THE INFLUENCE OF DEPRESSION ON OVERWEIGHT AND OBESE WOMEN: A SECONDARY DATA ANALYSIS OF THE RENO DIET-HEART STUDY

by

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Abstract

The purpose of this study was to investigate if baseline measures of depression, gender, weight group, and age in overweight and obese women predicted weight change over five years as compared to men. A secondary purpose of this study was to examine if depressive symptoms were positively correlated with weight change over five years. This study was a secondary analysis of archival data from the RENO Diet-Heart study (St. Jeor, 1997). The current study focused on women but used men as a comparison group. To screen for a homogeneous healthy sample, potential subjects were excluded if they reported a diagnosis of depression during the last five years, suffered from depressive symptoms within the last five years, or took antidepressants. The predictor variables for this study included depression scores (using the Center for Epidemiologic Studies-Depression scale, or CES-D) (Radloff, 1997), gender, weight group (normal weight, overweight, and obese based on current standards for body mass index), and age upon study entry. Obese subjects were in the minority for both females (26.9%) and males (23.9%). A hierarchical linear mixed model method was used to analyze data to predict weight change over time. Results indicated that depressive symptoms significantly contributed to weight gain over five years among obese women and obese men. In addition, twenty year-old women and men gained the most weight over time despite subclinical CES-D scores. Future research is needed to examine if depressive symptoms are related to obesity, especially among younger people.

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Dedications

For my father, who instilled in me the desire to continually learn and set new goals for myself, the fortitude to persevere, the strength to overcome adversity, the desire to teach others, and to be humble with the gifts God has given me.

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CHAPTER I

Background and Purpose

There is a paucity of research that examines obese individuals with mood disorders (Malhotra & McElroy, 2002). There is empirical evidence in the literature that women suffer from depression more than men (American Psychiatric Association, 2000; Ernst & Angst, 1985) both in the United States and other countries (Weissman, Bland, Joyce, Newman, Wells, & Wittchen, 1993). Women also experience earlier onsets of depression (American Psychiatric Association, 2000; Frank, Carpenter, & Kupfer, 1988; Kessler, McGonagle, Nelson, Hughes, Swartz, & Blazer, 1994; Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Kessler et al., 1994; Kornstein, 1997; Kornstein & McEnany, 2000; National Institute for Mental Health (NIMH), 2002; Nolen-Hoeksema, 1987; Parker & Brown, 2000; Schatzberg, Cole, & DeBattista, 1997; Weissman, Bland, Joyce, Newman, Wells, & Wittchen, 1993; Weissman & Klerman, 1977), have a tendency to become vegetative and overeat (Ernst & Angst; Frank, Carpenter, & Kupfer; Kornstein; Kornstein & McEnany), and experience more frequent exacerbations (Kessler, McGonagle, Nelson, Hughes, Swartz, & Blazer). Therefore, some women may gain weight as a symptom of depression, especially combined with excessive sleeping that underlies atypical depression (American Psychiatric Association, 2000).

The literature is replete with theories on causal factors of both obesity and depression. In general, few individuals are screened for depression in primary care (American Psychological Association, 2002a; Arnau, Meagher, Norris, & Bramson,

2002; Kettl, 2002; Parker & Brown, 2000; United States Preventive Task Force, 2002). National Institutes of Health recommend and support screening all clients for depression in primary care, yet few empirical studies were found that examined the incidence of depression among overweight and obese women, and how levels of depressive symptoms affects weight loss (Carpenter, Hasin, Allison, & Faith, 2000; Ernst & Angst, 1992; Frank, Carpenter, & Kupfer, 1988; Klesges, Klem, & Klesges, 1992; Nutzinger, Cayiroglu, Sachs, & Zapotoczky, 1985; Ross, 1994). There is convincing evidence that a subpopulation of overweight and obese women seeking treatment for obesity may suffer from underlying depressive symptoms that may cause weight gain over time.

This study focused on depression and obesity as major health problems in the United States. Experts agree that multiple causative factors are involved in the development of both obesity and depression, and depressive symptoms may contribute to weight gain over time. If a relationship exists between untreated depressive symptoms and weight gain, treatment guidelines for obese women could be impacted to consider obesity and depression as related co-morbidities and could contribute to existing knowledge about women, depression, and obesity.

Purpose and Research Questions

The purpose of this study was to investigate if baseline measures of depressive symptoms, gender, weight group, and age in overweight and obese women predicted weight change as compared to men. A secondary purpose of this study was to examine if depressive symptoms were positively correlated with weight change. This

study, a secondary analysis of archival data from the RENO Diet-Heart study (St. Jeor, 1997), focused on overweight and obese women, but compared women with men for possible gender differences.

Selected Review of the Literature

Healthy People 2010 Goals

Healthy People 2010 is a public health disease prevention and health promotion program for United States citizens (USDHHS, 2000). This program was created in 1990 (titled Healthy People 2000) by national healthcare experts to promote practitioner supported health promotion, disease prevention, and access to care for all citizens living in the United States. The goals of Healthy People were written to guide practitioners to create a healthier and more productive population and to decrease health disparities. For example, the mental health goal in Healthy People 2010 is to "improve mental health and ensure access to appropriate, quality mental health services" (p. 60). During 1997, only 23% of all depressed individuals received treatment for their depression (USDHHS). Individuals who suffer from depression cannot fulfill their responsibility to themselves, others, and to society. Women who are depressed and untreated may experience serious emotional and financial consequences, especially when women are primary caretakers for children, hold the financial responsibility for their family, or are single, with or without the responsibility of children. Depression also co-exists with and compounds other physical illnesses such as diabetes, cancer, heart disease, and renal disease (Bray, 1992; Khaodiar, McCowen, & Blackburn, 1999; NHLBI, 2000; Preskorn, 1999;

Weiser, Frishman, Michaelson, & Abdeen, 1997; USDHHS, 2000). The United States Preventive Services Task Force (2002) recently stated that primary care providers need to screen all of adult clients for depression. Their conclusion was based on a 6-year outcome study that found clients fared better when screening preceded follow-up care for depression.

Turning to obesity, the overall goal for Healthy People 2010 is to decrease the prevalence of obesity, and to "promote health and reduce chronic disease associated with diet and weight" (USDHHS, p. 60). Obesity is a major concern as baby boomers age, as they comprise the bulk of the population (Kuczmarski, Flegal, Campbell, & Johnson, 1994). The rate of obesity is highest for both men and women between the ages of 45 to 64 years of age (Schoenborn, Adams, & Barnes, 2002). Obesity rates have increased to 30.5% among adults (Flegal, Carroll, Ogden, & Johnson, 2002). The goal of Healthy People 2010 is to decrease the proportion of obese adults to less than 15%, half of what it is today.

Perhaps the strongest persuasion for researchers to examine obesity and depression was stated by Carpenter, Hasin, Allison, and Faith (2000), who stated that

> "the dearth of data is cause for concern given the public health significance of obesity and its potential relation to depression. Overweight and obesity, taken together, are very prevalent in the population, and results suggest that obesity is associated with psychiatric comorbidities. Given past epidemiological trends, these prevalence rates are expected to continue to rise" (p. 254).

It is plausible that depression affects weight change. Both obesity and depression have many heterogeneous causative factors that may interact synergistically to cause weight change in overweight and obese women. In the following sections, depression and obesity will be examined, discussed separately, and in combination.

