RUNNING HEAD: Obesity and mental disorders

Longitudinal Associations of Obesity with Mental Disorders and Suicidality in the Baltimore Epidemiologic Catchment Area Follow-Up Study

by

Amber Ann Mather

A thesis submitted to the Faculty of Graduate Studies of

The University of Manitoba

in partial fulfillment of the requirements for the degree of

MASTER OF ARTS

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THE UNIVERSITY OF MANITOBA

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Abstract

Obesity is on the rise worldwide, and researchers and clinicians are becoming aware of the serious physical and mental health consequences this trend carries. The objective of this study was to provide a comprehensive examination of the longitudinal associations between obesity and mental health variables (psychiatric diagnoses and suicidal behaviors). Data were from waves 3 and 4 of the Baltimore Epidemiologic Catchment Area study (ECA; N=1071). Earlier obesity predicted suicide attempts and panic disorder, and was protective against social phobia among women. Earlier depression predicted weight gain over the eleven-year follow-up period, as did suicidal ideation for women. The most important finding was of few significant predictive relationships between obesity and mental health variables. Because obesity and mental health were largely unrelated, clinicians and others should be wary of "weight-ism," and avoid making the assumption that higher body weight relates to mental health problems, particularly among middle-aged and older adults.

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Introduction

Overview

With obesity becoming the global "epidemic" that it is, research into the physical and mental health consequences of this condition is becoming increasingly important. Obesity has been clearly linked to increased risks of a number of physical health issues (World Health Organization, 2000), and research on the mental health correlates of obesity is indicating a similar trend. Cross-sectional studies have established associations between obesity and a number of mental disorders, though sometimes with conflicting results (e.g., Barry, Pietrzak, & Petry, 2008; Mather, Cox, Enns, & Sareen, 2009; Pickering, Grant, Chou, & Compton, 2007; Simon et al., 2006). Equivocal findings from cross-sectional research warrant longitudinal research to establish whether obesity functions as a causal agent in the development of these disorders. Some work of this sort has been conducted (e.g., Hasler et al., 2004; Herva et al., 2006; Taylor, Macdonald, McKinnon, Joffe, & MacQueen, 2008), but what is clearly needed, and what has yet to be provided, is a comprehensive examination of the relationship between obesity and a range of psychiatric diagnoses. This study will aim to fill this gap in the literature by determining whether earlier obesity is a risk factor for various later mental disorder diagnoses, and vice versa.

Trends in and Causes of Obesity

Obesity rates are on the rise world-wide (World Health Organization, 2000). Generally, obesity is defined as a body mass index (BMI; weight in kilograms divided by the square of height in metres) greater than or equal to 30 (Centers for Disease Control and Prevention, 2006; World Health Organization, 2000). With the healthy range for BMI considered to be between 18.5 and 24.9, BMI scores of 30 and above represent an unambiguous increase in body weight

per unit height. Currently the United States has one of the highest prevalences of obesity in the world, with nearly one third of the population meeting criteria (Ogden et al., 2006). Though not as high as in the U.S., other developed countries also report distressingly high rates of obesity. Canada, Europe, Scandinavia, and Australia generally estimate the prevalence of obesity among their general populations to be higher than 15% (Brown & Siahpush, 2007; Due, Heitmann, & Sorensen, 2007; Katzmarzyk & Mason, 2006; Lobstein, Millstone, & PorGrow Research Team, 2007; Neovius, Janson, & Rössner, 2006; Rennie & Jebb, 2005; Schokker, Visscher, Nooyens, van Baak, & Seidell, 2007). Obesity rates have historically been much lower, on the order of 5%, and the rapid rise over the past twenty-five years has led the World Health Organization to label obesity an epidemic (World Health Organization, 2000). Though once thought to be confined to the Western world, obesity has spread into other cultures, with prevalences also high and rising in many Asian, African, Middle Eastern, and Latin American countries (Asia Pacific Cohort Studies Collaboration, 2007; Filozof, Gonzalez, Sereday, Mazza, & Braguinsky, 2001; Galal, 2002; Obikili & Nwoye, 2006; Pan American Health Organization, 1994; Prentice, 2006; Rguibi & Belahsen, 2007; van der Merwe & Pepper, 2006). The World Health Organization estimates that approximately 400 million adults are obese worldwide and that, if current trends hold, this number will grow to 700 million by 2015 (World Health Organization, 2006).

The increasing worldwide prevalence of obesity is thought to be due in part to a "nutrition transition." In the first stages of a nutrition transition, there is a shift in the availability of food and patterns of consumption in a society such that famine and food scarcity are replaced by overabundance of food and increased intake of refined foods high in saturated fat and sugar (Popkin & Gordon-Larsen, 2004). Typically the result is a shift towards a decreased prevalence of underweight individuals and an increased prevalence of overweight and obese individuals that

affects the population in stages, starting with the wealthiest citizens and moving through progressively less economically advantaged strata until the entire population has shifted (World Health Organization, 2000). However, this pattern has disintegrated in recent years, as high-fat diets have become more affordable than in the past, so that all economic groups are facing increased overweight and obesity in developing countries (Canoy & Buchan, 2007). Accompanying or preceding this part of the transition is a demographic transition (a decrease in both mortality and fertility in a society) and an epidemiologic transition (a shift from predominating infectious diseases to predominating chronic and degenerative diseases that stems from improved nutrition and sanitation) that are a result of increasing industrialization and the lifestyle changes that necessarily follow from it (Popkin et al., 2004). In the final stage of the nutrition transition, the unhealthy patterns established in the beginning of the transition are broken via behavioral changes such that the population experiences improved health and further decreased mortality (Popkin et al., 2004). As indicated by the alarmingly high rates of obesity, the global population is reaching a point where this final stage must be actively sought, as much recent research, which I review below, has demonstrated that the consequences of population-wide excess consumption are very serious and wide-reaching.

Three general factors—societal, social and behavioral, and biological—contribute to obesity at different levels. At the population level, societal factors play a significant role in determining the body weights of a given population. The aforementioned global nutrition transition is one such factor. Other societal factors include relatively recent changes in population diet and physical activity. General dietary changes, such as an increased proportion of energy intake coming from fats, higher rates of consumption of caloric sweeteners (e.g., sugar, high-fructose corn syrup), increased fast food intake, and growing portion sizes, all contribute to

the high proportion of overweight and obese individuals (Bowman & Vinyard, 2004; Bray, Paeratakul, & Popkin, 2004; Drewnowski, 2007; Hill & Peters, 1998; Malik, Schulze, & Hu, 2006; Popkin, 2006; Prentice & Jebb, 1995; World Health Organization, 2000). This is particularly true for Western cultures, which explains the use of the phrase "Western diet" to describe eating habits consisting largely of high fat, high sugar, heavily processed foods (Bray et al., 2004; Canoy et al., 2007; Popkin et al., 2004). In addition, modernization has led to a decreased role for physical activity in the daily lives of most individuals in developed countries, which has also contributed to the current obesity problem (Lanningham-Foster, Nysse, & Levine, 2003; Prentice et al., 1995). Factors such as sedentary work environments, mechanized labour, motorized transport, and television have all contributed to decreased physical activity and therefore fewer calories being expended per day than in the past (Hill, Wyatt, Reed, & Peters, 2003; Lanningham-Foster et al., 2003). The result of this increased energy consumption and decreased energy expenditure for the vast majority of the population has been the net storage, or conversion to fat, of approximately 100 calories per day, which has been enough to fuel the rapid rise in the population rates of overweight and obesity that is currently being observed (Hill et al., 2003).

At the individual level, social and behavioral factors also contribute to obesity. Lifestyle choices, such as lack of leisure-time physical activity or excessive alcohol intake, are one example of this (Laurier, Guiguet, Chau, Wells, & Valleron, 1992; Risérus & Ingelsson, 2007; World Health Organization, 2000). Several social variables, such as lower adult and family-of-origin socioeconomic status, low education, urbanicity, and being from a single parent family have all been shown to be associated with increased rates of adult obesity in developed countries (Canoy et al., 2007; Laitinen, Power, & Järvelin, 2001; Laurier et al., 1992; Ogden, Yanovski,

Carroll, & Flegal, 2007; Olson, Bove, & Miller, 2007; Parsons, Power, Logan, & Summerbell, 1999; Shields & Tjepkema, 2006; Zhang & Wang, 2004). Experiencing abuse as a child also correlates with adult obesity, possibly because this abuse results in high long-term levels of psychological distress that lead to maladaptive eating habits (Alvarez, Pavao, Baumrind, & Kimerling, 2007; Williamson, Thompson, Anda, Dietz, & Felitti, 2002). Eating patterns show a high degree of family resemblance, indicating that parents may impart unhealthy eating behaviors on their children, which may contribute to obesity in the next generation (Provencher et al., 2005). Interestingly, recent work has determined obesity to be "socially contagious," with the onset of obesity within members of one's close social network significantly increasing an individual's own probability of becoming obese (Christakis & Fowler, 2007).

The biological aspect of obesity development and maintenance is also important to consider. Though experts agree their role is likely negligible in the current obesity epidemic (e.g., Hill et al., 2003; World Health Organization, 2000), certain physiological, genetic, and other biological factors may be significant at the individual level for the development and maintenance of an unhealthy body weight. For instance, many candidate genes have been identified that may be specifically involved in the development of obesity (Ahituv et al., 2007). In addition, the hormone leptin may play a role in obesity development. This hormone is produced by adipose tissue and regulates long-term food intake; resistance to leptin as a result of environmental factors (e.g., increased long-term food intake or dietary fat content), genetic mutations, or their interaction may be involved in the onset and maintenance of obesity for some individuals (Friedman & Halaas, 1998). Beyond these issues, several other biological factors influence the susceptibility to obesity. Obesity exhibits a curvilinear relationship with age, with obesity rates increasing until approximately 80 years of age and dropping dramatically thereafter

(Ogden et al., 2007). Also, female sex predisposes individuals to obesity, likely due to physiological processes that lead to increased fat storage compared to males (Clarke, O'Malley, Johnston, & Sculenberg, 2008; Ogden et al., 2006; World Health Organization, 2000). Additionally, ethnicity is linked to obesity, with certain ethnic groups, such as Mexican Americans, African Americans, and Aboriginal Canadians, having higher obesity rates than other groups, such as Caucasians, living in the same environment (Flegal, Carroll, Ogden, & Johnson, 2002; Garriguet, 2008; Ogden et al., 2006). This difference could be the result of genetic selection for obesity or thinness in these populations because of the historical patterns of food availability in their geographic regions of origin (Diamond, 1992). Finally, certain diseases and disorders, or the treatments for certain conditions, may make individuals prone to obesity. For example, hypothyroidism, hypothalamic tumours, corticosteroid therapy for rheumatoid arthritis, and antipsychotic medication for psychotic disorders may all lead to weight gain for some individuals and thus increase obesity rates (Kiraly, Gunning, & Leiser, 2008; Lieberman et al., 2005; World Health Organization, 2000).

