robust)* format (StataCorp, 2015, p. 2185). I ran the full regression model accounting for analytic weights and identifying the robust format, but I was not able to include the cluster variable in this analysis. Durbin's alternative test yielded $X^2(1) = 5.451$, $p = .020$ for the probability of serial correlation. These results indicate the possibility of serial correlation in this dataset, but due to the inability to fully account for the survey nature of the data by including *cluster* information, these results may be inaccurate.

Conclusion about meeting assumptions for the regression analysis for females. As identified in the discussion of meeting assumptions for male data, the use of survey data for female analysis presented a number of challenges with postestimation. In particular, determining the assumptions related to identification of outliers, normality of residuals, homoscedasticity, and linearity were hindered by this limitation. To address these potential problems, I note that robust tests in Stata using the *survey* command are recognized as attending to problems with data including normality, heteroscedasticity, and large residuals (Chen et al., 2015). Stata was useful in allowing me to address the assumptions of multicollinearity and model specification while accounting for survey data and these assumptions were met.

Research Question 2 (RQ2) for Male Participants

Research Question 2 was a consideration of adolescent depression (from Wave II data) as a possible mediator of the adolescent religiosity/adult obesity relationship with religiosity being measured at Wave I and adult obesity being measured at Wave IV. The *survey data analysis* option in Stata 14 was used in answering RQ2. This option

accounted for primary sampling units using the CLUSTER2 variable and for the sampling weight using the GSWGT4_2 variable provided with the Add Health datasets.

The first step in mediation analysis is to determine the significance of the relationship between the IV, religiosity in this case, and the DV, adult obesity. Using survey data options in Stata, I ran a regression analysis for adult Obesity on Religiosity with a result of 1,984 observations representing 9,282,390 U.S. males in Grades 7-12 in 1994-1995. According to Diebold (2013) the β derived from a simple regression represents the correlation of the two variables. In this case, the β for adult Obesity regressed on adolescent Religiosity was .124, $t(131) = 2.57$, $p = .011$. Therefore, in the most basic model (Model 1 from the previous section), there was a significant correlation between adolescent religiosity and adult obesity for males.

The next step of mediation analysis, according to Baron and Kenny (1986), is to identify the relationship that exists between the IV and the MV (path *a*). One way to accomplish mediation analysis with survey data using Stata 14 is through the SEM commands. Using Stata 14, the regression of Depression (Wave II) on Religiosity (Wave I) resulted in a significant relationship $t(131) = -2.45$, $p = .016$, with increases in Religiosity among males resulting in decreases in Depression symptomology scores. This relationship was determined using 1,425 males representing 6,653,785 U.S. males in Grades 7-12 in 1994-1995. The second portion of the SEM analysis combines an analysis of the dependent variable regressed on the mediator variable (path *b*; Obesity on Depression) and of the dependent variable regressed on the independent variable while accounting for the mediator (path *c*'; Obesity regressed on Religiosity through

Depression). Path *b*, Obesity regressed on Depression, was not significantly related, $t(131) = 1.46$, $p = .148$, while path *c*', Obesity regressed on Religiosity through Depression, remained significantly related, $t(131) = 2.72$, $p = .008$. Therefore, the primary model (Model 1) did not demonstrate mediation of Depression on the Religiosity-Obesity relationship for males.

However, as discussed with RQ1, the control variables of interest in this study had a profound impact on the relationships seen in the regression analyses. Therefore, it was prudent to run the analyses while accounting for the control variables of Age at Wave I, Race, BMI at Wave I, Depression at Wave I, Smoking Status at Wave I, Family Income at Wave I, Smoking Status at Wave IV, and Income Quintile at Wave IV. I first determined that the relationship between adolescent Religiosity (IV) and adult Obesity (DV) was significantly correlated using *svyset* linear regression analysis in Stata 14 and accounting for the stated control variables. The regression of BMI on Religiosity demonstrated a significant relationship between these two variables, $F(14, 118) = 47.92$, $p < .001$, and utilized 1,386 observations in the analysis while representing 6,506,860 males in Grades 7-12 in 1994-1995. The specific relationship between Religiosity and BMI was significant within this model at $t(131) = 2.97$, $p = .004$.Therefore, path *c*, as discussed by Baron and Kenny, was significantly correlated (see Figure 13).

Path *a*, the regression of Depression on Religiosity, was calculated using the GSEM commands to account for factor variables in the analysis. This analysis included evaluation of 1,064 observations accounting for 4,999,390 males and was not significantly related at $t(131) = -0.98$, $p = .328$.The second portion of the GSEM

commands in Stata provide the analysis for path *b* and for path *c*'. The regression between Wave II Depression and adult Obesity for males did not yield a significant correlation, $t(131) = 1.16$, with $p = .247$. Path *b* is not a significant relationship. The regression of Obesity on Religiosity while accounting for Wave II Depression was a significant relationship, $t(131) = 3.32$, $p = .001$. Nevertheless, because paths *a* and *b* were not significant, Wave II depression was not a mediator of the Religiosity-Obesity relationship for males. The null hypothesis for RQ2 was not rejected as it relates to the male population.

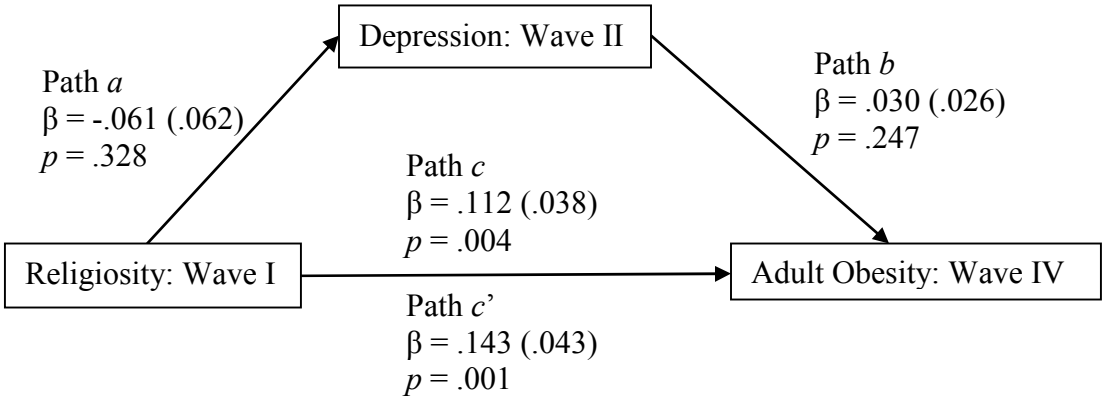


Figure 13. Betas (standard errors) and significance levels for paths a, b, and c of the GSEM analysis for mediation of the adult obesity (DV) and adolescent religiosity (IV) relationship by adolescent depression (MV) while accounting for control variables.

Research Question 2 (RQ2) for Female Participants

An analysis of female participants for adolescent Depression as a mediator of the Religiosity-Obesity relationship starts with the determination of the significance of the relationship between adolescent Religiosity and adult Obesity. For women, this relationship was not significantly correlated. The regression of adult Obesity on adolescent Religiosity was based on 2,219 females accounting for 8,669,080 females in

Grades 7-12 in 1994-1995 and was not significant, $F(1, 131) = 0.17$, $p = .678$. Therefore, the basic model for women did not meet the first criteria for consideration of mediation.

Similar to working with the male data, I attempted the mediation analyses for women while accounting for the control variables of Age at Wave I, BMI at Wave I, Depression at Wave I, Family Income at Wave I, Smoking Status at Wave IV, and Income Quintile at Wave IV. Adolescent Religiosity (IV) and adult Obesity (DV) were not significantly correlated in this analysis, $t(131) = 0.18$, $p = .858$. Therefore, the null hypothesis for RQ2 was not rejected for female participants.

Research Question 3 (RQ3) for Male Participants

Research Question 3 was a consideration of adolescent Social Support (from Wave II data) as a possible mediator of the adolescent Religiosity/adult Obesity relationship with Religiosity being measured at Wave I and adult Obesity being measured at Wave IV. The *survey data analysis* option in Stata 14, as outlined in the previous section, was used in answering RQ3.

As discussed with RQ2, the basic model, in which only Religiosity, Depression, Social Support, and Obesity were considered, the relationship between Religiosity (IV) and Obesity (DV) was significantly correlated, $t(131) = 2.57$, $p = .011$. Path *a*, Social Support regressed on Religiosity, was also a significant correlation, $t(131) = 4.64$, $p < .001$. However, path *b* was not a significant correlation, $t(131) = -0.66$, $p = .512$. The basic model (Model 1) did not demonstrate mediation of the Religiosity-Obesity relationship by Social Support.

As with the consideration of Depression as a mediator, I considered Social Support as a mediator when accounting for the identified control variables in this study. Path *c* (see Figure 14), the relationship between Religiosity and Obesity, was a significant relationship, $F(14, 118) = 47.92$, $p < .001$ and $t(131) = 2.97$, $p = .004$. The model for path *a*, the relationship between Religiosity and Social Support, included 1,040 observations and represented 4,896,121 males in Grades 7-12 in 1994-1995 and was significantly correlated, $t(131) = 2.45$, $p = .016$. Path *b* of the analysis, the relationship between Social Support and Obesity was not significantly correlated in this model, $t(131) = -1.16$, $p = .247$. Path *b* was not a significant correlation, therefore, Social Support was rejected as a possible mediator of the relationship between adolescent Religiosity and adult Obesity for males. The null hypothesis for RQ3 was not rejected as it relates to the male population.

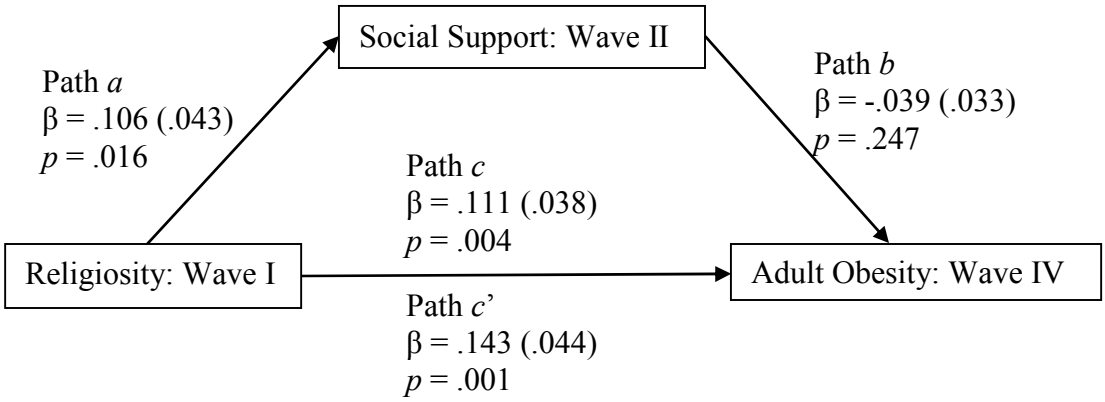


Figure 14. Betas (standard errors) and significance levels for paths a, b, and c of the GSEM analysis for mediation of the adult obesity (DV) and adolescent religiosity (IV) relationship by adolescent social support (MV) while accounting for control variables.

Research Question 3 (RQ3) for Female Participants

The null hypothesis for RQ3 was not rejected for females given that adolescent Religiosity was not significantly correlated with adult Obesity in neither the basic model (Model 1) nor the full model (Model 3). This basic requirement for mediation was shown to be missing in the discussion of RQ2 for females previously.

Changes in Obesity Over Time for Males Accounting for Religiosity, Depression, and Social Support in Adolescence

Measurements of BMI for Waves II and III were included in the analysis of the change in obesity over time. For males at Wave III, 10 of the respondents weighed more than 330 pounds, yet the specific scale used for measurement of participants did not extend past 330. Six of these males self-reported their weight to be less than 330 pounds despite the fact that the scale could not produce a weight for these individuals. Therefore, for these individuals, BMI was calculated based on a weight of 331 pounds. Two males self-reported weights above 330 and BMI was based on their self-reported weight. One male was changed to missing at Wave III because he exceeded the 330 pound threshold and did not provide a self-reported weight.

In the regression model presented in RQ1, adolescent Religiosity was shown to demonstrate a positive relationship with adult BMI, the chosen measure of obesity for this study. This relationship was true for Model 1 and for Model 3; Model 3 included control variables along with the primary study variables of Religiosity, Depression, and Social Support. The chosen analytic plan for explaining the change in BMI over four waves of data was repeated measures analysis of variance (ANOVA). However,

constraints of the use of survey data in Stata for this analysis prompted me to consider structural equation modeling (SEM) as an alternative since Stata allows for the analysis of survey data using the SEM commands.

The base model used to identify mean intercepts, linear slopes, and quadratic slopes separately for each race/ethnicity for males is shown in Figure 15. For this portion of the analysis, the intercepts for the four waves of data were constrained to be equal and were set at 1. Linear and quadratic slopes were used to account for the difference in time scale associated with the waves of data. The periods between waves varied as did the length of each data collection period for each wave. For instance, Wave I data was collected between April, 1995 and December, 1995, but Wave II data was collected between April, 1996 and August, 1996. In order to identify a time scale that accounted for these differences, I chose the middle of each collection period and scaled for time based on the years in relation to the first data collection. Table 19 represents the various data collection periods and appropriate scaling for linear and quadratic constraints for the SEM model. These same constraints are shown as constraints for paths from the linear slope and quadratic slope to each wave of BMI collection.