Depression

At least 19 million adults suffer from depression, a major contributor to disabilities (USDHHS, 2000). Individuals who experience depression may present with symptoms ranging from minor somatic complaints (feeling down, blue, no energy) to complete withdrawal from society and/or suicide (Preskorn, 1999). Generally, depression is defined as feeling sad and having a lack of pleasure in activities, a change in sleep habits, loss of energy, difficulty concentrating, and feeling worthless (NIMH, 2002). Specifically, depression is divided into different subtypes, as described in the Diagnostic and Statistical Manual of Mental Disorders IV, Text Revision (DSM-IV-TR; American Psychiatric Association, 2000). For the purpose of this study, only three depression subtypes (major depression episodes, major depressive disorders, and atypical depression) will be considered and are described in the following paragraphs. In addition, to provide background about depression, diagnostic criteria, physiological theories and treatments, psychosocial theories and treatments, along with instruments that measure depression and research on gender differences will be discussed and summarized. Although depression in

comorbid medical conditions is of great importance, this investigation will focus mainly on obesity and less on other comorbid conditions.

DSM-IV-TR diagnosis of depression.

According to the Diagnostic and Statistical Manual-IV-Test Revision (American Psychiatric Association, 2000), depressed individuals can have either a depressive episode, a major depressive disorder, or a major depressive disorder with atypical features. Individuals who experience a depressive "episode" experience a depressive mood change along with a minimum of four other symptoms including (a) a change in weight, appetite, sleep, or psychomotor activity, (b) difficulty making decisions and concentrating, (c) feelings of guilt and worthlessness, (d) generating ideas about committing suicide, attempts, or threats, and (e) a general lack of energy. Other symptoms of depressive episodes are when individuals decrease their involvement in activities of daily living that includes work and socializing with others.

In contrast, a major depressive disorder diagnosis is given for more than one major depressive episode without evidence of mania, hypomania, substance abuse, other medical conditions, delusions, psychoses, or schizophrenia. The prevalence of major depressive disorder is higher in women than in men, ranging from 10%-25% in women and 5%-12% in men (American Psychological Association, 2000a). Approximately one-third of all major depressive episodes develop into a chronic form of depression. Suicide can result from untreated depression (Preskorn, 1999). Up to 35,000 individuals per year who are depressed commit suicide (Preskorn). The

American Psychiatric Association (2000) stated that a "major depressive disorder is from one to three times more common among first degree relatives of persons with this order than among the general population" (p. 373). This is an important piece of information for the practitioner to obtain from clients.

Atypical major depressive disorder is characterized as having two out of four of the following list of symptoms, such as (a) significant weight gain or increased appetite, (b) sleeping more than ten hours per day, (c) experiencing a heavy feeling in legs or arms, and (d) sensitivity to rejection from others, including the diagnosis of major depressive disorder (American Psychiatric Association, 2000; Schatzberg, Cole, & DeBattista, 1997). This disorder is postulated to begin in childhood and progress through adult life. Atypical depression is up to three times more common in women (American Psychiatric Association, 2000; Asnis, McGinn, & Sanderson, 1995), and "the predictive value of atypical features is less clear with newer treatments, such as selective serotonin reuptake inhibitors or interpersonal or cognitive psychotherapies" (American Psychological Association, 2000, p. 421).

Chronic medical illness also contributes to depressive symptomatology, as illness interferes with individual goal achievement and role functioning (Aneshensel, Frerichs, & Huba, 1984). Self-esteem is decreased and normal healthy habits are limited (Frederick, Frerichs, & Clark, 1988). Preskorn (1999) ranked (from least to most severe) eight chronic medical illnesses that are linked to depression, including non-insulin dependent diabetes mellitus (NIDDM), myocardial infarction, Parkinson's

disease, epilepsy, chronic pain, kidney disease, strokes, and terminal solid-tumor cancers (p. 182).

There are numerous theories of depression that include both physiological and psychological causes and predisposing factors. The most prevalent biologic theories are the biogenic amine theory and the serotonin deficit theory, discussed in the next section.

Physiological theories.

Two prevalent physiological theories of depression are the biogenic amine theory and serotonin deficit theory. The biogenic amine theory was developed from observing client's responses to iproniazid, a medication for tuberculosis that proved to be an effective monoamine oxidase inhibitor (MAOI). Most clients with tuberculosis responded favorably to this drug, and experienced a remission of their depression (Bunney & Davis, 1965; Healy, 2000; Jones & Blackburn, 2002; Korn, 2002; Parker & Brown, 2000; Schatzberg, Cole, & DeBattista, 1997; Schildkraut, 1995). In animal studies, researchers found that monoamine oxidase degraded both norepinephrine and serotonin in the synaptic cleft, and these were considered "cornerstones of the biogenic amine theories of depression" (p. 32). By interfering with enzyme degradation, depression improved, hence the monoamine theory was developed (Korn).

The most prevalent theories today implicate a serotonin deficit as one cause of depression; however, the precise relationship between serotonin and depression remains unclear. There are several competing opinions as to how serotonin reuptake

inhibition occurs. Serotonin is released pre-synaptically, and during this process, some of the released serotonin is taken back into the nerve cell that released the serotonin, hence the term "reuptake". Goodwin (1996) stated that a group of drugs called selective serotonin reuptake inhibitors (or SSRIs) blocked reuptake of the neurotransmitter serotonin, and thus decreased depression. Weiser, Frishman, Michaelson, and Abdeen (1997) stated that SSRIs decreased depression by either stimulating the serotonergic system and/or blocking reuptake. Raap and Van de Kar (1999) and Westenberg (1999) stated that the reuptake process activated neuronal autoreceptors. These autoreceptors would sense the increase in serotonin levels in the synaptic cleft, and, through negative feedback, stimulate the neuron to produce less serotonin. Raap & Van de Kar also stated that after beginning SSRI treatment, the body adapts to the neural change within three weeks, and the brain decreases serotonin production as reuptake is inhibited. Schatzberg (2000) stated that instead of SSRIs acting on specific neurons, serotonin reuptake inhibition may be the first step in a chain of neurobiochemical events that result in antidepressant and/or antianxiety effects. Healy (2000) suggested that SSRIs may modify receptors and change the transmitter signal process. Obviously, researchers are still unclear about the details of how SSRIs inhibit reuptake. What researchers currently know from animal and human studies is that elevating serotonin in the synaptic cleft (by inhibiting serotonin reuptake) decreases depression (Cooper, Bloom, & Roth, 1996; Moller, 2000; Parker & Brown, 2000; Schatzberg, 2000; Schatzberg, Cole, & DeBattista, 1997).

Another theory linked to serotonin concerns a decrease in circulating plasma levels of L-tryptophan, the amino acid necessary for serotonin genesis (Cooper, Bloom, & Roth, 1996; Christensen, 1999). Tryptophan is obtained through eating food high in protein and carbohydrates, and "brain levels of tryptophan will be determined not only by the plasma concentration of tryptophan but also by plasma concentrations of competing neutral amino acids. Thus, dietary proteins and carbohydrate content can specifically influence brain tryptophan and serotonin levels" (Cooper, Bloom, & Roth, p. 354). Christensen suggested that tryptophan may have some antidepressant activity, or that tryptophan may increase the effects of antidepressant drugs.