Effects of Obesity

Just as obesity has many causes, it has many consequences for both body and mind. Obesity increases the risk of many physical health conditions, such as hypertension, type II (insulin-resistant) diabetes, dislipidemia (low high-density lipoprotein and high triglyceride levels), cardiovascular disease (e.g., heart attacks, stroke, congestive heart failure), osteoarthritis, and certain cancers (Ogden et al., 2007; World Health Organization, 2000). A recent study examined the proportion of the prevalence of several common physical health problems that can be attributed to obesity (the population attributable fraction). The authors estimated that currently 10% of colon cancer, 12% of post-menopausal cancer, 22% of endometrial cancer, 11% of

stroke, 19% of osteoarthritis, 23% of coronary artery disease, 35% of gallbladder disease, 39% of type II diabetes, and 45% of hypertension is attributable to obesity, which is an increase of 138% for men and 60% for women since 1970 (Luo et al., 2007). Obesity also increases the risk of mortality, both in general and as a result of many of the diseases mentioned above. Over 8000 Canadians die prematurely every year as a result of being overweight or obese (Luo et al., 2007); in the U.S. this number is an astonishing 300 000 (Allison, Fontaine, Manson, Stevens, & VanItallie, 1999). Surprisingly, even these high numbers may be underestimates of the true obesity-attributable mortality and morbidity in the population because of limitations of the BMI method of classifying obesity (Canoy et al., 2007; Greenberg, 2006). A series of studies have confirmed that obese people are at greater risk of death, both as a result of serious diseases and in general, and this risk is amplified for younger age groups (Calle, Thun, Petrelli, Rodriguez, & Heath, 1999; Calle, Rodriguez, Walker-Thurmond, & Thun, 2003; Stevens et al., 1998).

Impairments in quality of life are a concern for obese individuals. Much research has shown that both physical and mental health-related quality of life are negatively affected in overweight individuals (Fabricatore, Wadden, Sarwer, & Faith, 2005; Ford, Moriarty, Zack, Mokdad, & Chapman, 2001; Hassan, Joshi, Madhavan, & Amonkar, 2003; van Hout, van Oudheusden, & van Heck, 2004), and that obese people are more likely to report impairments in daily functioning, both in general (Lean, Han, & Seidell, 1999) and as a result of physical or mental health problems (Ford et al., 2001; Hassan et al., 2003). Longitudinal research has confirmed that being or becoming obese predicts impairments in daily functioning 10 and 20 years later (Ferraro, Su, Gretebeck, Black, & Badylak, 2002). Even more disconcerting was that these consequences of obesity do not appear to be significantly alleviated by weight loss (Ferraro et al., 2002). Importantly, sexual quality of life is decreased in obese individuals, as evidenced by

lack of sexual enjoyment and desire, avoidance of sexual encounters, and difficulty in sexual performance (Kolotkin et al., 2006). Well-being, a construct similar but not identical to quality of life, is similarly impaired in people with obesity. Obese individuals have lower physical, emotional, and social well-being (Doll, Petersen, & Stewart-Brown, 2000; Foreyt et al., 1995; Jorm et al., 2003), particularly when their obesity is coupled with one or more chronic physical health conditions (Doll et al., 2000).

Mental Health and Obesity

In addition to the physical health problems that often accompany it, recent research is demonstrating that mental health comorbidities are commonly associated with obesity as well. The most researched of the mental health correlates of obesity is depression, and a large body of literature demonstrates the cross-sectional link between obesity and both depressive symptoms (Cilli et al., 2003; Dixon, Dixon, & O'Brien, 2003; Heo, Pietrobelli, Fontaine, Sirey, & Faith, 2006; Johnston, Johnson, McLeod, & Johnston, 2004) and a depression diagnosis (Baumeister & Harter, 2007b; Carpenter, Hasin, Allison, & Faith, 2000; Dong, Sanchez, & Price, 2004; Mather et al., 2009; Onyike, Crum, Lee, Lyketsos, & Eaton, 2003; Palinkas, Wingard, & Barrett-Connor, 1996; Simon et al., 2006). There is some contradiction of these findings in the literature, as some research (e.g., John, Meyer, Rumpf, & Hapke, 2005) has found no such associations; however, the general consensus in the literature is that obesity and depression are indeed linked.

Longitudinal research has similarly supported a link between obesity and depression, generally suggesting that they are causally related to each other. Prospective studies strongly support a relationship where earlier obesity predicts later depression (Herva et al., 2006; Kasen, Cohen, Chen, & Must, 2008; Roberts, Strawbridge, Deleger, & Kaplan, 2002; Roberts, Deleger, Strawbridge, & Kaplan, 2003). Longitudinal designs following individuals through weight loss

also support this contention, as decreasing adiposity seems to be accompanied by diminished depressive symptomatology (Dixon et al., 2003; Nickel, Loew, & Bachler, 2007; Schowalter et al., 2008). Research into the opposite relationship, where earlier depression is examined as a predictor of later obesity, has generally reported positive findings as well. For example, Taylor et al. (2008) have reported that a significant fraction of incident obesity over a four-year period was attributable to depression in a group of never-treated patients with depression. Similarly, Richardson et al. (2003) report that earlier depression is significantly associated with later obesity in females. However, some other longitudinal work has found no such relationship (Hasler et al., 2004; e.g., Roberts et al., 2003). A recent meta-review (Blaine, 2008) supports the contention that earlier depression is a predictor of later obesity, particularly among adolescents. The author notes, however, that antidepressant medication use may account for part of this finding (i.e., antidepressants may cause weight gain themselves), as this variable is rarely taken into account when examining the longitudinal relationship between earlier depression and later obesity.

Disorders other than major depression have also begun to receive attention with respect to the comorbidity between obesity and mental health. For example, anxiety disorders are more recent topics of interest in the obesity literature. Overall, those with any anxiety disorder or anxiety symptoms have higher odds of being obese (Baumeister et al., 2007b; Jorm et al., 2003; Mather et al., 2009; Scott et al., 2007; Scott, McGee, Wells, & Oakley Browne, 2008). When analyses are stratified by gender, conflicting evidence is again seen concerning the associations in men, with some samples showing significant associations and others not. When examining individual anxiety disorders, a number of conditions show positive associations to obesity. Disorders that are consistently associated with obesity include panic disorder (Barry et al., 2008;

Mather et al., 2009; Scott et al., 2008; Simon et al., 2006) and specific phobia (Barry et al., 2008; Pickering et al., 2007; Scott et al., 2008), though some of these associations were specific to women in some studies. Agoraphobia seems to be unrelated to obesity when gender is unstratified (Mather et al., 2009; Scott et al., 2008), though when examining women separately, this relationship does attain statistical significance in some samples (Mather et al., 2009) but not others (Barry et al., 2008). Social phobia is another disorder that is significantly related to obesity only for women (Barry et al., 2008; Mather et al., 2009), but not in an overall, non-gender-stratified sample (Scott et al., 2008). The relationship of generalized anxiety disorder to obesity is unclear, with some research indicating significant associations in both genders (Barry et al., 2008), and other work finding no significant relationship for either gender (Pickering et al., 2007; Scott et al., 2008; Simon et al., 2006). Overall, though there are some general trends regarding the link between obesity and anxiety disorders, such as that women seem much more likely than men to evidence significant relationships across a range of anxiety disorders, there are still many areas where the findings are unclear.

Longitudinal research on anxiety-obesity comorbidity is extremely limited. Only two studies have addressed this issue using adult samples. Hasler et al. (2004) found that none of the anxiety disorders in their study (i.e., panic disorder, generalized anxiety disorder, or an "any phobic disorders" variable) were associated with weight gain over a 20-year follow-up period. Contradictory findings were reported by another group who examined the prospective relationship between body weight and generalized anxiety disorder (Kasen et al., 2008). These researchers uncovered significant associations among a community sample of women between being either overweight or obese at age 27 and meeting criteria for generalized anxiety disorder at age 59. Similarly, Nickel et al. (2007) conducted a prospective study of individuals

undergoing gastric banding and reported significant improvement in anxiety symptoms for these individuals six years after the surgery. However, further longitudinal work is still needed to help determine whether and how obesity and anxiety are longitudinally related.

Suicidality is arguably the least-researched of all the potential mental health correlates of obesity. Extant work suggests that obesity increases the risk of suicidal ideation and suicide attempts, both in the past year and over the lifespan (Dong, Li, Li, & Price, 2006; Mather et al., 2009). Carpenter et al. (2000) utilized a linear BMI variable and found a pattern of increasing suicidality with increasing body weight, which is in line with the results of the aforementioned research using a categorical obesity measure. Conversely, one study examining body weight on a continuous scale found that higher body mass index seems to be related to decreased suicide risk over a 16 year period in male healthcare professionals (Mukamal, Kawachi, Miller, & Rimm, 2007). However, this highly specialized sample does not generalize to women and is not representative of men in general. Further work is clearly necessary to examine the longitudinal relationship between obesity and suicidal behaviors.

The Role of Physical Health in Obesity-Mental Health Relationships

The issue of comorbidity of physical health conditions in the research on obesity and mental disorders is becoming increasingly prominent. Recent work by Pickering and colleagues (2007) used a large, nationally representative U.S. survey and found that obesity was positively related only to bipolar disorder and specific phobia in women, after adjusting for a myriad of physical health conditions. This study is the most recent in a growing body of research (Hach et al., 2007; Jorm et al., 2003) corroborating the hypothesis that the observed comorbidity between obesity and mental health may be due, at least in part, to the known high rate of comorbidity between physical and mental health problems (Fabricatore et al., 2005; Ross, 1994). However,

despite partial mediation of the obesity-mental health relationship by physical health conditions, the lack of full mediation suggests that some of this association is unexplained by a physical health mediation model. In fact, my own research (Mather et al., 2009) has demonstrated significant associations between obesity, various lifetime and past-year mental disorders (positive associations with depression, mania, panic attacks, social phobia, and agoraphobia; negative with drug dependence), and suicidal behaviors, independent of physical health conditions. As this research was conducted using a large, nationally representative Canadian sample, it provides reliable and generalizable evidence for a relationship between mental health and obesity that is not mediated by physical health.