Table 19

Data Collection Periods and Time Between Collection for Each Wave of Data Along With Scaling to Represent Linear and Quadratic Slope Factors

Wave	Collection period	Mid-point of collection	Time from first collection	Time scale for linear slope	Time scale for quadratic slope
I	4/95 to 12/95	8/95	0	0	0
II	4/96 to 8/96	6/96	10 months	.83 years	.69 years
III	7/01 to 4/02	11/15/01	6 years, 3.5 months	6.29 years	39.56 years
IV	1/08 to 2/09	6/15/08	12 years, 10.5 months	12.88 years	165.89 years

The SEM analysis was first run to determine the means for the intercept, linear slope, and quadratic slope based on the four waves of BMI data for males. Furthermore, the analysis was run using the *group (RACES4)* command to identify the differences in race for males. Table 20 details the noted means for males by race. Using the *test* command following the SEM analysis, I was able to test individual hypotheses that coefficients for a given race were significantly different from another race. For intercept, linear slope, and quadratic slope, there was no significant difference between or among any of the races. Similarly, there was no difference between or among the races based on Religiosity at Wave I or Social Support at Wave II. The only relationship that demonstrated significant difference was for Depression at Wave II in which White and Hispanic males were significantly different, $F(1,131) = 6.52$, $p = .012$ with the coefficient for depression symptomology for White males being significantly lower than the coefficient for depression symptomology for Hispanic males.

Table 20

Report of Regression Analytics for the SEM Analysis of Males Accounting for the Differences in Race

Variable	Race	Coefficient	SE	*t*-value	*p*-level	CI-95%
Religiosity	White	-.0137	.0200	-0.69	.493	[-.053, .029]
(Wave I)	Hispanic	-.0316	.0720	-0.44	.662	[-.174, .111]
	African Am	.0648	.0546	1.19	.238	[-.043, .173]
	Other	.2104	.1749	1.20	.231	[-.136, .556]
Depression	White	.0128[a]	.0108	1.18	.241	[-.009, .034]
(Wave II)	Hispanic	.0875[a]	.0267	3.28	.001	[.035, .140]
	African Am	.0515	.0233	2.21	.029	[.005, .098]
	Other	.0357	.0508	0.70	.483	[-.065, .136]
Social support	White	-.0086	.0061	-1.40	.165	[-.021, .004]
(Wave II)	Hispanic	-.0461	.0231	-2.00	.048	[-.092, -.000]
	African Am	.0135	.0206	0.66	.514	[-.027, .054]
	Other	.0482	.0509	0.95	.345	[-.053, .149]
Intercept	White	22.2963	.2990	74.56	.000	[21.70, 22.89]
(mean)	Hispanic	22.7621	1.1083	20.54	.000	[20.57, 24.95]
	African Am	21.5553	.6945	31.04	.000	[20.18, 22.93]
	Other	19.9103	1.8241	10.92	.000	[16.30, 23.52]
Linear slope	White	.7918	.0587	13.48	.000	[.676, .908]
(mean)	Hispanic	.8129	.2336	3.48	.001	[.351, 1.275]
	African Am	.8734	.1704	5.13	.000	[.536, 2.210]
	Other	1.4927	.4472	3.34	.001	[.608, 2.377]
Quad slope	White	-.0234	.0035	-6.62	.000	[-.030, -.016]
(mean)	Hispanic	-.0199	.0129	-1.54	.125	[-.045, .006]
	African Am	-.0235	.0094	-2.50	.014	[-.042, -.005]
	Other	-.0583	.0225	-2.60	.011	[-.103, -.014]

[a]Coefficients for depression were significantly different between the White and Hispanic male populations.

The general outcome of this SEM analysis demonstrated that males of each race (White, Hispanic, African American, and Other) were substantively similar in their pattern of development of obesity from adolescence to adulthood in relation to the role of religiosity, depression, and social support during adolescence. However, given that Age at Wave I was a significant contributor to the development of obesity at Wave IV as demonstrated by the linear regression analysis for males used to answer RQ1, I ran the SEM model (Figure 15) assuming no racial differences for males in their development of obesity. In this second analysis, age was accounted for by using the *group* function in SEM and by categorizing the controlled ages at Wave I as follows: ages 12 and 13 at Wave I were set to AgeCat 1, ages 14 and 15 were set to AgeCat 2, and ages 16 through 18 were set to AgeCat 3. The SEM analysis was then performed with the *group(AgeCat)* command to yield separate results for each age category. As expected, there were a number of significant differences between the ages groups related to their development of obesity over the time period of the study.

Reports of coefficients from the SEM analysis of males while accounting for the grouping of age categories are found in Table 21. The categorization of age for 12 and 13 year-olds yielded an n of 219; for ages 14 and 15, an n of 438; and for ages 16 through 18, an n of 434. Values indicating significant differences between and among age groups are provided in Table 19. Notably, there was a significant difference among the mean intercepts for the three categories, $F(2, 130) = 4.42$, $p = .014$. The mean intercept was also significantly different between the 12-13 and the 14-15 year olds, $F(1, 131) = 5.87$, $p = .017$, and between the 12-13 and 16-18 year olds, $F(1, 131) = 8.61$, $p = .004$. The mean

of the linear slope was significantly different between the 12-13 and 14-15 year olds, $F(1, 131)$, $p = 4.63$, $p = .033$. The mean of the quadratic slope was significantly different between the 14-15 and 16-18 year olds, $F(1, 131) = 4.77$, $p = .031$.

Table 21

Report of Regression Analytics for the SEM Analysis of Males Accounting for the Differences in Age Based on Age Categories

Variable	Age Group	Coef	SE	*t*-value	*p*-level	CI-95%
Religiosity	Ages 12 & 13	.0338	.0401	0.84	.401	[-.046, .113]
(Wave I)	Ages 14 & 15	-.0376	.0289	-1.30	.196	[-.095, .020]
	Ages 16-18	.0365	.0365	1.46	.147	[-.013, .086]
Depression	Ages 12 & 13	.0163	.0331	0.49	.624	[-.049, .082]
(Wave II)	Ages 14 & 15	.0183	.0147	1.24	.216	[-.011, .047]
	Ages 16-18	.0270	.0115	2.35*	.020	[.004, .050]
Social	Ages 12 & 13	.0042	.0132	0.32	.751	[-.022, .030]
support	Ages 14 & 15	-.0152	.0084	-1.81	.073	[-.032, .001]
(Wave II)	Ages 16-18	.0002	.0084	0.03	.980	[-.016, .017]
Intercept	Ages 12 & 13	20.7420	.5704	36.37[a]	.000	[19.61, 21.87]
(mean)	Ages 14 & 15	22.3865	.4125	54.27[a]	.000	[21.57, 23.20]
	Ages 16-18	22.7537	.3635	62.60[a]	.000	[22.03, 23.47]
Linear slope	Ages 12 & 13	1.0206	.1287	7.93***	.000	[.766, 1.275]
(mean)	Ages 14 & 15	.7043	.0724	9.73***	.000	[.561, .847]
	Ages 16-18	.8686	.0780	11.13***	.000	[.714, 1.023]
Quad slope	Ages 12 & 13	-.0296	.0073	-4.02***	.000	[-.044, -.015]
(mean)	Ages 14 & 15	-.0169	.0042	-3.98***	.000	[-.025, -.008]
	Ages 16-18	-.0299	.0048	-6.28***	.000	[-.039, -.040]

[a]The significance of this *t*-value is not relevant as it signifies a significant difference from 0, which is to be expected as a BMI value (Newsom, 2015).
*$p < .05$. ***$p < .001$.

Table 22

Adjusted Wald Test Results Between and Among the Three Categories of Age for the Male SEM Analysis

Variable	Among age categories ($df = 2, 130$)		Ages 12 & 13/ Ages 14 & 15 ($df = 1, 131$)		Ages 12 & 13/ Ages 16-18 ($df = 1, 131$)		Ages 14 & 15/ Ages 16-18 ($df = 1, 131$)	
	F-ratio	*p-value*	*F*-ratio	*p-value*	*F*-ratio	*p-value*	*F*-ratio	*p-value*
Religiosity (Wave I)	2.14	.122	2.08	.151	0.00	.955	3.82	.053
Depression (Wave II)	0.16	.849	0.00	.959	0.09	.763	0.25	.616
Social support (Wave II)	1.07	.345	1.37	.244	0.06	.803	1.68	.198
Intercept (Mean)	4.42*	.014	5.86*	.017	8.61**	.004	0.47	.496
Linear slope (Mean)	2.85	.062	4.63*	.033	0.99	.322	2.64	.107
Quadratic slope (Mean)	2.71	.070	2.17	.143	0.00	.970	4.77*	.031

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

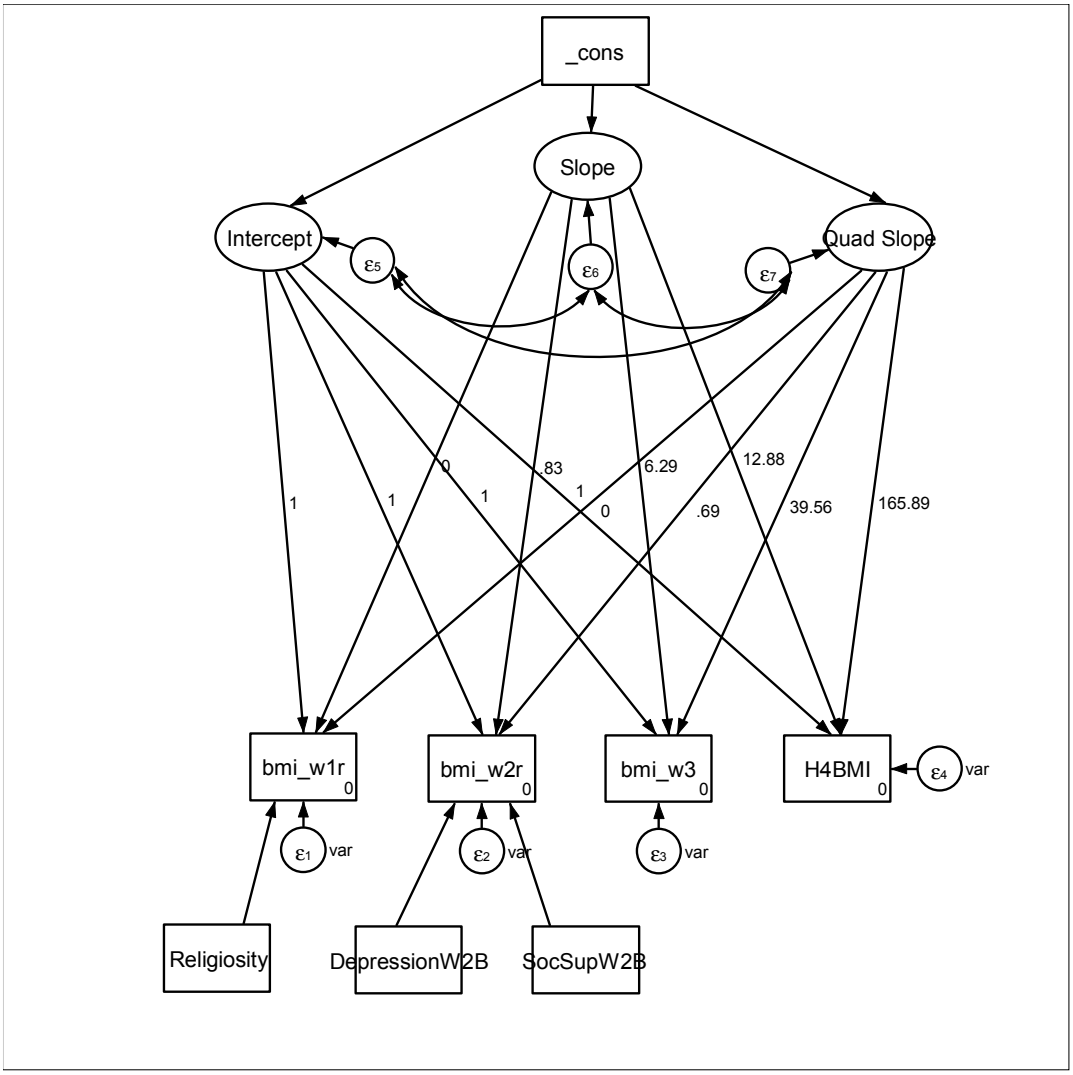


Figure 15. Structural equation model (SEM) yielding the intercepts, linear slope, and quadratic slopes while accounting for Religiosity at the Wave I data collection and Depression and Social Support at the Wave II data collection. This model was run while accounting for Race as a "group" parameter; this model was also used to run the analysis that did not distinguish between the races, but rather accounted for age categories.