Other proposed theories postulate alterations within the serotonin transporter gene (Cooper, Bloom, & Roth, 1996; Schatzberg, Cole, & De Battista, 1997), neurotransmitter dysfunction (Moller, 2000; Parker & Brown, 2000; Schatzberg, 2000), or a possible genetic cause for depression, as the risk for developing depression is increased among first-degree relatives diagnosed with depression. Other theorists postulate other causes that include hormonal changes, stress, trauma, and ruminative thinking (American Psychiatric Association, 2000). Stahl (2002) postulated that stressful events in the environment may influence multiple genes that act either alone or together to cause depression.

Medical treatments for depression.

In the 1930's, early treatments for depression included electroshock therapy (Matheney & Topalis, 1953) that resulted in memory disruption that freed the client

of depressive thoughts. Another early treatment was psychosurgery. Prefrontal lobotomies were performed to cut off communication with the thalamus, where emotions were thought to be centered. Somewhat less radical treatments were narcosis and narcoanalysis, used since the beginning days of psychiatry. Sodium pentothal or sodium amytal was given to render noncommunicative clients to become relaxed and uninhibited when talking with their therapist.

Pharmacotherapy.

Antidepressants were serendipitously discovered during the 1950's, when monoamine oxidase inhibitors (MAOIs) that were used to treat tuberculosis had an elevating effect on depressed mood (Goodwin, 1996; Healy, 2000; Moller, 2000; Schatzberg, Cole, & DeBattista, 1997; Westenberg, 1999). The first MAOIs developed are rarely prescribed today (Jones & Blackburn, 2002; Moller) due to their numerous dietary restrictions (e.g., no fermented foods such as cheese, wine, beer, yeast, smoked meats, and Chinese food) and physical side effects such as severe orthostatic hypotension (drop in blood pressure) upon rising from a sitting or lying position, constipation, weight gain, hypertensive crises, insomnia, impotence, and involuntary twitching (Schatzberg, Cole, & DeBattista).

During the 1960's, tricyclic antidepressants (TCAs) became the standard treatment for depressed clients who could not tolerate the side effects of the MAOIs. TCAs are equally effective as contemporary selective serotonin reuptake inhibitors (SSRIs), but TCAs have many side effects such as dry mouth (anticholinergic effects), impaired judgment and memory, hypotension, sleepiness (from stimulating

the histamine receptors), and weight gain that are unpleasant side effects that decreased patient compliance with their medication regimens (Berken, Weinstein, & Stern, 1984; Hirschfeld, 2000; Peretti, Judge, & Hindmarch, 2000). Overdosage can be lethal (Schatzberg, Cole, & De Battista).

Selective serotonin reuptake inhibitors (SSRIs) were developed in the late 1980's. The first SSRI developed was fluoxetine, or Prozac®. Coincidentally, fluoxetine was first developed for weight management, and researchers discovered that it elevated mood (Goldstein, 1992; Michelson et al., 1999; Orzack, Friedman, & Marby, 1990; Ward, Comer, Haney, Fischman, & Foltin, 1999). The goal of pharmacologic therapy is to correct serotonin dysregulation (Parker & Brown, 2000). All of the selective serotonin reuptake inhibitors (SSRIs) inhibit serotonin reuptake to some degree and exhibit reuptake inhibition within minutes, but antidepressant effects take as long as two weeks to exert their effects (Preskorn, 1999). This delayed response indicates that immediate reuptake inhibition in itself does not exert antidepressant effects, but researchers hypothesize that over time, serotonergic neuronal adaptation results in an antidepressant effect (Moret & Briley, 2000; Raap & Van de Kar, 1999; Schatzberg, Cole, & De Battista, 1997; Westenberg, 1999).

Prozac®, Paxil®, Lexapro®, and Zoloft® are lipid-soluble selective serotonin reuptake inhibitors (SSRIs) that are extensively bound to tissues (DeVane, 2000), including tissue in the central nervous system. Prescribing SSRIs to treat depression is popular due to the low incidence of side effects (Jones & Blackburn, 2002; Mendlewicz & Lecrubier, 2000; Preskorn, 1999; Schatzberg, 2000; Schatzberg, Cole,

& DeBattista, 1997) and lack of toxic effects at high doses (Rasmussen & Brosen, 2000). Side effects that may occur are usually transient (nausea, vomiting, and gastrointestinal distress); however, many individuals experience a loss of sexual function, especially with fluoxetine (Moller, 2000; Preskorn; Peretti, Judge, & Hindmarch,2000). Other potential side effects include insomnia, agitation, headache, and tremors (Schatzberg, Cole, & DeBattista). Healy (2000) stated that emotional blunting may be present in approximately 25% of patients treated with SSRIs. Some SSRIs, however, cause weight gain. Individuals who are already overweight will be less likely to comply with a drug that makes them gain more weight. Harvey and Bouwer (2000) found that Celexa® caused an increase in weight gain, while Fava (2000) found that individuals who were prescribed Paxil® and Zoloft® gained weight. Prozac® (Fava, 2000; Preskorn, 1999) does not cause weight gain, and new information on Lexapro® (2003) stated that no weight gain occurred among subjects in clinical trials.

In some cases, discontinuing an SSRI may suddenly produce uncomfortable side effects. Haddad (1998) listed common discontinuation effects such as dizziness, nausea, headache, and lethargy, and also emphasized the importance of educating patients to the possible side effects of medication withdrawal so that patients will be cognizant of potential side effects. Preskorn (1999) used the mnemonic FLUSH to assess for problems associated with drug discontinuation. FLUSH stands for "flulike [symptoms], lightheadedness, uneasiness, sleep and sensory disturbances, and headaches" (p. 101). Preskorn also stated that "the shorter the half-life, the more

likely the drug will wash out before the brain has had an opportunity to re-equilibrate (e.g., upregulating receptors), and hence, the more likely that withdrawal symptoms will occur after drug discontinuation" (p. 101). Another possible discontinuation symptom is rebound depression (Schatzberg, Cole, & DeBattista, 1997). The reason for this is unknown. Rebound depression may be more likely to occur with SSRIs that have shorter half-lives, whereas fluoxetine has the longest half-life (Preskorn). Fluoxetine may be discontinued abruptly if necessary because of its longer half-life, around 14 days.

Depression also influences whether individuals will remain compliant with medical interventions (DiMatteo, Lepper, & Croghan, 2000). Depressed clients have three times greater odds of becoming noncompliant because they feel hopeless and pessimistic that a prescribed pharmacologic intervention will work. Most patients will comply with SSRI treatment because of the minimal side effects associated with SSRIs (Roose, 1999). Long-term therapy is recommended for individuals who suffer from chronic depression (Preskorn, 1999).

Psychosocial theories and treatments of depression.

In general, psychosocial theories of depression focus on behavioral, cognitive, and cognitive-behavioral theories and strategies that have been developed over the years (Schwartz and Schwartz, 1993). Behavioral theories focus on how individuals learn, interpret and respond to situations in their environment, and their history of how they were reinforced for their behaviors. Cognitive theories postulate how individuals learn, interpret and respond to their environment. Cognitive-behavioral

theories include components from both the cognitive and behavioral theories (Wilson, 2000).

Behaviorists focus on "(1) a psychological model of human behavior that differs fundamentally from the traditional psychodynamic model [e.g., underlying unconscious reasons for behavior]" (Wilson, 2000). Behavior therapy is based on a learning or education model that uses short-term therapy to correct behaviors. In cognitive-behavioral therapy, the therapist assists the depressed client to self-monitor daily activities such as recording pleasant activities, mastering difficult skills, and to develop social skills with others (Wilson).