Issues in the Literature

Several gaps exist in the current knowledge concerning the relationship between obesity and mental disorders, which my study will attempt to address. First, although some longitudinal research exists examining a potential causal relationship between obesity and various mental health issues (i.e., Dixon et al., 2003; Hasler et al., 2004; Herva et al., 2006; Kasen et al., 2008; Mukamal et al., 2007; Nickel et al., 2007; Richardson et al., 2003; Roberts et al., 2002; Roberts et al., 2003; Schowalter et al., 2008; Taylor et al., 2008), very little such research has provided a comprehensive assessment of the relationship of multiple psychiatric disorders to obesity. Focusing on the relationship between a single mental health outcome and obesity is problematic because of the high degree of comorbidity between mental disorders (Baumeister & Harter, 2007a). Without examining multiple mental disorders in a single study, it is impossible to adjust for the effect of other disorders on the association between obesity and a given psychiatric diagnosis, which may impact the reliability of results. Second, there is a lack of longitudinal work examining the link between obesity and certain aspects of mental health, such as anxiety

disorders and suicidality. The current body of research has focused primarily on the link between obesity and depression, while leaving the relationships of obesity to these other mental health variables relatively unexplored. This issue becomes particularly important in light of the equivocal findings in the cross-sectional literature. Because both obesity and mental health problems are common in the general population, there are likely to be at least some significant results in cross-sectional work. However, only a longitudinal design has the power to determine the causal relationship between these variables. Third, the extant longitudinal work has consistently failed to adjust for physical health conditions despite suggestions from cross-sectional work that obesity, physical health, and mental health are interrelated. Finally, that longitudinal studies have produced inconsistent results with regard to certain mental disorders indicates that more work is needed to understand the true nature of the associations between body weight and these disorders. These inconsistencies may be due to an inability to control for psychiatric comorbidity or a lack of adjustment for physical health, and this study will endeavour to clarify these longitudinal relationships by taking these variables into account.

The Proposed Study

My proposed study aims to extend the literature on the potential relationship between obesity and mental health by comprehensively examining the bidirectional longitudinal associations between obesity and psychiatric disorders. By including traditionally under-researched mental health outcome variables in my analysis, I will expand current knowledge of how obesity and mental health are related. In addition, examining a wide range of mental health diagnoses and outcomes will allow me to adjust for psychiatric comorbidity, and including physical health in my analyses will enable me to determine whether or not the obesity-mental health relationship is independent of other somatic conditions. Both of these steps will help me to

assess the potential causal link between mental health and obesity more definitively than previous research has been able to. The five questions my proposed study will address are: 1) Does earlier obesity predict later mental disorder diagnoses? 2) Does earlier mental health predict later body weight change? 3) Do comorbid psychiatric disorders weaken these predictive relationships? 4) Do the associations between body weight and mental health remain when physical health is accounted for? 5) Are there any gender differences in the patterns of association? Due to the lack of relevant longitudinal research, it is difficult to speculate as to which particular disorders may be predicted by obesity and vice versa. However, a reasonable hypothesis for the predictive relationship of obesity with later mental health would be that results will be in line with the findings from cross-sectional research: obesity will likely be positively associated with panic disorder, GAD, social phobia, depression, and suicidality. Diagnoses that may or may not show associations with obesity include specific phobia, agoraphobia, and obsessive-compulsive disorder. For the opposite relationship, where mental health predicts change in body weight, I expect that there will be fewer significant results, in line with findings from the extremely limited extant longitudinal research. Depression will likely be positively related to weight gain, but anxiety disorders and suicidal behaviours may or may not show a relationship to weight change. In addition, for both sets of analyses (obesity predicting mental health, and mental health predicting weight change), I expect most of the relationships to remain significant after adjusting for other psychiatric disorders and physical health conditions, and I expect notable gender differences to emerge, with women showing significant associations more often than men.

Methods

Sample

Data were from the third and fourth waves of the Baltimore Epidemiologic Catchment Area (ECA) survey, collected by trained lay interviewers in 1993-1996 and 2004-2005, respectively. Wave 1 was collected in 1981 and probabilistically sampled 4238 individuals from the population of residents of the East Baltimore area. Of the selected individuals, 3481 completed the wave 1 interview. Wave 2, conducted one year later, collected data from 2768 original respondents. From 1993-1996, wave 3 data were collected (with most of the interviews taking place in 1993), with 1920 original respondents again participating (this time period will henceforth be identified as "wave 3" or "1993"). From 2004-2005, the most recent wave (wave 4) was conducted, with 1071 respondents participating (this time period will henceforth be identified as "wave 4" or "2004"). In this survey, attrition was cumulative, which means that a respondent who did not respond to wave 3, for example, would not have been recontacted for wave 4 data collection. In other words, all 1071 respondents included in wave 4 were also interviewed and have data available from waves 3 and earlier.

Eaton et al. (2007) describe the pattern of attrition from the original data collection in 1981 to the wave 4 data collection in 2004. Between 1981 and 2004, 1450 respondents had died, representing 42% of the original sample. An additional 524 could not be located at the time of data collection, accounting for 26% of surviving respondents (15% of original sample). Of the 1507 alive and located, 29% (436) refused the interview, leaving the wave 4 sample at 1071 respondents, or as Eaton et al. (2007) note, 53% of survivors from the original (1981) survey.

As a result of the extended period of time between the beginning of the longitudinal survey and wave 4 data collection, much of the sample in waves 3 and 4 consists of middle-aged

and older adults. Respondents at wave 1 were required to be at least 18 years of age, which theoretically makes the youngest possible wave 4 respondent approximately 41 years old. The advancing age of the sample as a whole can certainly be considered a factor contributing to the high rate of attrition, as a large number of original respondents had died and were therefore, obviously, not included in the wave 4 sample. The Baltimore site of the original ECA also oversampled older adults, additionally increasing the odds that original respondents would not survive to subsequent interview cycles. Another contributing factor to the high attrition rate (47% of survivors) is that, as mentioned above, those who were not interviewed for earlier waves were not contacted for interviews in later waves.

Obesity

At both waves 3 and 4, obesity status was determined by calculating a body mass index (BMI) for each participant. BMI scores were calculated using the standard formula that divides respondents' weight in kilograms by the square of their height in metres (Centers for Disease Control and Prevention, 2007). Respondents' BMIs were then classified into three categories: normal weight ($18 \leq BMI < 25$), overweight ($25 \leq BMI < 30$), or obese ($BMI \geq 30$) (Centers for Disease Control and Prevention, 2006). Individuals who are underweight ($BMI < 18$) were excluded from analyses, and overweight individuals were examined separately from normal-weight or obese individuals. These decisions were made because these weight categories shows different associations with mental health (e.g., de Wit, van Straten, van Herten, Penninx, & Cuijpers, 2009; Pickering et al., 2007), and their inclusion with normal-weight individuals in a heterogeneous "non-obese" category may have skewed results. A BMI change score was also calculated that characterized respondents' change in BMI score between waves 3 and 4. To calculate this variable, wave 4 BMI was subtracted from wave 3 BMI. This means that a positive

value for the BMI change score indicates that a respondent gained weight over the follow-up period; a negative value indicates that a respondent lost weight.

Mental Disorders

I examined several mental disorders in this study to determine their association with obesity. Diagnoses were made in waves 3 and 4 of the ECA survey using the Diagnostic Interview Schedule Version III Revised (DIS-III-R; Robins, Helzer, Cottler, & Goldring, 1989), which generates diagnoses based on the criteria of the DSM-III-R. The reliability and validity of the DIS have been examined previously, and have been reported to be acceptable or better than acceptable for all diagnoses (Robins, Helzer, Croughan, & Ratcliff, 1981). The exceptions to this are one-month diagnoses, which show lower concordance with physician-based diagnoses (Anthony et al., 1985) and therefore were not used in the current study. There is a tendency for under-diagnosis by the DIS, so it has been suggested that estimates of association between psychiatric disorders and their potential risk factors be considered to be conservative (Eaton, Neufeld, Chen, & Cai, 2000).

All Axis I mood and anxiety disorders assessed in both waves 3 and 4 of the ECA were examined. These disorders are major depression, panic disorder, agoraphobia, obsessive-compulsive disorder (OCD), generalized anxiety disorder (GAD), and social phobia. An error was present in the diagnostic algorithms for alcohol abuse and dependence, which misclassified respondents who did not meet criteria for each disorder as "missing," along with actual missing cases. Also, drug abuse and dependence had very low prevalences in the ECA sample (e.g., N = 2 for incident drug dependence between waves 3 and 4). Because of these issues, alcohol and drug abuse and dependence were not utilized in this study. However, as previous cross-sectional research has demonstrated few relationships between obesity and substance use disorders

(Mather et al., 2009; Scott et al., 2007), the inability to include these disorders in the current study is of little concern, since there would likely have been few significant findings. Small cell sizes also precluded analyses examining whether individuals who went from normal weight at wave 3 to overweight or obese at wave 4 showed different patterns of association with past-year wave 4 mental health variables. While 132 respondents went from normal weight to overweight and 23 respondents went from normal weight to obese over the follow-up period, only a maximum of 4 respondents within one of these categories met criteria for a past-year mental health problem at wave 4. I planned to conduct these analyses to test whether the onset of overweight or obesity is associated with mental health problems, but as indicated, cell sizes were too small on all variables to allow them to be conducted.

In order to allow comparisons to previous research, a first group of analyses was conducted examining the cross-sectional relationships between body weight categories and past-year mental disorders and suicidality at both waves 3 and 4 (see Analytic Strategy section below), which required the creation of past-year mental disorder variables at both waves 3 and 4. For the group of analyses examining the effect of obesity on mental health (see below), lifetime wave 3 diagnoses were utilized as covariates to adjust for the effect of a previous history of a given mental disorder when analyzing the impact of body weight on that disorder. For example, when investigating the relationship between wave 3 obesity and wave 4 depression, a lifetime diagnosis of depression at wave 3 (1993) was included as a covariate. This approach was selected, rather than simply excluding individuals from analysis who had previously met criteria for a given mental disorder, to try to maintain larger cell sizes (see Results section for a discussion of the issue of cell size in the current sample). Incident mental disorders (developing between waves 3 and 4) will be the dependent variables for which the predictive relationship of

obesity will be tested. For the group of analyses examining the effect of mental health on body weight change (again, see below), past-year diagnoses of mental disorders at wave 3 were utilized as predictors to determine whether mental health is related to body weight change.

In addition to examining mental disorders individually, summary "any anxiety disorders" variable were created to identify whether a respondent had met criteria for at least one anxiety disorder. For the group of cross-sectional analyses (see below), past-year "any anxiety disorder" variables were created at both waves 3 and 4. The wave 3 past-year "any anxiety disorder" variable was also utilized for the group of analyses examining the effect of mental health on body weight change, and it was employed in an analogous fashion to the other mental disorder variables in this group of analyses. For the group of analyses examining the effect of obesity on mental health (see below), these variables were created for both waves 3 and 4; the wave 3 variable characterized lifetime anxiety disorders, and the wave 4 variables characterized incident disorders. They were utilized in a similar manner to the individual mental disorder variables described above: the wave 3 variable was used to adjust for prior mental illness at wave 3, and the wave 4 variable was used as a dependent variable in analyses.

To account for the effects of comorbid psychiatric disorders on the associations between overweight/obesity and mental health variables, some analyses included an adjustment for the presence of other psychiatric diagnoses (see Analytic Strategy section below). For analyses concerning anxiety disorders, new variables were created to denote whether or not a respondent met criteria for any anxiety disorder other than the disorder currently being examined. By using these new variables, I was able to adjust the analyses examining anxiety disorders for depression and any other anxiety disorder. For the group of analyses examining the effect of obesity on mental health, these variables were created using incident mental disorder variables between

waves 3 and 4; for the group of analyses examining the effect of mental health on body weight change, these variables were created using past-year mental disorder variables at wave 3.

