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AFFECTIVE TRAITS AND ADIPOSITY: A PROSPECTIVE, BIDIRECTIONAL ANALYSIS OF THE AFRICAN AMERICAN HEALTH STUDY

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AFFECTIVE TRAITS AND ADIPOSITY:

A PROSPECTIVE, BIDIRECTIONAL ANALYSIS OF THE AFRICAN AMERICAN HEALTH STUDY DATA

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"We must find time to stop and thank the people who make a difference in our lives."

John F. Kennedy

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ABSTRACT

Hawkins, Misty A.W. M.S., Purdue University, August 2013. Affective Traits and Adiposity: A Prospective, Bidirectional Analysis of the African American Health Study Data. Major Professor: Jesse C. Stewart

Research indicates that negative affective traits (e.g., depression) are predictors and consequences of excess adiposity. Given that racial minorities and positive affective traits have been underrepresented in past investigations, more prospective studies are needed which examine multiple affective traits in relation to obesity in these populations. The objective of the current study was to investigate the prospective, bidirectional associations between multiple affective traits and multiple adiposity indicators in African Americans using data from the African American Health (AAH) study. The AAH study is a prospective cohort study of African Americans aged 49-65 years at baseline (N = 998). The longest follow-up period in the current study was 9 years (N = 579). Self-reported and measured body mass index (BMI; kg/m^2) and body fat percent (BF%) were used as adiposity indicators. Depressive symptoms were assessed with the 11-item Center for Epidemiologic Studies-Depression Scale (CES-D), and anxiety was assessed using the Generalized Anxiety Disorder-2 (GAD-2) scale. Positive affective traits were assessed with the Vitality subscale of the Short Form-36 and Positive Affect subscale from the CES-D. Latent variable path analysis, a structural equation modeling technique,

was conducted. Although fit statistics indicated that the models fit the data (RMSEA ≤ .06), examination of the structural paths revealed that the CES-D and GAD-2 were not predictors or consequences of self-reported BMI, measured BMI, or BF% (*p*s > .05). Likewise, Vitality and CES-D Positive Affect were not related to any adiposity indicator (*p*s > .05). The results of this prospective cohort study suggest that affective traits are not predictors or consequences of adiposity in middle-aged African Americans and that this group may require obesity prevention or intervention programs with little to no emphasis on affective traits. Possible explanations for the current results include ethnic differences in the mechanistic pathways between affective traits and adiposity.

INTRODUCTION

Adiposity, or body fat, can be represented by a spectrum, with the range for optimal human health located between insufficient adiposity on one end and excess adiposity on the other. This study focuses on the end of spectrum representing excess adiposity and places a special focus on obesity, a medical condition defined by excess adiposity. Obesity is a multifaceted disorder involving biological, psychological, and social factors in its causes and its consequences. Of the psychosocial variables related to obesity, affective traits have been increasingly emphasized in the literature, and the documented relationships between affective traits and obesity provided the impetus for this study. Affective traits, such as depression, anxiety, hostility, and trait positive affect, are emphasized because they have demonstrated overlap with one another and/or have been found to be related to various indicators of excess adiposity (Gariepy, Nitka, & Schmitz, 2010; Jorm et al., 2003; Luppino et al., 2010; Roberts, Strawbridge, Deleger, & Kaplan, 2002).

Unfortunately, the affective traits-obesity relationships have not been fully evaluated in regards to which affective traits are independent risk factors for obesity. In addition, the connection between affective traits and obesity among men and minorities is poorly understood, given the reliance on predominantly white and female samples.

Accordingly, the overall objective of the proposed project is to address these gaps in the literature by investigating the bidirectional associations between multiple affective traits and adiposity indicators in a large sample of community-dwelling African American males and females. This paper begins with a discussion of the extent and nature of the obesity problem. Next, traditional risk factors for obesity are reviewed, followed by a discussion of affective traits and the potential mechanisms by which they may influence obesity development and vice versa. After establishing the plausibility of the affective traits-obesity associations, the available empirical evidence examining these relationships is reviewed. Throughout this section, major limitations of this literature are identified, such as the issue of overlapping affective traits. A description of the project and how it addressed the limitations is then provided. The results of this prospective cohort study helped to clarify which affective traits are most predictive of changes in adiposity among middle-aged African Americans. Ultimately, this knowledge may inform the development of culturally sensitive interventions designed to prevent obesity. The present study also helped to identify whether adiposity is related to changes in specific affective traits over time, information which may be of use in developing interventions designed to minimize the psychosocial consequences of obesity in African Americans.

The Problem of Obesity

Obesity, a chronic disease characterized the excessive accumulation of adipose (fat) tissue (Bray, 2003; Stedman, 1995), has been traditionally defined as a body mass index (BMI) $\geq$ 30 kg/m^2 (World Health Organization, 1995). A BMI of 30 kg/m^2, roughly equivalent to 30 pounds of excess weight, is an established threshold at which

excess weight has been shown to confer increased health risk (National Heart, Lung, and Blood Institute; NHLBI, 1998). For this reason, the literature reviewed for the current study focuses primarily on excess adiposity as defined by the standard BMI cut-point for obesity. However, it should be noted that (1) BMI is not a direct measure of adiposity, (2) other indicators of adiposity are available, and (3) degree of excess adiposity has shown a more or less dose-response relationship with health risk, such that increasing amounts of body fat confer increasing risk of health problems, even for BMI ranges typically indicative of normal weight (Folsom et al., 1993; Willett et al., 1995; World Health Organization, 2000). Thus, although the term of obesity is usually reserved for individuals with BMIs ≥ 30 kg/m^2, evidence indicates that excess adiposity can occur in individuals with normal (18.5 – 24.9 kg/m^2) or overweight (25 – 29.9 kg/m^2) BMIs. Consequently, the term "adiposity" was also used in the current study. This term was used to capture the concepts of overweight and/or obesity, as both can be considered conditions of excess adiposity. Thus, "adiposity" can not only be used in reference to BMI but also to other obesity variables, such as waist circumference, waist-to-hip ratio, weight gain, and body fat.

In the United States, an estimated 32% of adult men and 36% of adult women suffer from obesity (Flegal, Carroll, Ogden, & Curtin, 2010). Obese individuals are at risk for numerous chronic medical conditions, including coronary artery disease, type 2 diabetes, dyslipidemia, and osteoarthritis (Kopelman, 2000; Must et al., 1999; NHLBI, 1998) and also have higher mortality risk compared to their normal weight peers (Lloyd-Jones et al., 2010; Rosamond et al., 2008). Accordingly, obesity-related health care expenditures are significant and estimated to be approximately 9.1% of the total U.S.

medical expenditures in 1998 (Finkelstein, Fiebelkorn, & Wang, 2003). Obesity is also associated with poorer psychosocial outcomes, including higher rates of depression and anxiety (Gariepy et al., 2010; Luppino et al., 2010). Obese individuals are also found to be less desirable marriage partners (Vener, Krupka, & Gerard, 1982), less likely to be employed, and receive lower salaries than their non-obese peers (Wadden et al., 2001). African Americans are at greater risk for some obesity-related health outcomes (e.g., type 2 diabetes and hypertension) but not all (e.g., dyslipidemia) (Allison, Edlen-Nezin, & Clay-Williams, 1997; Cossrow & Falkner, 2004). This heightened risk is concerning, given that African Americans have among the highest rates of obesity, with an estimated prevalence of 44% for black adults compared to 32% for whites and 39% for Hispanics (Flegal et al., 2010). Indeed, over half of African American women aged 40 or older are currently obese, the highest rate of any U.S. demographic group (Flegal et al., 2010).

Given the large percentage of obese individuals, especially among African Americans, and the associated personal and societal costs, the obesity epidemic has been declared a "public health crisis" (Wang, Beydoun, Liang, Caballero, & Kumanyika, 2008). Although evidence suggests that many obesity-related health complications may be reversible when excess weight is lost (Blissmer et al., 2006; NHLBI, 1998; Rippe et al., 1998), studies have shown that 80% of individuals in treatment programs are unsuccessful at achieving long-term weight loss (Wing & Phelan, 2005). The figures above highlight the importance of understanding and intervening on risk factors for excess adiposity in order to prevent the epidemic spread of obesity and its associated physical and psychosocial consequences. Efforts to identify the risk factors for obesity

have led to the discovery that a variety of physical, psychological, and social factors may play important roles in obesity development and progression.

Traditional Risk Factors for Obesity

Before discussing potential psychosocial risk factors, it is important to acknowledge more traditional risk factors for weight gain. Obesity is the natural result of a chronic energy imbalance in which an individual's energy intake exceeds his or her energy expenditure (World Health Organization, 2000; Wyatt, Winters, & Dubbert, 2006). A number of factors have been identified that influence energy balance and are thought to play an etiological role in obesity. Genetic predisposition is thought to account for 25-40% of the variance in BMI (Wadden, Brownell, & Foster, 2002). Because the gene pool has remained relatively stable over time (Price, 2002) and evidence supports gene-environment interactions in the etiology of obesity (Bouchard, 2008), the increased prevalence of obesity over the last three decades is better explained by a combination of biological, behavioral, psychological, and social factors rather than by genetic factors alone (Wadden et al., 2002; Wilding, 2011; Wyatt et al., 2006). Additional risk factors include biological factors (e.g., low basal metabolic functioning and younger age at pregnancy), as well as health behaviors (e.g., excessive calorie intake, minimal physical activity, heavy alcohol drinking, and past or current smoking) (Bamia, Trichopoulou, Lenas, & Trichopoulos, 2004; Kaye, Folsom, Jacobs, Hughes, & Flack, 1993; Lahti-Koski, Pietinen, Heliövaara, & Vartiainen, 2002; Rissanen, Heliovaara, Knekt, Reunanen, & Aromaa, 1991; Saarni, Pietilainen, Kantonen, Rissanen, & Kaprio, 2009; Shimokata et al., 1989; Tremblay et al., 1990; Wannamethee & Shaper, 2003;

Wyatt et al., 2006). Obesity is also strongly connected to social factors such as low socioeconomic status (SES) (Wang, 2001) and has been shown to propagate itself via social networks (i.e., obesity spreads from one person to other in social systems) (Christakis & Fowler, 2007). Unfortunately, these traditional risk factors leave a substantial percentage of the variance in obesity unexplained. For instance, Wing and colleagues (1991) found that the impact of health behaviors and weight history accounted for only 17% of the variance in waist-to-hip ratio in their sample of middle-aged women. Accordingly, research on traditional risk factors for obesity has been augmented by studies examining psychological factors. Among the psychological factors that have been linked with adiposity indicators are affective traits.

For the purposes of the current study, affective traits are conceptualized as pervasive, underlying affective dispositions that broadly influence individuals' cognitive, emotional, and behavioral experiences and are relatively stable across time (Rosenberg, 1998). In contrast, emotions (i.e., transient affective experiences that are related to specific purposes, which drive individuals' consequent responses) are not a focus (Gross, 2007; Rosenberg, 1998). Evidence regarding three particular negative affective traits, including depression, anxiety, and hostility/anger, is reviewed. These three traits were chosen because they are highly related and have demonstrated stable, trait-like qualities over time (See Table 1 for brief descriptions, example measures, and test-retest reliabilities of these traits). Even major depressive disorder, which is typically considered an episodic disorder, tends to be characterized by chronically recurrent episodes (Maj, Veltro, Pirozzi, Lobrace, & Magliano, 1992; Mueller et al., 1999). Similarly, many anxiety disorders tend to be chronic over time (Bruce et al., 2005;

Lovibond, 1998). Given their trait-like characteristics, depressive and anxiety disorders will be conceptualized as affective traits, along with continuous measures of trait depression and trait anxiety. Although some of these continuous measures are purported to assess symptom levels in the past week or two (e.g., Beck Depression Inventory, Beck Anxiety Inventory), they have also shown trait-like stability across time (*r*s = .59-.86 and .35-.81, respectively) (De Ayala, Vonderharr-Carlson, & Kim, 2005; Yin & Fan, 2000). As previously mentioned, affective traits may not only be involved in the prediction of obesity, they are also demonstrated consequences of obesity (e.g., Gariepy et al., 2010; Luppino et al., 2010).

Mechanisms of the Bidirectional Affective Traits-Obesity Relationships

Several candidate mechanisms have been put forth to explain how affective traits may contribute to obesity and vice versa. These explanatory factors are modeled in Figure 1 and described below. Of note, most of this research has examined mechanisms of the depression-obesity relationship; however, the other negative affective traits may be associated with obesity via similar pathways.

Affective Traits-to-Obesity

A number of possible mechanisms have been suggested to account for how affective traits may lead to obesity (Figure 1, Panel A). First, several biological pathways have been identified. For instance, depression has been associated with hypothalamic-pituitary-adrenal (HPA) axis dysregulation (e.g., increased cortisol secretion) (Björntorp, 2001; Bornstein, Schuppenies, Wong, & Licinio, 2006), and elevated cortisol secretion

has been purported to promote the development of adipose tissue (Asensio, Muzzin, & Rohner-Jeanrenaud, 2004; Ottosson, Lönnroth, Björntorp, & Edén, 2000). Another way that depression may contribute to obesity development is through increased systemic inflammation (e.g., elevated interleukin-6) (Miller, Freedland, Carney, Stetler, & Banks, 2003; Shelton & Miller, 2010), which can also cause HPA axis stimulation and corresponding obesity development (Yudkin, Kumari, Humphries, & Mohamed-Ali, 2000). A limitation of most studies regarding HPA axis dyregulation and inflammation is the use of a cross-sectional design, which prohibits the ability to clearly determine whether affective traits, obesity indicators, or both are responsible for promoting the HPA axis dysregulation or systemic inflammation. Due to the cross-sectional nature of this research and the inability to establish clear directionality, these pathways are presented in Panels A and B of Figure 1. Several neurotransmitter systems and neuropeptides (e.g., dopamine, serotonin, norepinephrine, and neuropeptide Y) have also been implicated in the regulation of mood and in the regulation of food intake and body weight (Bornstein et al., 2006; Meguid et al., 2000; Wang et al., 2001). These factors are also included as mechanisms in both Panels A and B (Figure 1), given that directionality has not been established.

Negative affective traits may also lead to obesity through their impact on health behaviors, such as emotional eating, sedentary behavior, and use of psychotropic medications, cigarettes, and alcohol. Konttinen et al. (2010) reported evidence that emotional eating (i.e., the tendency to eat in response to negative emotions) mediated the relationship between depression and obesity. Likewise, reviews support the longitudinal association between lack of physical activity and obesity development (Must & Tybor,

2005), and physical inactivity is a common characteristic of depressed persons (Paluska & Schwenk, 2000). Psychotropic medication use may also partially explain why negative affective traits ultimately results in obesity as one study suggests that use of antidepressants and antipsychotic medications may explain 86% of the variance between mood disorders and obesity and 32% of the variance between anxiety disorders and obesity (Smits et al., 2010). Depression and anxiety are also associated with increased risk of smoking and drinking (Strine et al., 2008), which have both been linked to increased risk of obesity or obesity progression (Bamia et al., 2004; Goodman & Whitaker, 2002; Rissanen et al., 1991; Wannamethee & Shaper, 2003).

Obesity-to-Affective Traits

Obesity may lead to increases in negative affective traits through some of same biological pathways mentioned above (Figure 1, Panel B). First, studies suggest that obesity may promote depression through HPA axis dysregulation, elevated inflammatory cytokines, and/or the dysregulation of various neurotransmitters and neuropeptides involved in food intake and weight regulation (Bornstein et al., 2006; Shelton & Miller, 2010; Yudkin et al., 2000). Obesity may also contribute to depression by promoting resistance to leptin, traditionally thought of as an anti-obesity hormone. Leptin elevations have been observed in obese individuals and are thought to reflect insensitivity to the hormone (Considine et al., 1996). Leptin insufficiency has been associated with depressive disorders (Jow, Yang, & Chen, 2006); therefore, obesity-related leptin resistance has also been hypothesized to promote depression (Lu, 2007).

Additional mechanisms have also been identified that may be unique for the pathway leading from obesity to affective traits, such as the stigma and discrimination that obese individuals face. Staffieri (1967) reported that children as young as 6 years old used terms such as "lazy," "stupid," "dirty," and "ugly" to describe silhouettes of obese children. A more recent prospective study by Thompson and colleagues (1995) has demonstrated that weight-related teasing (e.g., negative verbal commentary from others regarding one's weight) mediates the relationship between obesity and future depression in adolescents. Such teasing may lead to body dissatisfaction and pressure to be thin, both which have also shown to be mediators of the obesity-depression relationship (Chaiton et al., 2009; Gavin, Simon, & Ludman, 2010b). Obesity may also cause negative affect due to the development of physical morbidity and functional limitations. For instance, it has been found that the obesity-depression relationship is diminished after controlling for physical illness burden and/or functional impairments, a pattern consistent with meditation effects (Schieman, McMullen, & Swan, 2007).

Lastly, obesity may contribute to negative affective traits via its association with binge eating. Some obese individuals suffer from the one of the following disorders: (1) binge eating disorder, a syndrome characterized by episodes in which individuals eat large quantities of food in a short time while experiencing a lack of control and subjective distress, or (2) bulimia nervosa, a syndrome characterized by binge eating combined with compensatory behavior (e.g., purging or excessive exercise) (American Psychiatric Association, 2000; Castonguay, Eldredge, & Agras, 1995). Some research indicates that (a) obese individuals with binge eating disorder or bulimia are at increased risk for depression compared to obese individuals without binge eating behaviors and that (b)

binge eating may account for the relationship observed between obesity and affective consequences (Specker, de Zwaan, Raymond, & Mitchell, 1994; Telch & Agras, 1994). Another possibility is that binge eating reflects greater impulsivity (Rosval et al., 2006), a potential common cause for both obesity and negative affect (see below).

Common Causes

There are also potential common causes of both affective traits and obesity. First, a recent study by Afari and colleagues (2010) suggests that depression and obesity may be related to due shared genetic factors. These researchers found that 12% of the genetic factors associated with depression are shared with obesity (Afari et al., 2010). Conflicting results are also available, such those from Chi Choy et al. (2009), who found no evidence that shared genetic factors were responsible for the positive association observed between obesity indicators and depressive symptoms. Other plausible common causes include low SES (Wang, 2001), chronic physical diseases and physical disability (Liou, Pi-Sunyer, & Laferrere, 2005; Turner & Noh, 1988), older age (Crawford, Jeffery, Ball, & Brug, 2010; Mirowsky & Ross, 1992), female sex (Martin & Ferris, 2007; Paeratakul, Lovejoy, Ryan, & Bray, 2002), and high trait impulsivity (Granö et al., 2007; Mobbs, Crépin, Thiéry, Golay, & Van der Linden, 2010) , all of which have been linked with greater obesity and greater depressive symptoms.

Review of the Affective Traits and Obesity Literature

In defining the potential mechanisms of the bidirectional connections between affective traits and obesity, the plausibility of these relationships is established. The

following sections summarize the broad literature that has linked particular affective traits to obesity indicators and introduce important moderators of the relationships. Because most of the work that has been done focuses on negative affective traits, this topic is introduced first.

Negative Affective Traits and Obesity

Depression is likely the most researched negative affective trait in the obesity literature. Recent reviews and meta-analyses highlight depression's potential role as a cause of obesity, a consequence of obesity, or both (Blaine, 2008; de Wit et al., 2010; Luppino et al., 2010; Markowitz, Friedman, & Arent, 2008). Strong evidence has been compiled for depression as a risk factor for obesity, with reported odds ratios (OR) ranging from 1.18 to 2.57 (Blaine, 2008; Luppino et al., 2010). Depression as an emotional consequence of obesity has also been documented in studies linking obesity with greater risk of developing major depression or elevated depressive symptoms (OR = 1.55) (Carpenter, Hasin, Allison, & Faith, 2000; Gadalla, 2009; Luppino et al., 2010; Onyike, Crum, Lee, Lyketsos, & Eaton, 2003). In addition to depression, other negative affective traits have also shown positive, bidirectional relationships with obesity (Ahlberg et al., 2002; Gadalla, 2009; Gariepy et al., 2010). For example, prospective and cross-sectional research indicates that anxiety is associated with measures of obesity (Ahlberg et al., 2002; Nelson, Palmer, Pedersen, & Miles, 1999; Wing et al., 1991). Anxiety has also been identified as a possible consequence of obesity, as prospective studies have demonstrated increased risk for anxiety disorders in obese individuals (odds ratio = 1.02-1.46) (Gariepy et al., 2010). Although less research is available, investigators have found

positive associations between hostility/anger and increased BMI, waist-to-hip ratio, visceral fat, and waist circumference (Everson et al., 1997; Lewis et al., 2009; Niaura et al., 2002). In their meta-analytic analysis of cross-sectional studies examining hostility with BMI and/or WHR, Bunde and Suls (2006) reported Hedge's *g* effect sizes ranging from .15 to .25 using fixed and random effect models (Bunde & Suls, 2006). Prospective evidence also suggests that hostility and anger predict change in obesity outcomes over time (Midei & Matthews, 2009; Nelson et al., 1999; Räikkönen, Matthews, & Kuller, 1999); however, no prospective studies have been identified regarding obesity's potential influence on hostility/anger. It is also important to acknowledge that a contradictory line of research exists that does not support the positive association between obesity and negative affective traits. Indeed, these findings indicate that overweight and/or obesity have also been associated with reduced risk for depression and/or anxiety (Bin Li et al., 2004; Carr, Friedman, & Jaffe, 2007; Crisp & McGuiness, 1976; Palinkas, Wingard, & Barrett-Connor, 1996). When explicitly tested, these inverse relationships between increased adiposity and negative affective traits have failed to find support in some studies (Roberts, Kaplan, Shema, & Strawbridge, 2000; Roberts et al., 2002).

Potential Demographic Moderators of the Negative Affective Traits-Obesity Relationships

Although many studies have found positive associations of depression, anxiety, and hostility with obesity, research indicates that these relationships may be moderated by gender and race-ethnicity. Whereas a positive relationship is consistently found for women, some investigations find negative, weak, or null associations between depression

and obesity in men (Anderson, Cohen, Naumova, & Must, 2006; Carpenter et al., 2000; de Wit et al., 2010; Istvan, Zavela, & Weidner, 1992; Onyike et al., 2003). Similar patterns have been shown for anxiety and obesity, as positive relationships are more consistently detected in female samples than in male samples (Anderson et al., 2006; Jorm et al., 2003; Schieman et al., 2007). Evidence to the contrary has also been documented; some studies indicate that depression or anxiety are associated with obesity only in males (Bjerkeset, Romundstad, Evans, & Gunnell, 2008; Hach et al., 2007; Mustillo et al., 2003) or that no gender differences in the relationship have been detected (Haukkala & Uutela, 2000; Herva et al., 2005; Luppino et al., 2010). A limitation of the aforementioned research is that males are sometimes underrepresented in the study samples.

Race-ethnicity has also been identified as a potential moderator of the negative affective traits-obesity association. A recent study detected a depression by race interaction effect among women, such that obesity was associated with major depression in white women but not in African American, Hispanic, or Asian women (Gavin, Rue, & Takeuchi, 2010a). In this nationally representative sample of greater than 16,000 adults, obesity was not related to depression, and no depression by race interactions were found for men. Moderation by race-ethnicity has also been detected among adolescents. Anderson and colleagues (2011) found that depression was prospectively related to obesity and vice versa for white female adolescents but not for black or Hispanic females. These findings contradict evidence in which comparable effects have been found for whites and blacks (Carpenter et al., 2000; Heo, Pietrobelli, Fontaine, Sirey, & Faith, 2005) and studies that have found nonsignificant affective trait by race-ethnicity

interactions (Franko, Striegel-Moore, Thompson, Schreiber, & Daniels, 2005). As other researchers have noted (Gavin et al., 2010a), these discrepant findings may be due to the poor representation (i.e., small sample size) of minorities in studies that fail to find significant interaction effects. In conclusion, although the relationship appears to be strongest and most consistent among white, non-Hispanic women, extant evidence suggests that the negative affective traits-obesity relationship is not fully understood, especially for men and racial minorities, which are not as well-represented in the studies.

Positive Affective Traits and Obesity

An additional shortcoming of the affective traits-obesity literature is that research regarding positive affective traits is scarce and less developed than the available evidence on negative affective traits. The dearth of studies is somewhat surprising, given the suggested benefits of trait positive affect on other health outcomes, including increased longevity and less medical morbidity (Pressman & Cohen, 2005). The research that is available on the positive affect-obesity relationship is contradictory. Among the studies that have been conducted, some suggest that obesity is associated with less trait positive affect and optimism as well as poorer life satisfaction (Jorm et al., 2003; Roberts et al., 2002; Ryff et al., 2006). Another set of studies found no relationship between obesity and positive affective traits (Dierk et al., 2006; Doll, Petersen, & Stewart-Brown, 2000). Because affective traits are typically studied in isolation, it is not clear whether positive affective traits have associations with obesity that are independent of negative affective traits or vice versa.

The Issue of Overlapping Affective Traits and Obesity

The preceding section introduced an additional issue that remains unresolved within the affective trait-obesity literature: Although multiple negative and positive affective traits have been associated with obesity, these factors are rarely examined together in the same study. This limitation is important, as the considerable overlap among affective traits poses problems when attempting to interpret and apply evidence from this literature. Theoretically, depression, anxiety, and hostility/anger are distinct phenomena; however, these affective traits are moderately to highly correlated. For instance, Mook and colleagues (1990) observed the following correlations in a sample of 624 healthy adults: (1) $r = .73$ for depression and anxiety (2) $r = .22$-$.27$ for depression and hostility, and (3) $r = .37$-$.49$ for anxiety and hostility. These substantial correlations point to a common factor underlying the three traits. Indeed, evidence supports the existence of such a trait, often labeled negative affectivity (Marsland, Prather, Petersen, Cohen, & Manuck, 2008; Mook et al., 1990; Watson & Clark, 1984), which has been defined as the dispositional propensity to experience negative affect (Costa & McCrae, 1985; Suls & Bunde, 2005; Watson & Clark, 1984).

Examining depression, anxiety, and hostility separately ignores the substantial overlap of these traits and obscures these factors' relative and potentially additive and interactive influences on obesity development (Kaplan, 1995). Models outlining how risk factors might act together to influence an outcome have been proposed (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001), and these models can be adapted in order to evaluate the issues raised in the proposed study. First, the independent model suggests that negative affective traits each have unique effects on obesity development (Figure

2a). If this model is correct, obesity prevention efforts that contain modules for each negative trait would likely be more effective than those that focus on only one or two. In contrast, the overlapping model proposes that a common underlying factor, such as negative affectivity, is responsible for the associations between the individual affective traits and obesity development (Figure 2b). In this scenario, prevention efforts that target negative affectivity may be more successful than individual depression, anxiety, or hostility treatments. Lastly, the proxy model suggests that a particular negative affective trait is predictive of obesity development solely due to its strong correlation with another affective trait that is a true risk factor (Figure 2c). If one assumes that anxiety is a proxy risk factor and thus predicts obesity development only because of its close relationship with depression, then treating anxiety would likely not yield the same reduction in obesity risk, if any, as treating depression.

Similarly, obesity may have independent, overlapping, or proxy influences on negative affective traits. First, depression, anxiety, and hostility may each be unique consequence of excess adiposity (Figure 2d). Second, the overlapping model implies that excess adiposity does not directly influence each individual affective trait but instead may incite the general disposition to experience negative affect (Figure 2e). Third, the proxy model suggests that obesity may influence one trait in particular, a unique affective consequence (Figure 2f). Obesity only appears to influence another trait because of this trait's strong association with the unique affective consequence.

Although previous studies have linked depression, anxiety, and hostility with various indicators of adiposity, none have elucidated which of these three factors may be *independently* associated with these outcomes. The only available study which provides

a direct test of the aforementioned models examined depression and anxiety as predictors of obesity (van Reedt Dortland, Giltay, van Veen, Zitman, & Penninx, 2013). The authors found that anxiety was a proxy for depression which was the actual predictor of abdominal obesity (Figure 1c). Given this dearth of studies which directly test the aforementioned models with all three affective traits, a direct evaluation of the competing models is not possible. Instead, as a first step toward evaluating these models, I sought to answer the following question: do depression, anxiety, and hostility/anger have independent effects on excess adiposity? To accomplish this objective, I recently conducted and published a systematic literature review of studies examining multiple negative affective traits in relation to adiposity indicators (Figure 2a-c) (Hawkins & Stewart, 2012). Results indicating which emotional factors may independently contribute to obesity development were used as evidence to suggest which risk factor model may best represent the emotional factors–obesity relationships. Of note, the study by van Reedt Dortland et al. (2013) was published after the review and was not included. The methods and findings of this review are summarized below.