Interpretation of the SEM results was based on examples provided by Newsom (2015) in his text regarding longitudinal SEM. Newsom applied a nonlinear latent growth curve model to waves of BMI data from a health and aging data set, and his general applications are used to identify important information in the analysis for this current study. First, it should be noted that the use of Stata for SEM evaluation of complex survey data has limitations. The primary limitation in this case is a dearth of fit analytics that can be applied to survey data. In fact, there is only one fit measure that is allowed following SEM analysis of complex survey data in Stata and that is the absolute fit index known as the standardized root mean square residual (SRMR). For this analysis of males, the SRMR was .074 and was below the cutoff for a good fit of .08 as recommended by Hu and Bentler (1998). Therefore, the one fit index available in analysis of complex survey data using Stata demonstrated that this model was a good fit.

The mean intercept for each age group represents average BMI for males for each age category at Wave I. For ages 12 &13, the average BMI at Wave I was 20.74; the average BMI at Wave I for ages 14 &15 was 22.39; and the average BMI at Wave I for ages 16-18 was 22.75. As noted previously (Table 19), there was a significant difference for Wave I BMI between males aged 12 & 13 compared to males aged 14 & 15 and males aged 16-18. However, Wave I BMI levels show that for each age category, the average male was in the *normal weight* category for BMI.

The linear factor of the SEM analysis, which I have referred to as the linear slope, represents the instantaneous rate of change in BMI related to BMI at Wave I (Newsom, 2015). The linear slope for all three age categories was shown to be positive and to be

significant such that for males aged 12 &13, $t(131) = 7.93$, $p < .000$; for males aged 14 & 15, $t(131) = 9.73$, $p < .000$; and for males ages 16-18, $t(131) = 11.13$, $p < .000$. Standardized linear slope coefficients for each age category were high at 1.35 for ages 12 & 13, .95 for ages 14 &15, and 1.43 for ages 16-18. These coefficients represent large effects for rate of change in BMI for each age group. Furthermore, there was a significant difference in the linear slope for males ages 12 & 13 compared to males ages 14 & 15, $F(1, 133) = 4.64$, $p = .033$, such that males aged 12 &13 experienced a significantly increased rate of change in BMI compared to males aged 14 &15.

The quadratic factor of the SEM analysis, which I have referred to as quadratic slope, was negative for all three age categories, and represented a significant decline in the rate of change in BMI over time. Males aged 12 & 13 and 16-18 experienced similar slowing in their rate of change as demonstrated by coefficients of -.0296, $t(131) = -4.02$, $p < .001$ and -.0299, $t(131) = -6.28$, $p < .000$, respectively. The decline in the rate of change for males aged 14 & 15 was still significant, but less dramatic as demonstrated by the coefficient of -.0169, $t(131) = -3.98$, $p < .001$. Standardized values for the quadratic effect were large at -.5859, -.3361, and -.7089 for age categories of 12 & 13, 14 & 15, and 16-18, respectively. Standardized values indicated a large effect for ages 12 & 13 and 16-18 and a medium effect for ages 14 & 15 for the quadratic factor.

Finally, correlations between the linear factor and quadratic factor are of interest in nonlinear latent growth curve models. Correlations are derived from the standardized values of the covariances between the linear slope and quadratic slope. For this analysis, the correlations between the linear and quadratic factors were: -.90 for ages 12 & 13; -.92

for ages 14 & 15; and -.87 for ages 16-18. The results of this SEM analysis resembled the general outcomes presented by Newsom (2015) in his example evaluation of changes in BMI for adults aged 50-70.

For each of the age groups, average predicted values were determined using the formula $\hat{y}_t = \alpha_0 + \alpha_1\lambda_{t1} + \alpha_2\lambda_{t2}$ and were plotted to create the graph in Figure 16. There was a clear distinction between AgeCat1 and AgeCat3 at Wave I. Despite this difference, the predicted values indicated that individuals in AgeCat1 would attain a greater BMI at Wave IV than those in AgeCat3. All three age categories demonstrate a leveling off of the rate of BMI increase by Wave IV, with AgeCat3 participants finishing at a lower final BMI level.

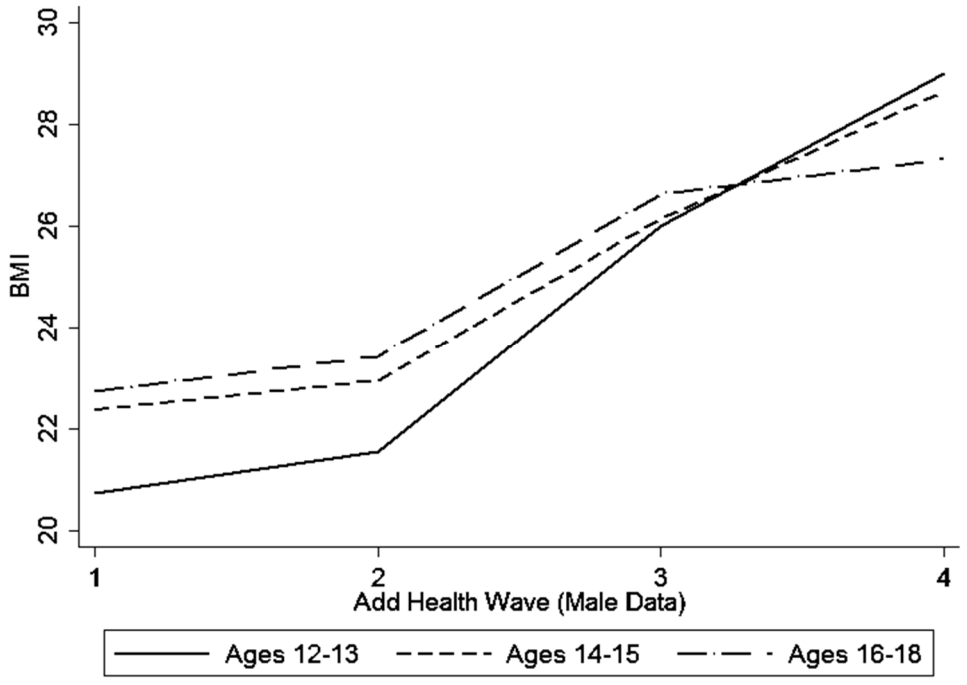


Figure 16. Predicted plots based on values from the nonlinear growth curve model of the change in BMI from adolescence to adulthood for males.

Changes in Obesity Over Time for Females Accounting for Religiosity, Depression, and Social Support in Adolescence

For this analysis, BMI scores for Wave II and Wave III were included. BMI scores at Wave III were based on self-reported height and weight, but height and weight were also measured by the interviewer. Seven of the female respondents exceeded the 330 pound weight limit of the scale being used for measurement. Therefore, BMI was based on 331 pounds for those who reported a weight less than 330 but who weighed more than 330 pounds according to the scale. Two women self-reported weights above 330 pounds and their BMI was based on these reports. One female's weight at Wave III was changed to missing because her weight exceeded the scale's threshold of 330 pounds and she did not provide a reported weight.

In the regression analysis used to answer RQ1 for females, Religiosity, Depression, and Social Support in adolescence were not significant individual contributors to the prediction of adult Obesity. However, together, these three independent variables added to the strength of the prediction of adult obesity. In particular, Social Support at Wave II was just beyond the .05 level of significance for the prediction of adult obesity. Unlike for the linear regression model for males, race/ethnicity in the female model was not a significant predictor of adult obesity; therefore, I did not run an SEM model for women in which I accounted for race/ethnicity. Rather, the SEM analysis for women accounted for religiosity, depression, and social support in relation to the BMI data collected at the same time (Figure 15) and accounting for age by using the *group(AgeCat)* command described in the analysis for men. The

AgeCat variable for women was similarly constructed so that AgeCat1 represented females ages 12 and 13, AgeCat2 represented females ages 14 and 15, and AgeCat3 represented females ages 16-18. Unstandardized values from the SEM analysis for females while accounting for age are presented in Table 23. For AgeCat1 (ages 12 and 13), $n = 253$; for AgeCat2 (ages 14 and 15), $n = 430$; and for AgeCat3 (ages 16-18), $n = 342$.

Table 23

Report of Regression Analytics for the SEM Analysis of Females Accounting for the Differences in Age Based on Age Categories

Variable	Age Group	Coef	SE	*t*-value	*p*-level	CI-95%
Religiosity	Ages 12 & 13	-.0005	.0399	-0.01	.989	[-.080, .078]
(Wave I)	Ages 14 & 15	.0028	.0344	0.08	.936	[-.065, .071]
	Ages 16-18	-.0822	.0323	-2.54*	.012	[-.146, -.018]
Depression	Ages 12 & 13	.0187	.0190	0.98	.327	[-.019, .056]
(Wave II)	Ages 14 & 15	-.0032	.0132	-0.24	.810	[-.029, .023]
	Ages 16-18	-.0200	.0153	-1.31	.194	[-.050, .010]
Social support	Ages 12 & 13	-.0002	.0127	-0.01	.990	[-.025, .025]
(Wave II)	Ages 14 & 15	.0038	.0127	0.30	.763	[-.021, .029]
	Ages 16-18	-.0160	.0128	-1.25	.213	[-.041, .009]
Intercept	Ages 12 & 13	20.6806	.6239	33.15[a]	.000	[19.45, 21.91]
(mean)	Ages 14 & 15	22.0015	.5023	43.80[a]	.000	[21.01, 23.00]
	Ages 16-18	23.5644	.5254	44.85[a]	.000	[22.52, 24.60]
Linear slope	Ages 12 & 13	.9044	.1573	5.75***	.000	[.593, 1.22]
(mean)	Ages 14 & 15	.8646	.1386	6.24***	.000	[.590, 1.14]
	Ages 16-18	.5810	.1250	4.65***	.000	[.334, .828]
Quad slope	Ages 12 & 13	-.0312	.0098	-3.19**	.002	[-.051, -.012]
(mean)	Ages 14 & 15	-.0284	.0075	-3.79***	.000	[-.043, -.014]
	Ages 16-18	-.0133	.0070	-1.91	.058	[-.027, .000]

[a]The significance of this *t*-value is not relevant as it signifies a significant difference from 0, which is to be expected as a BMI value (Newsom, 2015).
*$p < .05$. **$p < .01$. ***$p < .001$.

Further analysis of the coefficients for religiosity, depression, social support, mean intercept, mean slope, and mean quadratic slope was completed to determine differences among and between age categories for women. These results are detailed in Table 24. Mean intercept for the three age categories was the only variable demonstrating a significant difference. There was a significant difference among the three age categories for mean intercept, $F(2, 130) = 6.62$, $p = .002$. Specifically, AgeCat1 was significantly different from AgeCat3, $F(1, 131) = 12.54$, $p < .001$, and AgeCat2 was significantly different from AgeCat3, $F(1, 131) = 4.74$, $p = .031$. In both cases the mean intercept for AgeCat3 was significantly greater than the mean intercepts for AgeCat1 and AgeCat2.

Table 24

Adjusted Wald Test Results Between and Among the Three Categories of Age for the Female SEM Analysis

Variable	Among age categories (*df* = 2, 130)		Ages 12 & 13/ Ages 14 & 15 (*df* = 1, 131)		Ages 12 & 13/ Ages 16-18 (*df*= 1, 131)		Ages 14 & 15/ Ages 16-18 (*df* = 1, 131)	
	F-ratio	*p-value*	*F*-ratio	*p-value*	*F*-ratio	*p-value*	*F*-ratio	*p-value*
Religiosity (Wave I)	2.11	.125	0.00	.952	2.54	.113	3.32	.071
Depression (Wave II)	1.25	.291	0.86	.355	2.49	.117	0.71	.403
Social support (Wave II)	0.71	.495	0.04	.836	0.77	.382	1.23	.270
Intercept (Mean)	6.62**	.002	2.38	.125	12.54***	.001	4.74*	.031
Linear slope (Mean)	1.86	.160	0.03	.856	2.60	.109	2.46	.119
Quadratic slope (Mean)	1.69	.189	0.05	.828	2.23	.138	2.20	.141

*$p < .05$. **$p < .01$. ***$p < .001$.

The interpretation of the SEM analysis for females was also based on the example provided by Newsom (2015). First, the SRMR for the SEM model for women yielded a

value of .055. This value was below the cutoff of .08 recommended by Hu and Bentler (1998) and, therefore, indicated a good fit for this model.

The mean intercept in the SEM analysis represents the average BMI at Wave I for each of the age groups. The average BMI for AgeCat1 (ages 12 & 13) at Wave I was 20.68; the average BMI for AgeCat2 (ages 14 & 15) at Wave I was 22.00, and the average BMI for AgeCat3 (ages 16-18) at Wave I was 23.56. Although there was a significant difference between these values for each age category compared to AgeCat3 (as explained previously), the average BMI at Wave I for each age category is identified as a value for *normal weight*.

Linear slope, in a nonlinear growth curve model, represents the instantaneous change in BMI related to BMI at baseline (Newsom, 2015). Linear slope for all three age categories was positive and significantly different from zero (Table 20). AgeCat1 demonstrated the highest rate of change in BMI with a linear slope coefficient of .904, $t(131) = 5.75$, $p < .001$. The next highest rate of change was seen in AgeCat2 with a linear slope coefficient of .865, $t(131) = 6.24$, $p < .001$. The smallest rate of change was exhibited by AgeCat3 with a linear slope coefficient of .581, $t(131) = 4.65$, $p < .000$. Standardized coefficients for linear slopes indicated large effects as seen by values of 1.422, .922, and .661 for AgeCat1, AgeCat2, and AgeCat3, respectively.