Beck (1976) developed a cognitive theory of depression and therapy. Beck and Weisshaar (2000) stated that many factors contribute to depression,

> "such as hereditary susceptibility, diseases that cause persistent neurochemical abnormalities, developmental traumas leading to specific cognitive vulnerabilities, inadequate personal experiences that fail to provide appropriate coping skills, and counterproductive cognitive patterns, such as unrealistic goals, assumptions, or imperatives. Physical disease, severe and acute stress, and chronic stress are also precipitating factors" (p. 250).

Depressed individuals "respond to life events through a combination of cognitive, affective, motivational, and behavioral responses...based in human evolution and individual learning history" (Beck & Weishaar, p. 241). Beck introduced a term, "schema" that subsumes individual self-perceptions and perceptions about others, memories, goals, fantasies, expectations, and learning experiences. In general,

depressed individuals have a negative outlook on the world, themselves, and the future (the "cognitive triad"; Beck & Weisshaar, p. 250). When responding to another, depressed individuals tend to automatically activate previously learned behaviors based on their faulty automatic interpretations of what they envision reality to be. The purpose of cognitive therapy is to correct the depressed individual's misinterpretations, automatic thoughts and behaviors, by exploring and using reasoning to clarify how the individual processes information. Cognitive therapists use behavioral techniques such as homework, role playing, activity scheduling, and monitoring their personal thoughts and feelings to provide insight into their moods (Pendleton, Goodrick, Poston, Reeves, & Foreyt, 2002). Persons, Davidson, and Tompkins (2,001) reviewed randomized control experiments and concluded that cognitive-behavioral therapy is efficacious, cost-effective, and has demonstrated positive outcomes as compared to other treatment modalities, including medication therapy.

Research and measurement of depression.

Many instruments are available to assess depression that have demonstrated reliability and validity over time (Table 1). Different researchers use different measures based on the purpose of their inquiry. Some instruments take hours to complete, such as the Structured Clinical Interview for Depression, or SCID (Spitzer, Williams, Gibbon, & First, 1992); however, shorter questionnaires are available (Table 1). The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, and Erbaugh, 1961) is a 21-item self-report instrument that measures depressive behaviors

during the previous two weeks, and also reflects changes in depression during therapy. The Center for Epidemiologic Studies on Depression (CES-D; Radloff, 1977) measures depressive symptoms one would experience during the previous week, and was designed for use in a community population. In addition, the CES-D was designed to be administered by both experts and those without mental health expertise. The CES-D does not differentiate between types of depression nor should it be used to diagnose depression (Miller & Harrington, 1997; Radloff, 1977). The 20-item scale was developed from other established depression scales, and has demonstrated reliability and validity (Table 2).

The Hamilton Depression Scale (Hamilton, 1960) is a 21-item questionnaire developed to measure depression severity in individuals during therapy. The Zung Self-Depression Rating Scale (Zung SDS; Zung, 2000) is 20-item self-report questionnaire that measures depression in individuals diagnosed with depression.

Gender differences in depression.

During childhood, boys and girls demonstrate similar occurrences of depressive episodes (excluding mania) until around the age of ten. Kessler, McGonagle, Swartz, Blazer, and Nelson (1993) found that depressive episodes began to differentiate genders during adolescence. From late adolescence until late middle age, depressed females outnumber depressed males. Interestingly, this time roughly corresponds to women's reproductive years (Kornstein & McEnany, 2000). Kessler et al. (1994) examined data from the National Comorbidity Survey (NCS) and

concluded that women "have a higher risk of first onset of depression throughout the 15-54 age range" (p. 23).

There is unanimous agreement that the prevalence of mood disorders (excluding mania) is approximately twice the rate in women as it is in men in the United States and other countries (American Psychiatric Association, 2000; Anthony & Petronis, 1991; Blazer, Kessler, McGonagle, & Swartz, 1994; Blehar & Oren, 1995; Frank, Carpenter, & Kupfer, 1988; Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Kessler, McGonagle, Nelson, Hughes, Swartz, & Blazer, 1994; Kessler et al., 1994; Kornstein, 1997; Kornstein & McEnany, 2000; National Institute for Mental Health (NIMH; 2002); Nolen-Hoeksema, 1987; Parker & Brown, 2000; Schatzberg, Cole, & DeBattista, 1997; Weissman, Bland, Joyce, Newman, Wells, & Wittchen, 1993; Weissman & Klerman, 1977). The National Institute for Mental Health (2002) stated that 12% of women (compared to 7% of men) suffer from depression, and at least 20% of the total female population has experienced a treatable depressive episode. Women report more symptoms of depression than men, score higher on instruments that measure depression (Frank, Carpenter, & Kupfer; Nolen-Hoeksema, 1987), have longer periods of symptoms, and experience chronic re-occurrences of depressive episodes (Amenson & Lewisohn, 1981; Ernst & Angst, 1992; Kessler et al., 1994; Nolen-Hoeksema, 1987). In addition, married women have a higher prevalence of depression (American Psychological Association, 2002b). Goodwin & Blehar (1993) stated "gender is but a proxy term for a complex of psychosocial and biological variables" (p. 75) that contribute to depression.

Coryell, Endicott, and Keller (1992) found in 965 subjects recruited for a prospective, six year study that examined socio-economic status and the development of depression, that females were most likely to complete the six year program (54.7% versus 46% of males; $\chi^2 = 6.8$, $df = 1$, $p = .009$). However, 15% of female subjects ($n = 81$) who had no history of mental illness "developed major depression during the 6-year follow-up" (p. 120), twice the rate as in males. The researchers also found that living on a farm, higher education, and divorce and/or separation increased the risk of major depression among females who had no history of mental illness.

There are other gender issues involved in depression. Gender does not cause depression by itself, but combined with social role problems women face (e.g., discrimination, single parenting), lower socio-economic status (SES), psychological problems, environmental and developmental factors, a family history of depression, dieting failures, loss of self-esteem, and reproductive/hormonal issues may increase women's vulnerability to develop depression (Blehar & Oren, 1995; Foreyt, Brunner, Goodrick, Cutter, Brownell, & St. Jeor, 1995; Nolen-Hoeskema, 1987; Weiser, Frishman, Michaelson, & Abdeen, 1997).

There is empirical evidence to support that women suffer from depressive symptoms more than men, have earlier onsets of depression, and face psychosocial issues that put them at an increased risk for developing depression. In fact, women may gain weight or fail at losing weight as a symptom of underlying depression that supports the importance for practitioners to screen clients for depressive symptoms

not only in general, but for those who are overweight or obese, or individuals who have difficulty in losing weight.

Obesity

Obesity in the United States has reached epidemic proportions (Brownell, 2002; Drewnowski, 2002; Hill, 2002b; Mokdad, Serdula, Dietz, Bowman, Marks, & Koplan, 1999). According to current statistics (Flegal, Carroll, Ogden, & Johnson, 2002), the obesity prevalence increased from 22.9% (1988-1994 NHANES III data) to 30.5% (NHANES data collected on 4115 adults during 1999-2000). There were no ethnic group differences among men, but among women, African-American women were more overweight and obese than Caucasian, non-Hispanic women. The investigators found that in general, increases in overweight and obesity occurred in both genders, in all ages, and among all ethnic groups. The National Center for Health Statistics reported (2002) that more women are obese (33%) than men (28%), and among women, African-Americans have a higher prevalence of obesity (50%) as compared to Mexican-American women (40%) and non-Hispanic whites (30%).