Negative Affective Traits as Independent Predictors of Adiposity: A Review

A comprehensive literature search for articles was conducted to answer the following question: Are negative affective traits independent, overlapping, or proxy risk factors for increased adiposity? Studies examining affective traits and adiposity indicators were identified using two methods: (a) computer search of the PsycINFO, EMBASE, and MEDLINE databases using the keywords identified below; and (b) a manual search of the reference sections of previous reviews and empirical studies. The

set of keywords for depression was *depression, depressive, dysthymia, major depression,* and *dysphoria.* The set of keywords for anxiety was *anxiety, anxiety disorder, fear, phobia,* and *worry.* The set of keywords for hostility/anger was *hostility, anger,* and *aggression.* For adiposity indicators, the following set of keywords was used: *obesity, obese, adiposity, adipose, fat, body mass index (BMI), waist circumference, waist to hip ratio (WHR), body composition, body density, skinfolds, abdominal diameter, dual energy X-ray absorptiometry (DEXA), bioelectrical impedance (BIA), BOD POD, body mass, densitometry, hydrometry, hydrostatic weighing,* and *underwater weighing.*

I selected studies that examined the relationship between a negative emotional factor and an adiposity indicator while controlling for at least one other emotional factor. If a particular emotional factor remained associated with an adiposity indicator after adjustment for another emotional factor (i.e., adjusted analyses) I considered this evidence that the emotional factor had an independent effect on adiposity. I identified 3 prospective and 11 cross-sectional studies that met our inclusion/exclusion criteria (Hawkins & Stewart, 2012) . Of note, Chou and colleagues reported the associations of social phobia and panic disorder with BMI in two separate articles (Chou, 2009, 2010); however, because analyses were conducted on the same sample, they were counted as one study.

Sample sizes of the selected prospective studies ranged from 285 to 572 individuals with follow-up periods ranging from 1 to 6 years, whereas sample sizes of cross-sectional studies ranged from 145 to 78,866 individuals. Relevant characteristics of the selected studies are presented in Appendix A. The results of the unadjusted and adjusted analyses of these studies are shown in Appendix B and summarized below.

Independent Effects of Depression on Adiposity

Three prospective studies were identified that examined depression as a predictor of an adiposity indicator using adjusted analyses, two of which examined depression while controlling for anxiety (Bardone et al., 1998; Chiriboga et al., 2007). In a sample of adolescent girls, Bardone et al. (1998) observed that depressive disorder diagnosis at age 15 did not predict BMI at age 21 in the presence of anxiety disorders. Because the results of unadjusted analyses were not reported, it cannot be determined whether depression predicted BMI in this sample before adjustment for anxiety. Similarly, Chiriboga et al. (2007) found that depressive symptoms did not predict weight gain over 1-year period when controlling for anxiety symptoms in a sample of adult men and women. Depressive symptoms also failed to predict weight gain in a model that did not include anxiety symptoms. The remaining study examined the unique influence of depressive symptoms on 3-year change in BMI among middle-aged men and women (Haukkala, Uutela, & Salomaa, 2001). Results revealed that depression predicted BMI increases over time, even in the presence of hostility. In summary, only one prospective study reported that depression was an independent predictor of adiposity after controlling for hostility, while the results of the two other studies suggest that depression may not be a unique predictor in the presence of anxiety.

Eight cross-sectional studies have examined associations between depressive disorders or symptoms and adiposity indicators using adjusted analyses. A total of 75% of these studies (6/8) reported that depression was independently and positively associated with an adiposity indicator (i.e., overweight or obese BMI, waist circumference, body fat, or weight adjusted for height). Five of these studies found that

depression continued to be positively associated with adiposity indicators after adjustment for anxiety (Leventhal et al., 2010; Mather, Cox, Enns, & Sareen, 2009; Simon et al., 2006; Toker, Shirom, & Melamed, 2008; Williams et al., 2009), and one study observed that depression was positively related to visceral body fat after adjustment for hostility (Lewis et al., 2009). In contrast, 25% of the studies (2/8) did not detect an independent association between depression and adiposity indicators (see Appendix B). These two studies found that depressive disorders were no longer related to self-reported BMI when anxiety disorders were entered into the models (Pickering, Grant, Chou, & Compton, 2007; Scott, McGee, Wells, & Oakley Browne, 2008). Thus, most of the cross-sectional studies examining depression in the presence of anxiety or hostility indicate that depression has positive, independent associations with adiposity. In fact, 64% of all reviewed studies suggest that depression is independently associated with adiposity (see Appendix B).

Independent Effects of Anxiety on Adiposity

Two prospective studies examined anxiety as a predictor of changes in adiposity over time using adjusted analyses and provide mixed evidence regarding the independent effects of anxiety. On the one hand, Bardone et al. (1998) found that, among female adolescents, anxiety disorders did not predict BMI six years later in the presence of depressive disorders. On the other hand, Chiriboga et al. (2007) observed that anxiety symptoms predicted 1-year weight gain in models with and without depressive symptoms among men but not women. These results suggest that anxiety may be an independent predictor of BMI in men.

Seven cross-sectional studies were identified that have examined independent associations between anxiety disorders or symptoms and adiposity indicators, all of which adjusted for either depressive disorders or symptoms. Of these studies, 57% (4/7) indicated that anxiety remained related to overweight or obese BMI after controlling for depression (Mather et al., 2009; Pickering et al., 2007; Scott et al., 2008; Strine, Chapman, Kobau, & Balluz, 2005), whereas 43% (3/7) found that anxiety was no longer related to obese BMI, waist circumference, or body fat in the presence of depression (Chou, 2009; Simon et al., 2006; Williams et al., 2009). To summarize, the majority of the cross-sectional studies examining anxiety while adjusting for depression indicate that anxiety is independently and positively associated with adiposity indicators. Indeed, 56% of all prospective and cross-sectional studies indicate that anxiety is independently related to excess adiposity (see Appendix B).

Independent Effects of Hostility/Anger on Adiposity

Only one prospective study examined relationship between hostility/anger and adiposity indicators using adjusted analyses. In a sample of middle-aged adults, Haukkala et al. (2001) observed that hostility was not a predictor of 3-year change in BMI when controlling for depressive symptoms. Hostility also failed to predict BMI change in a model that did not include depressive symptoms. Two cross-sectional studies also performed adjusted analyses involving hostility/anger. In a sample of women, Lewis et al. (2009) determined that hostility remained positively related to visceral body fat after adjustment for depressive symptoms, although a separate analysis indicated that hostility was not related to subcutaneous fat. In contrast, Fassino et al. (2003) found that

trait anger was associated with obese BMI in unadjusted analyses; however, this relationship became nonsignificant in the presence of depressive symptoms. Collectively, these results suggest that hostility may not be independently related to adiposity, as only 33% of studies suggest that hostility is independently associated with excess adiposity (see Appendix B). However, the dearth of studies precludes one from drawing a firm conclusion and underscores the need for additional investigations in this area.

Review Conclusion

The results of the review (Hawkins & Stewart, 2012) support the notion that depression is independently associated with excess adiposity, as 64% of studies indicate that depression retained its relationship with adiposity indicators after controlling for either anxiety or hostility/anger. A smaller percentage (56%) of studies, although still a majority, found that anxiety remained associated with adiposity indicators in the presence of depression, suggesting that this construct may also have an independent effect on excess adiposity. In contrast, only 33% of the three identified studies reported evidence of an independent relationship between hostility/anger and an adiposity indicator. The unique role of hostility/anger, however, remains inconclusive due to an insufficient empirical literature.

This qualitative review makes an important contribution to the literature by indicating that both depression and anxiety may have unique influences on adiposity, consistent with the independent model in Figure 2a. A sizable minority of selected studies, however, do not support this model. Unfortunately, most of these studies did not

present all of the information needed to establish which of the competing models (i.e., the overlapping or proxy model) was supported. For example, several studies did not report the unadjusted and adjusted results for all examined factors (see Appendix B). Of the investigations that did include the required information, a few studies supported the proxy model (see Figure 2c), although the results were mixed as to which affective traits were proxies. For instance, the results of Simon et al. (2006) suggest that anxiety is a proxy for depression, whereas the results of Scott et al. (2008) indicate the opposite. These inconsistent findings highlight the need for further exploration of the overlapping and proxy models. Although the current review suggests that the independent model garners the most support with regards to depression and anxiety, the role of hostility/anger in these models has yet to be determined. Future studies may reveal that a hybrid model, which acknowledges the influence of both the shared and unique aspects of negative affective traits, may provide the most accurate representation of relationships between these factors and adiposity.

In addition to the conflicting results and small number of studies, which limit the ability to draw firm conclusions, other limitations of this review should be noted. First, with only three prospective studies identified, the directionality of independent associations between the affective traits and adiposity indicators could not be determined. Second, the majority of the included studies used self-reported BMI (see Appendix A). BMI is a crude estimate of total body fat and investigators have found that affective traits may be more strongly related to fat accumulation in particular regions, such as centrally located adiposity, than to total adiposity (Lewis et al., 2009; Wing et al., 1991). Thus, the ability to detect an independent effect between a particular affective trait and excess

adiposity may, in part, depend on the adiposity assessment employed. Third, because many of the samples were not racially diverse or were predominantly female, the generalizability of the available evidence may be called into question. Fourth, a review of the research examining the influence of positive factors on obesity when controlling for negative affective traits was not performed, given that the available evidence was too limited. The underdeveloped evidence regarding the independent effects of trait positive affect is a deficit in the literature for two reasons: (1) trait positive affect could be a potential protective factor against obesity, and (2) although positive and negative affective traits are distinct constructs, measures of trait positive affect have been shown to correlate inversely with trait negative affectivity and specific negative affective traits (Diener & Emmons, 1984; Watson, Clark, & Carey, 1988a; Watson, Clark, & Tellegen, 1988b). For instance, trait positive affect has shown moderate correlations with depressive disorders (r = -.38) and small correlations with anxiety disorders (r = -.12) (Watson et al., 1988a). In contrast, reported correlations between positive affect and hostility/anger are weak (r = -.03 to -.09) (Harmon-Jones, 2003). Nonetheless, the existing overlap suggests that researchers should examine trait positive affect in conjunction with overlapping negative affective traits.

Negative Affective Traits as Unique Consequences of Adiposity: A Summary

An issue not addressed by the above systematic review (Hawkins & Stewart, 2012) was whether excess adiposity has independent, overlapping, or proxy influences on

the expression of negative affective traits (Figure 2d-f). This issue was not covered in the review given the limited evidence available. A summary of the small number of existing studies is supplied below.

Depression as a Unique Consequence of Excess Adiposity

Only one prospective study was identified that performed adjusted analyses in which the influence of obesity on depression was examined while controlling for anxiety. In a sample of white children aged 9-16 years, BMI percentile cut-points for obesity, depressive disorders, and anxiety disorders were assessed. In models adjusting for age, sex, race as well as other psychiatric disorders (including anxiety disorders), Mustillo et al (2003) found that, among girls, obesity did not predict an increased risk for depressive disorders. In contrast, the authors found an increased risk for depressive disorders among chronically obese boys in similarly adjusted models. Because obesity remained a predictor of depression among boys after adjusting for anxiety, these results indicate that depression may be a unique consequence of obesity in males. For females, obesity was not a predictor of depression after adjusting for anxiety. However, because the authors did not report the unadjusted relationship between obesity and future depression, it is possible that depression was not related to obesity in this sample of females before anxiety adjustment. This study including only white children may not be applicable to minority children, especially in light of evidence that African Americans may experience less of weight-related stigma than Caucasians (Latner, Stunkard, & Wilson, 2005).

Anxiety as a Unique Consequence of Excess Adiposity

Two prospective studies were identified that examined obesity's influence on anxiety while controlling for depression. Specifically, Kasen and colleagues (2008) followed a sample of 544 women. BMI was assessed at baseline in 1975 and at follow-up in 2002-2005, whereas a diagnosis of generalized anxiety disorder (GAD) or major depressive disorder (MDD) was determined only at follow-up 2002-2005 using DSM-IV criteria. After adjusting for covariates, which included a continuous measure of depressive symptoms obtained in 1983, obesity (BMI $\geq$ 30 kg/m^2) still predicted an increased risk for GAD in 2002-2005. These findings suggest that obesity status has unique effects on anxiety, independent of previous depressive symptoms. In contrast, Mustillo did not find that obesity was a predictor of anxiety in males or females when examined in conjunction with depression and other psychiatric disorders. This pattern of results implies that obesity does not have any unique effects on anxiety after taking into account the influence of depressive symptoms. However, it also possible that anxiety was not related to obesity in unadjusted analyses, but these results were not reported. Because the Mustillo et al. (2003) study examined only white children and approximately 97% of participants in the Kasen et al. (2008) study were Caucasian and 100% were female, these findings may not generalize to minorities and men.

Hostility/Anger as a Unique Consequence of Excess Adiposity

No prospective studies were identified that examined the impact of adiposity indicators on hostility. In spite of the evidence that hostility is moderately correlated with depression and anxiety (Mook et al., 1990; Suls & Bunde, 2005), the absence of a

clear diagnostic category for hostility in the Diagnostic and Statistical Manual of Mental Disorders (fourth edition) (American Psychiatric Association, 2000) has likely led to a neglect of this construct in the literature.

Summary Conclusion

To conclude, the available literature on whether excess adiposity has independent, overlapping, or proxy influences on the expression of negative affective traits is very limited (three studies), does not examine hostility, and provides conflicting results. Consequently, the literature at this time does not lend itself to the identification of the most accurate risk model (Figure 2d-f).

To synthesize all the presented literature on affective traits and adiposity, the preliminary evidence suggests that depression and anxiety may be independent predictors of excess adiposity when controlling for other negative affective traits (Hawkins & Stewart, 2012). Additional studies are required to determine whether hostility/anger is an independent predictor of adiposity. Collectively, these findings are most consistent with the independent model of interrelated risk factors shown in Figure 2a, although conflicting findings support further evaluation of the competing overlapping and proxy models (see Figure 2b and c) and raise the possibility that a hybrid model may provide the best fit to the data. With regards to the adiposity-to-affective traits relationship, the paucity of evidence precludes identification of best model from Figures 2d, 2e, and 2f. More prospective studies are needed that (1) examine multiple negative and positive affective traits as predictors and consequences of adiposity, (2) utilize measurements of

adiposity other than BMI and (3) have adequate representation of minorities and males in order to establish which affective traits are independent predictors and consequences of obesity.

Present Study

This investigation seeks to address the identified gaps in the affective traits-obesity literature. Specifically, this study analyzed data collected as part of the African American Health (AAH) study to investigate the prospective, bidirectional associations between multiple affective traits and multiple adiposity indicators in African Americans. The AAH study is well-suited for this purpose, as it is a 9-year prospective cohort study of nearly 1,000 community dwelling African Americans that contains measures of several affective traits and adiposity indicators over time.

Primary Objectives

Given that racial minorities have been underrepresented in past investigations, more prospective studies are needed which examine affective trait-obesity relationships in these populations. Thus, *Objective #1* of the present study was to examine bidirectional associations between depressive symptoms and adiposity indicators over a 9-year period in African Americans. To achieve this aim, the following hypotheses were tested (see Figure 3):

> *Hypothesis #1: Depressive Symptom Severity as a Predictor of Adiposity*
> Greater baseline severity of depressive symptoms is associated with greater 9-year increases in adiposity as measured by BMI or body fat percent.

Hypothesis #2: Depressive Symptom Severity as a Consequence of Adiposity
Greater baseline BMI or body fat percent is associated with greater 9-year increases in depressive symptom severity.

A second limitation of this literature is that more prospective studies employing multiple measures of affective traits are needed in order to determine the independent associations of specific affective traits with adiposity indicators. Accordingly, *Objective #2* was to investigate whether depression and anxiety exhibit independent associations with changes in adiposity indicators in African Americans. Of note, only the 7-year and 9-year follow-up periods of the AAH study contain measures of depressive *and* anxiety symptoms, so 7-year follow-up scores served as "baseline" values predicting follow-up values two years later (i.e., 9-year follow-up period). To achieve this aim, the following hypotheses were tested (see Figure 4):

Hypothesis 3: Depressive Symptom Severity as an Independent Predictor of Adiposity.
Greater baseline severity of depressive symptoms is associated with greater 2-year increases in BMI, independent of anxiety symptoms.

Hypothesis 4: Anxiety Symptom Severity as an Independent Predictor of Adiposity.
Greater baseline severity of anxiety symptoms is associated with greater 2-year increases in BMI, but this association is not independent of depressive symptoms.

Hypothesis 5: Depressive Symptoms Severity as Unique Consequence of Adiposity.
Greater baseline BMI is associated with greater 2-year increases in depressive symptoms, independent of anxiety symptoms.

Hypothesis 6: Anxiety Symptoms Severity as Unique Consequence of Adiposity.
Greater baseline BMI is associated with greater 2-year increases in anxiety symptoms, but this association is not independent of depressive symptoms.

If the affective traits-obesity relationships are found to differ for African Americans, compared to other ethnic groups, this knowledge could be used to inform

racially-sensitive obesity prevention and treatment efforts. For instance, some studies summarized above suggest that the depression-obesity relationship may be weaker or not present among African Americans. If Hypotheses 1, 2, 3, and 5 are found to be null, it would suggest that African Americans may require obesity prevention or intervention programs with little to no emphasis on depressive symptoms. Similarly, if Hypotheses 4 and 6 are found to be null, the findings would suggest that anxiety may not be an important target for preventing or treatment obesity in this population.

Secondary Objectives

An additional topic that has received limited attention is whether positive affective traits are related to adiposity indicators, independent of negative affective traits. This deficit in the literature is unfortunate, as evidence suggests that positive affective traits may protect against disease risk (Pressman & Cohen, 2005; Steptoe & Wardle, 2005). Accordingly, the current study also had the following secondary objectives to determine whether: (1) measures of trait positive affect are predictors of change in adiposity indicators, (2) a reduction in trait positive affect is a consequence of excess adiposity, and (3) the associations, if present, are independent of the associations between negative affective traits and adiposity. The positive affective traits of interest are vitality (i.e., the presence of energy and absence of fatigue) and positive affect (see Figures 5 and 6).

METHOD

Participants

Sample Recruitment and Selection

The AAH study is a prospective cohort study of 998 African Americans aged 49-65 years at baseline living in the St. Louis metropolitan area. Two catchment areas of the city – a poor, inner-city location and the suburbs located northwest of the city – were sampled. Geocoding was used to identify area blocks to target for recruitment. Random selection was first used to select street segments within the identified area blocks and then used to select housing units within each street segment. In order to be eligible for the study, participants had to meet the following criteria: (1) self-reported black or African American race, (2) a birth date between January 1936 and December 1950, (3) a score ≥ 16 on the standardized Mini-Mental Status Examination (Molloy et al., 1996), and (4) completion of informed consent. If two or more eligible persons within a household were identified, then one was selected using Kish tables (Kish, 1965). Of the eligible persons identified, 76% (998 of 1,320) were enrolled in the study from 2000 to 2001 (baseline). Of the enrolled participants, 85% (n = 853) were reassessed in 2003-2004 for 3-year follow-up, 68% (n = 680) in 2007-2008 for 7-year follow-up, and 58%

(n = 579) in 2009-2010 for 9-year follow-up. For the purpose of this study, the baseline, 7-year, and 9-year follow-up periods were examined. The Institutional Review Board of Saint Louis University approved all procedures, and all participants signed written informed consents during the face-to-face interviews. Characteristics of participants across the study periods are presented in Tables 2 and 3 and their chronic disease history in shown in Table 4. Changes in adiposity and affective traits from baseline to the follow-up periods are also presented (see Table 5). As can been seen in Table 5, paired t-tests were run to examine significant changes between baseline affective traits and adiposity and their corresponding follow-up values.

Measures

Adiposity Assessment

The AAH study contains multiple measures of adiposity, including anthropometric (self-reported and measured BMI) and body composition measures (body fat percent; BF%). Only self-reported BMI data were obtained at baseline and all follow-up periods (see Table 6). Consequently, self-reported BMI was used as the primary indicator of adiposity when testing the hypotheses. When possible, analyses using self-reported BMI were rerun using measured BMIs and/or BF% when these data were available, as they provide more objective assessment of adiposity.

Participants' self-reported BMI was calculated as kg/m^2 using height and weight disclosed during the in-home evaluations at baseline, 7-year follow-up, and 9-year follow-up. Measured BMI was obtained at baseline and 9-year follow-up using measured

height and weight. Height (without shoes) was assessed by asking participants to stand erect against a doorframe to which a measuring tape had been applied, whereas weight (without shoes and stockings) was measured using a Tanita portable, electronic impedance scale (Tanita Corporation of America; Arlington Heights, IL) placed on a hard flat surface. BF% was measured using bioelectrical impendence analysis (BIA) at baseline and 9-year follow-up using the Tanita scale adjusting for sex, height, and weight. The Tanita scale passes a low level electrical current through the body, and the extent to which the current is impeded is used to estimate fat-free mass and fat mass. Given that fat mass is less conductive than fat-free mass, it is possible to derive an algorithm using the degree of impedence and participants' sex, height, and weight, which the Tanita scale uses to generate an estimate of body fat percent (Kyle et al., 2004). BF% was also measured for a subset of participants at baseline using dual X-ray absorptiometry (DEXA). A full-body scan of 204 participants (20.4%) was conducted using a DEXA scanner (Hologic QDR 4500W, Bedford, MA), which generated estimates of participants total mass, fat mass, fat-free soft mass, and bone mineral content. From these estimates, BF% was then calculated by dividing participants' fat mass (excluding head) by their total mass (excluding head).

Although BF% as measured by DEXA is considered a gold standard measure of adiposity due to its precision for assessing fat mass versus fat-free mass (Erselcan, Candan, Saruhan, & Ayca, 2000), it was not used to test the hypotheses for two reasons: (1) DEXA estimates of BF% were only available at baseline for a small percentage of participants and (2) BMI and BF% as measured by BIA provide adequate estimates of adiposity, a claim substantiated by previous data as well as the current study.

Specifically, correlations among adiposity indicators are generally high. Reported correlations between measures of BF% (i.e., BIA and DEXA) are strong, with *r*s typically greater than .90 (Svendsen, Haarbo, Heitmann, Gotfredsen, & Christiansen, 1991; Tyrrell et al., 2001). Correlations between BMI and body fat percent are more modest and can differ by age and sex groups (r = .40-.89) but are still moderate-to-strong (Deurenberg, Weststrate, & Seidell, 1991). In this sample, all adiposity indicators were strongly correlated with baseline DEXA scores (*r*s = .61 to .84) (see Table 7); thus, it was deemed reasonable to use BMI and BF% measured by BIA as adiposity indicators when testing the hypotheses. Given that BMI and BF% were operationalized using single values, no imputation for missing data was performed.

Importantly, obesity has been defined categorically using BMI or percent body fat cut-points (Gallagher et al., 2000; World Health Organization, 1995; Zhu, Wang, Shen, Heymsfield, & Heshka, 2003). However, the current study used continuous BMI and BF%. This approach is justified not only because it maximizes variation in the adiposity variables, but also because degree of adiposity has shown a more or less linear relationship with increased risk of health problems (Folsom et al., 1993; Willett et al., 1995; World Health Organization, 2000). Indeed, overweight BMI is such a strong predictor of subsequent development of obese BMI that it has even been termed "pre-obesity" (Guo et al., 2000; Magarey, Daniels, Boulton, & Cockington, 2003), and both overweight and obese individuals are considered to have excess adiposity.

Affective Traits Assessment

The AAH study also contains multiple measures of affective traits which were used in the current study. These traits and their associated measures are described below. Correlations among the affective traits are presented (Table 7) as well as the internal consistencies of the scales (Table 8). Appendix C contains the actual scale items and/or interview questions used to assess the affective traits measures.

Depressive symptoms

Depressive symptoms were assessed at baseline, 7-year follow-up, and 9-year follow-up with the 11-item Center for Epidemiologic Studies-Depression Scale (CES-D) (Kohout, Berkman, Evans, & Cornoni-Huntley, 1993). For all items, participants were asked to indicate how often they experienced various symptoms during the last week using a scale ranging from 0 (*rarely* or *none of the time*) to 3 (*most or all of the time*). Higher total scores indicate greater depressive symptom severity. The CES-D has been shown to have good construct validity, as it has strong correlations with clinician ratings and other self-report measures of depression (Radloff, 1977; Weissman, Sholomskas, Pottenger, Prusoff, & Locke, 1977). The 11-item CES-D has also demonstrated good internal consistency (Cronbach's α = .76-.81). The eleven items were selected from the original 20-item scale, given their high factor loadings on one of the four factors identified by Radloff (1977), including depressed affect, positive affect, somatic symptoms, and interpersonal problems. This four-factor structure of the 20-item scale has been confirmed in African American samples (Nguyen, Kitner-Triolo, Evans, & Zonderman, 2004; Williams et al., 2007), and the 11-item CES-D has demonstrated the

same four-dimensional structure (Kohout et al., 1993; Shafer, 2006). In this sample, a principal components analysis with orthogonal varimax rotation was used to extract and confirm the four factors, equivalent to the procedure used by Radloff (1977) and Kohout et al. (1993). The four-factor structure of the 11-item CES-D was mostly replicated (see Table 9) and explained 65.5% of the variance. Exceptions to the original structure were that the "get going," "effort," and "restless sleep" items loaded more strongly on the depressed affect factor than the somatic symptoms factor. Only the "appetite" item loaded strongly on the somatic symptoms subscale. All other factor loadings were similar to those reported in other studies.

For both follow-up periods, two additional CES-D items were added to the positive affect subscale resulting in 13 total CES-D items. Importantly, because the positive affect subscale of the CES-D was used as a separate indicator in the statistical models (see Trait Positive Affect section below), the items associated with this subscale were removed from the CES-D total score to avoid singularity in the models, resulting in a 9-item total subscale score for depressive symptoms. Cronbach's alpha for this 9-item depressive symptoms subscale ranged from .80 to .82 across baseline and the two follow-up periods (Table 8). For each participant missing ≤ two items, missing values were imputed with mean substitution using the available items from the 9-item scale.

Anxiety symptoms

Anxiety symptoms were assessed at the 7-year and 9 year follow-up periods using the Generalized Anxiety Disorder-2 (GAD-2) scale (Kroenke, Spitzer, Williams, Monahan, & Löwe, 2007). Participants were asked to indicate how often they felt

anxiety symptoms over the past two weeks using a scale ranging from 0 (*not at all*) to 3 (*nearly every day*). Higher total scores indicate greater anxiety symptom severity. The 2-item GAD-2 is a shortened version of the GAD-7 (Spitzer, Kroenke, Williams, & Lowe, 2006), a widely used screening tool for anxiety disorders. The two items from the GAD-2 were selected because they represent the core anxiety criteria (Criteria A and B) for GAD in the DSM-IV-TR (American Psychiatric Association, 2000). In support of its construct validity, the GAD-2 has shown adequate sensitivity (.59-88%) and specificity (81-.83%) for detecting anxiety disorders (Kroenke et al., 2007). In addition, area under the curve analyses indicate that the GAD-2 and GAD-7 have similar operating characteristics for detecting anxiety disorders, with the exception of PTSD for which GAD-7 is a better detector. The GAD-2 has shown adequate internal consistency in previous studies (Cronbach's $\alpha = .75$) (Löwe et al., 2010) as well as in the current study (Cronbach's $\alpha = .78$-$.79$) (Table 8). Participants with missing data were missing both items, so the missing data was not imputed.

Trait Positive Affect

Two measures were obtained that assessed aspects of trait positive affect. First, the 4-item Vitality subscale of the Short Form-36 (SF-36) from the Medical Outcomes Study (Ware, Kosinski, Dewey, & Gandek, 2000) was administered at baseline and both follow-up periods. The vitality construct has been conceptually defined as the presence of energy and "aliveness" and the absence of weariness or exhaustion. This conceptual definition overlaps with the definition of positive affect provided by Watson, Clark, and Carey (1988), which states that positive affect "is composed of…energy level, mental

alertness, interest…. whereas low PA [positive affect] is best denned by descriptors reflecting lethargy and fatigue" (p. 347). Although the SF-36 Vitality subscale focuses more strongly on the somatic activation component of positive affect, this subscale has demonstrated positive correlations (r = .29-.60) with measures tapping the cognitive-emotional aspects of positive affect (e.g., Trait Positive and Negative Affect Schedule and Mental Vitality) (Nagurney, 2007; Pincus, Williams, Vogel, & Field, 2004; Richman, Kubzansky, Maselko, Ackersond, & Bauer, 2009). The Vitality subscale consists of four items assessing how often the individual "felt full of life," "had a lot of energy," "felt worn out," and "felt tired" during the past 4 weeks using a scale ranging from 1 (*all of the time*) to 5 (*none of the time*). In previous literature, higher subscale scores typically indicate less vitality and more exhaustion; however, for the current study, scores were reversed so that higher scores represent greater vitality levels to promote easier interpretation of the results. The Vitality subscale has shown good internal consistency (Cronbach's α = .80) and adequate test-retest reliability coefficients (r = .61-.63) over a 2-3 week follow-up (Marx, Menezes, Horovitz, Jones, & Warren, 2003; Stadnyk, Calder, & Rockwood, 1998). In this sample, Cronbach's alpha ranged from .81 to. 83 (Table 8). For each participant missing ≤ one item, missing values were imputed with mean substitution using the available items from the 4-item scale.