The quadratic slope in a nonlinear growth curve model represents an increase or decrease in the rate of change in BMI over time (Newsom, 2015). For all three age categories, quadratic slope was negative indicating a decrease in the rate of change in BMI. As with linear slope, AgeCat1 demonstrated a more exaggerated decrease in the

rate of change than did the other two age categories. The coefficient for quadratic slope for AgeCat1 was -.031, $t(131) = -3.19$, $p = .002$. The quadratic slope coefficient for AgeCat2 was -.028, $t(131) = -3.79$, $p < .000$. The quadratic slope coefficient for AgeCat3 was not significant at -.013, $t(131) = -1.91$, $p = .058$. Standardized coefficients for each age category were -.636, -.431, and -.216 for AgeCat1, AgeCat2, and AgeCat3, respectively. The standardized coefficient for AgeCat1 was large and significant, $t(131) = -3.20$, $p = .002$. The standardized coefficient for AgeCat2 was medium and significant, $t(131) = -3.86$, $p < .000$. The standardized coefficient for AgeCat3 was small and nonsignificant, $t(131) = -1.83$, $p = .069$.

Finally, correlations derived from the standardization of the covariances between linear slope and quadratic slope were strong. The correlations between the linear and quadratic factors were as follows: -.85 for AgeCat1, -.90 for AgeCat2, and -.89 for AgeCat3.

For each of the age groups for female data, average predicted values were determined using the formula $\hat{y}_t = \alpha_0 + \alpha_1\lambda_{t1} + \alpha_2\lambda_{t2}$ and were plotted to create the graph in Figure 17. The graphs for females demonstrated a much more consistent development of BMI from adolescence through adulthood for each age category than was demonstrated with the male data. AgeCat3 females had a higher BMI at baseline than did females in the other two age categories and paths between the age categories did not cross throughout the prediction timeframe. This resulted in females in AgeCat3 ending with the highest BMI at Wave IV and females in AgeCat1 ending with the lowest BMI at Wave IV.

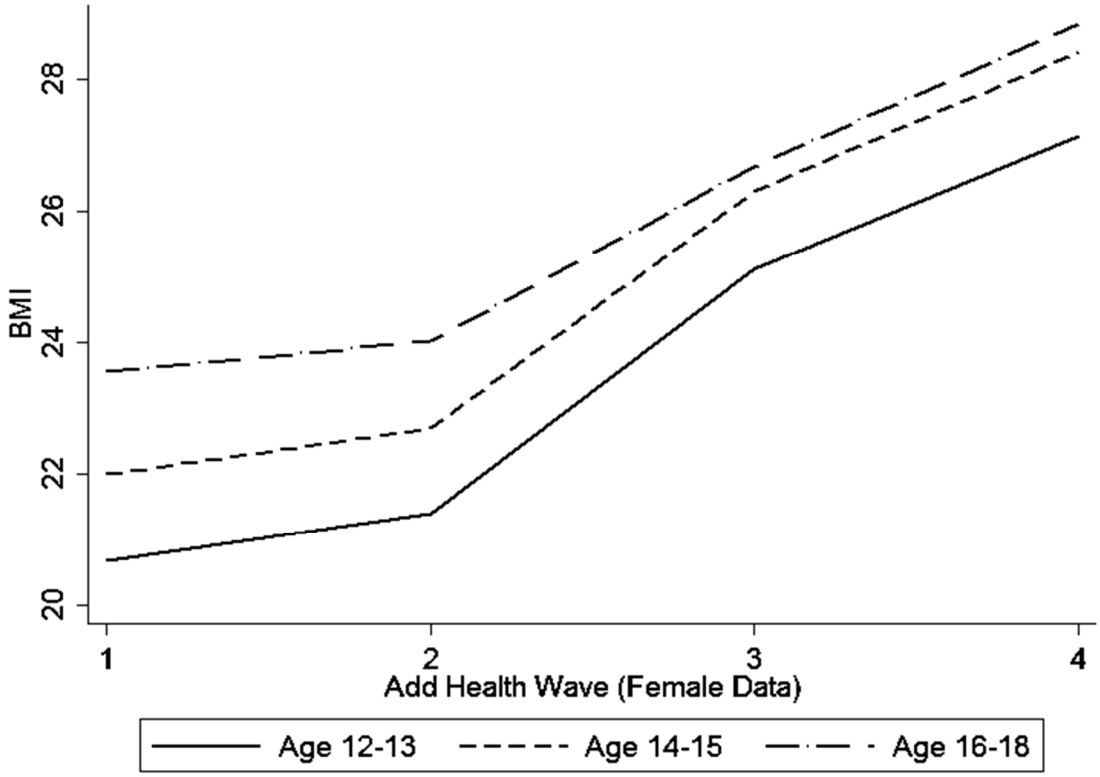


Figure 17. Predicted plots based on values from the nonlinear growth curve model of the change in BMI from adolescence to adulthood for females.

Summary

The results of multiple regression analysis indicate that adolescent religiosity contributed significantly to the prediction of adult obesity for males, but not for females. Furthermore, adolescent religiosity was positively and significantly related to adult obesity for males, but negatively related to adult obesity for females; however, the female religiosity-obesity relationship was not statistically significant. Depression at Wave II was positively related to adult male obesity but negatively related to adult female obesity (neither of the relationships were statistically significant). Social Support at Wave II was negatively related to adult male obesity but positively related to adult female obesity

(neither of the relationships were statistically significant). Therefore, adult male obesity was found to be greater for male adolescents with higher religiosity scores, higher depression scores, and lower social support scores. Conversely, female obesity was found to be greater for female adolescents with lower religiosity scores, lower depression scores, and higher social support scores. The inclusion of adolescent religiosity, depression, and social support enhanced the significance of the regression analysis for both males and females in predicting adult obesity. Among the independent variables, religiosity was negatively associated with later depression (but only significant for the female relationship) and religiosity was positively associated with later social support (significant for both sexes).

Adolescent male religiosity was significantly associated with adult male obesity; however, neither adolescent depression nor adolescent social support mediated the religiosity-obesity relationship. The direct relationship between adolescent female religiosity and adult female obesity was not statistically significant. For females, neither adolescent depression nor adolescent social support was considered for mediational effects.

Finally, SEM analysis indicated that the pattern of the development of obesity was consistent for males for each race/ethnicity category. Based on age categories, males tracked differently in their development of obesity. While all age categories demonstrated a leveling off of increase in the rate of BMI change by Wave IV, rates of change slowed more dramatically for the older adolescent males than for the younger adolescent males. Females racial differences were not indicated based on the regression analysis, but,

similar to the male data, females demonstrated a leveling off of the increase in the rate of change in BMI by Wave IV. Rates of change slowed more dramatically for the younger adolescent females than for the older adolescent females; however, females demonstrated more consistent obesity development in relationship to members of other age groups as compared to males.

Interpretations for these findings are discussed in Chapter 5. Specific demographic data is identified to be representative of adolescents in 1994-1995. Correlations between religiosity, depression, social support, and obesity are compared to findings from previous research and the primary findings from the regression analysis of adolescent religiosity, depression, and social support in the prediction of adult obesity are discussed in relation to prevention and treatment needs based on the sex of the developing adolescent.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

The purpose of this quantitative study was to identify the effect of adolescent religiosity in the longitudinal development of obesity and to do so while incorporating a multidimensional health model in which specific measures of spiritual, emotional, social, and physical health were considered. Furthermore, adolescent depression and adolescent social support were tested as mediational variables in the adolescent religiosity–adult obesity relationship. The relationships between religiosity and obesity, religiosity and depression, religiosity and social support, depression and obesity, and social support and obesity are also addressed for the adolescent population.

Child and adolescent obesity, as it relates to BMI, is based on the sex and age of the individual. Age and sex, therefore, needed to be accounted for in the statistical analyses related to this study. For these reasons, males and females were analyzed separately, and age was included as a covariate. For males, adolescent religiosity was shown to be a significant predictor of adult obesity in the model accounting for demographic and control data (Model 3). While adolescent depression and social support were not statistically significant predictors of adult obesity, they strengthened the model for predicting adult obesity as identified in the increase in R^2 for the full model. Specifically, increased level of religiosity in adolescence was statistically associated with a significant increase in obesity as an adult when accounting for depression, social support, and demographic variables. For females, adolescent religiosity, depression, and social support were not significant predictors of adult obesity (although, in Model 3 for

females, social support was just beyond the level of significance). However, when religiosity, depression, and social support were added to the demographic data, the prediction of adult obesity (as demonstrated by an increased R^2) was enhanced. For these reasons, the null hypothesis for RQ1 for both sexes was not retained.

Research Questions 2 and 3 (RQ2 and RQ3) were related to the roles of depression and of social support as mediators of the religiosity–obesity relationship. For males, adolescent religiosity was statistically significantly related to adult obesity. However, depression lacked a significant relationship with adolescent religiosity and with adult obesity, thereby nullifying the potential mediation by depression of the religiosity–obesity relationship. Social support for adolescent males demonstrated a significant relationship with religiosity, but not with obesity. Among females, the specific path from adolescent religiosity to adult obesity was not statistically significant; therefore, neither depression nor social support could be considered for mediational properties. The null hypothesis for RQ2 and for RQ3 for both sexes was retained.

As an extension to the research questions that were presented, a basic SEM analysis was included to identify the changes in obesity over time as related to adolescent religiosity, depression, and social support and accounting for four waves of collection for BMI. This analysis did not account for race/ethnicity but did account for the difference in age at Wave I using age categories to include ages 12 and 13, 14 and 15, and 16 through 18 separately. For males, all age groups exhibited significant positive linear slopes and significant negative quadratic slopes. The youngest age category was shown to exhibit the greatest rate of change for BMI while starting at a lower mean BMI at Wave I. The

projected BMI at Wave IV eclipsed the other two age categories despite starting at a lower value at baseline. For females, all three age groups demonstrated statistically significant increases in linear slope, with the youngest females exhibiting the highest rate of change in BMI and the oldest age group exhibiting the lowest rate of change in BMI of the three groups. Each age group demonstrated negative quadratic slopes, but the quadratic slope for the oldest age group was not statistically significant. Predicted changes in female BMI did not result in overlap with adjacent age groups.

Interpretation of the Findings

Interpretations for the findings of this study are organized such that a review of general demographic differences for religiosity, depression, social support, and obesity are addressed first in support or nonsupport of previous studies. Subsequently, interpretation of the findings related specifically to the relationships between religiosity, depression, and social support in the development of obesity is presented. Various covariates are discussed for any connections made to previous research. Finally, the theoretical foundation presented by Hawks (2004) is discussed and interpreted in light of the findings.

Demographic Findings Related to Religiosity, Depression, Social Support, and Obesity

The demographic makeup (refer to Table 5) of the respondents in the public-use data of the Add Health (Harris & Udry, 2014) study should be considered to be representative of students in Grades 7-12 in the United States in 1994-1995. Because the population of interest was chosen from the Wave IV public-use data, the *GSWGT4_2*

weight variable was applied to all analyses in this study, thereby representing adolescents in Grades 7-12 in the United States in 1994-1995. Males made up 50.5% of the population while females comprised 49.5%. The proportion of students in Grades 7-12 in the United States in the 1994-1995 school year who were categorized as White was 70%; African American students were the next largest racial/ethnic category at 15.4%; Hispanic students were the next highest at 11.1%; and the remaining 3.5% of the population was represented by the Other race/ethnicity category. The sex and racial breakdowns do not match exactly with specific percentages associated with other Add Health studies. Dunn, Milliren, Evans, Subramanian, and Richmond (2015) reported that in Wave I, 51% of participants were female and 58% were from the White population. The disparities in sex and age may reflect the difference in the primary wave of data used for the analyses as well as the use of the full data set versus the public-use data set. Most likely, choice of variables for each study limited the final participant pool used for analysis in each study, leading to variety in the overall demographics represented. Approximately 30% of students in Grades 7-12 in the United States in 1994-1995 were smokers as defined by having smoked at least one cigarette in the previous month.

Males at Wave I exhibited a significantly higher BMI compared to females, with average weighted BMIs recorded as 22.7 and 22.3, respectively. Due to the fact that BMI for children and adolescents is based on sex and age, mean values for adolescent BMI are not often provided. However, Ogden et al. (2012) reported that the mean value for BMI for boys ages 12-19 was 22.9 and for girls was 23.3 based on a 1999-2000 study. The values from the current study appear to be on par with these values presented by Odgen et

al. It should be noted that the Ogden et al. values were based on an unweighted sample, which may speak to the difference between the samples.