Suicidality

Suicidal ideation and attempts were measured in the depression section of the waves 3 and 4 surveys. Respondents were asked as part of the depression assessment two questions regarding suicidal behaviors: 1) "Have you ever felt [Did you feel] so low you thought about committing suicide?" and 2) "Have you ever attempted [Did you ever attempt] suicide?" Affirmative responses were followed up with queries regarding the onset and recency of these suicidal behaviors. Eight variables were derived from this raw data to indicate lifetime and past-year suicidal ideation and suicide attempts in wave 3, past-year suicidal ideation and attempts at wave 4, and incident suicidal ideation and suicide attempts between waves 3 and 4. These variables were used in an analogous way to the psychiatric diagnosis variables described above..

Physical Health

The ECA data contain information on a number of physical health conditions, based on respondents' self-reports. These conditions include diabetes, chest pain, heart troubles (rheumatic fever, rheumatic heart disease, angina pectoris, heart attack, or congestive heart failure), hypertension, joint pain, arthritis or rheumatism, back pain, bone breaks/fractures, stroke, cancer, loss of bladder control, fainting, seizures, dizziness, weakness, and severe headaches. To characterize the burden of physical health conditions on the individual, a variable was created that summarized the number of serious physical health conditions each respondent reported. For my purposes, I characterized seven of the available conditions (diabetes, heart troubles, hypertension, arthritis or rheumatism, stroke, cancer, and seizures) as serious; the remaining somatic conditions were not included in the physical health variable. From the data

regarding the seven serious conditions, a variable was created to characterize whether a respondent reported 0, 1, or 2 or more of these conditions.

Covariates

A number of wave 3 covariates were included in analyses. These included age, gender (in the analyses that are not gender-stratified), and race, all of which have been shown to moderate the depression-obesity relationship (Heo et al., 2006). Marital status and education were also adjusted for, as each of these variables has shown associations with both obesity (Everson, Maty, Lynch, & Kaplan, 2002; Pickering et al., 2007) and mental health (Everson et al., 2002; Patten et al., 2006). Age and education were entered as continuous variables for analyses; however, for table 1 these variables were broken down into categories to allow for frequencies and crosstabulations to be calculated. Age was broken down into six categories (30-39, 40-49, 50-59, 60-69, 70-79, or 80-86 years old), and education was broken down into three (high school or less, some college or college graduate, or at least some graduate studies).

Analytic Strategy

Crosstabulations were calculated to determine the prevalence of mental disorders and suicidal behaviors in 2004 overall, and then among those who are normal-weight, overweight, and obese in 1993, both in the whole sample and stratified by gender. The analyses were split into two groups: the first to test whether earlier obesity predicts later mental health, and the second to test whether earlier mental health predicts body weight change. Logistic regression analyses were conducted for the first two groups of analyses. To test the cross-sectional relationships between obesity and mental health, analyses were conducted examining whether body weight category predicted past-year mental disorder diagnoses and suicidal behaviors at both waves. These analyses were adjusted for age, gender, education, race, marital status, other

psychiatric disorders (see below), and physical health conditions, as is in line with the most recent cross-sectional research in the area. Because there has been some indication in the cross-sectional literature that there may be gender differences in the associations, these cross-sectional analyses were then stratified by gender to allow comparison between the ECA and other samples on this issue as well.

To test whether earlier obesity predicts later mental health, logistic regression analyses were first conducted examining whether wave 4 mental health (psychiatric disorders and suicidality) is predicted by wave 3 body weight, with the normal-weight category as the reference group and after adjusting for age, gender, education, race, and marital status. To test whether comorbid psychiatric disorders or physical health problems attenuate the obesity-mental health relationship, further analyses were carried out, and involved conducting the same analyses as above, first adding other psychiatric disorders to the list of covariates (see below), and then including both other psychiatric disorders and physical health. To determine whether any gender differences exist in the above associations, a final set of analyses in this group examined the gender-specific associations between wave 3 body weight and wave 4 mental health variables, after adjusting for other sociodemographic factors, other psychiatric disorders, and physical health.

For the group of analyses examining the effect of mental health on body weight change, analyses of covariance (ANCOVAs) were conducted. Analyses were first conducted examining whether past-year wave 3 mental health variables (psychiatric disorders and suicidality) predict a change in body weight, adjusting for sociodemographic variables (age, gender, education, race, and marital status). Further analyses were then performed to test whether these relationships were attenuated by the addition of other psychiatric disorders and the physical health conditions

variable to the model (similar to the analyses described above. Gender differences in the fully-adjusted model (adjusting for sociodemographics, other psychiatric disorders, and physical health conditions) were then examined.

To account for the presence of other psychiatric disorders, a variety of adjustments were made. Analyses concerning depression were adjusted for any anxiety disorder, and analyses concerning the "any anxiety disorder" variable were adjusted for depression. Those analyses involving suicidal behaviors (ideation and attempts) were adjusted for both depression and any anxiety disorder. Analyses concerning individual anxiety disorder variables were adjusted for depression, plus a variable that characterized whether the respondent met criteria for any anxiety disorder other than the one in question (see Mental Disorders section above). The reason for utilizing this relatively complex strategy for adjusting for comorbid psychiatric disorders is that it allowed me to take into account the impact of other psychiatric disorders on obesity-mental health relationships, while also preventing overspecification of the statistical model (constructing a model with degrees of freedom greater than those available in the data).

Results

Table 1 illustrates the distribution of respondents within the various sociodemographic variables, as well as within the physical health conditions variable. The majority of respondents at wave 3 were between 30-49 years of age, with the mean age being 47.6 years (standard deviation [SD] = 12.8) and no significant difference between the genders (χ^2 [5] = 3.93, p = 0.560). Being married was the most common marital status among respondents, though more men reported never having been married than women, who experienced all other marital status categories (married, widowed, separated, and divorced) at higher rates than men (χ^2 [4] = 31.56, $p < 0.001$). Pursuing an education beyond high school was relatively uncommon in the sample,

with women going on to college less frequently than men (χ^2 [2] = 23.58, $p < 0.001$). Non-Hispanic White was by far the most common race in the sample, with 61.8% of respondents endorsing this category. Over half (58.6%) of the respondents reported none of the seven serious physical health conditions examined in this study, though women were more likely to endorse at least one physical health condition than men (χ^2 [2] = 9.32, $p < 0.009$). The mean number of physical health conditions reported was 0.6 (SD = 0.8), further emphasizing the relatively good physical health of the sample.

Table 2 outlines the prevalence of mental disorders, suicidal behaviors, and the three body weight classifications at various timepoints within the ECA dataset. In the overall sample, lifetime anxiety disorder prevalences at wave 3 ranged from 1.1% for OCD to 5.7% for social phobia. As expected, past-year anxiety diagnoses at wave 3 and incident anxiety diagnoses between waves 3 and 4 had lower prevalences than their lifetime at wave 3 counterparts, but they followed a similar trend in terms of the relative frequency of the disorders. At wave 3, depression had lifetime and past-year prevalences of 6.5% and 2.3%, respectively, and 4.0% of the sample developed a major depressive episode between waves 3 and 4. Notably, suicidal ideation evidenced a relatively high prevalence in the sample, with 10.2% reporting lifetime ideation at wave 3 and 5.3% reporting incident ideation between the waves. When examining these prevalences by gender, women consistently demonstrated higher prevalences of all mental disorders and suicidal behaviours, though both genders exhibited similar patterns in relative prevalences to the overall sample. The low prevalence of certain mental health variables among men ($N < 5$) may have jeopardized the accuracy of statistical estimates, so a number of past-year mental health variables (depression, panic disorder, OCD, GAD, suicidal ideation, and suicide attempts) were not examined further in the second group of analyses exploring whether wave 3

mental health predicts weight change over the follow-up period. Suicide attempts in women were also excluded from further analyses, for the same reason. However, the excluded anxiety disorders were still included in calculating the "any past-year anxiety disorder" variable for wave 3.

Respondents in the overall sample were fairly evenly spread among the three body weight categories, with 37.6% being normal weight and 27.6% being obese at wave 3 (see Table 2). By wave 4, a shift toward higher adiposity had occurred, such that many more respondents (39.1%) were classified as obese at this timepoint. Comparing the genders, women demonstrated a lower prevalence of overweight compared to men, and higher rates of both normal weight and obesity. This was true for both waves 3 and 4, though both genders saw shifts toward higher rates of obesity at wave 4. Between waves 3 and 4, there was a mean BMI score change of 2.08 units (SD = 4.07), indicating that respondents in general gained weight over the course of the eleven-year (on average) follow-up. This mean BMI increase translates into a mean weight gain of 11.8 pounds (SD = 24.5), or just over one pound per year. The mean BMI score change for women was 2.27 (SD = 4.33) and 1.77 (SD = 3.58) for men, indicating women saw higher rates of weight gain than men over the follow-up period. However, with a BMI score change range from -16.40 to 27.88 in the overall sample, there was a large degree of variability in how much weight respondents gained or lost between the waves (these results not reported in table).

Table 3 describes the prevalence of incident mental disorders and suicidal behaviors among those who were normal weight, overweight, or obese at wave 3. With the exception of suicide attempts, chi-square tests indicate no differences in prevalence between the various body weight categories. The crosstabulations revealed small cell sizes ($N < 5$) for agoraphobia, obsessive-compulsive disorder, generalized anxiety disorder, and suicide attempts. Because

small cell sizes jeopardize the accuracy of point estimates in logistic regression, agoraphobia and obsessive-compulsive disorder were not analyzed further for the first group of analyses, examining whether wave 3 body weight predicts wave 4 mental health variables. For the same reason, the overweight category for generalized anxiety disorder and suicide attempts was excluded from further analyses in this analysis group as well. However, the excluded anxiety disorders were still included in the calculation of the "any anxiety disorder" variable. In addition, almost all of the respondents who attempted suicide are captured under the suicidal ideation variable, as further crosstabulations indicated that only one respondent reported an attempt without ideation.

Table 4 includes similar data to Table 2, broken down by gender. This table demonstrates gender differences in the prevalence of many mental disorders, with women generally showing higher percentages than men. Again, these crosstabulations indicate that many cell sizes are too small for reliable logistic regression analyses to be conducted. In men, all mental disorders and suicidal behaviours had cell sizes too small for analysis, so no further analyses examining wave 3 body weight and wave 4 mental health were conducted for men. For women, small cell sizes were noted for agoraphobia, OCD, GAD, and suicide attempt, and these variables were not analyzed further in this analysis group. Additionally, the overweight category for suicidal ideation was excluded from further analyses examining the relationship between earlier body weight and later mental health.