The second measure of positive affective traits is the four-item Positive Affect subscale from the CES-D. This four-item scale is available for the 7-year and 9-year follow-up periods. Participants were asked to indicate how often they experienced positive affective traits (e.g., happiness and life satisfaction) during the last week using a scale ranging from 0 (*rarely* or *none of the time*) to 3 (*most or all of the time*). Higher

subscale scores indicate greater positive affective traits. The presence of a positive affect factor for the CES-D has been supported in African Americans (Nguyen et al., 2004; Williams et al., 2007) and white samples (Hertzog, Van Alstine, Usala, Hultsch, & Dixon, 1990). Indeed, when a principal components analysis using varimax rotation to extract four factors was performed on this sample, the presence of a positive affect factor was supported. Rotated factor loadings of the two positive affect items on the positive affect factor were .68 and .88 (see Table 9). The Positive Affect subscale has been found to predict a variety of health outcomes, independently of the other CES-D subscales, which establishes its validity as a unique correlate of physical health outcomes (Moskowitz, 2003; Moskowitz, Epel, & Acree, 2008). Adequate internal consistency for the Positive affect subscale has been demonstrated (Cronbach's $\alpha = .78$) (Gatz & Hurwicz, 1990). For this sample, Cronbach's alpha ranged from .68 to .69 (Table 8). In this sample, the average cross-sectional correlation among Vitality and CES-D Positive Affect scale was moderate ($r = .45$). For each participant missing $\leq$ one item, missing values were imputed with mean substitution using the available items from the 4-item scale.

Covariates

Given their established associations with both negative affective traits and obesity, the following covariates were included in the analyses as potential confounders of any observed relationships between an affective trait and adiposity. First, older age, female sex, and low SES have been linked with greater obesity and depressive symptoms (Crawford et al., 2010; Martin & Ferris, 2007; Mirowsky & Ross, 1992; Paeratakul et al.,

2002). Thus, the following self-reported demographic factors from baseline are included: age (years), sex (male = 0, female = 1), education (years), and objective income (total annual household income ranging from less than $10,000 to $75,000 or more). Second, chronic physical diseases and physical disability and/or limitations can also contribute to negative affective traits and obesity (Katon & Sullivan, 1990; Liou et al., 2005; Turner & Noh, 1988). Thus, measures of chronic physical diseases and physical disability/limitations at baseline were included. The indicators of physical disease were a series of dichotomous variables reflecting the presence (score = 1) or absence (score = 0) of several chronic diseases and was determined by asking the participant if he or she had ever been told by a physician that he or she had a particular disease. The diseases of interest include the following: hypertension, diabetes mellitus, cancer, chronic kidney disease, and cardiovascular disease. Cardiovascular disease was a composite score of those with or without coronary artery disease, angina, stroke, and/or congestive heart failure. Of note, the hypertension variable was only available at baseline. The measures of physical disability/limitation are summary scores of self-reported performance on basic activities of daily living (ADLs), instrumental ADLs, and lower body functional tasks from the Second Longitudinal Study on Aging (National Center for Health Statistics, 1998) and Lawton and Brody (1969). Higher summary scores indicate greater physical limitation and disability.

Procedure

At baseline (September 2000-July 2001), participants received detailed, in-home baseline evaluations that averaged 2.5 hours and were conducted by one of 50

interviewers. Interviews for the 7-year follow-up period occurred by telephone and took an average of 1 hour, whereas 9 -year follow-up interviews were conducted in-home and took approximately 2 hours. Two thirds of the professional interviewers were African American. For each evaluation, interviewers asked questions from a standardized protocol that employed skip rules. For in-home evaluations, interviewer also set up the measuring tape and Tanita scale for measuring participants' height, weight, and body fat percent. During baseline, a random subsample (20%) of AAH participants was invited to complete additional assessments, including the DEXA testing. In addition to the interview evaluations, these participants went to a clinical testing center.

Data Analyses

Data Cleaning and Reduction

The data were screened to evaluate the assumptions of structural equation modeling (Kline, 2005; Tabachnick & Fidell, 2000) using SPSS 19 statistical software. The panel analysis was conducted using a latent variable path analysis (LVPA) approach in LISREL 8.8 (Jöreskog & Sörbom, 2008) and employed the full information maximum likelihood (FIML) estimation procedure. FIML uses all observed data points for parameter estimation; thus, cases with partially missing data are retained, and probable values for the missing data points are implied by the observed values (Enders & Bandalos, 2001). FIML has been demonstrated as a superior method of handling missing data compared to listwise deletion, which simply discards cases with any missing data, and pairwise deletion, which discards cases based on bivariate pairings of variables with

missing data (Enders, 2001; Enders & Bandalos, 2001). All relevant variables were calculated, and checks for computation errors were performed. Age, sex, and education were examined as predictors of missing data to determine whether systematic missingness existed for the relevant study variables. This assessment was done by creating two groups for each study variable (0 = no missing, 1 = missing). Next, logistic regressions were run to determine whether age, sex, or education (entered simultaneously in the models) were associated with a greater likelihood of missing data.

Frequencies for the data were run to check for out-of-range values; all variables were found to be within range. All outliers (z scores ≥ 3.3) in the data set were identified. At baseline, the variables and the corresponding frequency of z scores ≥ 3.3 were as follows: CES-D (n = 8), self-reported BMI (n = 6), measured BMI (n = 3), chronic disease summary (n = 4), basic ADLs (n = 30), and instrumental ADLs (n = 27). At 7-year follow-up, the following variables had outliers: CES-D (n = 8), self-reported BMI (n = 3), and chronic disease summary (n = 5). Finally, at 10-year follow-up, the following variables had outliers: CES-D (n = 8), self-reported BMI (n = 4) and measured BMI (n = 2). If outliers were identified for a particular variable, these outliers were not altered or deleted for three reasons: 1) these cases did not result in non-normal distributions, 2) some z scores ≥ 3.3 are expected when sample sizes are large, and 3) these cases are likely legitimate cases of the sample population (Tabachnick & Fidell, 2001). The assumption of normality was assessed using skewness and kurtosis statistics. All variables were normally distributed (skewness < 3.0 and kurtosis < 10.0); therefore, no transformations were performed. The assumption of multicollinearity was checked by

reviewing the squared multiple correlations between variables that were to be entered into the same model. In no case did the statistic exceed 0.90, which is the cutoff suggested by Kline (2005). These results indicate that deviations from normality and issues with multicollinearity were insubstantial so that standard SEM analysis and fit indices could be applied.

Body fat values for each wave were automatically calculated by the BIA and DEXA devices, whereas BMI values were manually calculated by dividing participants' weight (kg) by their height squared (m^2). Depressive symptoms for each period were calculated by summing the 9 CES-D items. Anxiety symptoms, vitality, and positive affect at each wave were also calculated by summing the corresponding items (2 items from GAD-2, 4 items from the SF-36 Vitality subscale, and 4 items from the CES-D positive affect scale). Cronbach's alphas were calculated to evaluate the internal consistency of each affective trait measure (see Table 8).

Analyses

To test Hypothese #1-6, cross-lagged panel analysis was used. This type of analyses is a structural equation modeling technique used when at least two variables (e.g., CES-D and BMI) were measured at two or more time-points. Panel data and LVPA are advantageous for testing potential bidirectional causal effects because 1) multiple outcomes can be predicted simultaneously and 2) both covariation of baseline variables and their simultaneous effects on multiple outcome variables can be accounted for. These key benefits are not available with techniques like multiple regression, which

solves for each outcome individually (Meyers, Gamst, & Guarino, 2006). LVPA is also advantageous because it can address the issue of missing data using estimation procedures, such as FIML.

Using the two-step approach advocated by Kline (2011), each hypothesized model was examined from a measurement and structural perspective. Measurement models are comprised of the measured variables, which serve as indicators for the latent variables. Arrows pointing from the latent variables to their indicators represent factor or item loadings. Error terms pointing to the indicators represent the variance unexplained by the latent factor. All latent variables are allowed to freely correlate in measurement models (i.e., two-headed arrows). Second, the structural model specifies the pattern of causal influence between latent variables using unidirectional arrows. Endogenous latent variables are those being predicted in the model, whereas exogenous variables do not have a causal source designated in the model. Similar to error terms, disturbance terms represent the variance of endogenous variables that is unexplained by the variables in the model. Two-headed arrows represent covariances between variables.

The measurement model for each analysis was first assessed to determine how well it fit the data. If the measurement model adequately fit the data, the structural model was then constructed. Because FIML was used, model fit was evaluated using the following two indicators: the chi-squared statistic (χ^2) and the root mean square error of approximation (RMSEA). Although other fit statistics exist, only χ^2 and RSMEA are available when using FIML estimation in LISREL 8.8. The χ^2 statistic indicates the absolute fit between the hypothesized model and the observed relationship patterns among the measured data. The χ^2 statistic should generally be small and non-significant

to indicate that there is no statistical difference between the hypothesized model and the observed patterns of relationships, and thereby indicating acceptable fit of the hypothesized model (Kline, 2011). However, the χ^2 statistic is also sensitive to sample size, with larger samples typically generating a significant χ^2 statistic, regardless of model fit (Kline, 2011). Thus, as recommended by Kline (2011), RMSEA was also considered. The RMSEA is a parsimonious fit statistic that takes into account the complexity of the hypothesized model and adjusts the absolute fit accordingly (Hu & Bentler, 1999). The RMSEA value should be less than 0.06 for acceptable fit (Byrne, 1998; Hu & Bentler, 1999). If a measurement model did not fit the data, re-evaluation and re-specification of the model was performed. If a structural model fit the data well, the beta weights (β) of the specific paths in the model were examined to determine whether the pattern of results matched the hypothesized paths (Little, 2013).

Objective #1 Analyses

To achieve Objective #1 (see Figure 3 for the conceptual model), a demographics-adjusted model (adjusting for age and sex) was constructed to determine whether bidirectional relationships existed between depressive symptoms and adiposity over the 9-year follow-up period. The model included the CES-D score and BMI or BF% measured at baseline and 9-year follow-up, as well as baseline age and sex. All variables except the CES-D score were modeled as latent variables with a single item-level indicator. Given that these variables had a single indicator, their error variances were set to zero. In contrast, the CES-D score had nine item-level indicators representing the items of the CES-D scale. For scaling purposes, the "I felt sad" item was used as the

reference item for baseline and follow-up, given that it has shown the highest factors loadings (Kohout et al., 1993). For the measurement model, all of the variables were freed to correlate with one another. The error variances of the baseline CES-D indicators were set to covary with their matching follow-up indicator, given that these indicators were worded identically and repeated at both waves. In the structural model, follow-up CES-D and BMI or BF% were adjusted for their corresponding baseline scores by adding structural paths between these variables, which created residualized change scores (i.e., 9-year change in depressive symptoms and 9-year change in BMI or BF%). The errors of follow-up CES-D and self-reported BMI, measured BMI, or BF% were set to covary. Finally, each of the baseline variables was connected to both of the follow-up variables with structural paths.

A second, fully-adjusted model was constructed to evaluate whether any relationships observed in the demographics-adjusted model persisted after adjustment for other potential confounders. The second model was identical to the demographics model except that the following baseline variables were added: socioeconomic variables (i.e., education and objective income), the presence of chronic physical diseases (i.e., hypertension, diabetes, cancer, cardiovascular disease, and kidney disease), and level of disability. Each of these variables was modeled as a latent variable with a single indicator except the disability factor, which had three indicators (i.e., summary scores for basic and instrumental activities of daily living and lower body functioning). Because each of these variables is a plausible predictor of increases in depressive symptoms and adiposity, each of these confounders were linked to both follow-up CES-D and BMI or BF% variables with structural paths.

Objective #2 Analyses

To achieve Objective #2 (see Figure 4 for the conceptual model), models were also examined to determine whether depression and anxiety exhibit independent associations with changes in adiposity and vice versa. The only adiposity variable available for these analyses was self-reported BMI. Thus, the demographics-adjusted model included age and sex measured at baseline as well as the GAD-2, CES-D, and self-reported BMI variables measured at 7-year and 9-year follow-up. Data at 9-year follow-up are referred to as "2-year follow-up" variables in these analyses, given that they occurred two years after the CES-D and GAD-2 assessment. The CES-D was again modeled with 9 indicators, and the GAD-2 was modeled with two indicators. The "nervous/anxious/on-edge" item was used as the reference item to scale the GAD-2 at baseline and follow-up. Error variances of the CES-D and GAD-2 indicators were set to covary with their follow-up indicators. For the measurement model, all of the latent variables were allowed to correlate with one another. For the structural model, paths were added from the baseline GAD-2, CES-D, and BMI variables to their corresponding follow-up scores, which created residualized change scores. The errors of follow-up CES-D, GAD-2, and self-reported BMI, measured BMI, or BF% were set to covary. Structural paths from all baseline variables to all of the follow-up variables were added. A fully-adjusted model was then conducted to determine whether any of the observed relationships were the result of the potential confounders assessed at baseline (i.e., education, income, chronic diseases, and disability). All structural paths are the same as described for the demographic-adjusted model, except that structural paths were added from the additional confounders to each follow-up variable. Of note, demographics-

adjusted models and fully-adjusted models were also run with the CES-D or GAD-2 separately to determine whether these emotional factors predicted self-reported BMI when included alone in the models.

Secondary Objectives Analyses

The secondary objectives were to determine whether: (1) measures of trait positive affect are predictors of change in adiposity, (2) a reduction in trait positive affect is a consequence of excess adiposity, and (3) the associations, if present, are independent of the associations between negative affective traits and adiposity. To meet these secondary objectives, each measure of positive affect (i.e., Vitality and CES-D Positive Affect) was examined in a separate set of models to avoid high multicollinearity (see Figures 5 and 6 for the conceptual models).

First, to determine whether Vitality was related to 9-year changes in adiposity, a demographics-adjusted model was constructed with paths equivalent to those described for the demographics-adjusted model of depression (Objective #1); however, this model used the Vitality subscale in place of the CES-D (Figure 5). Self-reported BMI, measured BMI, and BF% were each used as adiposity indicators. Vitality was modeled with 4 item-level indicators. The "full of energy" item was used as the reference item to scale the Vitality factor at baseline and follow-up. Error variances of the baseline Vitality indicators were set to covary with their corresponding follow-up indicator. A residualized change score was created for Vitality by including structural paths between baseline and 9-year Vitality. A second, fully-adjusted model was also constructed to evaluate whether any relationships observed between Vitality and BMI or BF% persisted

after adjustment for potential confounders. Because Vitality was also available at the 7-year follow-up period, demographics-adjusted and fully-adjusted models were also run using 7-year Vitality as the baseline predicting 2-year changes in Vitality (Figure 6).

Next, models were constructed with CES-D Positive Affect at 7-year follow-up and 9-year follow-up as the positive affective trait (Figure 6). In these analyses, only self-reported BMI was available. The "happy" item was used as the reference item to scale the CES-D Positive Affect factor at baseline and follow-up. Error variances of the baseline CES-D Positive Affect indicators were set to covary with their corresponding follow-up indicator. A residualized change score was created for CES-D Positive Affect by including structural paths between baseline and 9-year CES-D Positive Affect. An additional, fully-adjusted model with the aforementioned confounders was also analyzed. If Vitality or CES-D Positive was found to be associated with adiposity indicators in any of the fully-adjusted models, negative affective variables (e.g., CES-D and GAD-2) were added to the corresponding models to determine whether any relationships persisted in the presence of negative affective traits.

Complete Data Sensitivity Analyses

The FIML estimation technique used in the primary analyses does not delete cases with missing values and instead uses all available information in all observations to estimate parameters even for cases with partially missing data. Given that this procedure could result in estimating outcomes in participants who do not have follow-up data and thereby distort the accuracy of the results, all demographics-adjusted analyses were also run using only participants with complete data to ensure that results of FIML analyses

were comparable to those of participants with complete baseline and follow-up data. When using complete data, other fit statistics are available in LISREL 8.8. Thus, in addition to χ^2 and RMSEA, the standardized root mean square residual (SRMR) and comparative fit index (CFI) were examined in complete data models.

Power Analyses

Structural equation modeling, including LVPA, is generally understood to require large samples, with 200 cases as the suggested minimum (Kline, 2011). Increasingly complex models require larger samples as do certain estimation methods. As a rule of thumb, the general recommendation is to have a 20:1 ratio of participants to free parameters, although 10:1 may be a more realistic target (Kline, 2011). If the ratio falls below 5:1, the results may be untrustworthy. For the current study, the model with the most number of parameters to be estimated (Objective #2 Model) had 76 parameters to be estimated; therefore, 1,520 participants (20:1 ratio) would be ideal, and 760 (10:1) participants would be acceptable. With less than 380 participants, the results would become untrustworthy. The main analyses using FIML were run with 998 cases, which met and exceeded the 10:1 ratio. The complete data analyses were run in models with participants with no missing data, and the sample sizes of these models ranged from N = 430 to N = 540. The sample sizes for the complete data analyses were below the recommended numbers but well above the lower threshold of acceptability (N = 380). Alpha was set at .05 (2-tailed). Importantly, statistical significance testing is less relevant in structural equation modeling than in other data analytic techniques (e.g., multiple

regression). In SEM, the focus tends to be on the evaluation of entire models and the fit statistics, with the big-picture view typically taking precedence over attention to individual effects (Kline, 2011).

RESULTS

Characteristics of Participants

A total of 998 African American individuals consented to participate in this study at baseline. Of these participants, 680 participated at 7-year follow-up, and 579 participated at 9-year follow-up. In general, the sample at baseline was predominantly middle-aged, female, had less than six years of education, and made less than $25,000/year (see Table 2). On average, participants were obese at baseline (BMI > 30 kg/m^2) and had high a BF% (see Table 2). Participants' small average decrease in measured BMI was significant ($p = .01$), but the other adiposity variables did not significantly change, on average, over time (see Table 5). Participants' average baseline depressive symptom and anxiety severity was low, whereas their baseline levels of vitality and positive affect were high (see Table 2). At the group level, participants' average CES-D, GAD-2, Vitality, and CESD-PA did not change significantly over time ($ps \geq .12$) (see Table 5). Participants reported little difficulty with their baseline basic or instrumental ADLs and lower body functioning (see Table 2). With regards to medical comorbidities, a majority of participants had hypertension at baseline, and nearly a quarter had diabetes (see Table 4). Over 1 in 5 participants experienced cardiovascular disease at baseline, whereas few participants had kidney disease (see Table 4). Characteristics of a subset of participants with complete data for all the adiposity

variables (i.e., self-reported BMI, measured BMI, and BF%) for baseline and 9-year follow-up were also examined. The characteristics of this subset are presented in Table 3.

Age, sex, and education were examined as predictors of missing data across the study periods to assess for systematic missingness. At baseline, none of these variables predicted missingness. For the 7-year and 9-year follow-up periods, logistic regression indicated that those with greater years of education were typically more likely to have missing data across (see Table 9); however, it should be noted that the education variable may have poor validity, given that it was a double-barreled question (i.e., "How many grades of school or years of college have you completed?"). The wording of this question could have led participants to say they had 0 or 1 years of overall education when they meant to indicate that they had 0 or 1 year of college education. Females were more likely have missing data on the 7-year Vitality, GAD-2, and CESD-PA variables. Age was not associated with missingness at the 7-year or 9-year follow-up periods. These results indicate that data was likely not missing completely at random (MCAR) in this sample. Although FIML performs more poorly when MCAR assumptions are not met, FIML has been shown to strongly outperform listwise or pairwise deletion under all missingness conditions in Monte Carlo simulation studies (Newman, 2003).

Objective #1: Models for Depressive Symptom Severity and Adiposity over 9 Years

Self-reported BMI

The first objective of the current study was to determine whether bidirectional relationships existed between depressive symptoms and adiposity over the 9-year follow-up period. Hypotheses 1 and 2 – that greater baseline depression would predict greater 9-year increases in adiposity and that greater baseline adiposity would predict greater 9-year increases in depression – were evaluated in the same series of panel models. The first analysis used self-reported BMI as the adiposity indicator and included age and sex as covariates. The *a priori* measurement model was specified as planned, with all variables freely correlating. This model showed adequate fit, $\chi^2_{(189,\, N=998)} = 610.90$, ($p < .001$), RMSEA = .047, CI_{90}: 0.043- 0.052. Although the significance of χ^2 suggests the model failed an absolute-fit test, this finding is expected given that exact fit rarely occurs, especially in larger samples (Weston et al., 2008). It has been suggested that an over-reliance on the χ^2 statistic may lead to rejection of models with reasonably good fit (Kline, 2005).

Given that RMSEA indicated a good fit to the data (RMSEA $\leq .06$), I proceeded to test the structural model by freeing the structural paths as indicated by Figure 3. Structural model fit results were similar to those of the measurement model, $\chi^2_{(189,\, N=998)} = 610.90$, ($p < .001$), RMSEA = .047, CI_{90}= 0.043-0.052, indicating acceptable model fit. Consequently, the individual structural paths were examined and results are presented (see Figure 7). The structural path from baseline CES-D to self-reported BMI at 9-year follow-up was not significant ($\beta = 0.02$, $z = 0.64$, $p = .52$), suggesting that baseline

depressive symptoms did not predict change in adiposity over 9 years. The structural path from baseline self-reported BMI to CES-D at 9-year follow-up fell just short of significance ($\beta = 0.07$, $z = 1.83$, $p = .07$), indicating that baseline adiposity did not predict change in depressive symptoms over 9 years. Neither Hypothesis 1 nor 2 was supported by these results. As was expected, baseline CES-D and self-reported BMI strongly predicted their corresponding follow-up scores (βs = 0.59 and 0.83, ps < .001) (Figure 7). Baseline CES-D and self-reported BMI were not related, $r = 0.02$, $p = .65$, and nor were follow-up CES-D and BMI, $r = 0.04$, $p = .53$. With regards to the covariates, self-reported BMI was predicted by both baseline age ($\beta = -0.08$, z = -3.61, $p < .01$) and gender ($\beta = 0.07$, z = 3.11, $p < .01$), whereas CES-D was not associated with age or gender ($\beta = 0.02$, z = .57, $p = .57$ and $\beta = -0.01$, $z = -0.22$, $p = .83$, respectively). These results suggest that younger age and female gender are associated with greater 9-year increases in self-reported BMI. This pattern of results for the covariates – in which age and sex were unrelated to affective traits (e.g., the CES-D and GAD-2) and younger age and female gender predicted greater increases in adiposity (i.e., BMI measures and BF%) – remained across all remaining models and, thus, is not described again in the text. A fully-adjusted model was constructed containing the additional education, income, disease, and disability covariates. Fit statistics indicated that the measurement and structural models provided adequate, equivalent fit to the data, $\chi^2_{(375,\, N = 998)} = 1061.57$, ($p < .001$), RMSEA = .043, CI_{90}= 0.040-0.046). Neither the path from CES-D to self-reported BMI ($\beta = 0.01$, z = 0.50, $p = .62$) nor the path from BMI to CES-D ($\beta = 0.07$, z = 1.66, $p = .10$) was significant in this fully-adjusted model. Fewer years of education (β = -0.08, z = 3.21, $p < .01$) and the absence of hypertension at baseline ($\beta = -0.05$, z = 2.24,

p = .03) were related to greater self-reported BMI at 9-year follow-up. Lower baseline income was associated with 9-year increases in BMI (β = -0.11, z = -2.45, p = .01). No other covariates were related to follow-up CES-D or self-reported BMI.

Measured BMI and Body Fat Percent

When the demographics-adjusted model constructed for measured BMI or BF%, results were similar to those for self-reported BMI (see Figures 8 and 9). The measurement models indicated good fit for measured BMI $\chi^2_{(189,\, N=998)}$ = 610.23, (p < .001), RMSEA = .047, CI_{90}: 0.043-0.051 and BF% $_{(189,\, N=998)}$ = 594.48, (p < .001), RMSEA = .046, CI_{90}: 0.042-0.051. The structural model fit was also adequate for measured BMI, $\chi^2_{(197,\, N=998)}$ = 610.24, (p < .001), RMSEA = .047, CI_{90}: 0.043-0.051 (Figure 8), as well as for BF%, $\chi^2_{(189,\, N=998)}$ = 594.48, (p < .001), RMSEA = .046, CI_{90}: 0.042-0.051 (Figure 9). The path from baseline CES-D to measured BMI at 9-year follow-up was not significant (β = 0.01, z = 0.36, p = .72), nor was the path from CES-D to BF% (β = 0.01, z = 0.18, p = .86), indicating that baseline depressive symptoms scores did not predict changes in objectively-assessed adiposity. Similarly, the structural path from baseline measured BMI to CES-D at 9-year follow-up was not significant (β = 0.07, z = 1.65, p = .10), nor was the path from BF% to CES-D (β = 0.08, z = 1.62, p = .11), again suggesting that baseline adiposity is unrelated to changes in depressive symptoms over time. No correlation was detected between baseline CES-D and measured BMI (r = .02, p = .59) or BF% (r = .02, p = .66) or between follow-up CES-D and measured BMI (r = 0.05, p = .11) or BF% (r = 0.03, p = .34). These results also do not support Hypotheses 1 and 2. Baseline CES-D continued to predict its follow-up scores in these models with a

similar magnitude as in the self-reported BMI model (*p*s < .001). Measured BMI at baseline strongly predicted its 9-year follow-up score ($p < .001$) as did baseline BF% ($p < .001$). In the fully-adjusted models, fit statistics indicated that the measurement models provided adequate fit to the data for the measured BMI model, $\chi^2_{(375,\, N = 998)} = 1065.20$, ($p < .001$), RMSEA = .043, CI_{90}= 0.040-0.046) and the BF% model, $\chi^2_{(375,\, N = 998)} = 1024.43$, ($p < .001$), RMSEA = .042, CI_{90}= 0.039-0.045). Similarly, the structural models also fit the data for measured BMI, $\chi^2_{(375,\, N = 998)} = 1065.24$, ($p < .001$), RMSEA = .043, CI_{90}= 0.040-0.046) and for BF%, $\chi^2_{(375,\, N = 998)} = 1061.57$, ($p < .001$), RMSEA = .043, CI_{90}= 0.040-0.046). The paths from CES-D to measured BMI ($\beta = 0.03$, z = 1.15, $p = .25$) and BF% ($\beta = -0.02$, z = -0.44, $p = .66$) were not significant in the fully-adjusted models. Measured BMI ($\beta = 0.06$, z = 1.36, $p = .17$) and BF% ($\beta = 0.08$, z = 1.48, $p = .14$) did not predict 9-year changes in CES-D. Fewer years of education were related to greater measured BMI ($\beta = -0.11$, z = 4.70, $p < .001$) and BF% ($\beta = -0.06$, z = -2.00, $p < .05$) 9-years later. Lower baseline income was associated with 9-year increases in measured BMI ($\beta = -0.11$, z = -2.39, $p = .02$) and BF% ($\beta = -0.11$, z = -2.46, $p = .02$). No other covariates were related to follow-up CES-D or self-reported BMI.

Objective #2: Models for Depressive Symptom Severity, Anxiety Symptom Severity, and Adiposity over 2 Years

Self-reported BMI

The second objective was to determine whether depressive and anxiety symptom severity exhibit independent associations with changes in adiposity and vice versa. The

remaining hypotheses (Hypotheses 3, 4, 5, and 6) were evaluated in the same panel model. All analyses used self-reported BMI as the adiposity indicator. The first model contained both CES-D and GAD-2 and included age and sex as covariates. The *a priori* measurement model was specified as planned, with all variables freely correlating, and showed adequate fit, $\chi^2_{(264,\ N=998)} = 716.74$, ($p < .001$), RMSEA = .041, CI_{90}: 0.038, 0.045. Structural model fit was also acceptable, $\chi^2_{(264,\ N=998)} = 716.74$, ($p < .001$), RMSEA = .041, CI_{90}= 0.038-0.045. Thus, the individual paths were examined and are presented in Figure 10.

Like the results from Objective #1 analyses, CES-D did not predict self-reported BMI at 2-year follow-up ($\beta = 0.02$, $z = 0.49$, $p = .62$). The path from GAD-2 to BMI was also not significant ($\beta = -0.03$, $z = -0.76$, $p = .45$), suggesting that neither baseline depressive nor anxiety symptoms predict 2-year changes in self-reported BMI. Similarly, self-reported BMI did not predict changes in CES-D ($\beta = 0.06$, $z = 1.52$, $p = .13$) or GAD-2 ($\beta = -0.01$, $z = -0.22$, $p = .83$) at follow-up. These results indicate that adiposity did not influence 2-year changes in depressive or anxiety symptoms. The CES-D and GAD-2 were strongly correlated with one another at both baseline, ($r = 0.82$, $p < .001$) and follow-up ($r = 0.78$, $p < .001$), whereas self-reported BMI was not correlated with CES-D at baseline ($r = 0.06$, $p = .18$) or follow-up ($r = 0.06$, $p = .19$) or with GAD-2 at baseline ($r = -0.02$, $p = .28$) or follow-up ($r = -0.05$, $p = .11$).