While specific reports of mean adolescent BMI are not common, reports of the incidence of overweight/obese categories are common. When accounting for age, 14.9% of adolescent females at Wave I were classified as *overweight* and 9.8% were classified as *obese* based on BMI. For males, 17.8% of the adolescents at Wave I were classified as *overweight* and 13.6% were classified as *obese* based on BMI. These percentages are lower than those recorded for adolescents age 12-19 in the Ogden et al. (2002) study. Ogden et al. reported U.S. adolescent male and female rates of overweight to be 15.5%. The definition of *overweight* for adolescents at the time was BMI greater than the 95th percentile based on age and sex and is equivalent to the current definition of *obesity* for adolescents. Therefore, female rates of 9.8% and male rates of 13.6% in the current study are lower than reported by Ogden et al. However, the current study was based on BMI in 1994-1995, and the Ogden study was for 1999-2000. Ogden et al. reported an increase in the rate of overweight among adolescents of about 5% from the 1988-1994 National Health and Nutrition Examination Survey to their follow-up in 1999-2000. Therefore, Wave I obesity rates in the current study are within the expected range. With these differences in mind, each of the primary independent variables (religiosity, depression, and social support) and the dependent variable (adult obesity) is discussed based on demographic findings.

Demographic differences in religiosity. Adolescent males (ages 12-18 at Wave I) in the current study reported significantly lower religiosity scores than did females

(ages 12-18 at Wave I), $t(131) = -5.76$, $p < .001$. This finding is in agreement with a variety of studies that found females to exhibit higher levels of religiosity compared to males (Eliassen et al., 2005; Reeves et al., 2012). Another demographic comparison related to religiosity is for race/ethnicity. The adolescent male and female White population demonstrated lower scores on Religiosity than did the Hispanic, African American, and Other populations; the White population was significantly lower in Religiosity score than the African American population, $t(131) = -9.25$, $p < .000$, and the Other population, $t(131) = -2.03$, $p = .045$. The adolescent African American population also demonstrated significantly higher Religiosity scores than the Hispanic population, $t(131) = 5.36$, $p < .000$, and the Other population, $t(131) = 2.96$, $p = .004$. Brown et al. (2007) also reported significantly lower levels of religiosity (specifically related to prayer fulfillment and intrinsic religiosity) for the Caucasian college population compared to the other races/ethnicities.

Demographic differences in depression. Depression symptomology scores for adolescent males were significantly lower than for adolescent females in this study, $t(131) = -8.97$, $p < .000$. This finding is consistent with other studies of adolescents, including a study of Wave I data of the Add Health study (Dunn, Milliren, Evans, Subramanian, & Richmond, 2015), a meta-analysis of depression for 30 years of child/adolescent data (Costello, Erkanli, & Angold, 2006), a study of White adolescents from Iowa (Ge, Conger, & Elder, 2001), a study of adolescents from the Caribbean (Pilgrim & Blum, 2012), and a study of Chinese adolescents (Xie et al., 2005), and it is consistent with adult differences in depression (Reeves et al., 2012). In terms of

depression related to race/ethnicity, White adolescents demonstrated consistently lower rates of depression symptomology than all other race categories. Compared to White adolescents, Hispanic adolescents had significantly higher depression symptomology scores, $t(131) = 5.19$, $p < .000$; African American adolescents had significantly higher depression symptomology scores, $t(131) = 4.72$, $p < .000$; and, Other adolescents had significantly higher depression symptomology scores, $t(131) = 2.17$, $p = .032$. The findings support the general findings provided by Dunn et al. (2015), who reported that Black and Hispanic students in Wave I of the Add Health study demonstrated higher scores for depression symptoms. Respress, Morris, Gary, Lewin, and Francis (2013) also reported greater depression symptom scores for the Black and Other racial categories compared to White respondents based on the Wave II data of the Add Health study. While it is tempting to assume that the difference in depression among the races is due to feelings of discrimination, Respress et al. (2013) found that feelings of peer and teacher discrimination significantly influenced depression among White respondents but not Black respondents. In fact, for Black respondents, factors of parental education and poverty were significant influences on depression levels. This relationship is supported by data from the current study related to family income, which can be reflective of parental education as well, in which the White population had a significantly higher family income than both the Hispanic and African American populations (see Table 5).

Demographic differences in social support. Social support among adolescents showed fewer demographic differences than the previous two independent variables in this study. The only finding that held statistical significance was the difference between

levels of perceived social support for adolescent males versus adolescent females. Adolescent males perceived significantly lower levels of social support from teachers, family, and friends than did adolescent females, $t(131) = -2.30$, $p = .023$. The general finding of a higher level of social support for females than for males supports the finding of Brown et al. (2007), who reported that college females experience a significantly higher level of social support compared to college males. The gender difference among adolescents was not reported to be true for African American adults in the Reeves et al. (2012) study, but the difference in the scales used to measure social support and the demographic differences in the population are likely contributors to this difference. The current study does not align with the findings of Xie et al. (2005), who found that Chinese boys had significantly greater perceived social support than did Chinese girls. The reasons for the difference may be societal differences between the United States and China or differences in how social support was measured. For the Xie et al. study, adolescents were asked to list individuals who had provided specific types of social support, with the final score being the total number of individuals listed, while in the current study, social support included identification on a Likert scale for how much adolescents felt supported by parents, teachers, and peers. In the study of Chinese adolescents, value was placed on the number of supporters rather than on the quality (or perceived quality) of the support.

The adolescent population of African American males and females in the current study demonstrated lower levels of perceived social support than did any of the other three racial groups; however, none of these relationships were statistically significant.

Demographic differences in obesity. Adult obesity at Wave IV was not significantly different between males and females, with the mean for males at 28.96 and the mean for females at 29.20. Flegal et al (2012) reported similar values of 28.7 mean BMI scores for both males and females (when values were age adjusted). Adult BMI measures in the current study do not match fully with BMI measures for African American adults in the Reeves et al. (2012) study, as Reeves et al. reported a significant difference between men and women for BMI. However, the reported BMI means in the Reeves et al. study were higher than those reported in the current study for both men and women. The current study found the African American population to have significantly higher BMI scores than the White and Other populations; therefore, it is appropriate that the BMI scores in the African American-based study by Reeves demonstrated higher mean BMI than the overall population in the current study.

The percentage of females who were either overweight or obese at Wave IV was 63.2%; for males, this percentage was 71.4% (see Table 9). Flegal et al. reported lower rates of obesity in their report on obesity trends; Flegal et al. reported an overweight and obesity rate of 55.8% for women ages 20-39 (a similar age range for the participants in the current study at Wave IV) and 67.1% for men in the same age group. However, age-adjusted rates of overweight and obesity in the Flegal et al. study are much closer to the current percentages at 63.7% for women and 73.9% for men. In the current study, the Hispanic adult population and the African American adult population each demonstrated significantly higher levels of BMI than both the White and Other populations, with the African American population demonstrating the highest overall level of BMI compared

to all of the racial groups. This finding is consistent with the general findings of Flegal et al. who identified that the non-Hispanic Black population had the highest percentage of individuals in the obese category, followed by the Hispanic population and then the White population.

For females who had BMI information at Waves I and IV, 38.6% were categorized as obese based on BMI at Wave IV; for men, 35.7% were categorized as obese at Wave IV. These values exceed those reported by Ogden et al. (2012b), who reported female obesity rates of 31.9% and male obesity rates of 33.2% for those aged 20-39. Furthermore, in the current study, there was a 9.7% increase in the proportion of females who were overweight at Wave IV compared to those who were overweight at Wave I and a 27.4% increase in the proportion of those who were obese at Wave IV compared to those who were obese at Wave I. Males demonstrated increases in both proportions as well—a 17.8% increase in proportion for overweight and a 22.3% increase in proportion for obesity. The changes in obesity are consistent with evidence presented by other researchers. Juonala (2011) reported an increase from normal weight to obesity for 14.6% of participants in the four studies reviewed; Gordon-Larsen et al. (2010) detailed the doubling of the number of obese participants in their study of individuals transitioning from adolescence to early adulthood. In the current study, the increase in obesity is viewed a bit differently, but the result is the same. Over the approximately 12 years of the waves of data in the current study, 51.1% of female participants moved up in obesity category by at least one level (e.g., from normal weight to overweight). Furthermore, 55.3% of males moved up in obesity category by at least one level.

The Relationship Between Religiosity, Depression, and Social Support in the Development of Obesity

The findings of the relationship between religiosity, depression, social support, and obesity can be considered in a number of ways. The uniqueness of the current study was the multi-dimensional approach to including all of these facets of wellness into one substantive evaluation. However, much of the previous research has considered single connections between religiosity and obesity, depression and obesity, and social support and obesity. The multi-dimensional connection is discussed first.

Religiosity, depression, social support, and obesity. The role of religiosity in the development of depression and social support and subsequent obesity varies by sex. Adult obesity among males can be said to be greater when adolescent religiosity increases, adolescent depression increases, and adolescent social support decreases; conversely, adult obesity among females can be said to be greater when adolescent religiosity decreases, adolescent depression decreases, and adolescent social support increases. In order to better understand the relationship that occurs between these variables, further understanding of individual correlations is needed.

The relationship of the adolescent measures was informed by the correlations between independent variables. For males, adolescent religiosity was negatively correlated with every other independent variable except for social support, which was positively and significantly correlated (Table 12). Among females, adolescent religiosity was positively correlated with social support. Similar to males, females demonstrated a negative correlation between religiosity and depression and a positive correlation

between religiosity and social support; however, for females, both of these correlations were statistically significant.

Correlations of the independent variables in this study support the findings of other researchers. Pössel et al. (2011) and Pilgrim and Blum (2012) reported that religiosity was significantly correlated with reduced levels of depression among ninth grade students and English-speaking Caribbean adolescents, respectively. Both studies differ from the current study in that the current study found a significant correlation between religiosity and depression only for females. The current study did not confirm a significant correlation between religiosity and depression for males, however, the significance for the stated correlation for males in the current study was $p = .077$, which is still a fairly strong correlation. Therefore, the current study is in line with previous research.

In the current study, social support and religiosity were significantly and positively correlated for males and females. This finding is only partially confirmatory of the Moxey et al. (2011) study in which the older Australian adult female population demonstrated a significant relationship between measures of religiosity and increased social support. The Moxey et al. study was of a cross-sectional nature and included a much older, foreign population compared to the population used in the current study—thus, the potential reason for the differences observed.

In studies for which religiosity, depression, and social support were considered, results from the current study remain supportive of general findings by other researchers. Schnall et al. (2012), in their cross-sectional study of postmenopausal women, identified

that religious service attendance significantly reduced levels of depression symptomology and that religious service attendance led to higher levels of various social support measures—matching the current findings. Furthermore, the findings of Thomas and Washington (2012) related to these variables is partially supported by the current study. Thomas and Washington incorporated religiosity and social support in understanding mental and physical components of health-related quality of life among African American hemodialysis patients. Thomas and Washington found that religiosity provided a negative prediction for physical health-related quality of life and that social support provided a positive prediction for physical health-related quality of life. In my study, increased religiosity significantly predicted a negative health outcome of increased BMI for males, thus matching the religiosity findings of Thomas and Washington; however, my regression analyses did not present significant findings for social support related to the outcome as Thomas and Washington reported.

To my knowledge, there was only one other study that has considered the four main variables chosen for the current study and viewed in a similar role for the development of obesity. In the Reeves et al. (2012) study, African American adults were reported to exhibit decreases in depression and increases in social support in connection to increases in religious activities such as attendance and prayer. The results of the current study supported these general findings for males and females but did so for multiple races. However, Reeves et al. found no connection between religiosity (and spirituality) and BMI for African Americans. The current study demonstrated a significant relationship between adolescent religiosity and adult obesity for males, but not

for females, that applied to all racial groups. Regression analysis in the Reeves et al. study demonstrated social support to decrease BMI and depression to increase BMI; the current study demonstrated a difference in these relationships based on sex. Therefore, the findings of the current study do not support the findings of Reeves et al. related to social support and depression. The current study used a longitudinal view of the relationship, while Reeves et al. related religiosity and weight cross-sectionally and only for the central Mississippi, African American population. While health dimension relationships of this type are not apparent in cross-sectional data, there does appear to be a more definitive relationship that can be identified from longitudinal data.

The religiosity–obesity relationship. In the current study, adolescent religiosity was shown to be a statistically significant predictor of adult obesity among males, but not among females. For males, increased religiosity at baseline contributed significantly to increase BMI in adulthood. For females, religiosity at baseline was negatively related to adult BMI but not at a significant level. Neither adolescent depression nor social support were shown to be a mediator of the religiosity-obesity relationship. The findings of the current study match with general findings of some recent research yet conflict with others.