Obesity is described as excess adipose tissue that results from excess calories, high fat foods, reduced exercise, genetic predisposition, and metabolic problems (Goodrick & Foreyt, 1991; Guyton, 1991; Keller & Thomas, 1995; McCance & Huether, 1994; Weiser, Frishman, Michaelson, & Abdeen, 1997). The average weight for men is now 180 pounds, and the average height for men is 5 foot 9 inches. Computing body mass index, the BMI for "normal" men is 26.6, considered in the "overweight" category. The average weight for women is now 152 pounds, and the

average height is 5 foot 3 3/4 inches. Computing a body mass index (using the formula of weight/kg divided by height/m^2; NHLBI, 2000), the BMI for "normal" women is 26.1, again in the overweight" range. Overweight individuals have a BMI between 25.0 and 29.9, whereas obese individuals have a BMI greater than 30 (NHLBI, 2000).

Approximately 280,000 individuals die each year due to obesity and obesity-related conditions (Manson, Skerrett, & Willett, 2002). Martin, Robinson, and Moore (2000) stated that as body mass index (BMI) increases, so do medical expenditures. Grundy, Blackburn, Higgins, Lauer, Perri, & Ryan (1999) found that direct costs of a total lack of physical activity were around "24 billion dollars, or 2.4% of the U. S. health care expenditures. Direct costs for obesity defined as a BMI greater than 30 kg/m^2, in 1995 dollars, totals 70 billion dollars...overall, the direct costs of inactivity and obesity are estimated to consume some 9.4% of national health care expenditures in the United States" (p. S503).

Experts and researchers in the area of obesity agree that obesity is a chronic disorder that has many causative factors (Brownell & Wadden, 1992; Devlin, Yanovski, & Wilson, 2000; Friedman & Brownell, 1995; Istvan, Zavela, & Weidner, 1992; Kornstein, 1997; Lissner, 1993; National Task Force on the Prevention and Treatment of Obesity, 1996; Poston & Foreyt, 1999; Poston & Foreyt, 2000; Ross, 1994; Schlundt et al., 1991; Stunkard, 1996). To understand the complexities of obesity, one must understand the pathophysiology and theories of obesity,

comorbidities, psychosocial and social issues, physical exercise, obesity treatments, measuring obesity, and gender issues will be presented.

Pathophysiology and theories of obesity.

There are several competing theories about how obesity develops. The current most popular theory involves energy intake and output; however, the role of genetics in obesity will be discussed in this section.

In general, body weight is influenced by a feedback mechanism in the brain controlled by energy input and output (Pijl & Meinders, 1996; Ravussin & Swinburn, 1992). A chronic "positive" energy imbalance between an individual's energy intake and output results in obesity (Bray, 2002; Ravussin, 2002; Stunkard, 1996), caused by eating too much and exercising too little (Blundell, 2002). Food and adipose tissue send afferent signals to the hypothalamus about the body's energy needs, and in return, these efferent signals alter intake. Many different regulatory systems within the human body operate through a feedback mechanism designed to regulate energy balance and weight (Leibowitz, 2002), including numerous hormones (e.g., insulin, leptin, estrogen), peptides (e.g., cholecystokinin), steroids (estrogen and progesterone), and neurotransmitters (norepinephrine, dopamine, and serotonin) that stimulate the adrenal release of glucocorticoids and mineralocorticoids. The feedback system is initiated by ingesting food. Satiety signals inhibit eating, and are generated by not only the amount and quality of the food, but the act of eating.

Approximately 70% of our energy expenditure is accounted for by our resting metabolic rate (Ravussin, 2002), and the act of eating and digesting food only

accounts for about 10% of our total energy expenditure per day. Extra food that is eaten is stored as triglycerides in fat cells that become larger over time (Bray, 2002), and "big fat cells secrete more peptides and turn over more fatty acids" (p. 385) that lead to the development of gallstones (cholelithiasis), some cancers, diabetes, and arterial plaque formation. Women with breast cancer who gain or remain overweight or obese have less chance of surviving and an increased chance of recurrence (Krenkel & St. Jeor, 1995).

Another theory is the leptin theory. A gene has been identified, the obesity (ob) gene that is produced by fat cells (adipocytes) that bind with specific proteins when entering the circulatory system. These fat cells secrete a hormone called leptin (Considine et al., 1996), a peptide protein that belongs to the cytokine family (e.g., interleukin 6). Leptin also regulates insulin secretion, maintains energy stores in skeletal muscle and fat cells, and protects individuals from starvation. In addition, women have higher leptin levels than men (Campfield, 2002) that may explain why more women are overweight (National Center for Health Statistics, 2002). When an individual loses weight, serum leptin levels decrease. In a human study conducted by Considine et al. (1996) found that higher concentrations of leptin were positively correlated with a higher amount of body fat (r=.85, p<.001) (p. 292). They also found that hypertrophic adipose cells produced twice the amount of leptin as normal cells, and that a 10% weight loss produced a 53% reduction in serum leptin.

Hill (2002a) asserted that human energy balance is based on a "functional phenotype" (p. 68) that includes how an individual maintains energy balance and

weight in any type of environment. The functional phenotype subsumes the "metabolic" and "behavioral" phenotypes. The metabolic phenotype describes the individual's metabolism, and how internal signals effect energy balance. The brain interprets the need for eating and physical activity (energy intake and output) to keep an individual in optimal physical condition, while recognizing an instinctual behavior that allows individuals to stop eating when satiated, except for sweet food. The behavioral phenotype includes instinctive behaviors necessary for survival, but behaviors can be changed or modified when energy intake exceeds energy output. In other words, obesity may be caused by not only genetic predisposition but metabolic processes, behaviors, emotions, and interactions with the environment.

Physiological factors that contribute to developing obesity are partially based on genetics, including inheriting a possible genetic predisposition for excess fat cells and a higher hypothalamic set-point that increases appetite (Brownell & Wadden, 1992; Foreyt & Goodrick, 1995; Lichtman et al., 1992; McCance & Huether, 1994). Animal and twin studies support that genes have a role in developing obesity, and Poston and Foreyt (1999) stated that "genes may provide us with susceptibilities or vulnerabilities for obesity rather than acting as simplistic causal factors" (p. 202).

Human obesity may result "not from defective leptin but from a defect in the leptin receptor or in the chemical signals triggered by the receptor" (Weiser, Frishman, Michaelson, & Abdeen, 1997), or leptin resistance "since leptin is unable to generate an adequate response when its receptor is occupied" (Stahl, 1998, p. 448). Devlin, Yanovski, and Wilson (2000) emphasized that the genetics underlying obesity

are complex, that no one simple cause will be found, and "basic research findings identifying pathways that regulate food intake, adipocyte differentiation, and energy expenditure are likely to assist in the development of effective strategies for both prevention and treatment of obesity" (p. 855). Devlin, Yanovski, and Wilson also discussed "behavioral genetics", when individuals prefer a higher fat content in their food choices and individual preference for different forms of exercise behaviors (including no exercise) may be genetically determined. They postulated that if behaviors have genetic origins, they are more resistant to change as opposed to behaviors that are environmentally learned. Bouchard (2002) and Leibel (2002) stated that searching for an obese gene (based on the Mendelian law of genetic inheritance) may be difficult to locate as many genes may be involved and interact in yet an unknown manner. In other words, why and how obesity occurs and develops remains elusive.