In the fully-adjusted model, which added income, education, disease status, and disability as covariates, the results for the paths of interest remained essentially unchanged. The measurement model provided good data fit, $\chi^2_{(454,\ N=998)} = 1140.07$, ($p < .001$), RMSEA = .039, CI_{90}= 0.036-0.042, as did the structural model, $\chi^2_{(454,\ N=998)} =$

1139.93, ($p < .001$), RMSEA = .039, CI_{90}= 0.036-0.042. Neither the CES-D ($\beta = 0.01$, $z = 0.24$, $p = .81$) nor the GAD-2 ($\beta = -0.03$, $z = -.79$, $p = .43$) predicted 2-year changes in self-reported BMI in the fully-adjusted models. Of the added covariates, only the presence of kidney disease at baseline predicted increased self-reported BMI at follow-up ($\beta = 0.04$, $z = 2.33$, $p = .02$). Having kidney disease at baseline also predicted higher CES-D scores ($\beta = 0.16$, $z = 4.14$, $p < .001$). Greater disability ($\beta = 0.19$, $z = 3.37$, $p < .001$), fewer years of education ($\beta = -0.12$, $z = 2.71$, $p = .01$), and lower income ($\beta = -0.12$, $z = -3.06$, $p < .01$) at baseline also predicted greater 2-year follow-up CES-D scores. Of the covariates predicting follow-up GAD-2, only greater disability ($\beta = 0.17$, $z = 2.80$, $p = .01$) and lower income ($\beta = -0.12$, $z = -2.90$, $p < .01$) at baseline were associated with higher GAD-2 scores at follow-up.

In demographics-adjusted models that contained CES-D alone as the emotional predictor, CES-D and BMI were still unrelated. Measurement model fit was acceptable, $\chi^2_{(189,\, N=998)} = 615.17$, ($p < .001$), RMSEA = .048, CI_{90}= 0.043-0.052, as was structural model fit, $\chi^2_{(189,\, N=998)} = 615.17$, ($p < .001$), RMSEA = .048, CI_{90}= 0.043-0.052. CES-D did not predict 2-year changes in self-reported BMI ($\beta = -0.01$, $z = -.34$, $p = .73$), and BMI did not predict 2-year changes in CES-D ($\beta = 0.04$, $z = .88$, $p = .38$). Thus, Hypotheses 3 and 5, which posited that depression was an independent predictor and consequence of adiposity were not supported.

When GAD-2 was entered in the demographics-adjusted model as the only emotional predictor, GAD-2 and BMI were not related in either direction. Measurement model fit was excellent, $\chi^2_{(7,\, N=998)} = 3.09$, ($p = 0.88$), RMSEA = .000, CI_{90}= 0.00-0.019, as was structural model fit, $\chi^2_{(7,\, N=998)} = 3.09$, ($p = 0.78$), RMSEA = .000, CI_{90}= 0.00-

0.019. GAD-2 did not predict 2-year changes in self-reported BMI (β = -0.01, z = -.73, p = .47). Likewise, BMI did not predict 2-year changes in GAD-2 (β = 0.05, z = 1.10, p = .27). Thus, Hypotheses 4 and 6 were not supported by these results.

Secondary Objectives: Models for Positive Affective Traits over 9 years

Self-reported BMI

The secondary objectives were to determine whether positive affective traits are independent predictors of change in adiposity indicators and vice versa. The first analysis to address this objective used Vitality as the measure of positive affect and self-reported BMI as the adiposity indicator in a model adjusted for age and sex over 9 years. The *a priori* measurement model specified as planned did not show adequate fit, $\chi^2_{(39,\, N = 998)} = 347.14$, ($p < .001$), RMSEA = .089, CI_{90}: 0.081-0.098. The model was re-evaluated using the modification indices to guide re-specification of the model. Four correlations between error terms were freed based on the conceptual reasonableness of each change. Specifically, error terms were freed to correlate for the "tired" and "wornout" items and for the "full of life" and "a lot of energy" items at baseline and at follow-up, given that these pairs of items use synonymous phrasing, which could lead to systematic error among the pairs. This modification has been performed in past studies which have also cited the synonymic relationship between these item pairs (Wolinsky & Stump, 1996). The revised measurement model achieved good fit, $\chi^2_{(35,\, N = 998)} = 114.23$, ($p < .001$), RMSEA = .048, CI_{90}: 0.038, 0.057, so the structural model was examined.

Given that these modifications were theoretically founded and replicated in past research, the correlated errors among the item pairs were maintained in all other models with Vitality.

Structural model fit results were equivalent to those of the measurement model, $\chi^2_{(35,\, N=998)} = 114.24$, ($p < .001$), RMSEA = .048, CI_{90}= 0.038-0.057, indicating acceptable model fit (see Figure 11). The structural path from baseline Vitality to self-reported BMI at 9-year follow-up was not significant ($\beta = -0.01$, $z = -.37$, $p = .71$), suggesting that baseline Vitality did not predict 9-year change in self-reported BMI. Likewise, the structural path from baseline self-reported BMI to CES-D at 9-year follow-up was not significant ($\beta = 0.04$, $z = 0.95$, $p = .34$), indicating that baseline adiposity did not predict 9-year change in Vitality. Baseline Vitality and self-reported BMI were weakly correlated ($r = -0.21$, $p < .001$) and strongly predicted their corresponding follow-up scores (Vitality: βs = 0.68 and BMI: βs = 0.83, ps < .0001). Follow-up Vitality and BMI were also negatively correlated, $r = -0.16$, $p < .001$). These results indicate that positive affect, as measured by Vitality, is not associated with adiposity or vice versa.

Measured BMI and Body Fat Percent

When the demographics-adjusted model was constructed for measured BMI, results were similar (see Figure 12). The measurement model fit adequately, $\chi^2_{(35,\, N=998)} = 117.54$, ($p < .001$), RMSEA = .049, CI_{90}: 0.039-0.058. The structural model fit was equivalent, $\chi^2_{(35,\, N=998)} = 117.55$, ($p < .001$), RMSEA = .049, CI_{90}: 0.039-0.058. Follow-up BMI was predicted by baseline BMI ($\beta = 0.86$, $z = 36.75$, $p < .001$) but not by baseline Vitality ($\beta = -0.00$, $z = -0.06$, $p = .95$). Follow-up Vitality was predicted only by baseline Vitality

scores ($\beta = 0.69$, $z = 10.21$, $p < .001$). Vitality and measured BMI were weakly correlated at baseline ($r = -.19$, $p < .001$) and follow-up ($r = -0.16$, $p < .001$). The paths of interest from Vitality to measured BMI ($\beta = 0.01$, $z = 0.17$, $p = .87$) and from BMI to Vitality ($\beta = 0.01$, $z = 0.28$, $p = .78$) were non-significant in the fully-adjusted model.

When the demographics-adjusted model was constructed for BF%, a slightly different pattern emerged (see Figure 13). The structural model fit was acceptable for BF%, $\chi^2_{(35,\, N = 998)} = 99.29$, ($p < .001$), RMSEA = .043, CI_{90}: 0.033-0.053. Importantly, lower levels of Vitality emerged as a predictor of 9-year increases in BF% ($\beta = -0.07$, $z = -2.07$, $p = .04$). Baseline BF% predicted follow-up BF% ($\beta = 0.68$, $z = 19.13$, $p < .001$). The baseline Vitality score was the only predictor of 9-year Vitality ($\beta = 0.68$, $z = 10.20$, $p < .001$). Taken together, these results indicate that Vitality may predict adiposity, as measured by BF% but not by BMI.

The fully-adjusted model was then examined to determine whether the relationship between Vitality and BF% persisted in the presence of the following potential confounders: education, income, disability, and history of chronic disease (i.e., hypertension, diabetes, cardiovascular disease, cancer, and kidney disease). Results indicated good measurement model fit, $\chi^2_{(121,\, N = 998)} = 327.18$, ($p < .001$), RMSEA = .041, CI_{90}= 0.036-0.047, as well as structural model fit, $\chi^2_{(121,\, N = 998)} = 327.18$, ($p < .001$), RMSEA = .041, CI_{90}= 0.036.047). In the fully-adjusted model, Vitality no longer predicted 9-year increases in BF% ($\beta = -0.06$, $z = -1.17$, $p = .24$). Higher baseline BF% ($\beta = 0.68$, $z = 18.81$, $p < .001$) still predicted its 9-year follow-up score. Vitality at 9-year follow-up was predicted by baseline Vitality scores ($\beta = 0.70$, $z = 7.58$, $p < .001$). Greater 9-year Vitality was predicted by greater years of education at baseline ($\beta = 0.15$, $z = 3.29$,

$p < .01$). These results suggest that the initial relationship observed between Vitality and BF% in the demographics-adjusted model was likely due to confounding variables. Models adjusting for CES-D were not run, as these models were to determine whether the association between positive affect and adiposity, if present, were independent of the associations between negative affective and adiposity.

Secondary Objectives: Models for Positive Affective Traits over 2 years

Self-reported BMI

Models were then created to include positive affective traits at 7-year and 9-year follow-up (2-year change). Vitality and CES-D Positive Affect were examined in separate models. All analyses used self-reported BMI as the adiposity indicator. For Vitality, measurement model fit was good, $\chi^2_{(35,\, N=998)} = 73.85$, ($p < .001$), RMSEA = .033, CI_{90}= 0.023-0.044, as was structural model fit, $\chi^2_{(35,\, N=998)} = 73.85$, ($p < .001$), RMSEA = .033, CI_{90}= 0.023-0.044 (see Figure 14). Consistent with prior models, this model indicated that Vitality was not a predictor of 2-year changes in self-reported BMI ($\beta = -0.02$, $z = -.88$, $p = .38$). Similarly, BMI was not a predictor of 2-year changes in Vitality ($\beta = -0.04$, $z = -.92$, $p = .36$). Vitality and self-reported BMI were correlated at baseline ($r = -0.21$, $p < .001$) and at follow-up ($r = -0.18$, $p < .001$). In the fully-adjusted models, good measurement model fit, $\chi^2_{(113,\, N=998)} = 216.53$, ($p < .001$), RMSEA = .030, CI_{90}= 0.024-0.036, and structural model fit, $\chi^2_{(113,\, N=998)} = 216.53$, ($p < .001$), RMSEA = .030, CI_{90}= 0.024-0.036, were achieved. Greater baseline Vitality predicted greater self-reported BMI at 2-year follow-up, ($\beta = 0.08$, $z = 2.45$, $p = .01$), whereas BMI did not

predict 2-year changes in Vitality (β = -0.02, z = .41, p = .68). Greater BMI at 2-year follow-up was predicted by the following baseline covariates: greater disability (β = 0.08, z = .2.22, p = .03), lower education (β = -0.05, z = -2.01, p = .02), and the presence of cancer (β = 0.04, z = 1.97, p < .05) or kidney disease (β = 0.05, z = 2.46, p = .02). Of the covariate, only the presence of kidney disease at baseline predicted lower Vitality at 2-year follow-up (β = -0.09, z = -2.44, p = .01). In the fully-adjusted model, Vitality and self-reported BMI were still correlated at both baseline (r = -0.21, p < .001) and at follow-up (r = -0.18, p < .001).

For CES-D Positive Affect, measurement model fit, $\chi^2_{(39,\, N = 998)}$ = 66.56, (p < .01), RMSEA = .027, CI_{90}= 0.015-0.037 and structural model fit were good, $\chi^2_{(39,\, N = 998)}$ = 66.56, (p < .01), RMSEA = .027, CI_{90}= 0.015-0.037 (see Figure 15). Positive Affect was not a predictor of 2-year changes in self-reported BMI (β = 0.03, z = 1.56, p = .12), and self-reported BMI did not predict 2-year changes in CES-D Positive Affect (β = 0.00, z = .05, p = .96). Baseline Positive Affect did predict its 2-year follow-up score (β = 0.55, z = 9.06, p < .001) as did self-reported BMI (β = 0.90, z = 49.21, p < .001). Positive Affect and self-reported BMI were not correlated at baseline (r = -.04, p = .34) or follow-up (r = -.04, p = .21). In the fully-adjusted models, Positive Affect was not a predictor of self-reported BMI (β = 0.02, z = .73, p = .47) or vice versa (β = -0.03, z = -.53, p = .60). Of the baseline covariates, the presence of kidney disease predicted greater self-reported BMI (β = 0.04, z = 2.05, p = .04) and higher income predicted greater Vitality at 2-year follow-up (β = 0.12, z = 2.63, p < .01).

Complete Data Results

When the demographic-adjusted models were constructed only for participants who had complete data at baseline and follow-up, the results were essentially unchanged from those reported above using FIML (see Table 11). The only exception is that for models containing the CES-D, respecification of the measurement model was performed so that the model fit the data prior to examining the structural models. Specifically, modification indices indicated that the errors of the two interpersonal items of the CES-D should be correlated. Upon examination of the items, this modification seemed theoretically justified, given that the items are so similar that they may be synonymic. Thus, this change was retained for all additional models containing the CES-D. Once these items were freed to correlate, the measurement models fit the data, and the structural models were examined.

The paths from CES-D to self-reported BMI, measured BMI, or BF% over 9 years (Objective #1) did not become significant (all ps $\geq .59$) (see Table 11). The structural path from baseline self-reported BMI to CES-D at 9-year follow-up again fell short of significance ($\beta = 0.09$, $z = 1.88$, $p = .06$), and the paths from measured BMI and BF% to CES-D were still not significant (all ps $\geq .18$) (see Table 11). These results demonstrate that Hypotheses 1 and 2 are not supported in participants with complete data. Models containing the CES-D only or the GAD-2 only over 2 years failed to indicate that these affective traits were predictors (all ps $\geq .35$) or consequences (all ps $\geq .36$) of self-reported BMI. The model containing both CES-D and GAD-2 also indicated that self-

reported BMI was unrelated to CES-D or GAD-2 in either direction (all *p*s ≥ .28). Again, these results show that Hypotheses 1-6 are not supported in participants with complete data.

When examining Vitality as a predictor of self-reported BMI, measured BMI, or BF% over 9 years in participants with complete data, results were similar to those from the FIML models, such that Vitality did not predict self-reported or measured BMI (both *p*s > .10) (see Table 11). The adiposity variables did not predict 9-year changes in Vitality (all *p*s > .20). When examining Vitality and self-reported BMI over 2 years, Vitality was neither a predictor nor consequence of self-reported BMI (*p*s > .47). Lastly, the structural path from baseline CESD-PA to self-reported BMI at 2-year follow-up was significant (β = -0.04, z = -2.03, p = .04), whereas BMI did not predict 2-year changes in CESD-PA (p = .88). These results indicate that, with the exception of greater baseline CESD-PA predicting lower self-reported BMI at 2-year follow-up, positive affective traits are unrelated to adiposity in this sample.

DISCUSSION

Primary Objectives

Using structural equation modeling, the present study sought to determine whether prospective, bidirectional associations existed between multiple affective traits and adiposity indicators in a sample of middle-aged African American males and females. Objective #1 was to examine bidirectional associations between depressive symptom severity and adiposity indicators over a 9-year period. The hypothesis (Hypothesis 1) that greater baseline severity of depressive symptoms is associated with greater 9-year increases in adiposity was not supported, as baseline depressive symptom scores were not predictors of 9-year changes in BMI or body fat percent. When the reciprocal hypothesis (Hypothesis 2) was tested – that greater baseline BMI or body fat percent would be associated with greater 9-year increases in depressive symptom severity – it was also not supported. Baseline BMI and body fat percent were not predictors of 9-year changes in depressive symptom scores. The results were equivalent for both the demographics-adjusted models containing only age and sex and the fully-adjusted models which also included education, income, disease, and disability as covariates. In sum, no longitudinal associations were found between depressive symptom severity and adiposity in this large sample of African American adults.

Objective #2 was to investigate whether depression and anxiety exhibit independent associations with changes in adiposity indicators. The hypothesis (Hypothesis 3) that greater baseline severity of depressive symptoms predicts greater 2-year increases in BMI, independently of anxiety symptoms was not supported. Similarly, the inverse hypothesis (Hypothesis 5) that greater baseline BMI predicts greater 2-year increases in depressive symptom severity was also not supported. Indeed, depressive symptom severity was unrelated to adiposity in the models – whether included by itself or with anxiety. These findings are consistent with the aforementioned results and indicate that even when depressive symptoms were assessed more proximally to the adiposity outcomes and vice versa (2-year vs. 9-year follow-up), no associations were detected. With regards to anxiety symptoms, they were also unrelated to adiposity in either direction, regardless of whether the model adjusted for depressive symptoms. Again, the demographics-adjusted and fully-adjusted models yielded comparable results. Thus, Hypothesis 4 and 6 were also not supported. As a set, these findings do not support any of the risk factor models presented in Figure 2, as depressive and anxiety symptom severity did not emerge as risk factors or consequences of excess adiposity in this sample of middle-aged African Americans.

The absence of negative affective traits-adiposity relationships in this sample is both inconsistent and consistent with previous investigations. First, the current findings contrast evidence from two meta-analyses of prospective studies containing a total of 33,690 (Blaine, 2008) and 58,745 participants (Luppino et al., 2010), which found positive longitudinal associations between greater depression and future obesity (OR = 1.18 and 1.40, respectively) and obesity and future depression (OR = 1.57) (Luppino et

al., 2010). The present findings also contradict a meta-analysis of community-based, cross-sectional studies of 204,507 total participants (de Wit et al., 2010), which found a positive relationship between depression and obesity (OR = 1.18). Although these meta-analyses examined several thousand participants and included some studies with nationally representative samples, none performed subgroup analyses on different race-ethnicity groups. Therefore, it is unknown whether the observed associations would have been found among African Americans.

Four longitudinal studies are available that indicate greater depression at baseline predicts future obesity (Franko et al., 2005; Goodman & Whitaker, 2002; Pine, Cohen, Brook, & Coplan, 1997) and vice versa (Merten, Wickrama, & Williams, 2008) for both white and African American participants, with no significant affective trait x race-ethnicity interactions. Of note, none of these studies stratified by race and performed subgroup analyses. Additionally, all of these studies examined adolescent samples and may not generalize to the current participants who had a mean baseline age of 56 years. Large nationally representative studies have also shown that the relationship between depression and adiposity variables was comparable across whites and blacks (Carpenter et al., 2000; Heo et al., 2005); however, compared to this prospective cohort study, the cross-sectional design of these investigations is a key limitation and neither study performed subgroup analyses.

In contrast, the current study's findings are consistent with a growing number of studies that have detected depression x race interaction effects or no effects between depression and adiposity. For instance, in a prospective cohort study of older adults, Vogelzangs and colleagues (2008) found that baseline depression was associated with an

increase in abdominal obesity for men and white women, but not for African American women. When testing whether baseline obesity predicted depression onset, Vogelzangs et al. (2010) found that visceral fat (not BMI) predicted depressive symptom onset in African American and white men, but not African American or white women. In their longitudinal study of middle aged adults, Sutin and Zonderman (2012) found that the CES-D was not a predictor or consequence of adiposity indicators in their total sample, which was 20% African American, and they did not detect any depression x race interactions. Depression was also prospectively related to obesity and vice versa for whites but not for African Americans or Hispanics in a sample of adolescent females (Anderson et al., 2011). Lastly, a nationally representative study of 16,450 adults has also demonstrated that obesity is cross-sectionally associated with major depression in white women but not in African American, Hispanic, or Asian women (Gavin et al., 2010a). These findings suggest that, when depression-adiposity relationships are tested in well-powered samples of African Americans, the relationship is often not detected.

With regards to examining depression and anxiety concurrently in relation to adiposity, only three known prospective studies are available (Bardone et al., 1998; Chiriboga et al., 2007; van Reedt Dortland et al., 2013). First, Bardone and colleagues' (2008) findings are consistent with ours in that both depression and anxiety failed to predict 6-year changes in measured BMI. Unfortunately, their sample of adolescent females and use of depressive and anxiety disorder diagnoses preclude direct comparison with the current findings. Next, Chiriboga et al. (2008) observed that anxiety symptoms predicted a 1-year weight gain in models with and without depressive symptoms among men but not women. These results suggest that anxiety may be an independent predictor

of BMI in men, whereas depression was unrelated to BMI in either sex (Figure 2c with depression as proxy for anxiety). Unlike the present sample, the participants in that study were younger, predominantly white, and had higher education levels. Lastly, van Reedt Dortland et al. (2013) examined the longitudinal relationships between depression and anxiety symptom severity with waist circumference over a 2-year period. They determined that, when entered independently, baseline depression and anxiety both predicted 2-year increases in waist circumference. However, when entered simultaneously, only depression remained a predictor of adiposity, suggesting that anxiety is a proxy for depression (Figure 2c). While comparable in design, their study differs from the current study in several ways: (1) their sample consisted of primarily Dutch Europeans, (2) they used a measure of abdominal adiposity, and (3) they had more detailed assessments of depression and anxiety (30-item and 21- item scales). These three prospective studies also examined affective traits predicting obesity and not vice versa. The two prospective studies that have examined depression and anxiety as consequences of excess adiposity also provide conflicting results (Kasen, Cohen, Chen, & Must, 2008; Mustillo et al., 2003). One study found that obesity does not have unique effects on anxiety (Mustillo et al., 2003), whereas the other indicated that obesity status does have unique effects on anxiety, independent of previous depressive symptoms (Kasen et al., 2008). Mustillo et al. (2003) did find an increased risk for depressive disorders among chronically obese boys, but not girls, after adjusting for anxiety disorders. Again, these findings may not generalize to African Americans, as the Mustillo et al. (2003) study examined only white children and Kasen and colleagues' sample was 97% Caucasian.

Thus, it is apparent that studies examining negative affect traits in relation to adiposity have substantial methodological differences. First, dissimilarities exist in sample characteristics (e.g., examining only adolescents or not examining race-ethnicity subgroups). Second, measurement of negative affective traits varies from symptom severity inventories to psychiatric diagnoses. Lastly, adiposity is assessed using various indicators, such as BMI, waist circumference, and visceral fat. Given these discrepancies, conflicting findings across studies are not surprising and fail to offer clear consilience to the affective trait-adiposity literature. What is clear, however, from the current, well-powered, prospective study is the possibility that negative affective traits may not be predictors or consequences of adiposity among middle-aged African Americans. These findings, combined with supporting evidence from the aforementioned studies, have important clinical implications. Together, they suggest that, compared to Caucasians, African Americans may require obesity prevention or intervention programs with little to no emphasis on depressive or anxiety symptoms.

Secondary Objectives

The secondary objectives were to determine whether measures of trait positive affect are predictors of change in adiposity or vice versa. Overall, the findings suggest that positive affect variables do not predict changes in BMI over time or vice versa. Greater baseline positive affect, as indicted by higher vitality, was associated with 9-year decreases in body fat in models adjusted for age and sex, but this relationship was no longer significant after adjustment for potential confounders. With the exception of greater baseline CESD-PA predicting 2-year decreases in self-reported BMI in

participants with complete data. none of the other paths between trait positive affect variables (e.g., Vitality or CES-D Positive Affect) and adiposity indicators reached significance in either the 9-year or 2-year models. Although no prospective studies are available, these findings are consistent with those of a cross-sectional study which found no association between obesity and indicators of happiness and positive affect in a sample of adults 50 years or older (Roberts et al., 2002).

Explanation for Study Findings

Why might affective traits be unrelated to adiposity in this sample of middle-aged African Americans? Kazdin (2002) proposes two possible reasons for null findings: (a) they reflect the state of nature or (b) methodological issues prohibit the detection of true relationships among variables. To begin, it is possible that depressive and anxiety symptoms are truly unrelated to adiposity among African Americans. Evidence to support this supposition is provided by previous studies including African Americans in which negative affective traits were not associated with adiposity in this race-ethnicity subgroup (Anderson et al., 2011; Gavin et al., 2010a; Sutin & Zonderman, 2012; Vogelzangs et al., 2010; Vogelzangs et al., 2008). However, the reasons for why these traits are predictors of greater adiposity in Caucasians but not African Americans have yet to be identified.

A possible reason is that the proposed mechanisms underlying the affective traits-obesity relationship (Figure 1, Panel A) are not active among African Americans. As an example, a recent study of a nationally representative sample found that, in Caucasians, depression is related to C-reactive protein, a marker of systemic inflammation (Case &

Stewart, 2013). In contrast, this relationship was not detected in African Americans. Given that systemic inflammation is one pathway by which depression may contribute to obesity development (Miller et al., 2003), negative affective traits may not lead to greater adiposity in African Americans because they do not promote systemic inflammation. Of note, because the study by Case and Stewart (2013) was cross-sectional, the directionality of the depression-inflammation association was unclear. Ethnic disparity in SES is another potential reason why affective traits fail to predict adiposity in African Americans. Indeed, one study of racial/ethnic differences in weight gain in a community sample over 34 years found that the differences in weight gain between African American and Caucasian women can largely be attributed to differences in socioeconomic conditions and not depression (Baltrus, Lynch, Everson-Rose, Raghunathan, & Kaplan, 2005). In addition, obesity has been associated with greater depression only among women of high SES (Stunkard, Faith, & Allison, 2003). Thus, the lower SES of African Americans (Jackson & Knight, 2006), including the current participants who have an average income less than $25,000 and less than 6 years of education, may explain the lack of negative affective trait-to-obesity associations.

To explain why greater adiposity leads to increases in negative affective traits in Caucasians but not African Americans, it is also useful to examine whether the proposed mechanisms underlying this direction of the relationship (Figure 1, Panel B) might also be less active or inactive among African Americans. Given that the Case and Stewart (2013) study was cross-sectional, it can also be viewed as evidence that obesity does not lead to greater negative affective traits in African Americans because obesity does not promote the elevated inflammation that, in turn, contributes to depressive symptom

development. This idea should certainly be explored as a growing field of research supports the macrophage theory of depression, which postulates that systemic inflammation plays a causal role in depression pathogenesis (Raison, Capuron, & Miller, 2006). Therefore, if African Americans do not exhibit the inflammation-depression connection (Case & Stewart, 2013), the obesity-to-inflammation-to-depression pathway is short-circuited. Other reasons why obesity may not lead to increases in negative affective traits among African Americans may involve differences in body image dissatisfaction, weight-related stigma and discrimination, and perceived health risk of obesity. Body dissatisfaction has been associated with depression in both men and women (Olivardia, Pope, Borowiecki, & Cohane, 2004; Stice, Hayward, Cameron, Killen, & Taylor, 2000); however, African American women have been shown to have less body dissatisfaction than white women (Roberts, Cash, Feingold, & Johnson, 2006). Relatedly, evidence suggests that, compared to Caucasian men, African American men may prefer larger female body types and endorse more favorable and fewer unfavorable characteristics of obese women (Jackson & McGill, 1996). African American women have also indicated greater tolerance of larger body size (Molloy & Herzberger, 1998). Importantly, acceptance of a larger body habitus within a culture may be associated with less perceived health risk of obesity and less stress regarding overweight or obese status. An example is the finding that only 44% of African Americans families perceived their child's weight to pose potential health problems despite nearly 70% of the children in the sample being obese (Young-Hyman, Herman, Scott, & Schlundt, 2000). Thus, obesity may not have predicted negative affective traits in this sample due to greater body

shape/weight acceptance and satisfaction, less weight-related stigma and discrimination, and less perceived health risk and associated stress associated with obesity.

Because other studies involving African American samples have not found that race-ethnicity moderates the relationship between negative affective traits and adiposity (Carpenter et al., 2000; Franko et al., 2005; Goodman & Whitaker, 2002; Heo et al., 2005; Merten et al., 2008; Pine et al., 1997), the relationship between negative affective traits and adiposity in this population is currently unclear. The null findings of the current study could also be explained by methodological issues including: (1) suboptimal measurement of the independent or dependent variables, (2) insufficient power, (3) uncontrolled error variability, and (4) confounders accounting for too much variance in the outcome variables (Kazdin, 2002). Each of these potential sources is addressed below.

First, differences in the type and quality of assessment of study variables can contribute to inconsistent findings (Kazdin, 2002). In this study, the adiposity measures were self-reported BMI, measured BMI, and body fat percent, all of which are indicators of total body adiposity. These variables had high correlations with DEXA, a gold standard for adiposity measurement, which suggests that these indicators had strong validity (see Adiposity Assessment section and Table 7). However, the current indicators could not distinguish between different kinds of fat tissue (i.e., visceral versus subcutaneous) or certain areas of body fat distribution (i.e., abdominal fat versus total body fat). Given the findings that affective traits may only be related to visceral fat and abdominal fat (Lewis et al., 2009; Wing et al., 1991), the focus on total body fat could

have contributed to the null results. Although other studies using indirect measures of total body fat have detected affective trait-adiposity associations (Blaine, 2008; de Wit et al., 2010; Luppino et al., 2010).