Religiosity was measured as church attendance by Koenig and Vaillant (2009) in their longitudinal study of health factors for men. Koenig and Vaillant found that church attendance at age 47 was related directly to objective and subjective health at age 70 while church attendance at age 14 was not. These results do not match with the results of the current study, but religiosity and physical health were measured differently between

the two studies. Furthermore, Koenig and Vaillant identified *mood* to be a possible mediator of the relationship between church attendance and health. In the current study, depression was not shown to mediate the religiosity-obesity relationship; this difference is not surprising given that depression and mood are not equal measures and that obesity is a much more targeted measure than is objective or subjective health. Results from the Dodor (2012) study of African American adults and the Feinstein et al. (2010) study of a multi-ethnic, older population are partially supported by the current study. Dodor found a cross-sectional relationship to exist for religiosity and increased BMI, which was supported for the male analysis in the current study. Similarly, Feinstein et al. detailed increased odds for obesity development for various measures of religiosity. Where the current study differs from these two studies is in the scope of this relationship. Dodor and Feinstein et al. found the relationship to hold for males and females, but the current study only reported a significant relationship for males. The Dodor examination was limited to the African American population; however, because men and women were analyzed separately in the current study, the direct comparison between the genders for each race/ethnicity could not be generated. The Feinstein et al. study differed immensely from the current study for average age of participants and was based on cross-sectional analysis.

Another factor that may have affected the relationships being considered in the current study was the distinction that could be made between intrinsic and extrinsic religiosity. Brown et al. (2007) and Piruntisnky et al (2011) distinguished between intrinsic and extrinsic religiosity, which impacted the results of their studies. In the

current study, religiosity was identified using one continuous scale in which higher religiosity was demonstrated by a higher religiosity score derived from both facets of intrinsic and extrinsic religiosity. I did not make such a distinction in the current study because the Wave I participants of the Add Health study were shown to have high correlation between intrinsic and extrinsic religiosity scores (Le et al., 2007). In fact, in the current study, for those individuals retained in the regression analysis, the correlation between intrinsic and extrinsic religiosity scores was $r = .50$, $p < .001$. For a data set in which distinction could be made between the constructs of intrinsic and extrinsic religiosity, considering these two forms of religiosity separately might prove to be valuable.

The depression–obesity relationship. The depression-obesity relationship in the current study was viewed in terms of the longitudinal relationship between Depression at Wave II and BMI at Wave IV. This relationship was expressed by multiple regression analysis; for both males and females, Depression at Wave II did not significantly contribute to the prediction of BMI at Wave IV. In fact, Depression at Wave II was a positive contributor to BMI at Wave IV for males but a negative contributor to BMI at Wave IV for females. These findings are in accordance with Tanofsky-Kraft et al. (2006) who also found that depressive symptoms in childhood were not significant longitudinal predictors of increased fat mass. The timeframe of the Tanofsky-Kraft et al. study was shorter than the current study (average of 4.2 years compared to approximately 12.9 years for the current study) and did not use the same measure for obesity (dual energy x-ray absorptiometry versus BMI). Despite these differences, depression symptomology was

not longitudinally related to an increase in obesity in either study. Findings of the current study partially support the findings of Fowler-Brown et al. (2012) who determined that among young adults (mean age of 31 years) and with the exception of Hispanic women, symptoms of depression at baseline were unrelated to changes in obesity two years later. My findings match with the Fowler-Brown et al. findings with the exception that Hispanic females were not differentiated for this relationship in my study. The current study also partially supports the findings of the meta-analysis presented by Luppino et al. (2010). Luppino et al. reported that depression was a significant predictor of obesity but not for overweight. The current study supports the lack of a relationship with depression and overweight, but does not align with the Luppino finding of the relationship between depression and obesity. In the current study, I considered BMI as a continuous variable whereas the Luppino et al. report dealt with BMI as a dichotomous variable; this difference in the use of BMI is likely the reason for the different findings.

Results of the current study do not match with the findings of Wiltink et al. (2013) who determined that total depression score was related to BMI in men and women. Wiltink et al. specifically identified that somatic-affective aspects of depression symptomology (i.e., sleep, fatigue, appetite) demonstrated a strong, positive relationship with BMI while the cognitive-affective symptoms of depression (i.e., mood, feelings, concentration, suicide ideation) were not related to BMI. The differences between the current study and the Wiltink et al. study are likely due to the variability in handling depression scores (total score versus comparing the somatic- and cognitive-affective aspects) between the two studies as well as the fact that the Wiltink et al. study was a

cross-sectional study and was conducted on men and women rather than on adolescents. Pan et al. (2012) also reported a positive relationship between depression and obesity and did so using longitudinal data. However, depression in the Pan et al. study was identified as the use of antidepressant medications and depression as diagnosed by a physician and yielded a categorization of depressed/not depressed. In the current study, depression was viewed in terms of a self-reported scale of symptomology rather than categorization of depression. This may explain the different outcomes for the two studies. Furthermore, the Pan et al. study was conducted on women from the Nurses' Health Study with ages ranging from 54-79. The participants in the two studies represented distinctly different populations.

Depression has been shown to be a volatile predictor of future obesity for adolescent females in the current study as well as in at least one other study. When depression at Wave II was used alongside social support and religiosity to predict adult obesity, linear regression analysis of females indicated depression to be a significant predictor of adult obesity. However, when the full range of covariates was included in the analysis, depression at Wave II was no longer a significant predictor of adult obesity among females. In fact, the *p*-value for depression at Wave II in the full model was increased to .769. The same phenomenon was observed by Stice et al. (2005) in their evaluation of adolescent females. While depression was a significant univariate predictor of future obesity in the Stice et al. study, when covariates were included (i.e., dietary restraint, compensatory behaviors, and perceived parental obesity), depression was no longer a significant predictor of future obesity. In a related evaluation, Blaine (2008)

reported in his meta-analysis of depression-obesity studies that, among adolescents, girls differed from boys in their development of obesity such that depression was a predictor of obesity only for adolescent girls. The current study supports the concept of a different relationship for adolescent girls and boys in the role of depression on obesity, but in the current study, increased depression levels among girls predicted reduced obesity rather than increased obesity.

The definitive description of the connection between depression and obesity remains a challenge as demonstrated by the variety of studies that have been considered here. Although not as recent, the study by Vogelzangs et al. (2008) may provide the clearest understanding of the findings in the current study. Vogelzangs et al. concluded that for adults aged 70-79, increased depression symptoms, as measured by the CES-D, were related longitudinally to increased abdominal obesity. While the relationship did not hold for overall body obesity (such as identified by BMI), it did hold for abdominal obesity specifically. Vogelzangs et al. also observed that a categorical use of the CES-D yielded stronger results than did the continuous use of the CES-D measure. It may well be that an understanding of the longitudinal role of depression in the development of obesity is best identified by a measure of obesity, other than BMI, that accounts for abdominal or visceral obesity. This point is supported by Needham et al. (2010) who used growth curve models to demonstrate that symptoms of depression at baseline were more strongly associated with changes in waist circumference (abdominal obesity) than with BMI (overall obesity). Needham et al. reported an association for the White population between increased depression symptomology and increased rate of change in BMI but not

for the overall sample and therefore only partially agrees with the findings from the current study.

The social support–obesity relationship. Social support in adolescence was shown in the current study to interact differently in the prediction of adult obesity for males and females. Among males, social support was negatively related to obesity such that as social support scores increased, the prediction of adult obesity decreased. Among women, social support score increases in adolescence predicted an increase in adult obesity. The relationship between social support and obesity for males was not significant; for women, it was stronger, but still not significant. These findings appear to contradict the findings of two studies—Baskin et al. (2013) and Valente et al. (2009). Both studies reported on family social support; Baskin et al. reported that family social support significantly predicted an increase in the measure of daily physical activity among female African American adolescents and Valente et al. reported that positive family social support contributed significantly to increased obesity among boys and girls. However, the Baskin et al. study did not relate physical activity directly to obesity; furthermore, the Baskin et al. study was specific to African American adolescents in the South and the current study did not provide a separate measure for family social support. In regards to the Valente et al. study, the current study only found a social support-obesity relationship to be true for females and the relationship was not statistically significant. The population in the Valente et al. study consisted of a younger average age (12.7 years old) than for the current study and the racial/ethnicity makeup was incompatible as 36.14% of the Valente et al. participants were identified as being of

Asian descent. Findings from the current study support those by Siceloff et al. (2013) who reported that African Americans of various ages demonstrated no relationship between peer social support and BMI. However, the participants in the Siceloff et al. study were characterized as predominantly African American obese females—a population very different from the population in the current study. The current study also supports the general findings of Wiczinski, Döring, John, and von Lengerke (2009) of no relationship between obesity and perceived social support among men and women ages 35-74. As with other studies, the population in the Wiczinski et al. (2009) study was different from the current study population in terms of age. Furthermore, the social support measure was different from the one used in the current study primarily in that social support from teachers and parents would not have been particularly insightful for adults in the Wiczinski et al. study.

The current study demonstrated general support for a gender difference related to BMI and social support (Trogdon, Nonnemaker, & Pais, 2008). Trogdon, Nonnemaker, and Pais (2008) used the Add Health Wave I data to correlate adolescent BMI with mean peer BMI (Trogdon, Nonnemaker, & Pais, 2008). Trogdon et al. (2008) reported that as adolescent BMI increased, mean peer BMI also increased and that this was more apparent for females than for males. The Trogdon et al. (2008) study deals with social networks rather than social support; the current study is in agreement that the social connection to increased BMI is more significant for females than for males.

Depression and Social Support as Mediators of the Religiosity–Obesity Relationship

Adolescent depression and adolescent social support were not shown to be mediators of the adolescent religiosity-adult obesity relationship among either males or females. In the male mediation model, while there was a significant relationship between religiosity and obesity, the paths from religiosity to depression and from depression to obesity were not significantly correlated. For social support, the path from religiosity to social support was a significant correlation, but the path from social support to obesity was not. For females, the relationship between religiosity and obesity was not significant, therefore, social support and depression could not be considered as mediators.

Lack of mediation by depression in the religiosity-obesity relationship in the current study does not match with a recent mediational study by Son and Wilson (2011). Son and Wilson reported that emotional and psychological health mediated the relationship between home religiosity and self-rated health. However, variables of home religiosity and self-rated health are substantially different from the variables measured in the current study. Home religiosity is reflective of the home atmosphere while growing up rather than the specific value of religion to the adolescent as measured in the current study. Also, depression, as it was measured in the current study, is a focused aspect of the larger concept of emotional wellness measured by Son and Wilson.

The current study supports the Son and Wilson (2011) conclusion that social well-being did not mediate the home religiosity and health relationship. However, social well-being should not be confused to equate to social support and health should not be thought to be equal to obesity. Therefore, the variables of consideration are not well matched for

comparison. Variables from the current study align more closely with variables from the Diener et al. (2011) study, yet there is disparity between the findings of these studies. Social support was identified as a mediator of the relationship between religiosity and subjective well-being (SWB) in the Diener et al. (2011) study. Religiosity was measured somewhat similarly to the current study by including the level of importance of religion and recent religious service attendance; however, the outcome variables of SWB and obesity are not comparable. Furthermore, while Diener et al. report mediation of social support for the religiosity-SWB relationship, they do point out that some of the path *c* relationships were not significantly correlated. Social support was not statistically significantly correlated with life evaluation or positive emotions as a factor of SWB. Social support was only significantly related to the SWB category of negative emotions, thus calling into question mediation, at least as it was measured in the current study.

Consistent with the current study, neither social support nor mental health status (including feelings of depression) were shown to be mediators of the religiosity-substance use disorders relationship in the study completed by Edlund et al. (2010). Although the measure of physical health used in the Edlund et al. study is different from the physical health measure in the current study, it appears that for multiple examples of physical health outcomes (obesity and substance use disorders), social support and depression do not serve as mediators in the relationship with religiosity.

The lack of significant relationships between adult obesity and the main independent variables of religiosity, depression, and social support is a curious finding. On the one hand, this study concludes that adolescent religiosity, depression, and social

support are relevant and appropriate predictors of adult obesity, but that only adolescent religiosity among males is a significant predictor when accounting for other variables. Research regarding types of religiosity and the role of social support with depression may help to explain this paradoxical finding.

Religiosity was shown to be related to subjective well-being across four world religions and was shown to be mediated by social support (Diener et al., 2011). An interesting delineation made by Diener et al. (2011) was that this relationship was moderated by difficult life situations such that those experiencing more difficult life situations were also more religious. The current study did not account for life experiences, potentially explaining why social support was not shown to mediate the religiosity-obesity relationship. It could also be that social support is only a factor for main effect under certain circumstances as demonstrated by Brown et al. (2007) in their study of the relationship between religiosity and underage drinking as informed by social support. Brown et al. demonstrated that for multiple measures of substance abuse, social support did not present a main effect on underage drinking but there was an interaction effect between social support and extrinsic religiosity for the prediction of abuse problems. Interaction effects were not considered in the current study, nor did the current study account for the distinction between intrinsic and extrinsic religiosity.

Another possibility for the reason that social support and depression did not present a main effect on adult obesity in the current study may be due to the role of social support as a mediator of the religiosity-depression relationship as demonstrated by Ai et al. (2013). In their study of the Asian American population, Ai et al. identified that

religiosity (as measured by religious attendance) was significantly related to the prediction of major depressive episode but that the relationship became insignificant when social support was added to the regression analysis. The significant role of social support in depression was said to mediate the religiosity-depression relationship. An analysis of the role of religiosity, depression, and social support in predicting adult obesity may best be viewed when considering social support as a mediator of depression rather than for a main effect on obesity.