Comorbidities caused by obesity.

Obesity causes many comorbid conditions such as cardiovascular disease, dyslipidemia, gallbladder disease, gout, hypertension, insulin resistance, sleep apnea, type 2 diabetes mellitus, and osteoarthritis, (Bray, 1992; Hecker, Kris-Etherton, Zhao, Coval, & St. Jeor, 1999; Khaodiar, McCowen, & Blackburn, 1999; Manson, Skerrett, & Willett, 2002; NHLBI, 2000; Pi-Sunyer, 2002b; Weiser, Frishman, Michaelson, & Abdeen, 1997). Comorbid conditions result in increased time spent away from work, along with increased physician visits due to increased illnesses and disabilities (Wolf, 2002; Wolf & Colditz, 1998). In 1996, Wolf estimated that obesity "cost U. S.

businesses $12.7 billion...including $2.6 billion as a result of mild obesity and $10.1 billion due to moderate-to-severe obesity. Sixty-one percent of these costs were from health insurance expenditures, 19% from paid sick leave, 14% for life insurance expenditures, and 6% for disability insurance costs" (p. 457).

Goldstein (1992) examined research literature (using a meta-analysis technique) and found that individuals who lose 10% of their baseline weight positively influenced their comorbid conditions. Wing and Jeffery (1995) found that losing a modest amount of weight (10-15% of initial body weight) improved blood pressure and serum cholesterol in men (n=68) at eighteen months. They found that women (n=63) took longer to demonstrate improvements in serum cholesterol values and decreased their waist:hip ratio measurements, but demonstrated improved weight maintenance. Oster, Thompson, Edelsberg, Bird, and Colditz (1999) found similar results in both genders (using data obtained from the NHANES III and Framingham studies), that a 10% weight loss resulted in increased lifespan, reduction of comorbid conditions, and monetary savings on medical care. It appears that losing 10% of an individual's baseline weight has marked beneficial effects on improving health and lipid profiles (Goldstein; Hecker, Kris-Etherton, Zhao, Coval, & St. Jeor, 1999; Oster, Thompson, Edelsberg, Bird, & Colditz; Wing & Jeffery).

There is an empirically substantiated relationship between obesity and the development of cardiovascular diseases (Hecker, Kris-Etherton, Zhao, Coval, & St. Jeor, 1999). The NHLBI (2000) stated that obesity and other diseases place individuals at "absolute risk" for mortality (p. 1). Any three of the following risk

factors combined place an individual at an absolute risk for mortality that include decreased physical activity, smoking, hypertension, low levels of high density lipoprotein (HDL-C, or good cholesterol), and high levels of low density lipoprotein (LDL, or bad cholesterol), abnormal fasting glucose results (associated with type II diabetes), males over the age of 45 and females over 55 years, and a family history of cardiovascular disease. Obesity increases cardiac workload, as tissue oxygen demand is increased because there is more tissue to perfuse. When increased cardiac output is combined with hypertension, obese individuals may develop congestive heart failure (CHF; Khaodiar, McCowan, & Blackburn, 1999). In the Nurses' Health Study (Manson et al., 1995), body mass indices were strongly correlated with both cardiovascular and coronary heart disease than deaths attributed to other illnesses in women with BMIs >22.

There is a direct relationship between obesity and hypertension (Anderson & Forsstrom, 1998; Khaodiar, McCowan, & Blackburn, 1999; Mertens & Van Gaal, 2000; Pi-Sunyer, 1993). Hypertension is defined as blood pressure consistently greater than 140/90 (Mertens & Van Gaal). Research supports the contribution of obesity to hypertension, however, the underlying pathophysiology that creates cardiovascular changes are not totally understood. What is understood is when renal histology changes, increased sodium retention with subsequent fluid retention leads to increased cardiac output. Among individuals who lose weight, research supports positive physiologic changes (e.g., decreased blood pressure) that results in a decreased cardiac output, as the individual has less adipose tissue to perfuse. Mertens

and Van Gaal also stated that obese, normotensive individuals who lose weight can decrease their chance of developing hypertension.

Research on insulin resistance/type 2 diabetes mellitus (non-insulin dependent diabetes mellitus, or NIDDM) revealed that obese individuals with increased abdominal adiposity "causes insulin resistance with hyperinsulinemia" (Khaodiar, McCowan, & Blackburn, 1999, p. 22; Walker, 1995). Obesity increases insulin secretion from the islet cells in the pancreas, and large amounts of circulating insulin may affect sodium retention and hypertension in obese people (Bray, 1992). All the demand from overeating on the islet cells may cause the islet cells to stop producing insulin; therefore, the individual develops Type II diabetes. Pi-Sunyer (1996) stated since obesity causes NIDDM, this is a public health concern, as both are on the rise. Losing weight is an effective treatment, as individuals can delay or prevent the onset of NIDDM. Among individuals who already have NIDDM, losing weight can help control their glucose levels (Pi-Sunyer, 2002b), and decrease insulin resistance (Heymsfield et al., 2000; Pi-Sunyer, 1996; Walker, 1995). Grundy, Blackburn, Higgins, Lauer, Perri, & Ryan (1999) stated that exercise also decreases insulin resistance in obese individuals.

Up to 30% of individuals who have non-insulin-dependent diabetes mellitus (NIDDM) have a BMI >30 kg/m^2 (Walker, 1995). Elevated insulin levels indicate insulin resistance, as the insulin is not being used to transport glucose into the cells but instead remain in the circulatory system. Insulin sensitivity occurs more

frequently among individuals who have an android somatotype, or fat deposits around their middle and upper body.

Other comorbid conditions caused by obesity are dyslipidemia (or high serum cholesterol), characterized by high levels of low density lipoproteins (LDL), lower levels of high-density lipoproteins (HDL-C), and elevated serum triglyceride and total cholesterol levels (Bray, 1992; Khaodiar, McCowan, & Blackburn, 1999; Pi-Sunyer, 1993; Weiser, Frishman, Michaelson, & Abdeen, 1997). Increased cholesterol levels precipitate the formation of gallstones, as cholesterol secreted into bile is directly linked to the amount of fat an individual eats (Khaodiar, McCowan, & Blackburn, 1999; Weiser, Frishman, Michaelson, & Abdeen). Sleep apnea (Weiser, Frischman, Michaelson, & Abdeen) is caused by an upper airway obstruction that can be decreased by weight loss (McCance & Huether, 1994). Obese individuals also have the propensity to develop gout, as urate ions tend to precipitate in the great toes (Guyton, 1991; Khaodiar, McCowan, & Blackburn, 1999; Weiser, Frishman, Michaelson, & Abdeen, 1997). Obese individuals also tend to suffer from osteoarthritis, especially in the weight-bearing joints of the legs, hips, and also in the carpals and metacarpals. In general, comorbid conditions caused by obesity are not only uncomfortable, but in some cases are deadly.

Psychosocial and social issues.