The use of continuous measures of affective traits (i.e., depressive and anxiety symptom severity) rather than categorical diagnostic categories (i.e., mood or anxiety disorders) may have also diminished the ability to detect associations. For instance, a diagnosis of a mood and/or anxiety disorder may reflect more severe psychopathology, which may result in a stronger relationship with obesity. Specific mood and anxiety diagnoses, which were not assessed in the current study, may also be differentially related to obesity. As an example, Scott and colleagues (2008) found that the strongest relationship between anxiety disorders and obesity was for post-traumatic stress disorder (PTSD) (OR = 2.64), an anxiety disorder rarely examined in other studies. Next, the symptom measure used in a particular study may measure different affective, cognitive, behavioral, or somatic components of an affective trait, and the strength of the relationship between obesity and a particular affective trait may vary as a function of these differences. A study by Ahlberg et al. (2002) looked at the relationship between waist-to-hip ratio and the items of several depression and anxiety scales. The authors found that, compared to non-obese participants, participants with a waist-to-hip ratio > 1 (i.e., abdominal obesity) had significantly higher scores on many items assessing somatic problems but rarely had higher scores for items assessing cognitive or affective components of depression or anxiety. Because over half of the CES-D items are affective or behavioral in nature (Kohout et al., 1993), the 4-item assessment of somatic symptoms may been inadequate in this study. A similar problem exists for the measure of anxiety

symptoms by the GAD-2, given that only two items were available. These two items assessed only the cognitive (i.e., worrying) and affective (i.e., nervousness) components of anxiety, whereas typical somatic symptoms of anxiety, such as heart palpitations, sweating, or respiratory difficulties, were not assessed. Thus, although the GAD-2 was internally consistent at baseline and follow-up (see Table 8), this 2-item scale may also have lacked adequate content validity, particularly for the somatic symptoms, resulting in the failure to predict changes in adiposity and vice versa.

A final issue concerning the assessment of study variables is possible restricted ranges in the affective traits as predictors or outcomes. First, the levels of depressive symptom and anxiety symptom severity were low across the study periods in this sample. Although the standard deviations of each variable were indicative of individual differences in the variables, the possible range may have been restricted by the clustering of the affective traits variables at the lower values of their distributions. This potential restriction could have reduced the ability of affective traits to predict change in adiposity and be predicted by adiposity. Second, as shown in Table 5, affective traits changed little over time at the group level. As an example, the average change in depression from baseline to 9-year follow-up was -0.19 points on the CES-D with 18.8 percent of participants with a 0-point change over this period. For GAD-2, a similar pattern emerged with an average 2-year change of 0.04 points on the GAD-2 and 52.3 percent of participants with a 0-point change. When a substantial percentage of participants exhibit limited change in a study variable, change is consequently difficult to predict.

Although differences in the assessment, range, and change of the study variables are potential sources of the null findings of this study, Kazdin's (2002) other

methodological issues are not as plausible. First, Type II error caused by insufficient power is unlikely to have occurred in this study, given that the minimum sample size thresholds for analyses were met (Kline, 2011). Second, it is also unlikely that the protocol of the study was subject to excessive error variance. "Extra noise" caused by such factors as inadequate training of research assistants or differences in delivering instructions can lead to low reliability and consequent attenuation in effects; however, all research assistants were extensively trained using a structured, standardized protocol. Lastly, the possibility of confounders accounting for too much variance in the outcome variables is unlikely, given that the models were adjusted for age and sex only and that these variables explained between 7% to 40% of the variance in the adiposity indicators and generally no significant variance in the affective traits. Taken together, previous investigations' results and the proposed explanations for the study findings support the possibility that the current results reflect the state of nature rather than the failure to detect a true relationship. While the aforementioned methodological issues and other study limitations (described below) cannot be ignored as potential contributors of these null findings, the current study had several strengths which justify its conclusion that affective traits may be unrelated to adiposity in African Americans. These strengths include the following: (1) a prospective cohort design, (2) a large sample of African Americans, (3) multiple assessments of affective traits and adiposity indicators at baseline and follow-up, and (4) the use of advanced modeling techniques, which could not only ascertain the unique and shared contribution of affective traits on obesity outcomes and vice versa but also simultaneously examine the bidirectional relationships between affective traits and adiposity.

Limitations

In addition the study's strengths, several limitations should be acknowledged. Along with those mentioned in the preceding section (i.e., no assessment of type of body fat or distribution; suboptimal assessment of some symptoms of depressive and anxiety; restricted range and change in study variables), two other notable limitations exist. First, many latent variables had only one single-item indicator (e.g., BMI), whereas others had multiple indicators (e.g., CES-D items) but came from a single instrument. Using multiple indicators, especially multiple instrument-level indicators, is advocated, as it can provide a more reliable and valid assessment of latent constructs (Kline, 2011). Second, although men were well-represented in the total sample and sex was used as a covariate in the analyses, the present study did not have enough male participants to run the models stratified by sex. This limitation is unfortunate, given the extensive evidence that the negative affective trait-obesity relationship has been found to be present only in women or to be stronger in women (Anderson et al., 2006; Carpenter et al., 2000; de Wit et al., 2010; Istvan et al., 1992; Jorm et al., 2003; Onyike et al., 2003).

Future Directions and Recommendations

Although this study indicates that negative and positive affective traits are not related to measures of adiposity among African Americans, replication is needed. Several recommendations can be made with regards to this future research. To begin, no studies have simultaneously examined depression, anxiety, and hostility in African Americans. Thus, future prospective studies should be designed with the following features: (1) all three negative affective traits should be examined, (2) African American

and other race-ethnicities should be better represented so that moderation by race can be examined, and (3) statistical methods should be used which allow researchers to ascertain the unique and shared effects (e.g., structural equation modeling). The unique and shared effects of these three traits must be provided in order to accurately determine whether the independent, overlapping, or proxy model is most consistent with the data, although the present study suggests that these models may not apply to African Americans.

Next, to address the methodological concerns presented in earlier sections, it is also recommended that researchers attempt to use a variety of obesity measures, including those that assess type of adiposity and regional adiposity. Although more expensive, technologies such as CT scans can allow researchers to precisely distinguish between different kinds of fat tissue (i.e., subcutaneous versus visceral) instead of using indirect measures of total adiposity, such as BMI. Future studies should also seek to use multiple, well-validated measures of affective traits and mood and anxiety disorder diagnoses. It might be useful to employ scales which adequately assess somatic symptoms, given these symptoms' strong association with obesity (Ahlberg et al., 2002). Additionally, more studies are needed which account for the effects of sample characteristics, including sex and SES. Future studies should likely perform separate analyses for men and women and include adequate representation of members from all SES strata.

Conclusions

Altogether, the results of this prospective cohort study suggest that negative and positive affective traits are not predictors or consequences of adiposity in middle-aged

African Americans. Although a substantial literature contradicts the current findings, most of these investigations have been based on predominantly Caucasian samples and have not examined race-ethnicity subgroups. Possible explanations for the current results include ethnic differences in the mechanistic pathways between affective traits and adiposity, such as systemic inflammation, socioeconomic conditions, body dissatisfaction, weight-related stigma and discrimination, and the perceived health risk of obesity.

The scientific implications of the current findings are that more research is needed to identify and understand the reasons why negative affective traits are unrelated to adiposity in African Americans. As mentioned above, these reasons may reflect ethnic differences in physiological or psychosocial mechanisms or may be due to differences in study methodology. All of these possibilities should be empirically explored and highlight the critical importance of examining race-ethnicity as a moderator of any observed associations between affective traits and adiposity. With regards to clinical implications, the current findings imply that the independent, overlapping, and proxy models (Figure 2) may not be applicable to middle-aged African Americans. Thus, the results suggest that this group may require obesity prevention or intervention programs with little to no emphasis on affective traits. Instead, a closer examination of socioeconomic factors and perceptions of obesity-related health risk may be warranted, as these factors may play a greater role in obesity development and maintenance for this population than do affective traits. Because African Americans continue to have the highest rates of obesity in the nation, programs should focus on culturally sensitive

primary, secondary, and tertiary interventions for obesity. These efforts can result in programs that are relevant to and effective for this high risk group.

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TABLES

Table 1. Description of Negative Affective Traits and Their Measures

Negative Affective Trait	Brief Description of Construct	Example Measure	Stability over Time (Test-retest Reliabilities)
Depression	Feelings of sadness or lack of pleasure (anhedonia)	Beck Depression Inventory (BDI)	.59-.86 [1]
Anxiety	Feelings of tension or worry	Spielberger Trait Anxiety Inventory (STAI)	.86 [2]
Hostility/ Anger	Mistrustful and hostile feelings and thoughts towards others	Spielberger Trait Anger Expression Inventory (STAXI)	.70-.77 [3]

[1] Yin & Fan, 2000
[2] Spielberger, Gorsuch, & Lushene, 1970
[3] Jacobs, Latham, & Brown, 1988

Table 2. Characteristics of Participants

	Baseline		7-year follow-up		9-year follow-up	
Variable	Mean or N	SD or %	Mean or N	SD or %	Mean or N	SD or %
Demographic Factors						
Age (years)	56.3	4.4	--	--	--	--
Women	626	62.7	--	--	--	--
Education (years)	5.4	5.3	--	--	--	--
Income						
$10,000 or less	162	16.9	--	--	--	--
$10,000-$15,000	98	10.2	--	--	--	--
$15,000-$20,000	116	12.1	--	--	--	--
$20,000-$25,000	109	11.3	--	--	--	--
$25,000-$35,000	156	16.2	--	--	--	--
$35,000-$50,000	153	15.9	--	--	--	--
$50,000-$75,000	115	12.0	--	--	--	--
$75,000 or greater	52	5.4	--	--	--	--
Adiposity Indicators						
Self-report BMI (kg/m^2)	30.3	6.9	30.5	7.0	31.1	7.0
Measured BMI (kg/m^2)	31.0	7.0	--	--	31.7	6.8
Body Fat % (BIA)	36.2	11.2	--	--	36.5	10.3
Affective Traits (range)						
CES-D-9 (0-27)	5.2	5.1	4.6	4.9	4.6	4.8
GAD-2 (0-6)	--	--	1.1	1.6	1.1	1.7
Vitality (0-12)	10.2	3.6	10.2	3.7	9.8	3.4
Positive Affect (0-12)	--	--	10.3	2.5	10.4	2.4
Covariates (range)						
Basic ADLs (0-7) †	0.7	1.5	0.4	0.7	--	--
Instrumental ADLs (0-8) †	0.8	1.5	0.6	0.8	--	--
LBFL (0-5)	1.7	1.8	1.9	1.9	--	--

Note. CES-D = Center for Epidemiologic Studies – Depression Scale. GAD = Generalized Anxiety Disorder Scale. BMI = body mass index. BIA = bioelectrical impedance analysis. DEXA = dual X-ray absorptiometry. ADL = activities of daily living. LBFL = lower body functional limitations.

†Ranges for variables at 7-year follow-up are as follows: Basic ADLs (0-2) and Instrumental ADLs (0-2).

Table 3. Characteristics of Participants (Maximum N = 411)

	Baseline		7-year follow-up		9-year follow-up	
Variable	Mean or N	SD or %	Mean or N	SD or %	Mean or N	SD or %
Demographic Factors						
Age (years)	56.2	4.4	--	--	--	--
Women	269	65.5	--	--	--	--
Education (years)	2.4	2.9	--	--	--	--
Income						
$10,000 or less	49	12.2	--	--	--	--
$10,000-$15,000	26	6.5	--	--	--	--
$15,000-$20,000	45	11.2	--	--	--	--
$20,000-$25,000	40	10.0	--	--	--	--
$25,000-$35,000	70	17.5	--	--	--	--
$35,000-$50,000	77	19.2	--	--	--	--
$50,000-$75,000	64	16.0	--	--	--	--
$75,000 or greater	52	30	--	--	--	--
Adiposity Indicators						
Self-report BMI (kg/m^2)	30.6	6.3	30.7	6.3	30.8	6.3
Measured BMI (kg/m^2)	31.0	6.3	--	--	31.5	6.6
Body Fat % (BIA)	36.9	10.2	--	--	36.2	10.3
Affective Traits (range)						
CES-D-9 (0-27)	4.4	4.7	4.1	4.5	4.3	4.6
GAD-2 (0-6)	--	--	1.0	1.5	1.0	1.6
Vitality (0-12)	9.8	3.4	9.8	3.5	9.6	3.3
Positive Affect (0-12)	--	--	10.6	2.2	10.5	2.4
Covariates (range)						
Basic ADLs (0-7) †	0.5	1.2	0.3	0.6	--	--
Instrumental ADLs (0-8) †	0.6	1.3	0.5	0.7	--	--
LBFL (0-5)	1.3	1.7	1.6	1.7	--	--

Note. CES-D = Center for Epidemiologic Studies – Depression Scale. GAD = Generalized Anxiety Disorder Scale. BMI = body mass index. BIA = bioelectrical impedance analysis. DEXA = dual X-ray absorptiometry. ADL = activities of daily living. LBFL = lower body functional limitations.

†Ranges for variables at 7-year follow-up are as follows: Basic ADLs (0-2) and Instrumental ADLs (0-2).

Table 4. Participants Reporting a History of Chronic Diseases Across Study Periods

	Baseline		7-year follow-up	
	N	%	N	%
Disease/Events				
Hypertension	645	64.8	--	--
Diabetes	232	23.3	207	30.6
Kidney Disease	49	4.9	20	3.1
Cardiovascular Disease†	217	21.7	144	22.0

†Cardiovascular disease variable is a composite, including participants who endorsed any of the following symptoms: angina, heart attack, congestive heart failure, and/or stroke.

Table 5. Change in Adiposity and Affective Traits from Baseline to Follow-up in Participants with Complete Adiposity Data

	7-year Change† *Mean ± SD*	Paired t-test results	9-year Change† *Mean ± SD*	Paired t-test results
Self-reported BMI	-0.11 ± 3.7	$t(396) = 0.56$	0.21 ± 3.6	$t(410) = 1.17$
Measured BMI	--	--	-0.50 ± 3.5	$t(410) = 2.89$*
Body Fat %	--	--	0.67 ± 6.8	$t(410) = 1.96$
CES-D-9	0.23 ± 4.6	$t(394) = 1.00$	0.07 ± 4.4	$t(410) = 0.34$
Vitality	0.02 ± 3.5	$t(397) = 0.09$	0.20 ± 3.2	$t(409) = 1.24$
GAD-2	--	--	-0.01 ± 1.6	$t(391) = 0.10$
CESD-PA	--	--	0.15 ± 2.6	$t(393) = 1.14$

Note. BMI = body mass index. CES-D = Center for Epidemiologic Studies – Depression Scale. GAD = Generalized Anxiety Disorder Scale. CESD-PA = Center for Epidemiologic Studies – Depression Scale Positive Affect Subscale.

†Mean arithmetic change scores were computed by subtracting the baseline score from the follow-up score.

*$p < .05$

Table 6. Adiposity Indicators in the African American Health Study

Variable Name	Description of Variable	Established Cut-Points for Obesity	Maximum Sample at Baseline, 7-yr, and 9-yr Follow-up
Body mass index (BMI)	Weight in kilograms divided by height in square-meters (kg/m^2).	Class I: BMI = 30 – 34.99 kg/m^2* Class II: BMI = 35 – 39.99 kg/m^2 Class III: BMI ≥ 40 kg/m^2	Self-reported BMI 998, 680, 579 Measured BMI 998, (na), 579
Body fat percentage (BF%) as measured by bioelectrical impedance analysis (BIA)	The percentage of fat mass in the body as measured by the degree that an electrical current passed through the body is impeded. The impedance is used to estimate fat-free mass and fat mass because fat mass is less conductive than fat-free mass.	*Females*† 20-30 years: BF% ≥ 38 40-59 years: BF% ≥ 39 60-79 years: BF% ≥ 41 *Males*† 20-30 years: BF% ≥ 26 40-59 years: BF% ≥ 27 60-79 years: BF% ≥ 29	998, (na), 579

Note. (na) = variable data not available for a particular wave.
*Cut-points for BMI were taken from WHO (2005).
†Cut-points for BF% were taken from Gallagher et al. (2003).

Table 7. Correlations between Affective Traits, Adiposity Indicators, and Demographic Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Baseline																				
1. Age	--																			
2. Education	.02	--																		
3. CES-D-9	-.03	.03	--																	
4. Vitality	.02	.00	**-.57**	--																
5. Self-report BMI	-.02	**-.07**	.02	**-.17**	--															
6. Measured BMI	.01	-.03	.02	**-.15**	**.93**	--														
7. Body fat % (BIA)	.01	-.06	.01	**-.15**	**.67**	**.73**	--													
8. Body fat % (DEXA)	.00	-.14	-.04	**-.11**	**.65**	**.68**	**.84**	--												
7-year Follow-up																				
9. CES-D-9	-.04	**-.09**	**.49**	**-.36**	**.09**	.08	.01	.02	--											
10. Vitality	-.03	**.23**	**-.40**	**.45**	**-.17**	**-.17**	**-.12**	**-.15**	**-.54**	--										
11. CES-D PA	.04	**.09**	**-.36**	**.24**	-.01	.01	.03	.01	**-.54**	**.45**	--									
12. GAD-2	.01	-.06	**.38**	**-.27**	-.02	-.05	-.05	-.03	**.62**	**-.45**	**-.47**	--								
13. Self-report BMI	-.06	**.14**	.06	**-.20**	**.82**	**.86**	**.65**	**.63**	.05	**-.19**	.03	-.04	--							
9-year Follow-up																				
14. CES-D-9	-.02	**-.11**	**.53**	**-.39**	**.11**	**.10**	.07	.09	**.57**	**-.44**	**-.42**	**.43**	**.09**	--						
15. Vitality	.00	.07	**-.46**	**.51**	**-.19**	**-.15**	**-.14**	**-.16**	**-.49**	**.63**	**.42**	**-.41**	**-.20**	**-.59**	--					
16. CES-D PA	-.03	**.09**	**-.38**	**.27**	.02	.04	.02	.02	**-.39**	**.29**	**.42**	**-.37**	.01	**-.50**	**.45**	--				
17. GAD-2	.01	-.07	**.42**	**-.27**	.02	-.02	-.01	-.02	**.51**	**-.39**	**-.35**	**.50**	.03	**.65**	**-.47**	**-.44**	--			
18. Self-report BMI	**-.09**	-.09	.07	**-.22**	**.85**	**.86**	**.62**	**.61**	.08	**-.19**	-.01	-.04	**.91**	.06	**-.18**	.02	.00	--		
19. Measured BMI	-.06	**-.15**	.07	**-.21**	**.83**	**.86**	**.65**	**.65**	**.09**	**-.19**	-.05	-.04	**.90**	.08	**-.14**	.03	.03	**.97**	--	
20. Body fat % (BIA)	-.03	**-.16**	.08	**-.22**	**.56**	**.61**	**.77**	**.80**	.04	**-.18**	-.03	.01	**.63**	.05	**-.13**	.02	.01	**.71**	**.74**	--
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

Bolded, shaded coefficients are significant at $p < .05$ level. Bolded only coefficients are significant at $p \leq .07$

Note. CES-D = Center for Epidemiologic Studies Depression Scale. BMI = body mass index. BIA = bioelectrical impedance analysis. DEXA = dual

Table 8. Internal Consistency of Multi-Item Measures

	Baseline		7-year follow-up		9-year follow-up		Temporal Reliability
	Cronbach's α	# of Items	Cronbach's α	# of Items	Cronbach's α	# of Items	Average 8-year Test-retest *r* [a]
Affective Traits							
CES-D-9	.82	9	.80	9	.82	9	.53
Vitality	.81	4	.83	4	.82	4	.53
GAD-2	--	--	.78	2	.79	2	.50
CESD-PA	--	--	.68	4	.69	4	.42

Note. CES-D = Center for Epidemiologic Studies Depression Scale. GAD = Generalized Anxiety Disorder Scale. CESD-PA = Center for Epidemiologic Studies Positive Affect Scale.

[a]Average test-retest reliability was calculated as the average of the correlation between baseline and 7-year follow-up scores and the correlation between baseline and 9-year follow-up scores.

Table 9. Rotated Factor Loadings[a] for the 11-item Form of the CES-D in the AAH Study Sample at Baseline

Items	Factor 1 Depressed Affect	Factor 2 Positive Affect	Factor 3 Somatic Symptoms	Factor 4 Interpersonal
I felt sad.	**.67**	.36	-.05	.26
I felt depressed.	**.62**	.44	.16	.26
I felt lonely.	**.54**	.33	-.05	.36
I enjoyed life.	.05	**.88**	.13	.07
I was happy.	.38	**.68**	.12	.05
I could not get going.	.65	.15	**.32**	.11
I felt everything I did was an effort.	.54	.09	**.44**	.07
I did not feel like eating; my appetite was poor.	.15	.16	**.89**	.12
My sleep was restless.	.75	.01	**.12**	.07
People were unfriendly.	.08	.07	.09	**.86**
I felt that people disliked me.	.24	.06	.09	**.78**

[a] Loadings greater than .40 are underlined.

Note. CES-D = Center for Epidemiologic Studies Depression Scale. AAH = African American Health. Bolded items represent the items that have typically had the highest loadings for each factor in previous studies (Radloff, 1977 and Kohout et al., 1993).

Table 10. Correlates of Missingness among Study Variables

	Age			Sex			Education		
	Baseline	7-year Follow-up	9-year Follow-up	Baseline	7-year Follow-up	9-year Follow-up	Baseline	7-year Follow-up	9-year Follow-up
Self-reported BMI	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	↑	↑
Measured BMI	n.s.	--	n.s.	n.s.	--	n.s.	n.s.	--	↑
Body Fat %	n.s.	--	n.s.	n.s.	--	n.s.	n.s.	--	↑
CES-D-9	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	↑	↑
Vitality	n.s.	n.s.	n.s.	n.s.	Females	n.s.	n.s.	↑	↑
GAD-2	--	n.s.	n.s.	--	Females	n.s.	--	↑	↑
CESD-PA	--	n.s.	n.s.	--	Females	n.s.	--	↑	↑

Note. BMI = body mass index. CES-D = Center for Epidemiologic Studies – Depression Scale. GAD = Generalized Anxiety Disorder Scale. CESD-PA = Center for Epidemiologic Studies – Depression Scale Positive Affect Subscale. "↑" indicates that higher age or education levels predicted greater missing data. "↓"indicates that lower age or education levels predicted greater missing data. "Females" or "Males" indicates the group that had more missing data. "n.s." indicates that age, education, and sex did not predict missing data.

Table 11. Fit Statistics for Demographics-adjusted (Age and Sex) Structural Models of Participants with Complete Data

Objective	*N*	χ^2	RMSEA (CI_{90})	SRMR	CFI	Path: Affect → Adiposity	Path: Adiposity → Affect
Objective #1: Depressive Symptom Severity and Adiposity over 9 years							
Self-reported BMI (Figure 7) [a]	459	342.85*	.044 (.036-.051)	.041	.97	$\beta = 0.02, z = 0.54$	$\beta = 0.09, z = 1.88$†
Measured BMI (Figure 8) [a]	460	404.91*	.051 (.044-.057)	.046	.97	$\beta = 0.01, z = 0.38$	$\beta = 0.06, z = 1.33$
BF% (Figure 9) [a]	431	375.75*	.049 (.042-.056)	.046	.97	$\beta = -0.00, z = -0.08$	$\beta = 0.03, z = 0.45$
Objective #2: Depressive Symptom Severity, Anxiety Symptom Severity, and Adiposity over 2 years							
Self-reported BMI (CES-D & GAD-2) (Figure 10) [a]	488	564.05*	.050 (.045-.056)	.048	.98	CESD $\beta = 0.03, z = 0.64$ GAD $\beta = -0.04, z = -0.94$	CESD $\beta = 0.04, z = 0.91$ GAD $\beta = -0.02, z = -0.49$
Self-reported BMI (CES-D only) [a]	494	451.64*	.055 (.049-.061)	.052	.97	$\beta = -0.00, z = -0.25$	$\beta = 0.03, z = 0.60$
Self-reported BMI (GAD-2 only)	488	6.88	.000 (.000-.055)	.014	1.00	$\beta = -0.02, z = -1.08$	$\beta = 0.04, z = 1.01$
Secondary Objectives: Positive Affective Traits (Vitality) and Adiposity over 9 years							
Self-reported BMI (Figure 11) [b]	540	73.43*	.045 (.031-.060)	.029	.99	$\beta = -0.01, z = -0.31$	$\beta = 0.05, z = 1.03$
Measured BMI (Figure 12) [b]	459	85.81*	.055 (.040-.070)	.034	.98	$\beta = -0.00, z = -0.13$	$\beta = -0.07, z = -1.29$
BF% (Figure 13) [b]	430	58.12*	.039 (.020-.057)	.028	.99	$\beta = -0.06, z = -1.65$	$\beta = -0.03, z = -0.40$
Secondary Objectives: Positive Affective Traits (Vitality and CESD-PA) and Adiposity over 2 years							
Self-reported BMI (Vitality only) (Figure 14) [b]	497	75.52*	.048 (.033-.063)	.033	.99	$\beta = -0.01, z = -0.63$	$\beta = 0.03, z = 0.72$
Self-reported BMI (CESD-PA only) (Figure 15) [b]	484	69.64*	.041 (.025-.056)	.037	.98	$\beta = -0.04, z = -2.03$*	$\beta = -0.01, z = -0.15$

Note. BMI = body mass index. BF% = body fat percent. CES-D = Center for Epidemiologic Studies – Depression Scale. GAD = Generalized Anxiety Disorder Scale. CESD-PA = Center for Epidemiologic Studies – Depression Scale Positive Affect Subscale.

[a]Figures show the results for models using the full information maximum likelihood (FIML) estimation procedure. For participants with complete data, specification of the models was exactly equivalent to the FIML models except that the errors of the two interpersonal items of the CES-D were freed to covary.

[b]Figures show the results for model using the FIML estimation procedure. For participants with complete data, specification of the models was exactly equivalent to the FIML models.

*$p \leq .05$, †$p \leq .07$

FIGURES

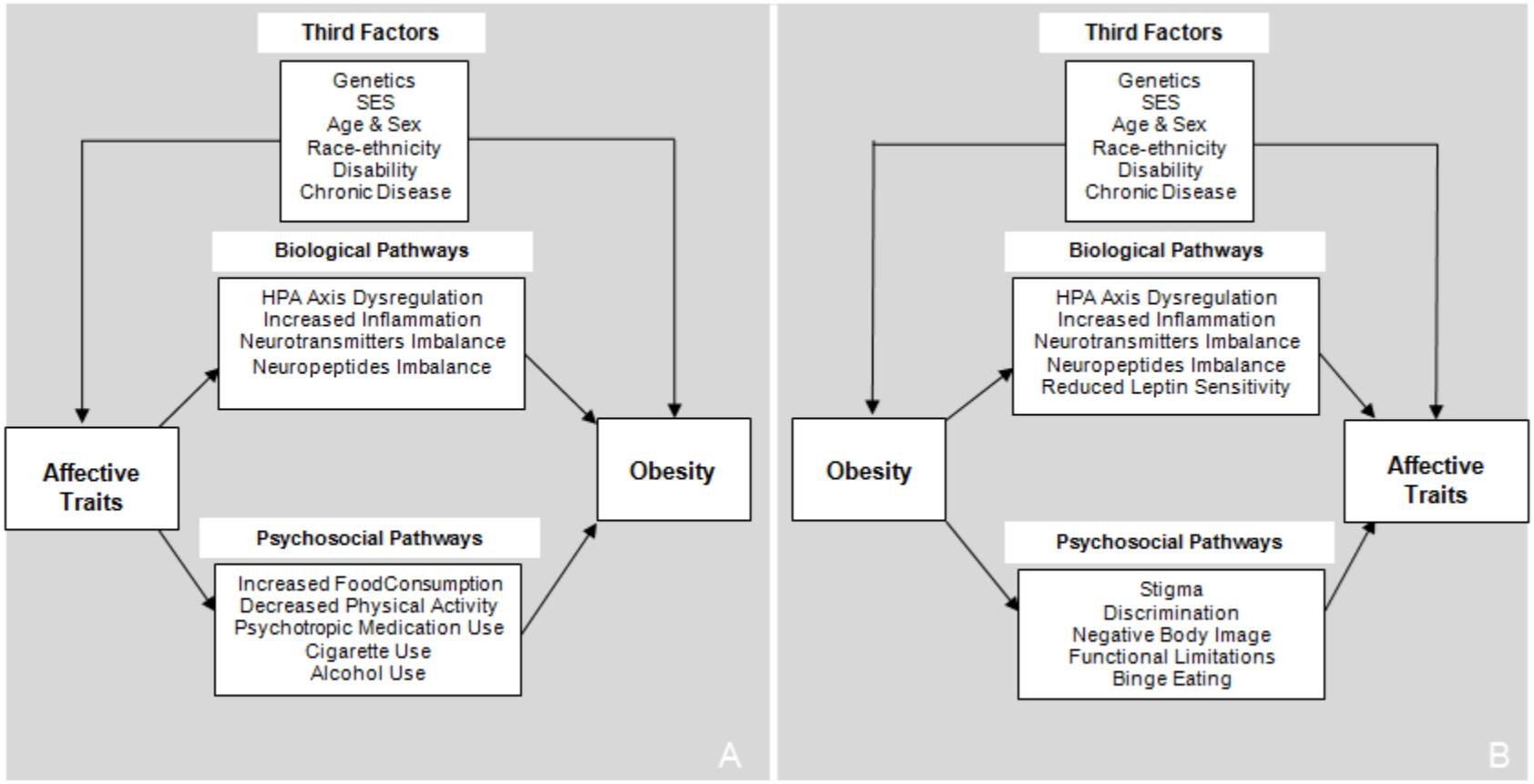

Figure 1. Biopsychosocial models of affective traits and obesity. Panel A depicts affective traits as predictors of obesity and the potential mechanisms. Panel B depicts the affective consequences of obesity and the potential mechanisms.