Alternatively, it could be that depression is not a mediator of the religiosity-obesity relationship but rather that religiosity is a moderator of the depression obesity relationship. Pirutinsky et al. (2011) determined that the relationship between physical health and depression among a Jewish population was moderated by intrinsic religiosity; further analysis indicated mediation by social support of the interaction among a certain segment of the studied population. Obesity is not equivalent to an overall measure of physical health, but a proposed parallel between the two constructs seems reasonable. While the paths and constructs are not the same between the Pirutinsky et al. and current study, it is conceivable that social support should be considered as a mediator of relationships other than the specific relationship between religiosity and obesity.

Other Independent Variable Relationships With Adult Obesity

The addition of adolescent religiosity, depression, and social support to the regression model strengthened the prediction of both adult male obesity and adult female obesity as reflected by the increase in the value of R^2 compared to models without these three variables. However, it remains that demographic information contributes largely to

the prediction of adult obesity. Disparities related to these factors are considered independently in the discussion to follow.

Smoking status. Smoking status has been included as a covariate in a number of obesity studies; in the current study, smoking status at Wave I was an insignificant but positive predictor of adult obesity for males but was dropped from the female model because it did not contribute to the model in a meaningful way. The observed positive association between smoking status (at Wave I) and adult obesity is counter to the typical role of smoking as related to obesity, which is usually represented by a decrease in BMI among those who smoke (Fidler, West, Van Jaarsveld, Jarvis, & Wardle, 2007). Smoking status at Wave IV aligned with previous research in that for both men and women, smoking at Wave IV was statistically significant and negatively related to adult obesity for both genders. It is possible that the definition used to identify smokers at Wave I and Wave IV limited the usefulness of this variable for the Wave I analysis. Smoking was defined as those individuals who had smoked at least one cigarette in the previous month. Potentially, some adolescents who fit into the *smoking* category based on this definition were only categorized this way due to recent experimentation with smoking rather than a true status as a smoker. It is less likely that adults would be incorrectly categorized as a smoker due to experimentation.

Race and ethnicity. Race is a common point of distinction with obesity studies, and results have been inconsistent. Race/ethnicity was accounted for in the current study as an independent variable in which categories included White, Hispanic, African

American, and Other. Race has been discussed in previous sections related to obesity and to religiosity.

Initial age. The prediction of male obesity in the current study demonstrated a negative relationship with age at Wave I for both men and women. In other words, as age at Wave I increased, BMI at Wave IV decreased. This relationship was significant for males, but not for females. Differences in males and females for the trajectory of BMI growth were likely reflective of the growth and development differences between males and females. Females are typically about two years ahead of males in their maturation process by adolescence. Some girls will have completed puberty by age 12 while some boys will just be starting puberty by age 12. It should be expected that females will be more stable in their growth patterns beginning at age 12 than will males. Given that the average age of participants at Wave I was 15.4, the likelihood that females in this analysis would be more comparable to each other compared to males is reflective of the differences likely to exist in the maturational process. Because this study did not include sexual maturation as a variable, the influence of puberty on the obesity growth curves is unknown.

Initial BMI (BMI at Wave I). The strongest contributor to the prediction of adult obesity (at Wave IV) was BMI at Wave I for both males and females. For males, each unit increase in BMI at Wave I resulted in a predicted increase of 1.096 BMI points at Wave IV. For females, each unit increase in BMI at Wave I resulted in a predicted increase of 1.298 BMI points at Wave IV. This outcome is supported by semipartial correlation analysis, which was not included in the official results for this study because

the analysis could not be applied definitively to complex survey data and because the factor variables could not be accounted for in the analysis. However, I was able to generate semipartial correlations of Wave IV BMI accounting for all continuous independent variables that existed in my analysis. Semipartial correlations are indicative of the variance attributed to predictor variables (Diebold, 2013). For males, BMI at Wave I held a semipartial correlation squared value of .4949 and for females, the semipartial correlation squared value, for BMI at Wave I, was .4915. These values indicated that initial BMI accounted for 49.5% and 49.2% of the variance in the prediction of adult male and female obesity, respectively.

Initial depression (depression at Wave I). The effect of initial depression on adult obesity varied by gender; however, neither measure of Wave I depression was significantly related to adult obesity.

Family income at Wave I. Family income at Wave I was determined using responses from the Parent Survey for the Add Health study. For both males and females, each unit increase in family income (one unit representing $1,000), predicted BMI at Wave IV was reduced by .002 BMI points for males and by .007 BMI points for females. These results indicate that male and female adolescents raised in families with higher annual income had and took advantage of more opportunities for lifestyles conducive to reduced BMI levels, but that females from families with more money did so at a more significant level. These findings are similar to the general conclusions from Fradkin et al. (2014); however, Fradkin et al. defined socioeconomic status (SES) in relation to parental educational attainment. Fradkin et al. reported that the negative relationship between SES

and obesity levels was most true for White adolescent girls. While educational attainment and income are distinct measures related to SES, there does appear to be some overlap in the results in that higher attainment (educational or income-based) is associated with lower rates of obesity and that females tend to be a population for whom the connection is strongest.

Adult income. Income for participants was evaluated and included in the regression analyses based on quintiles reported by adults at Wave IV. For males, all quintiles contributed significantly to the positive increase in obesity compared to the lowest quintile with coefficients ranging from 1.600 to 2.669. For females, the opposite was true. Compared to the lowest quintile for income, all quintiles contributed to the level of obesity by reducing BMI with coefficients ranging from -.920 to -2.261. The third quintile was the only quintile that was not significantly different from the first quintile in this relationship for women. Furthermore, while the third quintile for income among women exhibited the smallest effect on adult obesity (reduction of .920 BMI points), the third quintile for men exhibited the largest effect on adult obesity (increase of 2.669 BMI points) compared to the first quintile for each sex. These results support the general findings of Ogden et al. (2010) who reported a trend towards increased obesity with increased income among males and a trend towards decreased obesity with increased income among females for the data collection period of 2005-2008. Similarly, Ogden et al. demonstrated that obesity rates among men were highest in the middle range of the poverty income ratio (their measure of socioeconomic status); in the current study, males in the third quintile for income had the highest coefficient contributing to the prediction

of obesity. For women, Ogden et al. demonstrated that obesity rates were highest for the lowest level of the poverty income ratio and in the current study all income quintiles contributed to the lowering of obesity rates compared to the first quintile.

The Theoretical Foundation Proposed by Hawks (2004)

Spiritual wellness was hypothesized by Hawks (2004) to impact physical and intellectual wellness through the conduits of emotional and social wellness. In order to test this theory, I chose single measures to represent each dimensions of wellness included in the study (with the exception of intellectual wellness) and did so accounting for temporal relationship of these measures. Religiosity at Wave I was shown to have a positive correlation with social support at Wave II and a negative correlation with depression at Wave II (all correlations were significant for both sexes except for religiosity and depression for males). Furthermore, religiosity was shown to significantly and positively predict male obesity at Wave IV but was negative and an insignificant predictor of adult female obesity. Therefore, Hawks' model is supported for male relationships between the four dimensions and supported at a statistically significant level for the spiritual and physical dimensions. Hawks' model is not supported by the outcome of the female relationship—at least not from a statistically significant level. However, for both males and females, the addition of religiosity, depression, and social support increased the strength of prediction of adult obesity despite the fact that male religiosity was the only significant association in the predictions. Therefore, while the spiritual, emotional, and social health dimensions do not always hold a direct relationship to the physical health dimension, there does appear to be an interaction, or interactions,

associated with these variables that allows them to strengthen the overall prediction of adult obesity when they are included in the regression models.

In this study, I evaluated the role of adolescent religiosity, depression, and social support in the prediction of adult obesity as a way to test for the relationship that exists between multiple dimensions of health. While the general premise that spiritual health has a significant effect on physical health was upheld for males, none of the other relationships was directly supported for males or for females. In general, when results from my study did not match those of previous research, either the specific aspects of the variables being considered were not a close enough match to make a definitive judgment, or the variables used in other studies were an overarching construct related to the more specific variables used in my study. For instance, social well-being, physical well-being, and emotional well-being typically demonstrated different reactions compared to the more specific constructs of social support, depression, and obesity that were used in the current study.

Despite these differences, there was a clear distinction between the ways in which these four health dimensions relate based on sex. In particular, males and females lacked congruency in the combined effect of adolescent religiosity, depression, and social support on adult obesity. The combination differences can be identified by individual differences for a given variable, such as depression. For instance, Ge, Conger, and Elder (2001) determined that depression symptoms followed a different path for adolescent girls and boys based on puberty status (especially puberty status in 7th grade). These varied paths might speak to the different way in which depression in adolescence affected

females compared to males in the current study. Furthermore, social support appeared to provide a different level of protection from obesity for men and women (reduced obesity for men, but increased obesity for women). The difference in social support effect and its connection to depression is supported by Lim et al. (2011) who reported that the interaction of perceived social support and peer victimization was a moderator of depressive symptoms for obese girls but not for obese boys. From the Lim et al. study, the clear distinction between social support and depression for males and females is apparent. The relationship between religiosity and depression is also varied by sex among adolescents as witnessed by Rasic, Asbridge, Kisely, and Langille (2013). Rasic et al. (2013) differentiated the role of religiosity on future depression by identifying the role of self-efficacy among females and level of depression at baseline among males as indicators of later depression. While the current study identified specific relationships to exist between religiosity, depression, social support, and obesity, the mechanisms behind these relationships were different for males and females and were not fully explained by the current study.

Limitations of the Study

This study provided a longitudinal view of the development of obesity from adolescence into adulthood using the Public Use data of the Add Health study. Appropriately applying weights to account for the complex survey data of the Add Health study allowed for the study to be representative of students in Grades 7-12 in the United States in the 1994-1995 school year. The design of this study allowed for the temporal determination of cause and effect related to a number of relationships including

appropriately designing mediational analysis with variables from multiple time-frames. Despite the strengths of the study, there were also a number of limitations.

One limitation of the study was that the complex survey design of the data set limited the post-estimation analyses that could be accomplished using Stata 14.0. In particular, the traditional checks for assumptions were not readily available when using survey data. This limitation, however, should not be too deleterious given that a strength of Stata is its application of robust analyses for survey data, which can account for such problems as heteroscedasticity, and problems with large residuals and normality, to name a few.

Another limitation of the study was that the dataset used was collected without my particular study/methods in mind. Because the data were collected without my input, I had to search for the best way to conduct my study given the variables that were defined by the study researchers. In some cases, the scales and variables were easily identified and recognizable for validity determination. The depression scale used by the Add Health researchers is an example of a validated scale that was used for identifying depression symptomology in the form of the CES-D. On the other hand, the scales used to represent religiosity and social support were not recognizable scales with established support for validity. In these cases, I was able to use the scales constructed by other researchers for use with the Add Health data and derived validity measures from their research. Perhaps the most problematic limitation related to the variables used was related to a measure of obesity. BMI is constructed using the height and weight of an individual. At Waves I and II, BMI was based on self-reported height and weight. Wave III used both self-reports of

height and weight and interview-conducted measures for height and weight (but with limitations on weight). Wave IV used measured values for height and weight. This study may have been strengthened had all values for height and weight been measured by the interviewer using standardized equipment. The Add Health researchers included a measure of waist circumference in their Wave IV data. Such measurements beginning in adulthood (and perhaps even in adolescence) would have strengthened the data set for the current study.

There were a number of covariates that could have been considered in the study; however more covariates required larger sample sizes to meet statistical power. There was one variable I would like to have added to this analysis—the use of psychotropic drugs was a variable missing from the Add Health data. Because psychotropic drugs are typically associated with weight gain (Dent et al., 2012; McCloughen & Foster, 2011), this would have been a useful variable to control for in this study.

Another limitation of this study was the unequal time period between data collection of the Add Health waves as well as inconsistency from participant-to-participant in the length of the time period. Wave II was collected within about 10 months of the first wave; the gap between Wave II and III was approximately 5-6 years, and the gap between Wave III and IV was approximately 6-7 years. Due to the fact that adolescents were at different stages of development (ages 12-18 in the sample used for this study) at the start of the study, the role of early depression, BMI, and religiosity cannot be assumed to be evenly evaluated for all participants. In other words, the role of

religiosity in the development of depression for a 12-year old can be assumed to be much different than that for a 16-year old—yet both were evaluated in this study.

Related to the depression scale (the CES-D), in particular, it should be noted that responses were based on experiences of the respondent during the seven days previous to the interview. As such, the CES-D should not be viewed as a measure of depression, but rather as a measure of depression symptoms. Such a measure will not be as consistent over time as would be a clinical determination of depression. Similarly, two questions used to construct the religiosity measure were bound by actions within the past 12 months. Those questions were related to the concept of extrinsic religiosity and may not be as consistent a measure as the questions related to intrinsic religiosity in this study. In all, the three main independent variables of religiosity, social support, and depression were measured at distinct points in time and were not evaluated over the span of the entire study or even over the span of adolescence for each participant. While temporal relationships between these variables allow for a greater understanding of the cause-effect relationship that exists, the cause-effect relationships are not as strong as they could be had more comprehensive growth curve models been used for analysis of all of the variables.