Society values thinness and equates thinness with success (Christensen, 1999; Melcher & Bostwick, 1998). Our society stigmatizes obese individuals who overeat to maintain their obesity as lazy, out of control, and weak-willed (Devlin, Yanovski,

and Wilson, 2000; Garner & Wooley, 1991). Psychological issues caused by obesity range from feelings of failure and poor self-image, to the development of eating disorders and depression. FitzGibbon and Kirschenbaum (1990) stated that although the etiology of obesity is heterogeneous, subgroups may evidence psychological disturbances. They suggested that weight loss treatment programs screen clients for psychological issues, as this may influence their treatment outcome. Anderson & Wadden (1999) also advocated screening obese individuals for depression.

Individuals fail at weight loss because they return to former habits of overeating and decreasing their amount of exercise. Individuals can also experience stress, negative moods and emotions (including depression) that may be caused by a decreased self-esteem (Goodrick & Foreyt, 1991). Individuals who spend time alone often overeat, as well as those who experience negative moods such as depression, rejection, and frustration. The food preference of choice among overeaters appears to be carbohydrates (Christensen, 1999). Researchers have suggested that low levels of serotonin may stimulate a preference for carbohydrate ingestion (Ericsson, Poston, & Foreyt, 1996). Christensen stated that two hypotheses have been developed that may explain why carbohydrate craving occurs in obese individuals. First, individuals eat carbohydrates to elevate their serotonin level, and second, carbohydrate metabolism is impaired.

Weiser, Frishman, Michaelson, and Abdeen (1997) stated that low socio-economic status (SES) contributes to obesity. Impoverished individuals do not have the money to buy fresh fruits and vegetables, but instead resort to eating high fat

foods. In addition, families share culture and eating habits learned from each other (Ravussin & Swinburn, 1992). In perhaps the earliest study on the social aspects of obesity, Moore, Stunkard, and Srole (1962) examined relationships between obesity, social class, and mental illness in a population of 1660 residents of New York City and found that obesity was seven times more common among women of lower SES, although the investigators did not define what a "lower SES" represented. Adler and Coriell (1997) found that the risk for morbidity and mortality was higher in lower SES women than those with a higher SES, attributed in part to limited access to health services. Women in a lower SES were more likely to delay their own health care and present with more severe disease symptoms than women in higher SESs. Adler and Corriell also stated that "SES is a clear risk factor for disease and should be part of an assessment of a patient's profile of risk factors" (p. 20).

During the last twenty years, our society in the United States has become less active, due in part to labor saving devices (such as garage door openers, elevators, and escalators). People watch television instead of exercising, and people do not monitor what they eat (Hill, 2002a; Poston & Foreyt, 1999). Poston and Foreyt labeled this activity change "'New World Syndrome'" (p. 204). Poston and Foreyt and James (2002) stated that the environment, socio-economic status, and geographic areas contribute to obesity. Individuals living in a lower socio-economic status usually have to perform more physical, blue-collar labor as compared to white-collar workers, and may be healthier. Hill (2002a) stated that researchers investigating

obesity do not understand the complex interactions between humans in their environment to determine who is at risk for becoming obese.

More people eat out now as compared to twenty years ago, choose to eat calorie-dense foods, and are served huge quantities (Poston & Foreyt, 1999). Foods highest in fat are termed "energy dense food" (Jebb, 2002) and are most easily stored in the body as fat because they are oxidized at a lower rate than proteins or carbohydrates. Weiser, Frishman, Michaelson, and Abdeen (1997) also stated that foods high in fat content make food more palatable and stimulate the individual's need to consume more dietary fat.

There is a national trend in the fast food industry to supersize food (Brownell, 2002; Martin, Robinson, & Moore, 2000), serving individuals extremely large portions of calorie, carbohydrate, sugar, and fat-laden food and drink. For example, in the 1950's, soft drinks were bottled in 8-ounce portions. Today, "the 7-Eleven chain has the Big Gulp (32 ounces) and the Double Big Gulp (64 ounces)" (Brownell, 2002, p. 436). Candy and other snacks and sweets are also packaged in "Big Grab" bags for consumers (Brownell, p. 436). Brownell also found that one quarter of all the vegetables eaten in the United States are french fries. Also contributory to our obese nation is that school children are driven to school instead walking a reasonable distance. The percentage of students taking physical education classes in the United States has dropped from 42% in 1991 to 25% in 1995, yet only one state in the United States requires mandatory physical education (Brownell). Today's children watch approximately 10,000 commercials for junk food on television every year, and

traditional school cafeteria food has been replaced by fast food (Brownell). Many schools contract with soft drink companies to install vending machines that dispense soft drinks that are easily accessible to students. In addition, children are not playing outside during their leisure time, but instead play computer and video games that are sedentary activities.

Overeating is also considered an addictive behavior (Goodrick & Foreyt, 1991; Turner, Knosby, & Popkess-Vawter, 2002). The pattern of repeated relapses after dietary modifications "fits a model of substance dependence in the sense that volitional efforts to control eating seem to be in conflict with psychological defenses [and] parallel those for addictive behavior" (Goodrick & Foreyt, p. 1245). In some cases, eating is paired with rewards and good feelings one experiences during childhood. Cognitive restructuring is a strategy used in therapy to replace detrimental self-talk that occurs in some obese individuals (e.g., "I am bad because I eat too much", or "I am bad because I gained back the weight I lost") with positive self-talk.

Obesity treatments.

More that 66% of the adults in the United States are trying to either lose or maintain their weight (Serdula, Mokdad, Williamson, Galuska, Mendelein, & Heath, 1999). Only 20% of adults trying to lose weight reportedly exercise and eat less. Obesity treatments vary in type and duration. Treatments for obesity include pharmacological, surgical, behavioral, psychological, cognitive and behavioral, and dieting (Devlin, Yanovski, & Wilson, 2000). Individuals are denied many medical and psychological interventions for obesity because insurance companies are

reluctant to pay for obesity treatments (Martin, Robinson, & Moore, 2000). The Health Care Finance Agency (HCFA) considers gastric bypass surgery a cosmetic surgery for individuals who are morbidly obese, and "less that 1% of eligible morbidly obese Americans receive surgical therapy in any given year" (Martin, Robinson, & Moore, p. 347).

Most information on weight loss treatments is found in medically designed and supervised weight loss programs (Brownell & Rodin, 1994; Garner & Wooley, 1991). The majority of weight reduction programs focus on limiting dietary intake (energy intake) with some form of behavioral modification, exercise, or pharmacological intervention; however, a recent meta-analysis of randomized clinical trials (RCT's) investigated the effects of anti-obesity medications combined with lifestyle changes (Poston, Haddock, Dill, Thayer, & Foreyt, 2001) and found that less than 10% of all studies documented any type of lifestyle change in combination with pharmacotherapy. Less than 30% of RCT's included behavior modification and less than 1% included cognitive-behavioral strategies with pharmacotherapy. In addition, less than 17% included any form of exercise and pharmacotherapy. Most studies were conducted on women who were over the age of 40, with body mass indices (BMIs) up to 35 kg/m^2. Among lifestyle treatments mentioned, a balanced-deficit diet (1200-1500 kcal balanced diet) was used most often in combination with pharmacotherapy (41.4%), followed by low-calorie diets (24.5%). One behavior modification strategy, self-monitoring, was used more than any of the other strategies (23.5%).

Pharmacologic treatments.