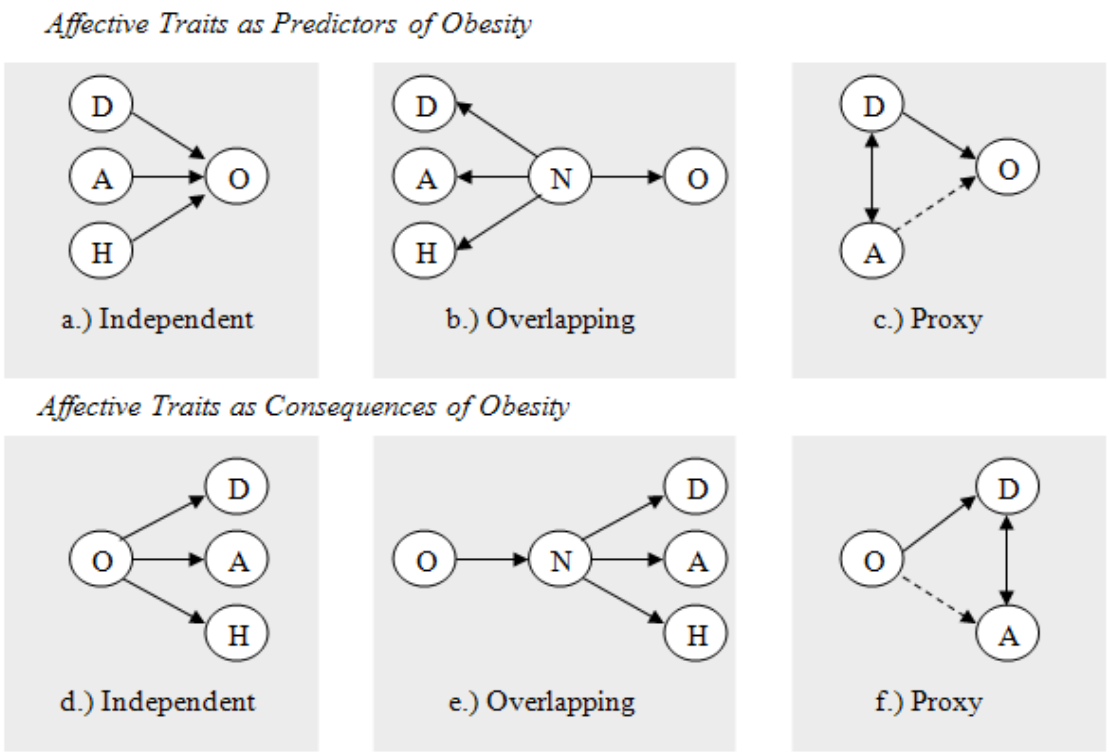

Figure 2. Different ways in which negative affective traits may be related to obesity. D = depression; A = anxiety; H = hostility; O = obesity; N = negative affectivity/neuroticism

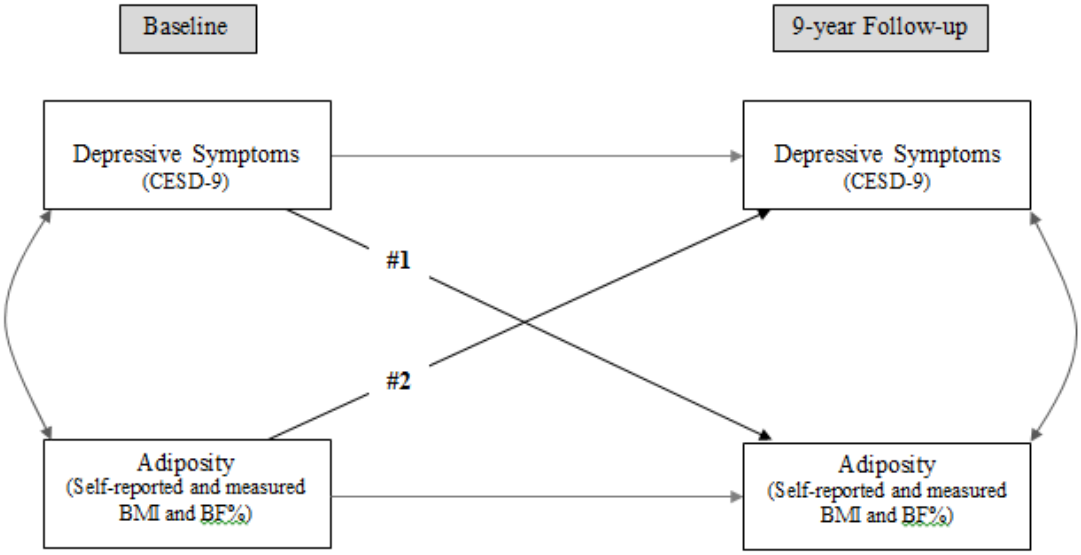

Figure 3. Hypotheses #1 and #2 for Primary Objective #1: Depressive symptom severity as a predictor and a consequence of adiposity.

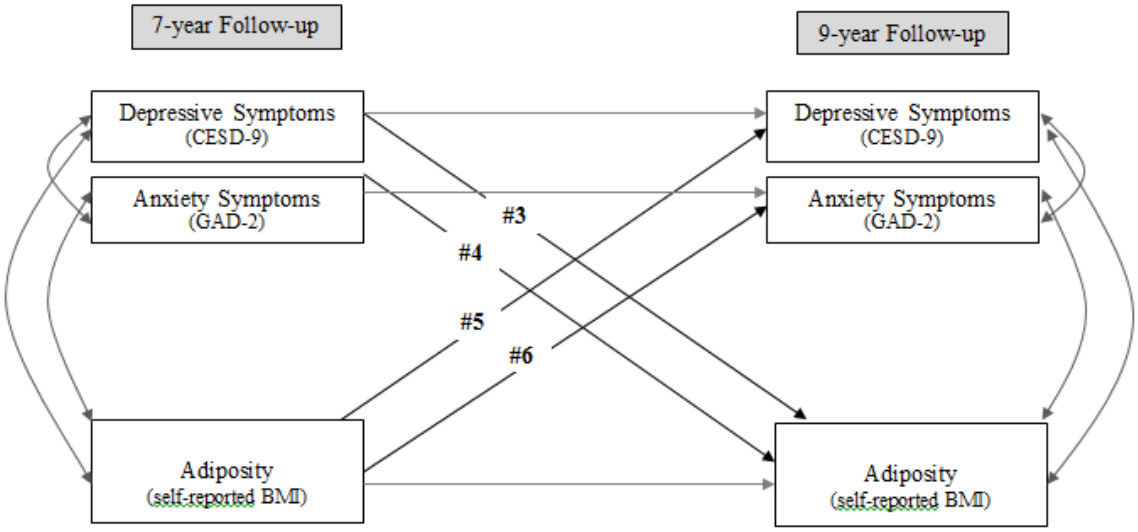

Figure 4. Hypotheses #3 through #6 for Primary Objective #2: Depressive and anxiety symptom severity as independent predictors and consequences of adiposity.

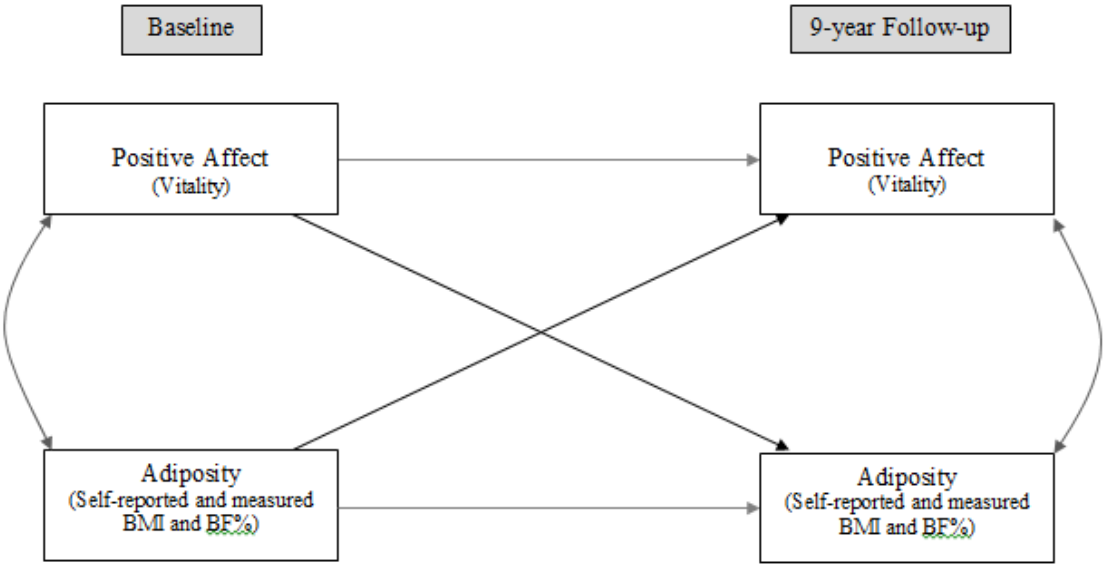

Figure 5. Secondary Objectives with 9-year follow-up data: Positive affect variables as predictors and consequences of adiposity.

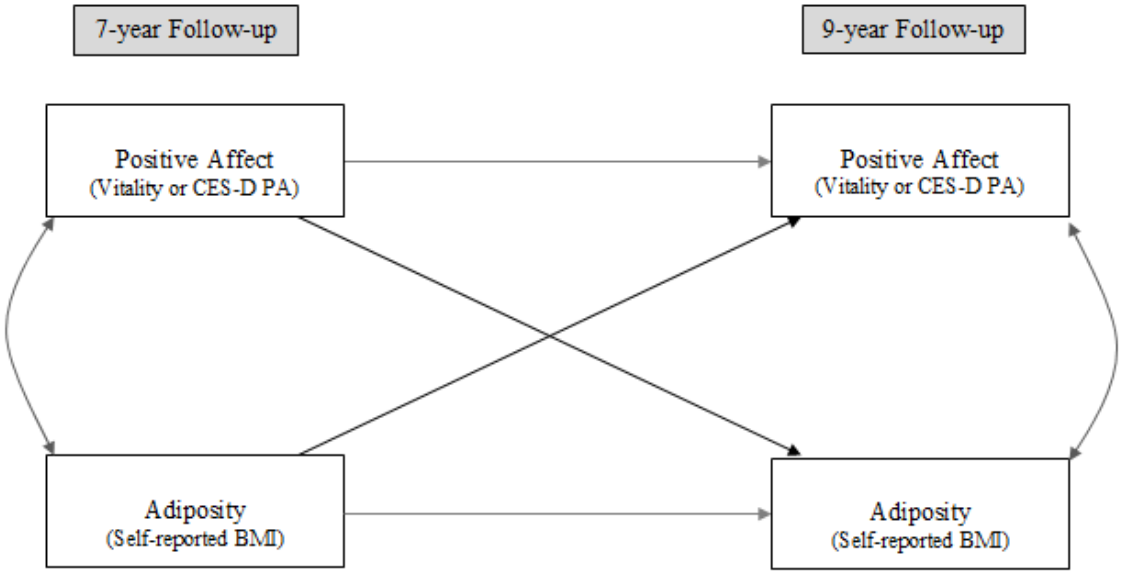

Figure 6. Secondary Objectives with 2-year follow-up data: Positive affect variables as predictors and consequences of adiposity.

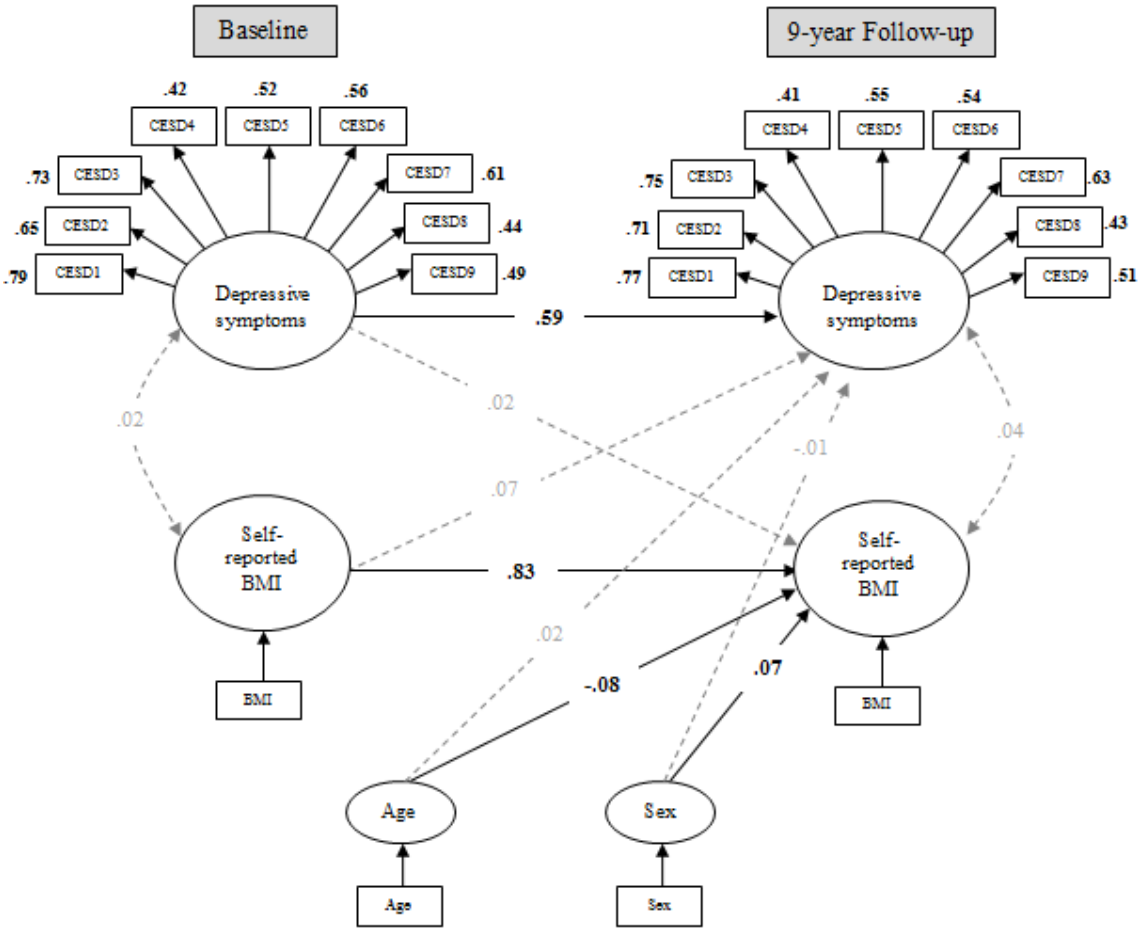

Figure 7. Demographics-adjusted model for Hypotheses 1 and 2 using self-reported body mass index over 9 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

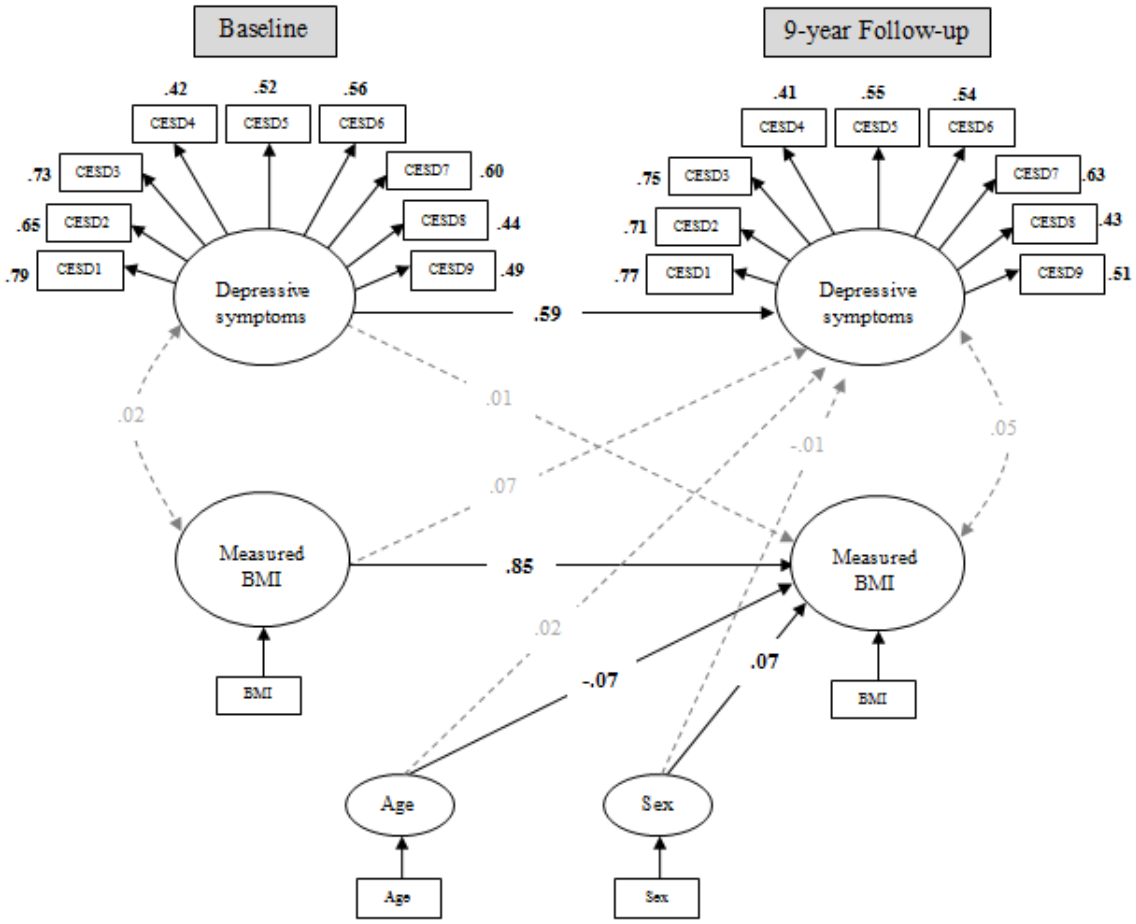

Figure 8. Demographics-adjusted model for Hypotheses 1 and 2 using measured body mass index over 9 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

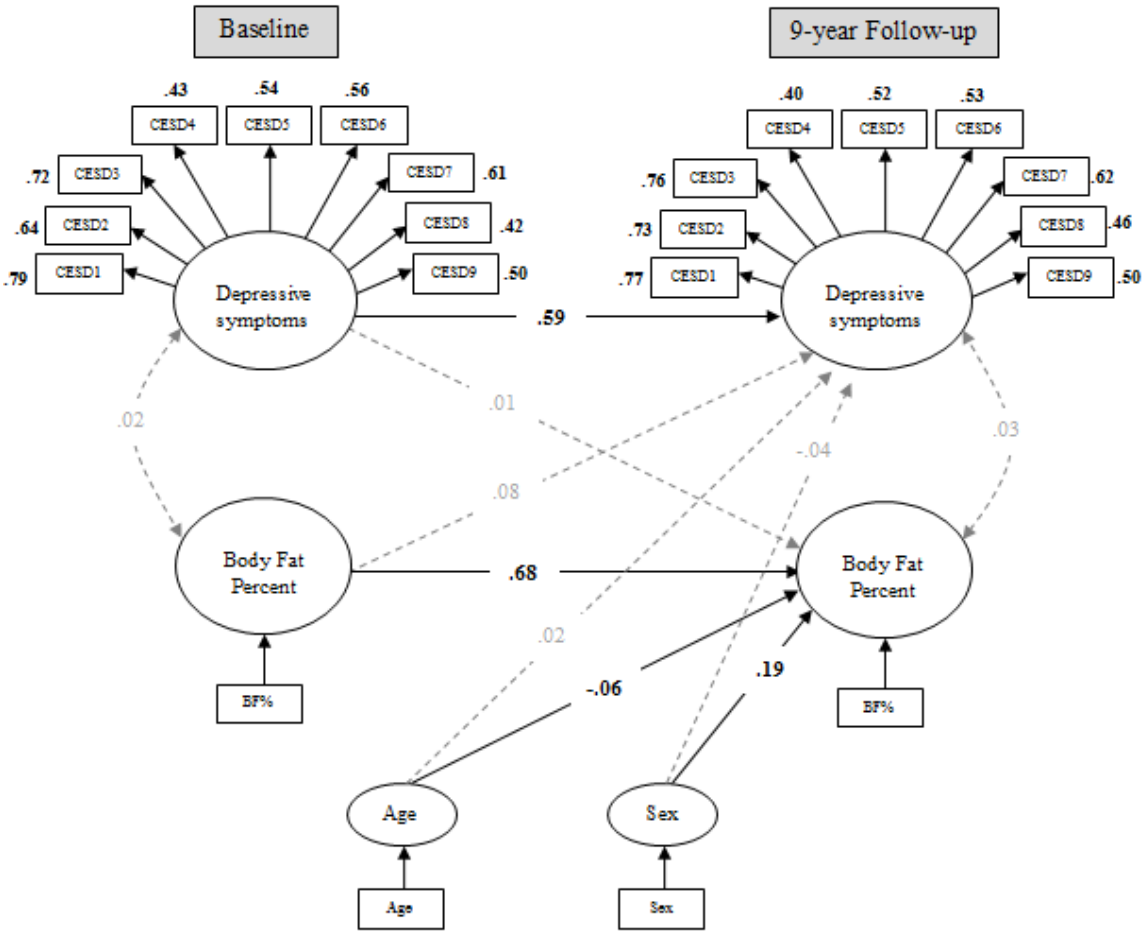

Figure 9. Demographics-adjusted model for Hypotheses 1 and 2 using body fat % over 9 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

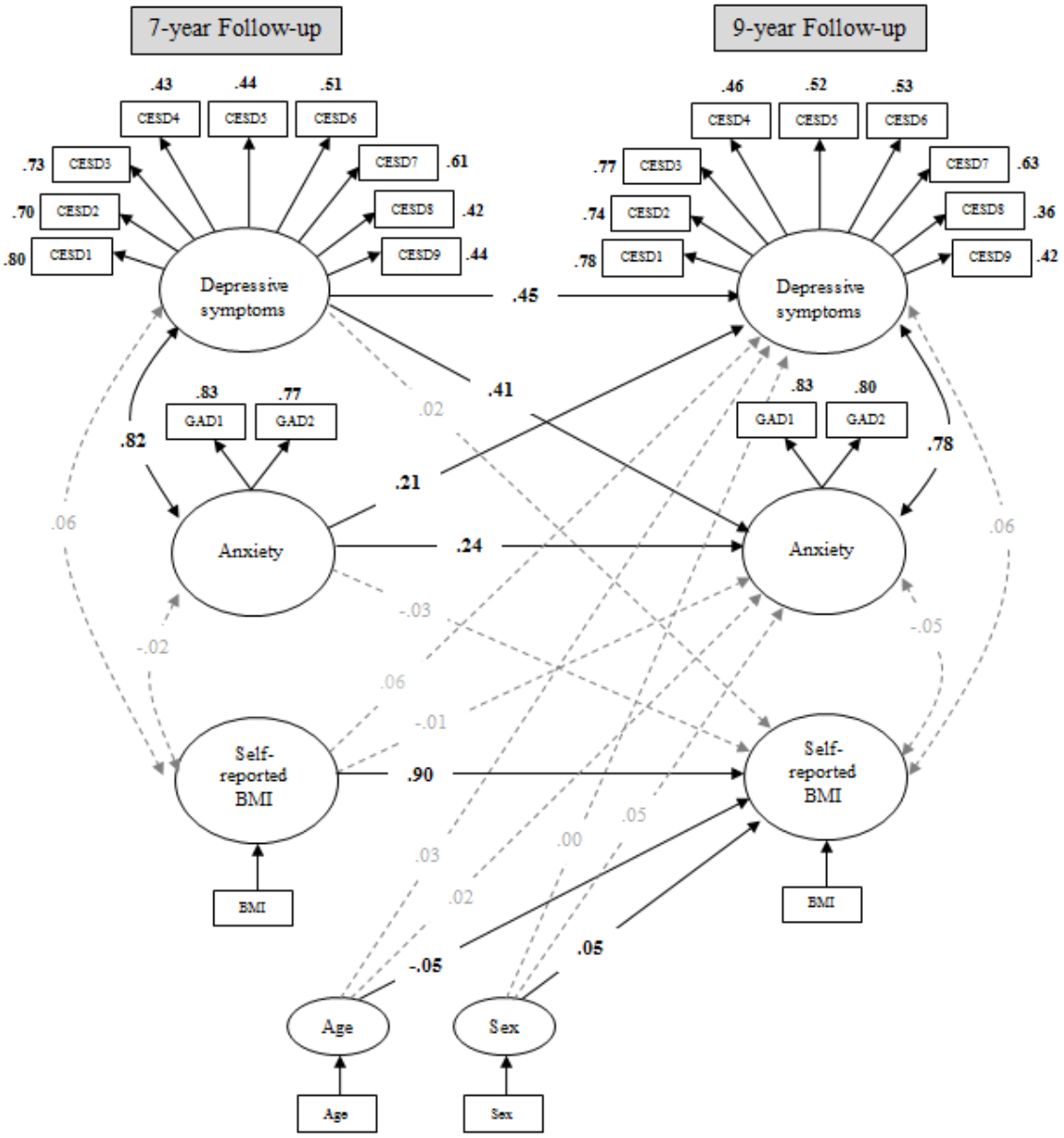

Figure 10. Demographics-adjusted model for Hypotheses 3 through 6 using self-reported body mass index over 2 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

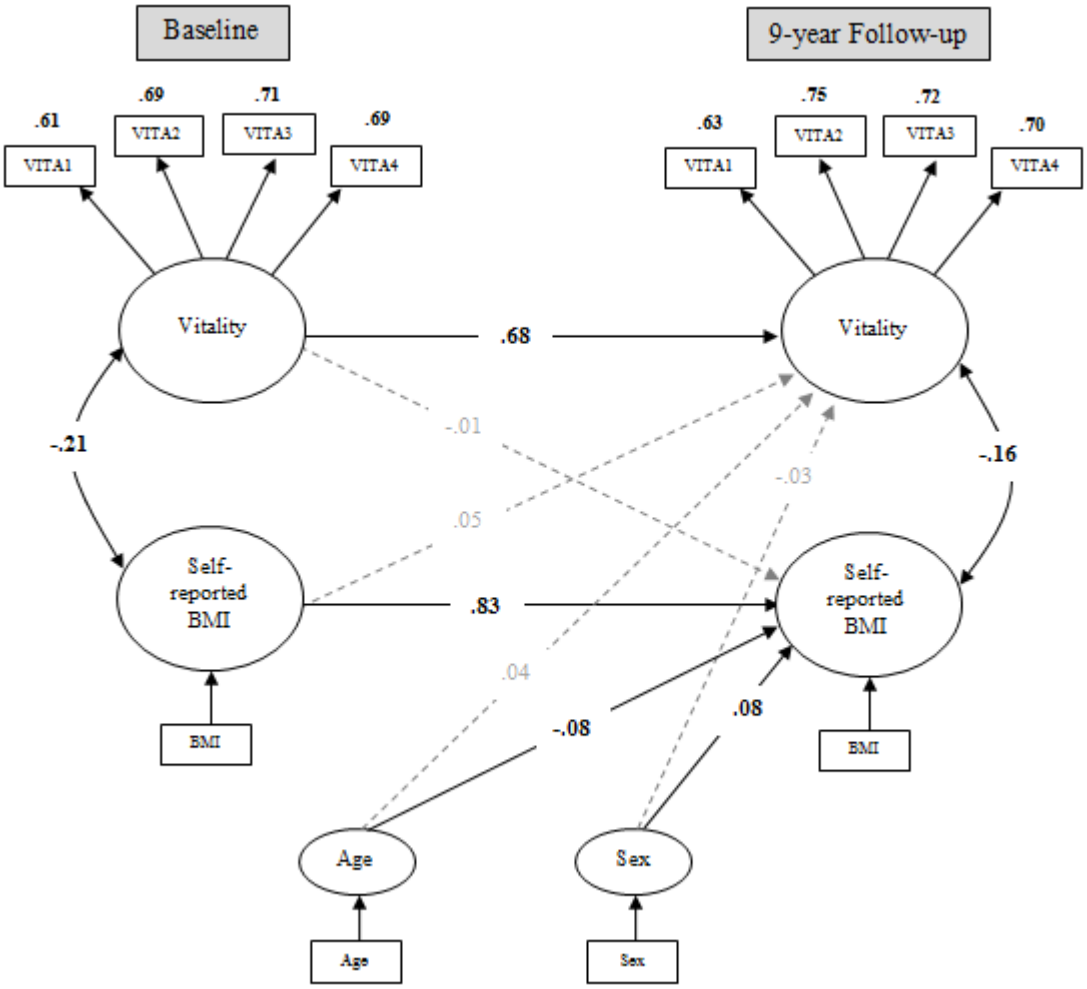

Figure 11. Demographics-adjusted model for secondary objectives using Vitality and self-reported body mass index over 9 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