Generalizability for the current study must be considered as well. While the study reflects students in Grades 7-12 in the United States in 1994-1995, the demographics in U.S. society have changed considerably. Researchers must not assume that adolescents in 2016 are similar to adolescents in 1994. Even changes in technology leading to greater opportunities for screen time in the past 20 years are likely to have affected obesity rates

over this timeframe. Certainly the religious demographics can be assumed to have morphed over the past two decades in the United States. Furthermore, the psyche and experiences of today's adolescents cannot be presumed to be the same as for those from the mid-1990s. For all of these reasons, while this study is reflective of U.S. culture, it cannot be said to be reflective of today's youth.

Recommendations

This study has demonstrated that an understanding of the development of obesity from adolescence through adulthood can be further understood using a holistic approach to health. By considering self-reports of level of religiosity, depression, and social support in adolescence, the profile for the development of obesity can be enhanced. Unfortunately, the specific interconnection between religiosity, depression, social support, and obesity is still not clear. Increased religiosity was shown to relate to a significantly higher level of obesity in adulthood for males but not for females. Despite this finding, the mechanism for this relationship is unclear given that religiosity has a significant positive relationship with social support among adolescent males and females and religiosity has a significant negative relationship with depression for females and a strong but nonsignificant relationship with depression for males. The logical connection of correlations would imply that if religiosity reduces depression and increases social support among adolescents, and if depression correlates positively with obesity and positive social support correlates with increased health behaviors, then religiosity should be connected to reductions in obesity. This does not appear to be true for U.S. adolescents transitioning into adulthood. For males, the role of increased religiosity was significantly

related to increased obesity and for females there was no significant relationship, but the relationship was opposite of that for males.

The exact mechanisms accounting for the disparity in the role of religiosity, depression, and social support related to obesity for males compared to females is not yet known; however, there are some possibilities to consider. In general, adult males exhibit higher levels of activity than do females (Kruger, Yore, & Kohl, 2008). Given that the average BMI of males at Wave IV was 28.96 and categorized as *overweight*, it is possible that for a portion of these men, their elevated BMI is a reflection of muscle mass rather than fat mass. Therefore, although this study considered changes in BMI, changes in BMI could be caused by increases in fat or nonfat portions of the body. Use of a different measure of *obesity* may have identified a different relationship for men for the main independent variables.

Assuming that there are distinct differences between men and women in the ways in which religiosity, social support, and depression relate to adult BMI, there are some possibilities that should also be considered. The difference between men and women for religiosity scores was demonstrated at Wave I of the Add Health study. However, Ploch and Hastings (1994) identified a trend in which for White and African American respondents to the General Social Survey (GSS) the gender gap for church attendance continued to widen with age. Therefore, more consistent church attendance by women, which is generally associated with greater social support, may be what led to the negative relationship between religiosity and BMI for women in this study. However, since social support, in this study, was positively associated with increased BMI, it may be that

increased *religious social support* is what led to the decrease in overall BMI for the women in the current study. Furthermore, Silverstein et al. (2013) identified a gender difference for somatic depression in which the disparity in male and female depression prevalence was accounted for by higher levels of somatic depression (disordered eating, body image, fatigue, and insomnia are examples) among women. If depression symptomatology scores for women are more indicative of physical symptoms, this could explain some of the difference between the depression interaction with a physical outcome between men and women.

There are a few steps that can be taken to illuminate the relationships between religiosity, depression, social support, and obesity in future research. Future research should consider the changes in obesity from a different perspective. BMI tends to be the default measure for obesity in obesity studies; this is likely due to the ease with which measures for BMI can be collected. However, there is evidence that different forms of measurements for obesity yield different results in obesity studies. Needham et al. (2010) reported that the use of BMI resulted in significant relationships only for the White population whereas the use of waist circumference demonstrated population-wide relationships. Needham et al. posit that a measurement specific to central adiposity rather than relative weight is the difference in connecting depression to obesity due to metabolic factors. This point is reflective of an earlier study completed by Vogelzangs et al. (2008) in which depression was associated with visceral fat (internal fat in the abdominal region) despite a lack of overall fat accumulation.

Future research should consider the trend towards religious social support as an independent variable. Religious social support specifically measures aspects of support given and received through organized religious organizations. This type of support has been found to add benefit beyond that received from general social support for certain health behaviors including consumption of fruits and vegetables, activity, and substance abuse (Debnam et al. 2012). Holt et al. (2013) further applied the concepts of religious social support and determined that the relationship between religiosity (religious behaviors in the Holt et al. study) and depression was mediated by religious emotional support. Finally, religious social support was shown to have distinguishable features for older Mexican American men compared to older Mexican American women (Krause &Hayward, 2013). Krause and Hayward (2013) discovered that older Mexican American men were more impacted by all religious social support factors than were older Mexican American women. Therefore, religious social support can potentially delineate between the effects of social support and religion among the sexes.

Finally, future research should focus on potential interactions that may occur between and among the independent variables used in this study. Social support, depression, and religious social support and interactions of these variables should be considered for moderation properties in the link between religiosity and obesity. The specific ways in which depression, social support, and religious social support interact within each sex should be considered. Furthermore, intrinsic and extrinsic religiosity should be considered separately for other populations to see how this distinction affects the relationship with obesity.

Positive Social Change

The current study contributes to the understanding of the development of obesity from adolescence to adulthood and does so while considering the role of multiple dimensions of wellness including the spiritual, emotional, and social dimensions. By accounting for the multidimensional nature of humans, this study provides further insight into the interplay that occurs between dimensions as it relates to health outcomes, thus allowing a platform for the propagation of the interrelatedness of health dimensions that is so common to health education. The specific relationships related to adult obesity remain somewhat elusive; however, adolescent religiosity was shown to relate significantly to adult obesity among males. The specific ways in which religiosity, depression, and social support interact will need to be refined in future longitudinal models related to obesity, but a general framework has been established.

This study supports the relationships that exist between various health dimensions and does so from a longitudinal perspective. The strength of the relationships between adolescent religiosity and decreased depression and between adolescent religiosity and increased social support is enhanced from a longitudinal perspective. The significant ties associated with these dimensions (other than for religiosity and depression among males) can foster the development of support resources for adolescents as they navigate the transition from adolescence to adulthood. Similar to the way in which adults are considering multiple dimensions for enhancing health (socially, emotionally, and even spiritually), this study provides support for a multi-dimensional approach that can be enacted for adolescents. Decisions made in adolescence are key indicators of health

behaviors later in life; therefore, support for making better decisions and developing better health habits can have an immense impact on future generational health. If some aspect of religiosity (intrinsic or extrinsic) can decrease depression and increase social support among adolescents, such an aspect should be made known to caregivers and practitioners so that those offering help and support can make available all resources that may be relevant to the patient.

An important generalization of the current study is that dimensions of wellness confer differential relationships for males and females. The roles of religiosity, depression, and social support in the development of obesity are not congruent between the sexes. This generalization provides a rationale for differentiating the support and resources made available to males and females in appropriate and structured ways. Specific types of religiosity, depression, and social support need to be identified for the specific pathways that lead to increased or decreased obesity for each sex. In any case, the type and format of obesity prevention enacted at the adolescent stage needs to be shrouded in an understanding of the differential interactions that occur by sex. The provision and form of resources should be informed by relevant research specific to each sex.

The results of the current study also add to the understanding of the relationship that exists between and among dimensions of wellness as presented by health educators. This study demonstrates that multiple dimensions of wellness can be considered together as influences of at least one other dimension. Adolescent religiosity, depression, and social support were shown to enhance the prediction of the development of obesity over

time. The health education sector needs continued examples of such relationships in order to support the general premise that dimensions of wellness are interrelated. Interrelatedness of health dimensions is a concept that tends to be promoted without sufficient evidence of the specific relationship. Furthermore, many of the associations that are presented are supported by minimal one-to-one connections (i.e., depression and obesity) using cross-sectional designs that limit the nature of cause and effect between the dimensions. Health educators now have an example of the framework for the interrelatedness of spiritual, emotional, and social dimensions in predicting a physical outcome and using a model of interpretation that accounts for a longitudinal perspective.

Conclusions

Obesity is a prominent social problem in the United States. Much of the obesity research conducted has connected obesity to a variety of health dimensions but has done so cross-sectionally and for a limited number of dimensions. Recent obesity research has been focused on the longitudinal understanding of this development and this type of research is growing. The nature of the Add Health data set allows for an understanding of the development of obesity from a longitudinal perspective and allows for the evaluation of multiple dimensions of wellness. This study identifies the longitudinal relationship between adolescent religiosity and future depression and social support. Furthermore, adolescent religiosity, depression, and social support have been shown to enhance the prediction of adult obesity for both men and women while religiosity was the only significant contributor for a direct effect on adult male obesity. The specific mechanisms by which religiosity, depression, and social support interact to predict adult obesity is not

known, but neither depression nor social support were shown to mediate the religiosity-obesity relationship that exists for men. The relationships of the chosen predictors in the current study were shown to be different for males and females. An understanding of the gender differences is important when considering the support and prevention approaches that will be implemented with youth.

The longitudinal focus of the current study is an important quality that will continue to influence the direction of obesity studies. Furthermore, the multidimensional focus takes advantage of the wider spectrum of influence evident in the development of health behaviors—even those behaviors that influence obesity. Future research can build on the design utilized in the current study to further identify the specific relationship that occurs between religiosity, depression, and social support in the longitudinal development of obesity. Social support should be considered for the role of mediation in the religiosity-depression relationship and religious social support should be included as a variable in the overall relationship between religiosity and obesity. Future research should include the use of measures of obesity other than BMI as there is evidence that visceral adiposity is more commonly associated with depression than is overall weight, which is measured by BMI.

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Appendix: IRB Approval Email

Gmail by Google

Trini Rangel <trini.rangel@waldenu.edu>

IRB Materials Approved - Trini Rangel
6 messages

IRB <IRB@waldenu.edu> Mon, Jul 6, 2015 at 12:15 PM
To: Trini Rangel <trini.rangel@waldenu.edu>, IRB <IRB@waldenu.edu>
Cc: Jesus Tanguma <jesus.tanguma@waldenu.edu>

Dear Ms. Rangel,

This email is to notify you that the Institutional Review Board (IRB) confirms that your doctoral capstone entitled, "The Role of Religiosity, Social Support, and Depression in the Development of Obesity from Adolescence to Adulthood: An Archival Study," meets Walden University's ethical standards. Since this project will serve as a Walden doctoral capstone, the Walden IRB will oversee your capstone data analysis and results reporting. Your IRB approval number is 07-06-15-0169976.

This confirmation is contingent upon your adherence to the exact procedures described in the final version of the documents that have been submitted to IRB@waldenu.edu as of this date. This includes maintaining your current status with the university and the oversight relationship is only valid while you are an actively enrolled student at Walden University. If you need to take a leave of absence or are otherwise unable to remain actively enrolled, this is suspended.

If you need to make any changes to the project staff or procedures, you must obtain IRB approval by submitting the IRB Request for Change in Procedures Form. You will receive confirmation with a status update of the request within 10 business days of submitting the change request form and are not permitted to implement changes prior to receiving approval. Please note that Walden University does not accept responsibility or liability for research activities conducted without the IRB's approval, and the University will not accept or grant credit for student work that fails to comply with the policies and procedures related to ethical standards in research.

When you submitted your IRB materials, you made a commitment to communicate both discrete adverse events and general problems to the IRB within 1 week of their occurrence/realization. Failure to do so may result in invalidation of data, loss of academic credit, and/or loss of legal protections otherwise available to the researcher.

Both the Adverse Event Reporting form and Request for Change in Procedures form can be obtained at the IRB section of the Walden website: http://academicguides.waldenu.edu/researchcenter/orec

You are expected to keep detailed records of your capstone activities for the same period of time you retain the original data. If, in the future, you require copies of the originally submitted IRB materials, you may request them from Institutional Review Board.

Both students and faculty are invited to provide feedback on this IRB experience at the link below:

http://www.surveymonkey.com/s.aspx?sm=qHBJzkJMUx43pZegKImdiQ_3d_3d

Sincerely,

Libby Munson

Research Ethics Support Specialist

Office of Research Ethics and Compliance

Email: irb@waldenu.edu

Fax: 626-605-0472

Phone: 612-312-1283

Office address for Walden University:

100 Washington Avenue South, Suite 900

Minneapolis, MN 55401

Information about the Walden University Institutional Review Board, including instructions for application, may be found at this link: http://academicguides.waldenu.edu/researchcenter/orec

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Jesus Tanguma <jesus.tanguma@waldenu.edu> Mon, Jul 6, 2015 at 5:39 PM
To: Trini Rangel <trini.rangel@waldenu.edu>

Trini,

Congratulations on IRB approving

If you would, please post a message so that other students might be encouraged.

Thank you and congrats again,
[Quoted text hidden]

Trini Rangel <trini.rangel@waldenu.edu> Mon, Jul 6, 2015 at 7:10 PM
To: Jesus Tanguma <jesus.tanguma@waldenu.edu>

Thank you - I will post a message! I have downloaded Stata and I have downloaded the dataset from ICPSR - time to see if I can figure this thing out :)

Trini
[Quoted text hidden]