Results from the analyses of the cross-sectional relationship between obesity and mental health variables at both waves were non-significant, with the lowest p value from the wave 3 analyses being 0.200 and the lowest p value from the wave 4 analyses being 0.123. Because of the absolute non-significance of these findings and the fact that these were auxiliary analyses

only conducted to allow for comparison of my data to other cross-sectional research, they are not presented in tables here.

Tables 5 through 7 address the first question of this study: whether obesity predicts the onset of mental health problems. In Table 5, I demonstrate that there are very few relationships between earlier body weight and later mental health in both unadjusted models and after adjusting for sociodemographic variables. Of the incident mental disorders with sufficient cell sizes for analysis, none showed a significant relationship with either earlier overweight or earlier obesity. Among the suicidal behaviors, only incident suicide attempt showed a significant association with being obese at wave 3 (adjusted odds ratio [AOR] = 3.76, 95% CI 1.11-12.76). Table 6 demonstrates the same associations after additionally adjusting first for sociodemographics and other incident psychiatric disorders (AOR1), and then adjusting for these variables plus physical health conditions (AOR2; see Table 6). Again, only incident suicide attempts showed a significant relationship to wave 3 obesity (AOR = 10.42, 95% CI 1.84-59.00). However, after adding the physical health conditions variable as a covariate, this association was no longer statistically significant. Interestingly, after this additional adjustment, incident panic disorder evidenced a significant relationship to wave 3 obesity (AOR = 4.50, 95% CI 1.08-18.70). No other mental health variable showed significant associations after physical health conditions were adjusted for. When the fully-adjusted set of analyses (adjusting for sociodemographics, other incident mental disorders, and physical health conditions) were broken down by gender (Table 7), an interesting finding emerged. Although the data could not be analyzed for men due to small cell sizes, women demonstrated a single significant relationship where earlier obesity predicted lower odds of incident social phobia (AOR = 0.12, 95% CI 0.02-0.71). No other significant relationships were found.

Tables 8 through 10 contain the results of analyses examining the second question for this study, namely, whether mental health variables have an effect on body weight change. Tables 8 and 9 indicate that few past-year mental disorders at wave 3 are predictive of change in body weight. Adjusting for sociodemographic variables, only depression showed a relationship with body weight, predicting a significant weight gain over the follow-up period (F [1, 956] = 8.063, $p = 0.005$; see Table 8). This relationship remains significant after adjusting for both other psychiatric disorders and physical health conditions (F [1, 853] = 3.962, $p = 0.047$; see Table 9). Table 10 demonstrates the results from analyses on mental health variables and body weight change, stratified by gender. As mentioned above, many mental disorder and suicidal behavior variables could not be analyzed due to low prevalences. However, among those that could be analyzed, two differences from the unstratified analyses arose. First, depression evidenced only a marginally significant positive relationship with weight change among women (F [1, 521] = 2.996, $p = 0.084$). Second, suicidal ideation demonstrated a significant association with body weight change among women (F [1, 520] = 3.977, $p = 0.047$), even though ideation was not a significant predictor in the unstratified analyses. Again, this group of analyses indicates that the overarching pattern is for mental health to be statistically unrelated to body weight change over an eleven-year follow-up period.

Discussion

Limitations and Considerations

Two of the major variables in this study were obesity status and BMI change. Both of these variables were calculated using respondents' BMI scores, and this use of BMI scores raises an important issue to consider. Though BMI is the accepted measure for determined obesity (Centers for Disease Control and Prevention, 2007), it still only provides a rough estimate of an

individual's adiposity. Certain factors, such as gender, age, and ethnicity, are known to influence the degree of adiposity at a given BMI (Gallagher et al., 1996). A well-known example of the inexactness of BMI is in reference to professional athletes, whose high proportion of muscle often leads them to be classified as having a high BMI. A high BMI would normally be suggestive of higher adiposity, but in the case of these individuals actually reflects their increased muscle mass. Although other measures, such as waist-to-hip ratios and skinfold thickness, have been suggested as more precise measures of adiposity, BMI has been accepted as the best measure for determining obesity status at the population level (World Health Organization, 2000). One must be aware that fluctuations in body proportions means that similar BMI levels for any two individuals does not correspond to equal adiposity and therefore equal risk of physical and mental disorder for them. However, the frequent inclusion of weight and height in population surveys and the fact that a BMI of over 30 is almost always indicative of excess body fat make this a common and satisfactory measure for determine the health correlates, including psychological health associates, of obesity.

In addition to the above issue, the current study has several limitations that are important to consider when interpreting the results. First, the surveys were conducted by trained lay interviewers, and these assessments may not achieve the accuracy of clinicians' diagnoses. Second, the fact that 42% of the original ECA sample had died by wave four, and that only 53% of surviving respondents were interviewed at wave 4, suggests that survivor and attrition biases may skew the remaining sample towards being mentally healthier. This would lower the observed prevalences of mental disorders, and may have therefore impacted the results in this study. Third, height and weight were self-report in wave 3, and this may introduce some inaccuracies to the classifications made of respondents into body weight categories at wave 3

(most respondents consented to having height and weight measurements taken at wave 4). There is a slight tendency for individuals to overestimate their height and underestimate their weight, which would distort BMI scores such that the calculated scores would be slightly lower than the actual values (Roberts, 1995). When classifying BMI into body weight categories, this bias means that some individuals will be mistakenly placed in an incorrect category. However, this distortion is likely small and the risks it poses to analyses are minimal enough that BMI-based body weight classifications are used in most epidemiological research in the field. Fourth, eating disorders (most importantly, bulimia nervosa and binge eating disorder) and body dysmorphic disorder were not assessed in the ECA survey, and results may be affected by the inability to adjust for conditions that show relationships to both body weight (American Psychiatric Association, 2000) and other facets of mental health (Kaye et al., 2004; Mitchell & Mussell, 1995; Phillips et al., 2005). Fifth, and relatedly, information on psychotropic medication use was not available in the ECA survey, and these variables may mediate some body weight-mental health relationships by causing weight gain themselves (e.g., Blaine, 2008). Sixth, the Baltimore ECA survey utilized DSM-III-R diagnoses. The use of these diagnoses was necessary in wave 3 since data collection began before the DSM-IV was published; their use was continued in wave 4 to maintain consistency between the two waves (Eaton, Kalaydjian, Scharfstein, Mezuk, & Ding, 2007). However, the DSM-III-R definitions of certain disorders (e.g., depression, panic disorder) differ somewhat from the currently-accepted definitions identified in the DSM-IV. Seventh, the length of time between waves 3 and 4 varied between respondents, and this may have an impact on results (e.g., longer lengths of time between waves would leave more time for respondents to gain or lose weight, which would have affected the analyses on the impact of mental health on body weight change). Eighth, the results of this study may not be generalizeable to younger

adults because, due to its longitudinal nature, the sample I utilized consists solely of middle-aged and older adults (ages 41 years and older). Ninth, a large number of variables could not be analyzed for men, due to small cell sizes. Because of this, I was largely unable to determine the nature of the obesity-mental health relationship specifically for men, and the results of this study cannot be fully generalized to this group as a result. Finally, because the ECA dataset did not contain information on every mood and anxiety disorder, I was unable to examine the effect of certain disorders (e.g., bipolar disorder, specific phobia) that have shown relationships to obesity in cross-sectional research (e.g., Barry et al., 2008; Pickering et al., 2007). As a result, I was unable to fully account for psychiatric comorbidity in my analyses, and it therefore remains unknown what impact these disorders may have had on the results of this study.

As described above, cross-sectional analyses were carried out to allow for results of the ECA to be compared with results from other samples, as cross-sectional work is much more common in the literature on obesity and mental health. No significant results were uncovered at all. This is in contrast with the findings of previous studies using American samples (e.g., Barry et al., 2008; Pickering et al., 2007; Simon et al., 2006) which have shown cross-sectional relationships between obesity and mental health variables. This could possibly be attributed to the age differences between the ECA sample (primarily middle-aged and older adults) and most general population surveys, which is likely the explanation for why many of the longitudinal associations examined in this study were non-significant as well (see below for full explanation). Another possible contributory factor could be the survivor effects and attrition bias discussed above, as these effects would likely lessen the significance of relationships between obesity and mental health.

Finding 1: Prevalence of sociodemographic variables, obesity, and mental health variables compared to the general population

In light of these issues, the current study has several different findings to consider. First, the prevalences of sociodemographic variables, obesity, psychiatric disorders, and suicidal behaviours in the ECA were often similar to those for the general population, though differences between this sample and the general population also emerged. With regard to sociodemographics, the ECA sample had a higher proportion of never married individuals (31% versus 23%) and a lower level of currently married individuals (45% versus 61%) than the general US population at the time (Saluter, 1994). The ECA sample was also generally less well-educated than the general US population, with 45% of the general population ages 25 and over having completed some college or more (Kominski & Adams, 1993), compared to only 30% of the ECA sample. However, this could likely be attributed to the higher average age of the ECA sample, as higher education has become more common in more recent years and is therefore more common among younger generations (Stoops, 2007). The distribution of ECA respondents among the race categories varied distinctly from the general population, with far fewer non-Hispanic White respondents in the ECA (62% versus 76%), and proportionally more minorities (non-Hispanic Black, American Indian, Asian/Pacific Islander, and Hispanic) respondents, than was found in the US population at that time (Gibson & Jung, 2002).

The rate of obesity at wave 3 (1994) was in line with that in the general US adult population at the same time, though overweight was more prevalent in the ECA sample (33.5%) than in the general US population at this time (25.1%; Ogden et al., 2007). Though the rates of overweight and obesity increased in both the ECA and the general US population over the follow-up period, these rates seem to have increased more quickly in the ECA sample than in the

population in general: Both overweight and obesity rates were higher in 2004 (wave 4) in my study than in the general population (30.7% and 39.1%, respectively, compared to 27.4% and 34.0% for the general US population). This difference in rates is likely because the ECA sample is older, on average, than the general US population, as obesity rates tend to increase with age (Ogden et al., 2007). The difference is likely also due to regional sociodemographic differences, because, as discussed above, adults in Baltimore have a somewhat different sociodemographic profile than those from other areas of the US. For instance, variables such as minority race status, never having married, and lower education levels, which are more common in the Baltimore area, are factors that are known to relate to obesity (Everson et al., 2002; Pickering et al., 2007).