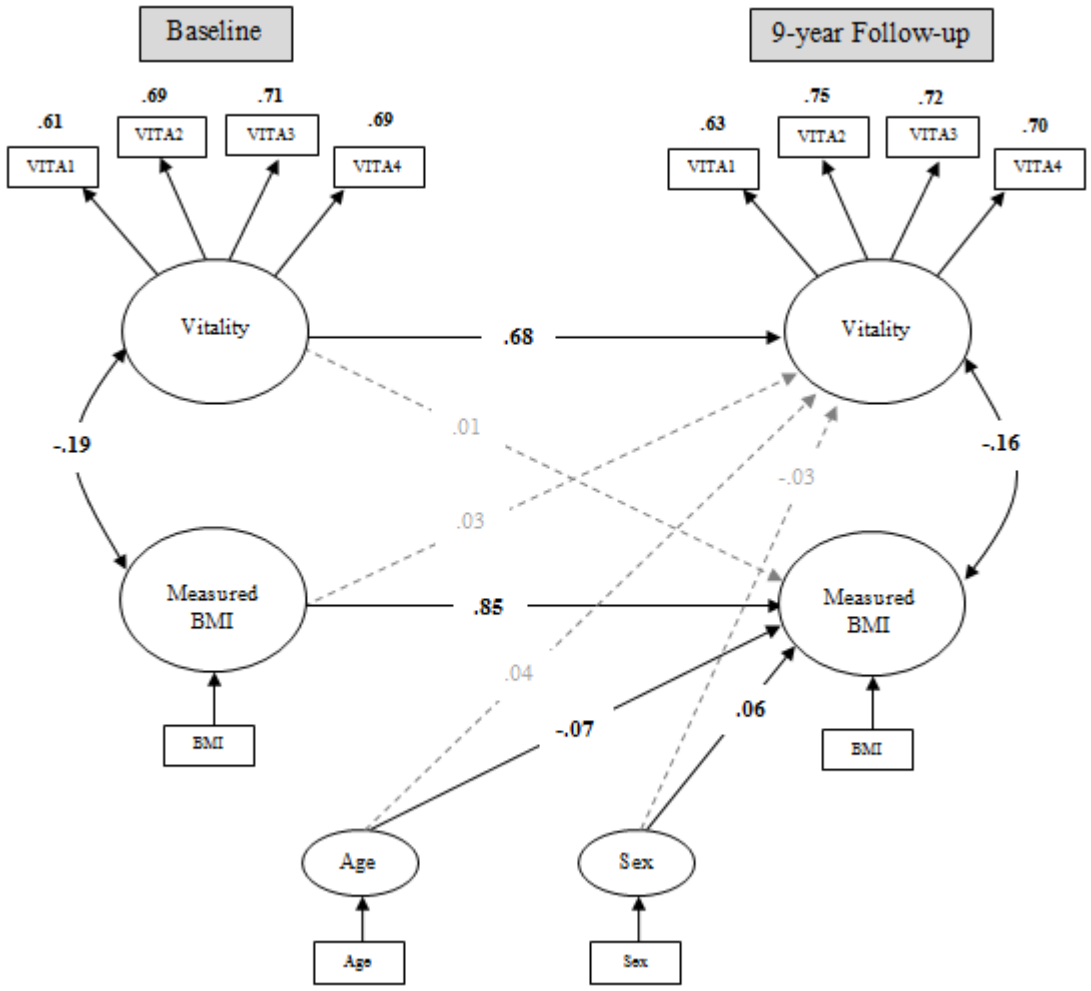

Figure 12. Demographics-adjusted model for secondary objectives using Vitality and measured body mass index over 9 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

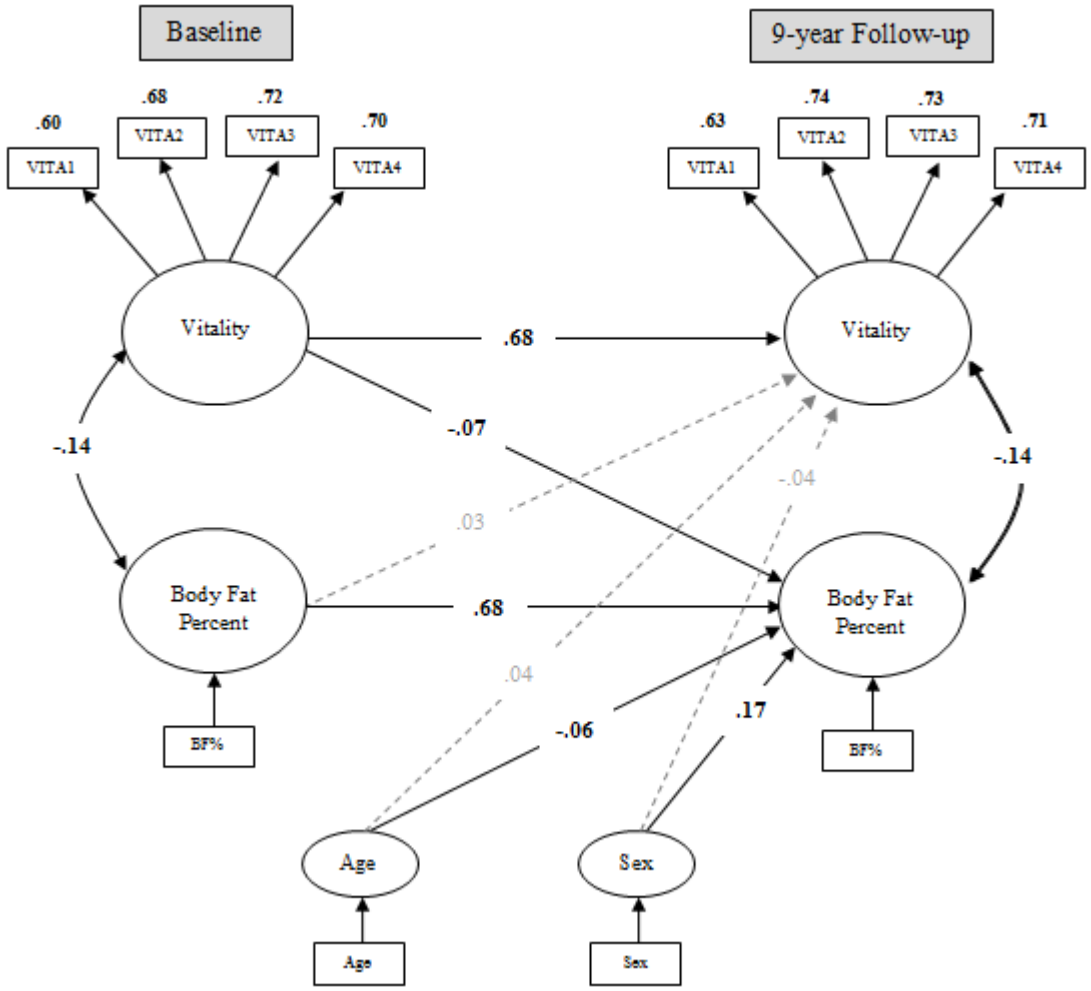

Figure 13. Demographics-adjusted model for secondary objectives using Vitality and body fat percent over 9 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

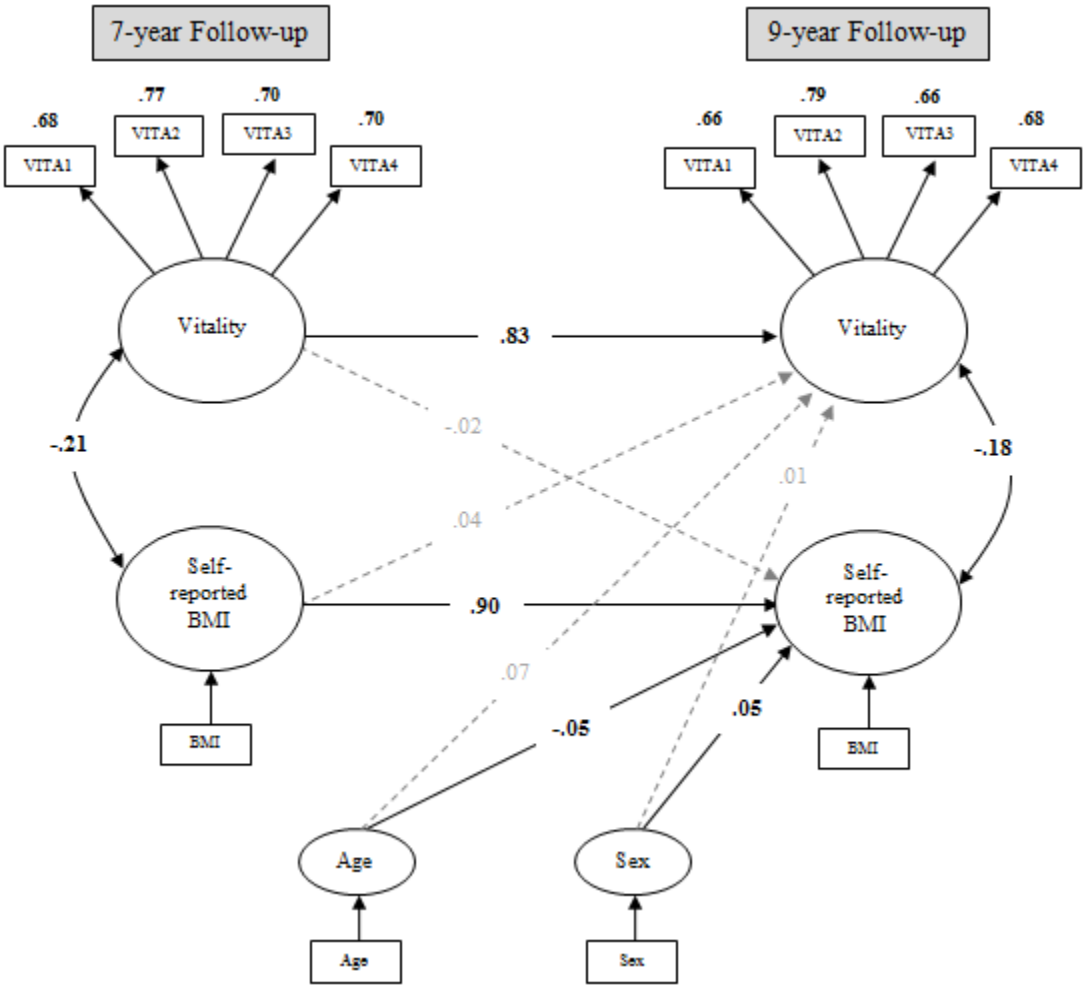

Figure 14. Demographics-adjusted model for secondary objectives using Vitality and self-reported body mass index over 2 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

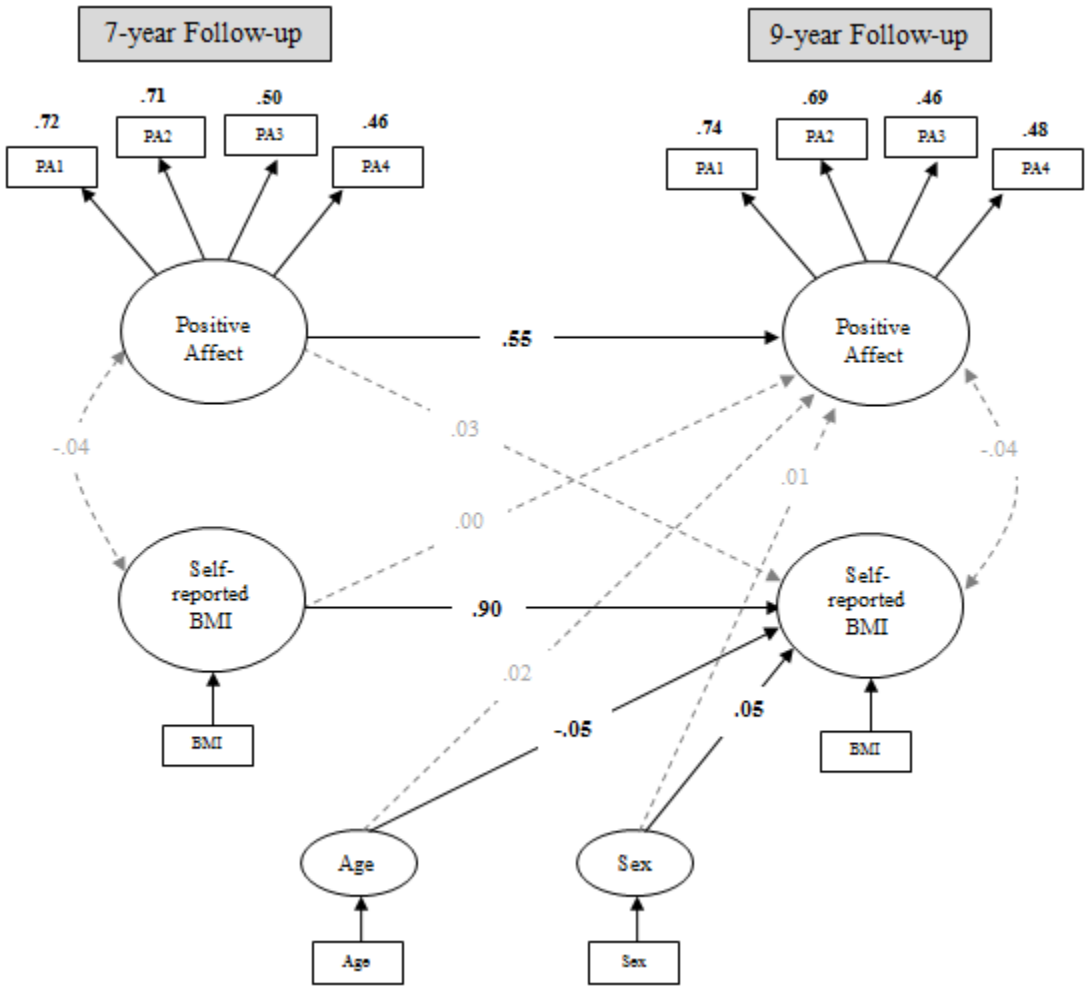

Figure 15. Demographics-adjusted model for secondary objectives using Positive Affect and self-reported body mass index over 2 years. Values for unidirectional arrows (structural paths) are standardized regression coefficients or factor loadings; values associated with bidirectional arrows are Pearson correlation coefficients. Black, solid paths are significant ($p < 0.05$); grey, dashed paths are not significant. Error terms and correlations with baseline age and sex are omitted for clarity.

APPENDICES

Appendix A: Characteristics of Studies

Characteristics of Studies Examining Multiple Affective Traits and Adiposity Indicators

Study (First author, year)	Total Sample (% female, % non-white, if reported)	Affective Trait Measures	Adiposity Indicator(s)	Covariates
Depression & Anxiety				
Bardone, 1998*	459 adolescents (100% female)	Composite scores: Any mood disorder (MDD or dysthymia) or any anxiety disorder (overanxious disorder, separation anxiety, simple fears, simple phobia, social phobia) at age 15 assessed by the DISC-C.	Measured BMI at 6-year follow-up	Conduct disorder, age at menarche, maternal BMI, parental SES, absence of father figure, parental smoking, childhood health, maternal health
Strine, 2005	78,866 adults	≥ 14 days of depression symptoms or anxiety symptoms in the past 30 days	Self-reported BMI ≥ 30	Age, sex, race/ ethnicity, education, marital status
Simon, 2006	9,125 adults (51% female, 27% non-white)	Composite scores: Any lifetime and past-year mood disorder (MDD, dysthymia, or bipolar I or II) or any lifetime and past-year anxiety disorder (GAD, panic disorder, or agoraphobia without panic disorder) assessed by the CIDI	Self-reported BMI > 30	Age, sex, smoking, substance use disorders
Pickering, 2007	40,388 adults (55% female)	Past-year MDD, dysthymia, GAD, panic disorder, social phobia, or specific phobia assessed by the AUDADIS-IV	Self-reported BMI 25 to 29.9 and BMI ≥ 30	Age, race-ethnicity, marital status, income, education, region, urbanicity, physical conditions, impairment, & stressful life events, alcohol, drug, & tobacco use, substance use disorders, bipolar I or II, personality disorders
Chiriboga, 2008*	572 adults (48% female, 13% non-white)	BDI & BAI	Measured weight adjusted for height at 1-year follow-up	Height, age, education, marital status, energy intake, alcohol use, physical activity, smoking, seasonality

Characteristics of Studies Examining Multiple Affective Traits and Adiposity Indicators (Appendix A continued)

Study (First author, year)	Total Sample (% female, % non-white, if reported)	Affective Trait Measures	Adiposity Indicator(s)	Other Covariates
Depression & Anxiety				
Scott, 2008	12,992 adults & adolescents (52% female)	Composite scores: Any past-year mood disorder (MDD, dysthymia, bipolar I or II, mania, or hypomania) or any past-year anxiety disorder (GAD, panic disorder, agoraphobia without panic disorder, social phobia, specific phobia, PTSD, or OCD) assessed by the CIDI	Self-reported BMI ≥ 30	Age, sex, race/ethnicity, education, smoking status
Toker, 2008	3,880 adults (39% female)	PHQ-9 & 4-item scale for anxiety	Waist circumference > 120cm for men, > 88 cm for women	Age, number of daily cigarettes, physical exercise, education, burnout
Chou, 2009 Chou, 2010	13,420 older adults (55% female, 20% non-white)	Composite scores: Any lifetime and current mood disorder (MDD or dysthymia) or any lifetime and current anxiety disorder (GAD, panic disorder, agoraphobia without panic disorder, social phobia, or specific phobia) assessed by the AUDADIS-IV	Self-reported BMI > 30	Age, gender, race, martial status, education, employment status, personal income, personality disorders except obsessive-compulsive, OCD, alcohol abuse or dependence, nicotine dependence, bipolar I or II
Mather, 2009	34,900 adults & adolescents (56% female)	Composite scores: Any lifetime mood disorder (MDD or mania) or any lifetime anxiety disorder (panic disorder, agoraphobia without panic disorder, panic attack, or social phobia) assessed by the CIDI	Self-reported BMI ≥ 30	Age, sex (in analysis of total sample), educational attainment, physical illness burden, substance use disorders
Williams, 2009	979 adults (100% female)	Composite scores: Any lifetime mood disorder (MDD, dysthymia, minor depression, bipolar disorder, mood disorder due to medical condition, or substance-induced mood disorder) or any lifetime anxiety disorder (GAD, panic disorder,	Measured BMI ≥ 25 and ≥ 30, waist circumference ≥ 80 cm, body fat mass, body fat percentage, weight	Age, cigarette use, alcohol consumption, physical activity

Characteristics of Studies Examining Multiple Affective Traits and Adiposity Indicators (Appendix A continued)

Study (First author, year)	Total Sample (% female, % non-white, if reported)	Affective Trait Measures	Adiposity Indicator(s)	Other Covariates
Depression & Anxiety				
Leventhal, 2010	41,654 adults	Past-year major depressive episode & lifetime history of anxiety disorders assessed using AUDADIS-IV	Self-reported BMI ≥ 30 and continuous BMI	Age, sex, race-ethnicity, marital status, education, urbanicity, lifetime history of personality disorders, alcohol use and drug use disorders, mania
Depression & Hostility				
Haukkala, 2001*	285 adults (58% female)	BDI & Cynical Distrust Scale	Measured BMI gain >2 units, 3-year follow-up	Age, gender, baseline BMI, dieting history, education, smoking status, alcohol consumption, exercise, sweet, fat, and cereal consumption
Fassino, 2003	145 adults (100% female)	BDI & STAXI-Trait Anger	Measured BMI ≥ 30	Obese individuals with and without binge eating disorder (BED) were compared to normal weight control group. Only comparisons between controls and non-BED obese participants were used for this review
Lewis, 2009	418 adults (100% female, 45% non-white)	CES-D & CMHS	Visceral fat, subcutaneous fat	Total body fat or BMI, age, race-ethnicity, time since assessment

Note. MDD = major depressive disorder. DISC-C = Diagnostic Interview Schedule. BMI = body mass index. GAD = generalized anxiety disorder. CIDI = Composite International Diagnostic Interview. AUDADIS-IV = Alcohol Use Disorder and Associated Disabilities Interview Schedule. BDI = Beck Depression Inventory. BAI = Beck Anxiety Inventory. PTSD = post traumatic stress disorder. OCD = obsessive-compulsive disorder. PHQ = Patient Health Questionnaire. NOS = not otherwise specified. SCID/NP = Structured Clinical Interview for DSM-IV-TR Research Version, Non-patient Edition.

Appendix B: Results of Studies

Results of Unadjusted and Adjusted Analyses from Studies Examining the Associations of Multiple Affective Traits with Adiposity Indicators

Study (First author, year)	Unadjusted for other traits(s)[a]			Adjusted for other traits(s)[b]		
	Depression	Anxiety	Hostility/ Anger	Depression	Anxiety	Hostility/ Anger
Depression & Anxiety						
Bardone, 1998*	…	…		0	0	
Strine, 2005	…	+		…	+	
Simon, 2006	+	+		+	0	
Pickering, 2007	+	…		0	+	
Chiriboga, 2008*	0	+		0	+	
Scott, 2008	+	+		0	+	
Toker, 2008	+	…		+	…	
Chou, 2009/2010	…	+		…	0	
Mather, 2009	+	+		+	+	
Williams, 2009	+	0		+	0	
Leventhal, 2010	+	…		+	…	
Depression & Hostility						
Haukkala, 2001*	+		0	+		0
Fassino, 2003	+		+	…		0
Lewis, 2009	+		+	+		+
Proportion of studies reporting significant effects	**10 of 11** (91%)	**6 of 7** (86%)	**2 of 3** (67%)	**7 of 11** (64%)	**5 of 9** (56%)	**1 of 3** (33%)

Note. A plus sign (+) indicates that a significant positive relationship between the negative affective trait and an adiposity indicator was detected. A zero (0) indicates that no relationship was detected. Three periods (…) indicates that the affective trait was assessed but the relationship between that affective trait and adiposity was not reported. [a]Unadjusted analyses refer to those in which a negative affective trait was examined in relation to an adiposity indicator without adjusting for other affective trait. [b]Adjusted analyses refer to those in which a negative affective trait was examined in relation to an adiposity indicator while adjusting for one or more other affective trait. *Prospective study

Appendix C: Scales

Center for Epidemiologic Studies – 11-item Depression Scale (CES-D)

(Available at Baseline, 7-year follow-up, and 9-year follow-up)

Interviewer: "Next, I will read a list of ways you might have felt or behaved. For each statement, think about how often you have felt or behaved this way during the past week. Was it rarely or none of the time, some or a little of the time, occasionally or a moderate amount of time, or most or all of the time?"

RESPONSE OPTIONS
0 = Rarely or none of the time (less than 1 day)
1 = Some or a little of the time (1 - 2 days)
2 = Occasionally or a moderate amount of time (3 - 4 days)
3 = Most or all of the time (5 - 7 days)
8 = Don't know
9 = Refused

Interviewer then read the statements below. After each statement, the participant was asked: "Did you feel this way rarely or none of the time, some or a little of the time, occasionally or a moderate amount of time, or most or all of the time?"

1. I did not feel like eating; my appetite was poor.
2. I felt depressed.
3. I felt that everything I did was an effort.
4. During the past week, my sleep was restless.
5. I was happy.
6. I felt lonely.
7. People were unfriendly
8. I enjoyed life.
9. I felt sad.
10. I felt that people disliked me
11. I could not get "going."

Appendix C (continued)

Generalized Anxiety Disorder-2 scale (GAD-2)
(Available at 7-year follow-up and 9-year follow-up)

Interviewer: "Now some questions about how you have been feeling in the past two weeks."

RESPONSE OPTIONS
0 = Not at all
1 = Several days
2 = More than half the days
3 = Nearly everyday
8 = Don't know
9 = Refused

Interviewer then read the questions below. After each question, the participant was asked: "Would that be not at all, several days, more than half of the days, or nearly every day?

1. In the past two weeks, how often have you been bothered by feeling nervous, anxious, or on edge?

2. In the past two weeks, how often have you been bothered by not being able to stop or control worrying?

Appendix C (continued)

Vitality Subscale of the SF-36
(Available at Baseline, 7-year follow-up, and 9-year follow-up)

Interviewer: The next questions are about how you feel and how things have been with you during the past 4 weeks. As I read each statement, please give me the one answer that comes closest to the way you have been feeling; is it all of the time, most of the time, some of the time, a little of the time, or none of the time?

RESPONSE OPTIONS
1 = All of the time
2 = Most of the time
3 = Some of the time
4 = A little of the time
5 = None of the time
8 = don't know
9 = refused

Interviewer then read the questions below. After each question, the participant was asked:
All of the time, most of the time, some of the time, a little of the time, or none of the time?

1. How much of the time during the past 4 weeks did you feel full of life?

2. How much of the time during the past 4 weeks did you have a lot of energy?

3. How much of the time during the past 4 weeks did you feel tired?

4. How much of the time during the past 4 weeks did you feel worn out?

Appendix C (continued)

Positive Affect scale from the CES-D
(Available at 7-year follow-up and 9-year follow-up)

Center for Epidemiologic Studies – 4-item Positive Affect Subscale (CES-D)

Interviewer: "Next, I will read a list of ways you might have felt or behaved. For each statement, think about how often you have felt or behaved this way during the past week. Was it rarely or none of the time, some or a little of the time, occasionally or a moderate amount of time, or most or all of the time?"

RESPONSE OPTIONS
0 = Rarely or none of the time (less than 1 day)
1 = Some or a little of the time (1 - 2 days)
2 = Occasionally or a moderate amount of time (3 - 4 days)
3 = Most or all of the time (5 - 7 days)
8 = Don't know
10 = Refused

Interviewer then read the statements below. After each statement, the participant was asked: "Did you feel this way rarely or none of the time, some or a little of the time, occasionally or a moderate amount of time, or most or all of the time?"

1. I was happy.*
2. I enjoyed life.*
3. I felt that I was as good as other people.
4. I felt hopeful about the future.

*Items 1 and 2 were also available at baseline.

VITA

VITA

MISTY A.W. HAWKINS, Ph.D.

EDUCATION AND TRAINING

2009-2013 INDIANA UNIVERSITY-PURDUE UNIVERSITY INDIANAPOLIS
APA-Accredited
Ph.D.
Specialization: Clinical Health Psychology
Advisor: Jesse C. Stewart, Ph.D.
Dissertation: Affective Traits and Adiposity: A Prospective, Bidirectional Analysis of the African American Health Study Data
Preliminary Exam
Systematic Review: Bidirectional Relationships between Overlapping Negative Traits and Obesity

2012-2013 WEST VIRGINIA UNIVERSITY SCHOOL OF MEDICINE CHARLESTON
APA-Accredited
Internship
Department of Behavioral Medicine and Psychiatry
Charleston Area Medical Center

2007-2009 INDIANA UNIVERSITY-PURDUE UNIVERSITY INDIANAPOLIS
APA-Accredited
M.S.
Specialization: Clinical Health Psychology
Advisor: Jesse C. Stewart, Ph.D.
Thesis: The Influence of Cognitive Emotion Regulation Strategies on Cardiovascular Recovery from Stress

2003-2007 INDIANA UNIVERSITY BLOOMINGTON
B.S.
With Honors
With Highest Distinction
Major: Psychology; Minors: Biology, French; Certificate: Neuroscience
Advisor: Edward R. Hirt, Ph.D.
Undergraduate Honors Thesis: Mental Illness Stigma and the Law of Contagion

GRANTS, FELLOWSHIPS, SCHOLARSHIPS, AND AWARDS

2013	Outstanding Resident Researcher Award – West Virginia University Department of Behavioral Medicine and Psychiatry
2013	Clinical Psychology Award for Research Excellence – Honorable Mention – IUPUI Psychology Department
2013	Research Travel Grant – IUPUI Graduate Student Office ($500)
2010-2012	National Science Foundation Fellowship - GK-12 Program ($30,000/year plus tuition and conference travel funds for 2 academic years)
2012	Award for Outstanding Research Oral Presentation – Indiana Psychological Association
2012	Award for Research in Diversity - Indiana Psychological Association
2012	Outstanding Student Research Poster - Indiana Psychological Association
2011	Research Travel Grant – IUPUI Graduate Student Office ($500)
2010	Outstanding Student Research Poster - Indiana Psychological Association
2010	Research Travel Grant – IUPUI Graduate Student Office ($500)
2010	Outstanding Graduate Student Teaching Award - IUPUI School of Science
2010	Outstanding Graduate Student Instructor Award - IUPUI Psychology Department
2010	Favorite Professor Award - IUPUI Athletics Department
2009	Research Travel Grant – IUPUI Graduate Student Office ($500)
2007	Outstanding Undergraduate Research Award - Indiana University ($100)
2006	Phi Beta Kappa - Indiana University
2006	International Experiences Grant – Indiana University Honors College ($2000)
2005	Burnett Master Scholarship - Indiana University Honors College ($1000)
2005	Capstone Undergraduate Research Grant - Indiana University ($300)
2003-2007	Valedictorian Scholarship - Indiana University ($4000)
2003-2007	Freese Academic Scholarship - Indiana University ($4000)
2003-2007	Eli Lilly Academic Merit Scholarship ($ Full College Tuition for 4 years)

PEER-REVIEWED PUBLICATIONS

Hawkins, M.A.W., & Stewart, J.C. (2012). Do negative affective traits have independent associations with excess adiposity? *Journal of Psychosomatic Research, 73*, 243-250.