When compared to results from representative US surveys, many of the mental disorders in the ECA appear to have lower prevalences in 1993 (wave 3) than in the general population at the same time. For instance, depression had a lifetime prevalence of 6.5% in wave 3, compared to 7.5-8.3% for the general population at around the same time (Kessler, Zhao, Blazer, & Swartz, 1997). GAD also evidenced lower lifetime prevalences in 1993 in the ECA (2.0%) compared to the general population (5.1%; Wittchen, Zhao, Kessler, & Eaton, 1994), as did social phobia (5.7% versus 13.3%; Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996). The prevalences of panic disorder and agoraphobia were also lower in the ECA (2.8% and 4.7%, respectively) than in the general population (3.5% and 5.3%, respectively; Kessler et al., 1994) though the estimates for these disorders are more similar than for other disorders. However, these prevalence estimates are from the National Comorbidity Survey (NCS), and at this time-point, the sample contained only US adults ages 18-54 years (Kessler et al., 1994). This makes the sample substantially younger, on average, than the ECA sample at wave 3, which is significant because younger groups tend to show higher prevalence rates of mental disorders than their older

counterparts (Kessler & Wang, 2008). In addition, and as mentioned above, a survivor effect may have skewed the ECA sample such that those still included by wave 4 were more likely to be mentally (and physically) healthy than those who had died. This would obviously lower the observed prevalences of mental disorders in the ECA sample. Attrition bias is likely to have had the same effect, as respondents who did not complete subsequent surveys after wave 1 (e.g., due to homelessness, severe illness, etc.) are likely to be less healthy than those who participated in all four waves. Though US population estimates are unavailable for the prevalence of OCD in 1993 (no US epidemiological surveys besides the ECA had included a diagnosis of OCD at this point), the prevalence estimates in other countries at this time ranges from 1.1% to 1.8% (Weissman et al., 1994), indicating that the ECA wave 3 estimate of 0.5% is low, likely due to the aforementioned survivor effects and attrition bias.

Generally, suicidal behaviors evidenced similar prevalences in the ECA and the general population. In the NCS, 4.6% of respondents reported a lifetime suicide attempt, as did 4.2% of respondents in wave 3 of the ECA. Suicidal ideation was somewhat lower in wave 3 of the ECA than in the general population, with 10.2% reporting lifetime suicidal ideation in the ECA and 13.5% reporting the same in the NCS (Kessler, Borges, & Walters, 1999). Incident suicidal ideation has similar prevalences between waves 3 and 4 of the ECA (5.3%) and between the NCS and its second wave, the NCS-Replication, conducted ten years after the first NCS (6.2%; Borges, Angst, Nock, Ruscio, & Kessler, 2008). The same is true for incident suicide attempts, with a prevalence of 1.7% in the ECA and 0.9% in the NCS (Borges et al., 2008). Together, the general consistencies in the prevalences of obesity, psychiatric disorders, and suicidal behaviors between the ECA and the general US population indicate that the ECA is largely similar to the

general population, which bolsters the confidence with which results from my study can be generalized to the population at large.

Finding 2: Body weight and mental health are largely unrelated

Besides suicide attempts, panic disorder, and social phobia (discussed below), no mental health variables were predicted by body weight category at wave 3, which is in contrast with the findings of cross-sectional studies in this area. Particularly notable is the lack of relationship between earlier obesity and later depression, as this relationship has been previously demonstrated in both cross-sectional and longitudinal research (Mather et al., 2009; Roberts et al., 2003; Roberts et al., 2002; Simon et al., 2006). Because of the issue of small cell sizes, I elected to simply adjust for mental health variables at wave 3, rather than remove individuals with a lifetime history of the given mental disorder from analyses. Roberts et al. (2003) compared these two analytic approaches when examining the relationship between earlier obesity and later depression, and found that the exclusion model generated significant results, while the adjusted model did not. This may partially explain the findings here. Another more general explanation for why obesity did not predict mental disorders in this study is psychological. A common explanation for why obesity and mental health might be causally related involves the experience of stigma. Overweight and obese individuals commonly experience stigma because of their weight (Myers & Rosen, 1999; Puhl & Brownell, 2006). Exposure to stigmatization negatively impacts psychological well-being and may thereby increase the propensity for a variety of mental disorders to develop (Myers et al., 1999). However, the frequency of stigmatization is significantly related to degree of psychological distress (Myers et al., 1999), and the frequency of experiencing stigmatizing situations decreases with age (Puhl et al., 2006). In fact, older individuals are at particularly lower risk of

stigmatizing experiences such as nasty comments from children, being avoided or excluded, and confronting physical barriers and obstacles (Puhl et al., 2006) that, at least on the surface, appear to be the most psychologically damaging. Given these associations, it makes sense that a sample comprised largely of adults over age 40 at wave 3 (1993) may evidence fewer relationships between obesity and mental health, as these individuals are experiencing fewer of the stigmatizing situations that might induce mental health problems. Another possible explanation for the overall lack of relationship between obesity and later mental health is that people develop better emotion regulation abilities with age (Blanchard-Fields & Coats, 2008) and experience fewer mental disorders and less distress (Kessler et al., 2008; Orpana, 2008) as they get older, as well. This increased resilience, combined with the aforementioned experience of fewer stigmatizing situations, may weaken the relationship between earlier obesity and later mental health with age to the point that obesity is no longer a significant player in the development of mental health problems among middle-aged and older adults. This further suggests that the predictive relationship between obesity and mental health issues that has been uncovered in previous research may be largely attributable to the younger respondents in the samples employed.

As in the analyses examining obesity's predictive relationship to mental health, the analyses investigating the relationship between mental health and body weight change uncovered few significant relationships. Anxiety disorders were notably absent from the list of significant predictors of body weight change. A total lack of associations in this regard is somewhat surprising, given the cross-sectional findings (Barry et al., 2008; Mather et al., 2009; Pickering et al., 2007; Simon et al., 2006). However, the only other extant study to examine the relationship between anxiety disorders and body weight change had similar results, finding that none of panic

disorder, GAD, nor an "any phobic disorders" variable was associated with weight gain over a twenty year period (Hasler et al., 2004). My work expanded on that of Hasler et al. (2004) by examining a larger number of anxiety disorders, and my findings suggest that although obesity may be causally related to at least one anxiety disorder (panic disorder), the converse relationship is likely not true. This further indicates that anxiety disorders likely have no association to the current obesity epidemic, as these disorders are not contributing to weight gain. One explanation for this finding immediately jumps to mind. In contrast to depression, which has symptoms such as psychomotor retardation and increased appetite, anxiety disorders are often accompanied by symptoms such as insomnia, hypervigilance, and lack of appetite. Obviously, symptoms like these do not lend themselves well to gaining weight, and as such would preclude anxiety disorders from causing weight gain or obesity, at least among middle-aged and older adults. Because of the novelty of these findings, they should be replicated in future studies for confirmation.

Finding 3: Obesity is significantly related to specific mental health variables

Although there was an overall lack of significant findings in the analyses examining the impact of obesity on mental health, a small number of significant relationships were uncovered. First, obesity was a significant predictor of suicide attempts, but this association lost significance after adjusting for psychiatric comorbidity and physical health conditions. Notably, the association was still significant when adjusting only for comorbid psychiatric disorders, which suggests that physical health affects the relationship between obesity and suicide attempts. The relationship between obesity and other physical health conditions is well-documented (Ogden et al., 2007; World Health Organization, 2000), which explains part of this finding. A recent review article on suicide (Hawton & van Heeringen, 2009) explains that suicidality is associated with a

variety of serious physical health conditions, including cancer, epilepsy, HIV/AIDS, and Huntington's disease. This makes intuitive sense, as individuals suffering from devastating, painful, degenerative, and/or fatal physical diseases may choose to escape their eventual fate due to the disease via suicide; the current euthanasia debate illustrates this point nicely. Interestingly, overweight and obese individuals showed no higher propensity to ideate about suicide, compared to normal-weight respondents. This indicates that perhaps overweight and obese individuals are simply more likely to act on their thoughts of committing suicide, though they experience these thoughts at a rate comparable to that of normal-weight individuals. It is important to note that the lack of relationship between obesity and suicidal ideation is contrary to the results of cross-sectional work (Carpenter et al., 2000; Dong et al., 2006; Mather et al., 2009) indicating such a relationship exists. This suggests that obesity and suicidal ideation share a common risk factor, but that the two are not causally related when one examines the relationship longitudinally. One example of such a risk factor is previous physical abuse, which increases the risk of both suicidality (Hawton et al., 2009) and obesity (Bentley & Widom, 2009).

Interestingly, obesity seems to be protective against social phobia among women, even after adjusting for psychiatric comorbidity and physical health conditions. This is a surprising finding that is in opposition to what has been previously established in the cross-sectional literature (Barry et al., 2008; Mather et al., 2009). One hypothesis as to why obesity protects women from developing social phobia is that perhaps the coping strategies obese women utilize to combat stigmatizing experiences, such as using humor, witty comebacks, or jokes to deflect negative comments, seeking social support from others, or refusing to hide (Puhl et al., 2006), may empower these women to cope effectively with social situations in general. As a result of this, obese women may feel less fear of social situations, and consequently these women may put

themselves at lower risk of social phobia. Whether men experience a similar protective effect from obesity remains unknown, since the cell size was too small in the ECA sample to analyze this relationship. However, the gender-specificity of previous cross-sectional findings leads me to hypothesize that this may have been the case in the ECA as well. Reasons for a discrepancy between the genders could include the fact that men are less likely to internalize negative stigmatizing experiences (Grover, Keel, & Mitchell, 2003), which may reduce their need to enact the coping strategies, outlined above, that contribute to a reduced risk of social phobia.

Overweight and obesity showed no predictive relationship to panic disorder in any analyses, until physical health conditions were adjusted for. After adjusting for health, panic disorder was strongly predicted by being obese (but not overweight), compared to the normal-weight category. The most likely reason for this finding is statistical: it is possible that adjusting for physical health conditions accounted for "noise" in the data (i.e., some of the error variance in the model) that was previously concealing a true significant relationship. When this predictor was added to the statistical model, the error variance was more accurately characterized, and the true predictive relationship between obesity and panic disorder was revealed. Future work could explore further the relationship between earlier obesity and later panic disorder, to determine what the true nature of the association between these variables is. Regardless, one possible explanation for why obesity increases the odds for panic disorder relates to the hypothesis above for why social phobia is less common in obesity. Perhaps the stigmatizing situations often experienced by obese individuals (Myers et al., 1999; Puhl et al., 2006) lead to a sort of "sink or swim" phenomenon whereby obese individuals either learn how to cope successfully with these situations, and social situations in general (thereby decreasing their risk of social phobia), or they fail to cope effectively with these situations and develop an increased risk for panic disorder due

to their understandable anxiety over entering into these demeaning and humiliating situations. The specificity of this finding to panic disorder, rather than to a wider array of anxiety disorders, may relate to the similarities between the symptoms of panic attacks and some of the medical comorbidities of obesity (Pickering et al., 2007), which may make obese individuals more likely to experience panic attacks instead of other forms of anxiety.

Finding 4: Select mental health variables predict body weight change

When examining whether mental disorders and suicidality predict a change in body weight, very few significant results were found. One disorder that consistently stood out as a predictor, however, was depression, even in analyses adjusting for comorbid psychiatric disorders and physical health conditions. Previous work has been mixed, with some finding associations between earlier depression and later obesity, and others not. In a comprehensive review, McElroy et al. (2004) suggest that adult-onset depression is not predictive of later obesity. Our results are not in line with this; however, much previous research has utilized a dichotomous obesity variable, rather than the more sensitive BMI change measure I employed in this study. This slight difference in methodology may have allowed me to detect a true relationship that others have missed due to the nature of their data. Reasons why depression may be causally related to weight change are apparent. First, one of the features of depression is weight gain, potentially due to the decreased level of physical activity associated with the disorder (American Psychiatric Association, 2000). Second, depressed people are prone to emotional eating (eating in the absence of hunger as a method of emotion regulation), which could very easily lead to increased weight (Ouwens, van Strien, & van Leeuwe, 2009). Finally, the antidepressant medications that are often prescribed for depression can themselves induce weight gain (World Health Organization, 2000). Any or all of these mechanisms may be at play

in a given individual with depression to increase the risk of gaining weight over a period of time. Regardless of mechanism, the finding of higher rates of weight gain in depression is relevant to the current obesity epidemic. An average weight gain over eleven years of almost four BMI points would be enough to put most American adults, who are already overweight (BMI > 25) on average, into the obese category (BMI > 30). This, combined with the relatively high prevalence of depression in the population in general (see above), may partially explain the increase in average adiposity and the rampant spread of obesity among adults in the US and worldwide.

When stratifying analyses by gender, depression was not significantly associated with weight gain for women (results could not be analyzed for men due to small sample sizes). However, this is most likely explained statistically, as a smaller sample size in the gender-stratified analyses reduced the statistical power of the model to detect a true effect. Suicidal ideation was significantly protective against weight gain for women, even though this relationship was not found for the overall sample. One explanation for this may be found in the positive association between suicidal ideation and both eating disorders (Franko & Keel, 2006) and body dysmorphic disorder (Phillips et al., 2005). Both of these disorders, by their nature, decrease the likelihood of weight gain, and as such are potential mediators of the relationship between suicidal ideation and lower risk of weight gain. Eating disorders are also much more common in women (American Psychiatric Association, 2000), which could explain the gender-specificity of this relationship. As discussed above, my inability to control for eating disorders or body dysmorphic disorder constitutes a significant limitation of my work, and future research that takes these disorders into account would be welcome.

Future Directions

Future research could build on this study in a variety of ways. First, and as already mentioned, replication of the findings presented above is important, as little longitudinal research has been conducted of this type. This is particularly important given the novelty of my overarching finding of few predictive relationships between mental health and body weight. Replication in other samples would bolster the validity of these findings and help solidify our understanding of the nature of the obesity-mental health relationship (or lack thereof). Second, selection of a methodology that allowed for larger sample and cell sizes would benefit the understanding of how the obesity-mental health relationship differs between men and women. A matched-control design is one option that comes to mind for increasing cell sizes without needing to conduct an exorbitant number of survey interviews (this sample had N > 1000, but because it relied on chance for individuals with a given diagnosis to be included, the resultant *n*'s for the psychiatric diagnoses were quite small). Third, including eating disorders, body dysmorphic disorder, and psychotropic medication use in the survey would allow future researchers to adjust for these variables, as they may have confounded some of the findings presented here. Finally, future researchers should continue to investigate known and discovered factors that impact the body weight-mental health relationship, to expand on the investigation of physical health conditions presented here and to add to our understanding of the nature of the association between body weight and mental health.

Implications

My study has many important implications. Though the significant relationship of obesity to later suicide attempts was attenuated by the inclusion in the statistical model of physical health conditions, this relationship is still relevant from a clinical perspective. Obesity could serve as an indicator of risk for suicidal behavior, even though the suicidal behavior is not itself due to the

obesity. Clinicians working with obese patients should be vigilant in monitoring these individuals for suicidality, in the hopes of identifying and treating this problem before the patient attempts suicide in the future. This is especially important given the finding that obese individuals do not ideate more, as a group, than those in other body weight categories. Obesity also predicted the onset of panic disorder, and similar monitoring of obese patients would be warranted to mitigate the effects of this disorder as well. That depression is a significant risk factor for weight gain is well-known, and the results of this study should re-emphasize for clinicians the importance of mental health in the current obesity epidemic, and the potential for obesity rates to be reduced by intervening at the level of the underlying depression that may be behind increased adiposity.

The most important finding in my study was that body weight and mental health are, for the most part, unrelated. While I do suggest that clinicians pay attention to their obese patients' mental health because of the increased risk of suicide attempts and panic disorder, it is important to keep in mind that obese individuals, on the whole, are not less mentally healthy than normal-weight individuals, at least in the over-40 age group. Clinicians, and others, should be wary of "weight-ism," or making negative assumptions about obese individuals (in this case, specifically regarding their mental health) simply because of their weight. Importantly, obese individuals identify doctors as the second most common source of stigma in their lives (Puhl et al., 2006); in light of this, my findings indicate that clinicians in particular should take care to avoid weight bias in their interactions with obese individuals.

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Table 1. Distribution of respondents within the sociodemographic variables and on the number of serious physical health conditions at wave 3 (1993).

Variable	Overall N (%)	Men N (%)	Women N (%)	χ^2	df	*p*
Age				3.93	5	0.560
30-39	337 (31.5)	133 (33.5)	204 (30.3)			
40-49	375 (35.0)	137 (34.5)	238 (35.3)			
50-59	158 (14.8)	58 (14.6)	100 (14.8)			
60-69	102 (9.5)	39 (9.8)	63 (9.3)			
70-79	82 (7.7)	23 (5.8)	59 (8.8)			
80-86	17 (1.6)	7 (1.8)	10 (1.5)			
Marital status				31.56	4	<0.001
Married	479 (44.8)	185 (46.6)	294 (43.7)			
Widowed	57 (5.3)	8 (2.0)	49 (7.3)			
Separated	96 (9.0)	21 (5.3)	75 (11.1)			
Divorced	103 (9.6)	34 (8.6)	69 (10.3)			
Never married	335 (31.3)	149 (37.5)	186 (27.6)			
Education				23.58	2	<0.001
High school or less	753 (70.3)	245 (61.7)	508 (75.4)			
Some college or college graduate	260 (24.3)	128 (32.2)	132 (19.6)			
At least some graduate studies	58 (5.4)	24 (6.0)	34 (5.0)			
Race				17.98	4	0.001
American Indian	17 (1.6)	6 (1.5)	11 (1.6)			
Asian/Pacific Islander	9 (0.8)	4 (1.0)	5 (0.7)			
Hispanic	9 (0.8)	4 (1.0)	5 (0.7)			
Non-Hispanic Black	374 (34.9)	107 (27.0)	267 (39.6)			
Non-Hispanic White	662 (61.8)	276 (69.5)	386 (57.3)			
Number of physical health conditions				9.32	2	0.009
0	556 (58.6)	217 (62.2)	339 (56.5)			
1	245 (25.8)	94 (26.9)	151 (25.2)			
2 or more	148 (15.6)	38 (10.9)	110 (18.3)			

Table 2. Prevalence of mental disorders, suicidal behaviours, and body weight classifications in the ECA.

	Lifetime at wave 3			Past-year at wave 3			Incident between waves 3 and 4		
	Overall	Men	Women	Overall	Men	Women	Overall	Men	Women
Disorder	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)
Depression	67 (6.5)	16 (4.1)	51 (7.9)	24 (2.3)	2 (0.5)	22 (3.4)	41 (4.0)	7 (1.8)	34 (5.3)
Anxiety disorders									
Panic disorder	29 (2.8)	5 (1.3)	24 (3.6)	15 (1.4)	0	15 (2.3)	26 (2.5)	4 (1.0)	22 (3.4)
Agoraphobia	49 (4.7)	9 (2.3)	40 (6.1)	36 (3.4)	6 (1.5)	30 (4.5)	7 (0.7)	0	7 (1.1)
OCD	11 (1.1)	2 (0.5)	9 (1.4)	5 (0.5)	0	5 (0.8)	6 (0.6)	1 (0.3)	5 (0.8)
GAD	20 (2.0)	5 (1.3)	15 (2.4)	10 (1.0)	2 (0.5)	8 (1.3)	17 (1.8)	3 (0.9)	14 (2.4)
Social phobia	58 (5.7)	16 (4.2)	42 (6.5)	47 (4.6)	13 (3.4)	34 (5.3)	40 (3.9)	10 (2.6)	30 (4.7)
Any anxiety disorder	129 (12.6)	30 (7.8)	99 (15.4)	92 (9.0)	19 (5.0)	73 (11.4)	75 (8.1)	16 (4.6)	59 (10.2)
Suicidal behaviors									
Suicidal ideation	107 (10.2)	35 (9.0)	72 (10.9)	21 (2.0)	3 (0.8)	18 (2.7)	54 (5.3)	12 (3.1)	42 (6.5)
Suicide attempt	44 (4.2)	12 (3.1)	32 (4.8)	4 (0.4)	0	4 (0.6)	17 (1.7)	4 (1.0)	13 (2.0)

	Wave 3			Wave 4		
	Overall	Men	Women	Overall	Men	Women
Body weight category	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)
Normal weight	385 (37.6)	127 (32.7)	258 (40.6)	251 (23.4)	88 (23.2)	163 (26.3)
Overweight	343 (33.5)	164 (42.3)	179 (28.1)	329 (30.7)	154 (40.6)	175 (28.2)
Obese	296 (27.6)	97 (25.0)	199 (31.3)	419 (39.1)	137 (36.1)	282 (45.5)

N (%): Number and percentage of respondents within the given gender group (men, women, or overall) who met criteria for the given mental disorder, suicidal behavior, or weight category.

Table 3. Prevalence of wave 4 (2004) mental disorders and suicidality within wave 3 (1993) body weight categories.

Disorder	Normal weight N (%)	Overweight N (%)	Obese N (%)	χ^2	df	*p*
Depression	21 (5.7)	8 (2.4)	11 (3.9)	4.81	2	0.09
Anxiety disorders						
Panic disorder	10 (2.7)	7 (2.1)	9 (3.1)	0.619	2	0.73
Agoraphobia	2 (0.5)	2 (0.6)	3 (1.0)	0.672	2	0.72
Obsessive-compulsive disorder	2 (0.5)	3 (0.9)	1 (0.4)	0.824	2	0.66
Generalized anxiety disorder	6 (1.9)	4 (1.3)	5 (1.8)	0.336	2	0.84
Social phobia	20 (5.4)	11 (3.3)	8 (2.8)	3.472	2	0.18
Any anxiety disorder	30 (9.4)	23 (7.7)	20 (7.4)	0.997	2	0.61
Suicidal behaviors						
Suicidal ideation	25 (6.8)	11 (3.3)	15 (5.2)	4.321	2	0.12
Suicide attempt	6 (1.6)	1 (0.3)	9 (3.2)	7.910	2	0.02

N(%): Number and percentage of respondents within the given weight category at wave 3 who developed the given mental disorder or suicidal behavior between waves 3 and 4.