Stewart, J.C., Zielke, D.J., **Hawkins, M.A.W.,** Williams, D.R., Carnethon, M.R., Knox, S.S., & Matthews, K.A. (2012). Depressive symptom clusters and 5-year incidence of coronary artery calcification: The Coronary Artery Risk Development in Young Adults Study. *Circulation, 126*, 410-417.

Redelman, C.V., **Hawkins, M.A.W.,** Drumwright, F.R., Ransdell, B., Marrs, K., & Anderson, G.G. (2012). Inquiry-based examination of chemical disruption of bacterial biofilms. *Biochemistry and Molecular Biology Education, 40,* 191-197.

PEER-REVIEWED PUBLICATIONS (CONTINUED)

Hawkins, M.A.W., Stewart, J.C, & Fitzgerald, G.J. (2011). Combined effect of depressive symptoms and hostility on autonomic nervous system function. *International Journal of Psychophysiology, 81,* 317-323.

Stewart, J.C, Rand K.L., **Hawkins, M.A.W.,** & Stines, J. (2011). Associations of the shared and unique aspects of positive and negative emotional factors with sleep quality. *Personality and Individual Differences, 50,* 609-614.

CONFERENCE PRESENTATIONS

National

Hawkins, M.A.W., Stewart, J.C., & Miller, D.K. (2013, March). *Depressive symptom clusters as predictors of body mass index and body fat percent in African Americans over a 9-year period*. Oral presentation to be presented at the 70th annual meeting of the American Psychosomatic Society. Received "Newsworthy" designation.

Hawkins, M.A.W., & Stewart, J.C. (2011, August). *Do overlapping negative affective traits have independent associations with obesity?* Poster presentation at the 2011 American Psychological Association Convention, Washington, D.C.

Hawkins, M.A.W., Redelman, C.V., Marrs, K., Stewart, J.C. (2011, March). *Behavioral medicine in the high school classroom: A lesson in psychology and physiology.* Poster presentation at the 2011 National Science Foundation annual meeting of the GK-12 Program, Washington, D.C.

Hawkins, M.A.W., Stewart, J.C., & Marrs, K.A. (2011, March). *Learning about the connections between depression, stigma, and obesity*. Poster presentation at the 2011 Annual Meeting of the National Science Foundation GK-12 Program, Washington, D.C.

Stewart, J.C., Zielke, D.J., Hawkins, M.A.W., Williams, D.R., Carnethon, M.R., Knox, S.S., & Matthews, K.A. (2011, March). *Depressive symptom clusters as predictors of 5-year incidence of coronary artery calcification: The CARDIA study.* Paper presentation at the 69th annual meeting of the American Psychosomatic Society, San Antonio, TX.

Hawkins, M.A., Stewart, J.C., & Fitzgerald, G.J. (2010, March). *Combined effect of depressive symptoms and hostility on autonomic nervous system function.* Poster presentation at the 68th annual meeting of the American Psychosomatic Society, Portland, OR.

CONFERENCE PRESENTATIONS (CONTINUED)

Stewart, J.C., Hawkins, M.A., & Zielke, D.J. (2009, March). *Associations of positive and negative psychological factors with indices of cardiac autonomic balance and regulatory capacity*. Poster presentation at the 67th annual meeting of the American Psychosomatic Society, Chicago, IL.

Regional

Hawkins, M.A.W., Selby-Nelson, E., Fields, S. (2013, April). *Promoting patient engagement in primary care: The role of "warm hand-offs."* Poster presented at the 2013 Charleston Area Medical Center/West Virginia University Research Day, Charleston, WV.

Hawkins, M.A.W., Chelf, M., Sirbu, C. (2013, April). *Evaluation of the Herridge Cardiopulmonary Questionnaire in cardiac patients."* Poster presented at the 2013 Charleston Area Medical Center/West Virginia University Research Day, Charleston, WV.

Hawkins, M.A.W., Stewart, J.C., & Rand, K.L. (2012, October). *Why is hope helpful? The influence of hope on emotional reactions to stress.* Poster presented at the annual meeting of the West Virginia Psychological Association, Pipestem, WV.

Hawkins, M.A.W., Stewart, J.C., & Miller, D.K. (2012, October). *Positive affect predicts future body mass index: A 2-year prospective analysis of the African American Health Study.* Poster presented at the Indiana Psychological Association Annual Conference, Indianapolis, IN.

Hawkins, M.A.W., Pearce, R., Judd, M. & Marrs, K. (2012, April). *IUPUI GK-12 Program: Health psychology meets freshmen biology.* Poster presented at the 2012 IUPUI Research Day, Indianapolis, IN.

Hawkins, M.A.W. & Stewart, J.C. (2011, April). *Are overlapping negative affective traits independent predictors of obesity?* Poster presentation at the 2011 IUPUI Research Day, Indianapolis, IN.

Hawkins, M.A. (2007, May) *Mental illness stigma: Mechanisms involving the contagion effect and implications*. Poster presentation at the Annual Indiana University Honors Thesis Banquet, Bloomington, IN.

Hawkins, M.A. (2005, April). *Characteristics of social defeat associated with immune response in Siberian hamsters* (*Phodopus sungorus*). Poster presentation at the 12th annual Indiana University Animal Behavior Conference, Bloomington, IN.

RESEARCH EXPERIENCE

Fall 2011-Present — PRE-DOCTORAL INTERN RESEARCH
Supervisor: Cristian Sirbu, M.D., Ph.D., Scott Fields, Ph.D., and Emily Selby-Nelson, Psy D
I was involved in two separate, ongoing research studies at my internship site. The first study investigates the relationship between psychosocial factors in cardiac patients participating in a Cardiac Rehabilitation program. I have been asked to create and prepare the dataset with the intent of conducting analyses on this ongoing study. I am also currently working on a manuscript examining how hostility, depression, and social support change over time during Cardiac Rehabilitation. For the second project, I am working on a study examining how "warm hand-offs" and other variables predict follow-up appointments in primary care patients. I am working with the study investigator to identify the study variables, prepare the dataset, and conduct analyses using archival patient medical record data. For this project, I will assist with writing of the manuscript for publication.

Fall 2011-Present — DISSERTATION RESEARCH
"Affective Traits and Adiposity: A Prospective, Bidirectional Analysis of the African American
Health Study Data"
Chair: Jesse Stewart, Ph.D.
The purpose of this study is to evaluate whether overlapping emotional factors are associated with changes in adiposity and vice versa. I have already obtained a limited use dataset from the African American Health study, a prospective cohort study of 1,000 African Americans, which contains multiple measures of affective traits and adiposity (e.g., body mass index, dual x-ray absorptiometry, and bioelectrical impedance analysis). I am using latent variable path analysis in Lisrel to determine whether specific affective traits are independently related to measures of adiposity in African Americans. I anticipate completing data analysis by January 2013 and defending by May 2013.

Fall 2010-Spring 2012 — GK-12 RESEARCH ON STUDENT LEARNING
Supervisor: Kathleen Marrs, Ph.D.
I have prepared an Institutional Review Board (IRB) application to conduct research on high school student learning and reactions to having a scientist-practitioner in the high school classroom. The sample was inner city students, predominantly African American or Hispanic. I designed a project that exposed students to health psychology research (a heart rate reactivity lab) and have collected data to determine whether their ideas about the mind-body connection. changed after the activity. In addition, I also acted as a statistical consultant for another fellow (a microbiologist) in the program.

RESEARCH EXPERIENCE (CONTINUED)

Fall 2007-Present — RESEARCH ASSISTANTSHIP, LABORATORY COORDINATOR
Supervisor: Jesse Stewart, Ph.D.
I have prepared Institutional Review Board (IRB) applications for a variety of projects. I have helped prepare and write grant applications. I have been involved in the preparation and writing of manuscripts. I have supervised undergraduate research assistants and coordinated lab meetings. As a complement to my dissertation, which examines archival data, I have also designed an independent data collection study to examine a potential mechanism of the negative emotion factors-adiposity relationship. My hypothesis that attentional biases toward food-related stimuli may partially explain the path from depression to obesity development will be tested in a sample of approximately 50 depressed and non-depressed overweight individuals. I will be using eye-tracking equipment to assess attentional biases. I anticipate completing data collection by August 2013.

Fall 2007-Fall 2009 — MASTER'S THESIS RESEARCH
"The Influence of Cognitive Emotion Regulation Strategies on Cardiovascular Recovery from Stress"
Chair: Jesse Stewart, Ph.D.
I participated in all stages of a study evaluating how the cognitive regulation of emotion may delay cardiovascular recovery in response to a psychological stressor. I formulated the design and protocol of the study. I have collected data using psychophysiological recording of cardiovascular and autonomic nervous system responses to stress, including EKG and impedance cardiography. I have scored psychophysiological data using Mindware Technologies software. Coordinated data collection. Subjects included introductory psychology students. I trained and supervised undergraduates in running experimental sessions including electrode placement and psychological survey data collection. Data analysis, interpretation, and final thesis completed and presented.

Spring 2005-Spring 2006 — HONORS THESIS RESEARCH
"Mental Illness Stigma and the Law of Contagion"
Chair: Ed Hirt, Ph.D.
I participated in all stages of a study evaluating potential cognitive mechanisms by which mentally ill populations are stigmatized. I formulated the design of the study. I administered questionnaires, and coordinated data collection. Subjects included introductory psychology students. I trained and supervised three undergraduates assisting data entry. Data analysis, interpretation, and final thesis write-up completed. Poster presentation given at Honors Thesis Banquet.

RESEARCH EXPERIENCE (CONTINUED)

Spring 2005 ANTIEPILEPTIC DRUGS AND COGNITIVE IMPAIRMENT EXPERIMENT
Supervisor: Preston Garraghty, Ph.D.
I participated in animal behavior shaping and data collection of an experiment examining how the antiepileptic drug phenytoin affects cognitive abilities of rats. I learned how to operate Skinner boxes in an appetitive-to-aversive transfer task. I authored a funding proposal and was awarded Capstone research grant and laboratory stipend for the study.

Fall 2004 – Spring 2005 SOCIAL DEFEAT STRESS AND IMMUNE FUNCTION EXPERIMENT
Supervisor: Greg Demas, Ph.D.
I helped implement study examining how social defeat affects immune function in the Siberian hamster. I ran a social defeat paradigm, collected and centrifuged blood samples, did ELISA assays of samples to assess levels of stress hormone and antibodies present. I analyzed and interpreted data.
Poster presentation given.

CLINICAL EXPERIENCE

Internship 2012-2013 FAMILY MEDICINE CLINIC - MEMORIAL HOSPITAL
Supervisor: Scott Fields, Ph.D.
Dates: July 2012-September 2012

Worked in a Family Medicine Clinic housed in Memorial Hospital. Acted as a member of a multidisciplinary team, including a psychologist, primary care physicians, nurses, and pharmacists. Conducted intake interviews for patients referred or "handed-off" by their primary care physician to triage regarding patients' needed level of mental health care. When indicated, provided brief, time-limited individual therapy sessions. Monitored patient progress by administering symptoms inventories. Taught seminar to medical residents. Weekly individual supervision. Primary therapeutic modalities were Cognitive-Behavioral Therapy, supportive therapy, and psychoeducation.
Tests given: Mood Disorder Questionnaire (MDQ), Patient Health Questionnaire (PHQ-9)

CLINICAL EXPERIENCE (CONTINUED)

RURAL PRIMARY CARE HEALTH CLINICS – CABIN CREEK HEALTH SYSTEMS
Supervisors: Scott Fields, Ph.D. and Emily Selby-Nelson, Psy D
Dates: July 2012-September 2012

Worked in two rural primary care Health Clinics located in Clendenin, WV and Sissonville, WV. Acted as a member of a multidisciplinary team, including a psychologist, primary care physicians, and nurses. Conducted intake interviews for patients referred or "handed-off" by their primary care physician to triage regarding patients' needed level of mental health care. When indicated, provided brief, time-limited individual therapy sessions. Monitored patient progress by administering symptoms inventories. Weekly individual supervision. Primary therapeutic modalities were Cognitive-Behavioral Therapy (CBT), supportive therapy, and psychoeducation.
Tests given: Generalized Anxiety Disorder Questionnaire (GAD-7), Insomnia Severity Index (ISI),
Mood Disorder Questionnaire (MDQ), Patient Health Questionnaire (PHQ-9)

WEST VIRGINIA UNIVERSITY OUTPATIENT PSYCHIATRY CLINIC
Supervisor: John Linton, Ph.D.
Dates: July 2012-June 2013

Conducted initial intakes of adult patients presenting at an outpatient clinic. Conducted individual therapy sessions for patients with depression, anxiety, bipolar disorder, adjustment disorders, grief reactions, and relational problems. Monitored patient progress by administering symptoms inventories. Weekly individual supervision. Primary therapeutic modalities were Cognitive-Behavioral Therapy (CBT) and Acceptance and Commitment Therapy (ACT).
Tests given: Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI-II), Generalized Anxiety Disorder Questionnaire (GAD-7), Patient Health Questionnaire (PHQ-9), Valued Living Questionnaire

CLINICAL EXPERIENCE (CONTINUED)

DIALETICAL BEHAVIOR THERAPY PROGRAM – WEST VIRIGINIA UNIVERSITY DEPARTMENT OF BEHAVIROAL MEDICAL AND PSYCHIATRY
Supervisor: Patrick Kerr, Ph.D.
Dates: July 2012-December 2012

Co-led skills training groups for patients with pervasive emotional regulation problems, self-injury, suicidal behaviors, and/or diagnosis of borderline personality disorder. Developed mindfulness activities and distress tolerance module activities to correspond with Marsha Linehan's Cognitive Behavioral Treatment of Borderline Personality Disorder. Participated in weekly consultation team meetings with other psychologists and psychiatrists involved in DBT program. Weekly individual supervision.

CARDIAC REHABILITATION PROGRAM – CHARLESTON AREA MEDICAL CENTER
Supervisor: Melisa Chelf-Sirbu, Ph.D.
Dates: October 2012-December 2012
Worked as a team member in a multidisciplinary program for patients who have experienced cardiac events. Participated in initial consultation and assessment of cardiac patients. Co-taught psychoeducational classes on psychosocial factors associated with cardiac disease development and progression. Provided problem-focused, brief individual therapy. Provided brief screening assessments for mood and memory impairment. Trained in Dean Ornish program strategies for reversing cardiac disease. Observed a coronary artery bypass graft surgery procedure. Weekly individual supervision.
Tests given: Herridge Cardiopulmonary Questionnaire, Neurobehavioral Cognitive Status Examination (Cognistat/NCSE)

FAMILY RESOURCE CENTER – CHARLESTONE AREA MEDICAL CENTER
Supervisor: Susan Walker-Matthews, Ph.D.
Dates: October 2012-December 2012

Conducted initial intakes of adolescent patients presenting at an outpatient clinic. Conducted individual therapy sessions with patients with depression, adjustment disorders, and parent-child relational problems, and attention problems as well as their family members. Monitored patient progress by administering symptoms inventories. Administered tests to assess presence of attention deficit disorder. Weekly individual supervision. Primary therapeutic modalities were Cognitive-Behavioral Therapy (CBT).*Tests given*: Child's Depression Inventory (CDI), Connors Test, Patient Health Questionnaire for Adolescents (PHQ-9A), Test of Variables of Attention (TOVA)

CLINICAL EXPERIENCE (CONTINUED)

SLEEP CENTER CHARLESTON AREA MEDICAL CENTER
Supervisor: Elisa Drake, Ph.D. and George Zaldivar, M.D.
Dates: November 2012-December 2012

Participated in initial consultation and assessment of sleep disorders, such as insomnia and sleep apnea. Learned how to deliver evidence-based psychotherapy treatments for insomnia, including sleep restriction, stimulus control, relaxation, sleep hygiene, and cognitive behavioral therapy for insomnia (CBTi). Provided individual co-therapy sessions to patients with sleep disturbances. Observed overnight sleep studies assessing polysomnographic data and participated in feedback sessions.
Tests given: Epworth Sleepiness Scale, Sleep Diaries

Spring 2011 ROUDEBUSH VA MEDICAL CENTER, PRIMARY CARE CLINIC
Supervisor: Jennifer Lydon-Lam, Ph.D., HSPP
Dates: January 2011-August 2011

Worked as a member of a multidisciplinary team, including a clinical psychologist, primary care physicians, nurses, and pharmacists. Conducted intake interviews for veterans referred by their primary care physician. Conducted brief neuropsychological and cognitive testing as well as personality and symptom inventories with veterans. Provided individual therapy sessions to veterans on issues of chronic pain, PTSD, and mood/anxiety disorders. Monitored patient progress by administering symptoms inventories. Conducted phone screens for potential Chronic Pain group members. Led or co-facilitated 12-week MOVE groups for weight loss and 8-week Chronic Pain Management groups with veterans. Weekly individual supervision. Primary therapeutic modalities were Cognitive-behavioral therapy (CBT) and Acceptance and Commitment Therapy (ACT). I have written 15 integrated reports of psychological, neurological, and cognitive findings for referring physicians at this site.
Tests given: Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI-II), Neurobehavioral Cognitive Status Examination (Cognistat/NCSE), Geriatric Depression Scale (GDS), Minnesota Multiphasic Personality Inventory-Second Edition (MMPI-2), Montreal Cognitive Assessment (MOCA), Patient Health Questionnaire (PHQ-9), Post-traumatic Stress Disorder (PTSD) Check List (PCL-M), Shipley Institute of Living Scale, Wechsler Memory Scale -IV (WMS- IV)

CLINICAL EXPERIENCE (CONTINUED)

Summer 2010 INDIANA UNIVERSITY NEUROPSYCHOLOGY CLINIC
Practicum Supervisor: Daniel Rexroth, Psy.D.
Dates: May 2010-November 2010

Administered, scored, and interpreted tests for neuropsychological evaluations of patients presenting with cognitive impairments associated with the following disorders: depression and anxiety, Parkinson's disease, essential tremor, multiple sclerosis, Asperger's disorder, general mild cognitive impairment, and dysexecutive syndromes. Gained testing experience assessing the following cognitive domains: orientation, intelligence, processing speed, executive functioning, verbal memory, language, spatial memory, motor skills. Observed interview and feedback process for patients and family members. Attended Neurology Grand Rounds by Indiana University School of Medicine. Weekly individual supervision. I have written 6 integrative reports for this placement. *Tests given*: Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI), Boston Naming Task, Controlled Oral Word Association Test (Fluency), Finger Tapping/Grip/Grooved Pegboard, Geriatric Depression Scale (GDS), Mini Mental Status Exam (MMSE), MMPI, Rey Auditory Verbal Learning Test (RAVLT), Wechsler Stroop Task, Test of Memory Malingering (TOMM), Trails A & B, Test of Adult Reading (WTAR), Wechsler Adult Intelligence Scale (WAIS), Wechsler Memory Scale -Revised (WMS-R), Wide Range Achievement Test (WRAT), Wisconsin Card Sorting Task

Spring 2010 INDIANA UNIVERSITY OUTPATIENT PSYCHIATRY CLINIC
Practicum Supervisor: Jeffrey Lightfoot, Ph.D.
Dates: January 2010-June 2010

Conducted initial intakes of patients presenting at an outpatient clinic.
Conducted individual therapy sessions for patients with depression, anxiety, panic disorder, social phobia, and dependent personality disorder. Primary therapeutic modalities were cognitive-behavioral therapy (CBT).
Modified level of CBT techniques to be appropriate to cognitive level of patient, as I had a patient with mild mental retardation. Monitored patient progress by administering symptoms inventories.
Weekly group supervision with medical residents and other psychology graduate students.
Tests given: Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI-II), Center for Epidemiologic Studies –Depression (CES-D)

CLINICAL EXPERIENCE (CONTINUED)

Spring & Fall Practicum

INDIANA UNIVERSITY HEALTH BARIATRIC CENTER
Supervisor: William Hilgendorf, Ph.D., HSPP
Dates: May 2009-December 2010

Worked as a member of a multidisciplinary clinic which included a clinical psychologist, bariatric surgeons, nurses, and dieticians. Conducted biopsychosocial assessments for individuals seeking bariatric surgery to determine their readiness for surgery. I also researched, created materials for, and led a 4-week group designed to help patients develop pre-surgical coping skills, including Modifying Eating Habits, Motivation for Physical Activity, Interacting with Friends and Family about Your Surgery, and Body Image Concerns. Led or co-led Coping Group for post-surgical patients. Provided individual therapy with clients to improve their readiness for surgery. Primary therapeutic modalities were cognitive-behavioral therapy and motivational interviewing. Was invited to give pre-surgical group lecture about Body Image to patients. Published blog entries on Clarian website about psychosocial factors in bariatric surgery. I have written 17 integrative reports for this placement.
Tests given: Beck Depression Inventory (BDI-II), Generalized Anxiety Disorder-7 (GAD-7), Impact of Weight on Quality of Life Questionnaire-Lite (IWQOL-Lite), Millon Behavioral Medicine Diagnostic (MBMD), Patient Health Questionnaire (PHQ-9), World Health Organization Quality of Life-Bref (WHOQOL-BREF)

Fall 2008 Practicum

LARUE CARTER INPATIENT PSYCHIATRIC HOSPITAL
Supervisors: Tim Lines, Ph.D. and Natalie Blevins-Datillo, Ph.D.
Dates: August 2009-January 2010

Conducted assessments, group therapy, and individual therapy with individuals suffering from severe mental illnesses including schizophrenia, bipolar disorder, dementia, and mental retardation. Led or co-led Assertive Communication Group. Provided individual therapy for individuals deemed incompetent to stand trial (ICST). Provided individual therapy to individual on specialized unit for Borderline Personality Disorder. Primary therapeutic modalities were cognitive-behavioral therapy and supportive therapy. Attended treatment team meetings with clinical psychologists, psychiatrists, nurses, social workers.
Tests given: MMSE, WAIS, MMPI

CLINICAL TRAINING WORKSHOPS AND SPECIALITY TRAINING

Fall 2011	HELPING THE SUICIDAL AND SURVIVIORS OF SUICIDE LOSS Workshop Leader: Thomas Joiner, Ph.D. Professor, Department of Psychology Florida State University
Fall 2011	CONSULTATION: THE COMPASS MODEL WORKSHOP Workshop Leader: Lisa Ruble, Ph.D. Associate Professor, Educational, School, and Counseling Psychology University of Kentucky
Spring 2011	SCHEMA THERAPY FOR BORDERLINE PESRONALITY DISORDER WORKSHOP Workshop Leader: Joan Farrell, Ph.D. Training Director of the Center for BPD Treatment & Research, Department of Psychiatry Indiana University School of Medicine
Spring 2010	EVIDENCE-BASED PRACTICE: WHAT PSYCHOLOGISTS NEED TO KNOW AND WHY WORKSHOP Workshop Leader: Barbara Walker, Ph.D. Professor, Department of Psychological and Brain Sciences Indiana University Bloomington
Spring 2010- Present	PROSEMINAR IN CLINICAL PSYCHOLOGY Department of Psychology, IUPUI Professional development course covering advanced clinical training topics. Relevant clinical training topics: "Depression and Diabetes: What We Know and What We Need to Know" Presenter: Mary de Groot, Ph.D. Associate Professor, Division of Endocrinology and Metabolism Indiana University School of Medicine
Spring 2009	ACCEPTANCE AND COMMITMENT THERAPY TRAINING WORKSHOP Workshop Leader: Rhonda M. Merwin, Ph.D. Assistant Professor, Department of Psychiatry and Behavioral Sciences Duke University Medical Center
Spring 2008	MOTIVATIONAL INTERVIEWING WORKSHOP Workshop Leader: John Wryobeck, Ph.D. Assistant Professor, Department of Psychiatry University of Toledo, School of Medicine

TEACHING EXPERIENCE

Fall 2010-Spring 2012 TEACHING FELLOW (NATIONAL SCIENCE FOUNDATION GK-12 PROGRAM)
Helped bring scientific principles of health psychology to high school curriculum. Developed independent lesson plans to teach students about principles of health psychology. Facilitated class discussions. Developed performance assessments and provided students with feedback.

Fall 2009-Spring 2010 INSTRUCTOR (300-LEVEL ABNORMAL PSYCHOLOGY)
Instructed class of approximately 75 upper level undergraduates. Independently developed course structure and presented class sessions via Powerpoint. Facilitated class discussions and incorporated active learning activities. Independently developed performance assessments including homework, exams, and a written project.

Fall 2009-Spring 2010 INSTRUCTOR (100-LEVEL INTRODUCTORY PSYCHOLOGY)
Instructed class of approximately 50 undergraduates. Followed a pre-developed classroom pedagogy which emphasized collaborative and active learning activities. Implemented and/or developed active learning activities and facilitated class room discussion.

Spring 2009 GRADUATE TEACHING ASSISTANT (NANCY BADIA-ELDER, PH.D)
Served as teaching assistant for Introductory Psychology as a Biological Science. Class size was approximately 65 students. Tutored students, prepared grades. Led weekly laboratory sessions and review sessions.

Fall 2007-Spring 2008 GRADUATE TEACHING ASSISTANT (JESSE STEWART, PH.D)
Served as teaching assistant for Clinical Research class of approximately 30 students. Tutored students, prepared grades. Developed and presented SPSS training sessions via Powerpoint.

Fall 2007 GRADUATE TEACHING ASSISTANT (CHARLES GOODLETT, PH.D)
Served as teaching assistant for Introductory Psychology as a Biological Science. Class size was approximately 65 students. Tutored students, prepared grades. Led weekly laboratory sessions and review sessions.

AVAILABLE STUDENT SATISFACTION RATINGS

Each semester, IUPUI students complete standardized evaluation forms. For most courses, the form yields three subscales (exams, student motivation, and rapport with students) and a global score.

Response scale is a 5-point Likert-type scale (5 = strongly agree, 4 = agree, 3 = undecided, 2 = disagree, 1 = strongly disagree), with higher score indicating more positive ratings.

In the following table, scores are presented for courses in which I was the primary instructor.

Term	Course	Course Title	N	Global Score Mean	Exams	Motivation	Rapport
Fall 2009	B104	Introductory Psychology	32	4.45	N/A*	N/A*	N/A*
Fall 2009	B380	Abnormal Psychology	29	4.66	4.79	4.52	4.79
Spring 2010	B104	Introductory Psychology	28	4.59	N/A*	N/A*	N/A*
Spring 2010	B380	Abnormal Psychology	21	4.77	4.55	4.56	4.75

Note. 5 = strongly agree, 4 = agree, 3 = undecided, 2 = disagree, 1 = strongly disagree

*N/A = Scores not available because course uses evaluation form for which these specific subscales are not created.

COLLOQUIA AND INVITED LECTURES

Hawkins, M.A.W. (2012, December). *Serving U.S. military personnel: A guide for mental health providers.* A two-hour seminar for the West Virginia University Charleston Division Department of Behavior Medicine and Psychiatry faculty, residents and medical students, Charleston, WV.

Hawkins, M.A.W. (2012, September). *Helping patients get the message: The physician as communicator.* A one-hour seminar for the Charleston Area Medical Center Department of Family Medicine medical residents and medical students, Charleston, WV.

Hawkins, M.A.W. (2011, April). *Finding and using empirically supported treatments for your clients: A case example.* A one-hour case presentation at the ProSeminar for the Psychology Department graduate students and faculty, Indianapolis, IN.

COLLOQUIA AND INVITED LECTURES (CONTINUED)

Hawkins, M.A. (2010, September). *The influence of cognitive emotion regulation strategies on cardiovascular recovery from stress.* A one-hour research presentation at the ProSeminar for the Psychology Department graduate students and faculty, Indianapolis, IN.

Hawkins, M.A. (2009, April). *Body image before and after bariatric surgery.* A one-hour presentation for the pre- operative bariatric patients of Clarian Bariatric Center, Indianapolis, IN.

UNIVERSITY AND PROFESSIONAL SERVICE

2011	Programming Committee Member for Division 38 (Health Psychology),
2011	American Psychological Association Convention, Washington, D.C.
2011	Abstract Reviewer for the 2011 APA Division 38 Conference, Washington, D.C.
2010	Abstract Reviewer for 2011 Annual Meeting for American Psychosomatic Society, Portland, OR.
2007	Psi Chi National Honor Society, President of the Indiana University Chapter, Bloomington, IN.

PROFESSIONAL MEMBERSHIPS

2011- Present Obesity Society, Student Member
2010- Present American Psychosomatic Society (APS), Student Member
2010- Present American Psychological Association (APA), Student Member
2010- Present Indiana Psychological Association (IPA), Student Member