Table 4. Prevalence of wave 4 (2004) mental disorders and suicidality within wave 3 (1993) body weight categories, by gender.

	Normal weight N(%)		Overweight N(%)		Obese N(%)	
Disorder	Men	Women	Men	Women	Men	Women
Depression	2 (1.6)	19 (7.7)	3 (1.9)	5 (2.9)	2 (2.1)	9 (4.8)
Anxiety disorders						
Panic disorder	0	10 (4.1)	2 (1.3)	5 (2.9)	2 (2.1)	7 (3.6)
Agoraphobia	0	2 (0.8)	0	2 (1.1)	0	3 (1.6)
Obsessive-compulsive disorder	0	2 (0.8)	1 (0.6)	2 (1.2)	0	1 (0.5)
Generalized anxiety disorder	2 (1.8)	4 (1.9)	0	4 (2.5)	0	5 (2.7)
Social phobia	3 (2.4)	17 (6.9)	4 (2.5)	7 (4.0)	2 (2.1)	6 (3.1)
Any anxiety disorder	4 (3.7)	26 (12.4)	7 (5.0)	16 (10.0)	4 (4.4)	16 (8.8)
Suicidal behaviors						
Suicidal ideation	3 (2.5)	22 (8.9)	6 (3.7)	5 (2.9)	3 (3.1)	12 (6.3)
Suicide attempt	0	6 (2.4)	1 (0.6)	0	3 (3.1)	6 (3.2)

N(%): Number and percentage of respondents within the given weight category at wave 3 who developed the given mental disorder or suicidal behavior between waves 3 and 4.

Table 5. Associations of wave 3 body weight with wave 4 mental disorders and suicidal behaviours.

Disorder	Normal weight	Overweight	Obese
Depression	1.00	0.83 (0.34-2.06)	0.90 (0.38-2.11)
Anxiety disorders	1.00		
Panic disorder	1.00	1.27 (0.45-3.60)	1.615 (0.593-4.40)
Agoraphobia	1.00	---	---
Obsessive-compulsive disorder	1.00	---	---
Generalized anxiety disorder	1.00	---	2.792 (0.60-12.96)
Social phobia	1.00	0.755 (0.33-1.75)	0.515 (0.20-1.30)
Any anxiety disorder	1.00	1.241 (0.63-2.44)	0.971 (0.48-1.95)
Suicidal behaviors	1.00		
Suicidal ideation	1.00	0.77 (0.34-1.71)	0.99 (0.47-2.09)
Suicide attempt	1.00	---	**3.76 (1.11-12.76)**

Note: All odds ratios presented are adjusted for age, gender, education, race, and marital status, and lifetime diagnosis of the given disorder at wave 3.

--- Odds ratio could not be calculated due to small cell sizes.

Table 6. Associations of wave 3 body weight with wave 4 mental disorders and suicidal behaviours, adjusted for psychiatric comorbidity and physical health.

	Overweight		Obese	
Disorder	AOR1	AOR2	AOR1	AOR2
Depression	0.61 (0.21-1.78)	0.55 (0.16-1.92)	0.93 (0.36-2.40)	0.61 (0.19-1.96)
Anxiety disorders				
Panic disorder	1.76 (0.52-6.00)	2.31 (0.53-10.12)	1.86 (0.57-6.07)	**4.50 (1.08-18.70)**
Agoraphobia	---	---	---	---
Obsessive-compulsive disorder	---	---	---	---
Generalized anxiety disorder	---	---	2.82 (0.61-13.10)	2.39 (0.50-11.28)
Social phobia	0.70 (0.27-1.80)	0.75 (0.28-2.00)	0.36 (0.12-1.09)	0.30 (0.09-1.03)
Any anxiety disorder	1.26 (0.63-2.52)	1.33 (0.63-2.78)	0.91 (0.45-1.87)	1.02 (0.46-2.23)
Suicidal behaviors				
Suicidal ideation	0.70 (0.24-2.05)	0.78 (0.26-2.33)	1.21 (0.47-3.12)	1.02 (0.36-2.89)
Suicide attempt	---	---	**10.42 (1.84-59.00)**	5.48 (0.89-33.68)

Note: All odds ratios use the normal body weight category as the reference category.
AOR1: Odds ratio adjusted for sociodemographics (age, gender, education, race, marital status), the given lifetime psychiatric diagnosis at wave 3, and other incident psychiatric disorders at wave 4.
AOR2: Odds ratio adjusted for sociodemographics (age, gender, education, race, marital status), other psychiatric disorders, and physical health conditions at wave 3.
--- Odds ratio could not be calculated due to small cell sizes.

Table 7. Associations of wave 3 body weight with wave 4 mental disorders and suicidal behaviours, stratified by gender.

	Overweight		Obese	
Disorder	Men	Women	Men	Women
Depression	---	0.41 (0.09-1.86)	---	0.56 (0.15-2.05)
Anxiety disorders				
Panic disorder	---	1.49 (0.31-7.10)	---	2.11 (0.47-9.56)
Agoraphobia	---	---	---	---
Obsessive-compulsive disorder	---	---	---	---
Generalized anxiety disorder	---	---	---	---
Social phobia	---	0.61 (0.17-2.14)	---	**0.12 (0.02-0.71)**
Any anxiety disorder	---	1.31 (0.54-3.17)	---	0.81 (0.32-2.05)
Suicidal behaviors				
Suicidal ideation	---	0.66 (0.16-2.75)	---	1.29 (0.37-4.48)
Suicide attempt	---	---	---	---

Note: All odds ratios use the normal body weight category as the reference category. All odds ratios are adjusted for sociodemographics (age, gender, education, race, marital status), the given lifetime psychiatric diagnosis at wave 3, other incident psychiatric disorders at wave 4, and physical health conditions at wave 3.

--- Odds ratio could not be calculated due to small cell sizes.

Table 8. Association between wave 3 past-year mental health variables and respondents' change in BMI between waves 3 and 4.

	Mean BMI change score (SE)			
Disorder	Disorder present	Disorder absent	F	*p*
Depression	**3.807 (1.030)**	**1.476 (0.631)**	**8.063**	**0.005**
Anxiety disorders				
Panic disorder	2.382 (1.218)	1.510 (0.634)	0.707	0.401
Agoraphobia	1.220 (0.944)	1.491 (0.636)	0.150	0.698
Obsessive-compulsive disorder	2.023 (1.897)	1.471 (0.644)	0.095	0.757
Generalized anxiety disorder	0.614 (1.527)	1.492 (0.646)	0.385	0.535
Social phobia	1.945 (0.878)	1.458 (0.644)	0.629	0.428
Any anxiety disorder	1.708 (0.773)	1.461 (0.645)	0.305	0.581
Suicidal behaviors				
Suicidal ideation	0.717 (1.075)	1.507 (0.635)	0.809	0.369
Suicide attempt	0.745 (2.099)	1.496 (0.635)	0.141	0.707

Note: All analyses are adjusted for age, gender, education, race, and marital status. All mean BMI change scores are estimated marginal means.

Table 9. Association between wave 3 past-year mental health variables and respondents' change in BMI between waves 3 and 4, adjusted for psychiatric comorbidity and physical health.

	AA1				AA2			
	Mean BMI change score (SE)				Mean BMI change score (SE)			
Disorder	Disorder present	Disorder absent	F	*p*	Disorder present	Disorder absent	F	*p*
Depression	**3.741 (1.037)**	**1.389 (0.676)**	**7.650**	**0.006**	**3.132 (1.132)**	**1.238 (0.713)**	**3.962**	**0.047**
Anxiety disorders								
Panic disorder	3.602 (1.373)	2.318 (0.779)	1.071	0.301	2.427 (1.498)	2.123 (0.834)	0.047	0.829
Agoraphobia	1.997 (0.992)	2.791 (0.783)	1.122	0.290	1.739 (1.020)	2.385 (0.847)	0.712	0.399
OCD	3.117 (1.939)	2.483 (0.771)	0.127	0.721	2.816 (1.966)	2.168 (0.818)	0.132	0.716
GAD	3.052 (1.663)	2.479 (0.774)	0.143	0.706	3.126 (1.942)	2.155 (0.820)	0.291	0.590
Social phobia	2.347 (0.947)	2.548 (0.800)	0.095	0.758	1.865 (1.005)	2.255 (0.841)	0.327	0.568
Any anxiety disorder	2.498 (0.832)	2.632 (0.768)	0.080	0.777	2.007 (0.890)	2.362 (0.820)	0.500	0.480
Suicidal behaviors								
Suicidal ideation	1.421 (1.140)	2.755 (0.778)	1.831	0.176	0.522 (1.237)	2.492 (0.835)	3.205	0.074
Suicide attempt	4.266 (2.385)	2.527 (0.767)	0.567	0.452	5.703 (2.906)	2.069 (0.823)	1.592	0.207

Note: All mean BMI change scores are estimated marginal means.

AA1: Analysis adjusted for sociodemographics (age, gender, education, race, marital status) and other past-year psychiatric disorders at wave 3.

AA2: Analysis adjusted for sociodemographics (age, gender, education, race, marital status), other past-year psychiatric disorders at wave 3, and physical health conditions at wave 3.

Table 10. Association between wave 3 past-year mental health variables and respondents' change in BMI between waves 3 and 4, adjusted for psychiatric comorbidity and physical health, by gender.

	Men				Women			
	Mean BMI change score (SE)				Mean BMI change score (SE)			
Disorder	Disorder present	Disorder absent	F	*p*	Disorder present	Disorder absent	F	*p*
Depression	---	---	---	---	3.459 (1.410)	1.544 (1.009)	2.996	0.084
Anxiety disorders								
Panic disorder	---	---	---	---	2.764 (1.734)	2.438 (1.112)	0.046	0.831
Agoraphobia	1.834 (2.126)	1.541 (1.632)	0.039	0.843	1.933 (1.312)	2.785 (1.127)	0.837	0.361
OCD	---	---	---	---	3.061 (2.218)	2.488 (1.095)	0.089	0.766
GAD	---	---	---	---	2.774 (2.387)	2.492 (1.098)	0.017	0.897
Social phobia	0.926 (1.975)	2.108 (1.701)	1.318	0.252	2.534 (1.328)	2.419 (1.121)	0.016	0.899
Any anxiety disorder	1.077 (1.768)	2.043 (1.535)	1.320	0.251	2.462 (1.172)	2.541 (1.106)	0.015	0.901
Suicidal behaviors								
Suicidal ideation	---	---	---	---	**0.399 (1.538)**	**2.941 (1.113)**	**3.977**	**0.047**
Suicide attempt	---	---	---	---	---	---	---	---

Note: All analyses adjusted for sociodemographics (age, education, race, marital status), other past-year psychiatric disorders at wave 3, and physical health conditions at wave 3. All mean BMI change scores are estimated marginal means.
--- Statistic could not be calculated due to small cell sizes.