Medications were developed to assist individuals to lose weight targeted at producing metabolic changes, blocking chemical actions that contributed to weight gain, and suppressing appetite (Kordik & Reitz, 1999; Weiser, Frishman, Michaelson, & Abdeen, 1997). Some drugs used to treat obesity have been withdrawn due to deleterious side effects and addictive properties such as amphetamines (Campbell & Mathys, 2001; National Task Force on the Prevention and Treatment of Obesity, 1996). The National Heart, Lung, and Blood Institute (NHLBI) recommended that pharmacotherapy be supported with other adjunct treatments such as a low-calorie diet, exercise, and behavior therapy (2000). Pharmacotherapy is recommended for all individuals whose BMI $\geq$30, and for individuals whose BMI is $\geq$27 who present with comorbid diseases or risk factors, or when an individual makes the required lifestyle changes and cannot lose weight (Pi-Sunyer, 2002a).

Historically, the group of drugs known as amphetamines were prescribed during the 1950's and 1960's (National Task Force on the Prevention and Treatment of Obesity, 1996). Throughout the next twenty years (1970-1990), there were no pharmacologic interventions for obesity other than amphetamines, and treatments focused on behavioral modification and exercise. Fluoxetine (Prozac®) was first developed for weight management when researchers discovered that it elevated mood (Goldstein, 1992; Michelson et al., 1999; Orzack, Friedman, & Marby, 1990; Ward, Comer, Haney, Fischman, & Foltin, 1999). In 1996 (National Task Force on the Prevention and Treatment of Obesity), fenfluramine (Pondimin®) and

dexfenfluramine (combined, known as fen-phen) were prescribed for many obese people. These two drugs are serotonin releasing agents as opposed to reuptake inhibitors (Jones & Blackburn, 2002). They were banned by the Food and Drug Administration (FDA) in 1997 as they caused cardiac valve disorders and primary pulmonary hypertension.

Sibutramine (Meridia®) is a norepinephrine-serotonin-dopamine reuptake inhibitor first marketed as an antidepressant (Jones & Blackburn, 2002). Sibutramine is similar to monoamine oxidase inhibitors (MAOIs) as it inhibits reuptake of all three neurotransmitters listed above. By decreasing the natural reuptake of norepinephrine and serotonin in the central nervous system, satiety is reached sooner and the individual consumes less calories. One can expect to lose up to 10% of baseline weight using sibutramine (Pi-Sunyer, 2002a). Sibutramine should not be prescribed if an individual is already taking another selective serotonin reuptake inhibitor (SSRI, e.g., Prozac®, Paxil®, Lexapro®, Zoloft®, etc.). Many studies support the safety and efficacy of sibutramine not only with weight loss but in reducing waist:hip ratios, LDL cholesterol, and blood pressure (Dujovne, Zavoral, Rowe, & Mendel, 2001; James et al., 2000; Ross et al., 2000; Smith & Goulder, 2001; Wirth & Krause, 2001).

Orlistat is a nonabsorbable lipase inhibitor that inhibits fats from systemic absorption in the colon (Weiser, Frishman, Michaelson, & Abdeen, 1997). Jones and Blackburn (2002) stated that researchers are currently moving away from serotonin therapy and focusing more on orlistat as an anti-obesity treatment. Individuals treated with orlistat can expect to lose 10% of their baseline weight (Pi-Sunyer, 2002a). The

most common side effects among individuals taking orlistat are flatulence, diarrhea, fatty stools, increased stool production, and uncontrolled bowel movements, especially if fat intake exceeds 30% of the total daily calories. These unwanted side effects occur in 95% of the individuals taking orlistat. Orlistat also blocks the absorption of the fat-soluble vitamins A, D, E, and K. Finer, James, Kopelman, Lean, and Williams (2000) compared the efficacy of orlistat compared to a placebo in 228 obese individuals. All subjects were instructed to eat a low-fat diet plus taking either orlistat or a placebo. At the end of one year, the orlistat group lost 8.5% of their initial body weight in comparison to 5.4% in the placebo control group. The orlistat group also showed a significant decrease ($p<.05$) in serum cholesterol, LDL, and increased HDL levels as compared to the placebo group. Rossner, Sjostrom, Noack, Meinders, and Noseda (2000) found similar results. Many investigators have found that orlistat was appropriate to use for both initial and long-term weight loss as it improves cholesterol levels, thereby decreasing some of the risk factors associated with obesity (Davidson et al., 1999).

Another recent meta-analysis was published by Haddock, Poston, Dill, Foreyt, and Ericsson (2002) that evaluated 40 years of randomized clinical trials (RCTs) on medication efficacy used in the treatment of obesity. Results indicated that weight loss does not continue after six months on any type of pharmacotherapy, and that obese individuals usually lost an average of 4 kg. (8.8 pounds). The authors did suggest that pharmacotherapy may assist individuals with weight maintenance over time.

Surgical treatments.

Surgery offers a solution for individuals who are approximately 100 pounds overweight, or roughly exceeding a BMI of 40 kg/m^2 (Pories & Beshay, 2002). Types of surgical procedures performed are gastric partitioning, Roux-en-Y, and vertical banded gastroplasty. Surgery offers a solution for individuals who are around 100 pounds overweight, or roughly exceeding a BMI of 40 kg/m^2 (Pories & Beshay, 2002). One procedure performed is gastric partitioning, that 'partitions' a portion of the stomach so the stomach will be reduced in size. Most weight loss occurs during the first year; however, this type of surgery can result in complications such as gastric outlet obstruction, ulcer formation in the pouch segment, and problems with the staple lines. Gastric banding involves the surgeon applying a type of belt distal to the cardiac valve that creates a small pouch out of the upper portion of the stomach (Pories & Beshay). The belt can be adjusted by a physician as an outpatient procedure. One major advantage to this surgery is that it can be performed using laparoscopic technique, as opposed to the gastric partitioning that requires a major abdominal incision. Another advantage is that the client does not suffer from vitamin or mineral malabsorption that occurs after gastroplasty. These two types of surgeries result in the highest percentage of weight loss (15%) maintained over ten years in obese individuals, and may become the treatment of choice in the future (Torgerson & Sjostrom, 2002).

Surgery is not a panacea, however, as individuals who still consume high fat and high carbohydrate foods (such as milkshakes and ice cream) fail to lose weight.

Psychological therapies.

During the 1970's, obesity was considered to be a psychological problem that individuals could modify. Other beliefs during this time were that obese individuals preferred sweet foods, were picky eaters, and exerted no self control when eating these foods (Drewnowski, 2002). Most current psychological therapies are based on behavioral, cognitive, and cognitive-behavioral theories. Behavioral approaches focus on stimulus control of individual behaviors that contribute to weight gain (Jebb, 2002). Behavior modification focuses on outcomes rather than any underlying cognitive origins of the stimulus.

Behavior therapies are used in combination with very low caloric diets (VLCDs) that include principles and techniques to change eating and exercise behaviors (Kushner & Hopson, 1998; Wing, Epstein, Marcus, & Kupfer, 1984). Behavioral modification is based in learning theory, where individuals are taught to be aware of their intake and attend to environmental cues that stimulate the desire to eat or overeat (Wing, 1992). Behavioral therapy has been successful in some individuals, but most relapse to former behaviors and re-gain their weight (Goodrick & Foreyt, 1991). Therefore, maintaining compliance with treatment therapy is difficult (Wadden, 1993), and behavioral therapy appears to only work for a limited amount of time (Wilson, 2000). Wilson stated that "the pattern of weight loss and regain in behavioral treatment is consistent. The rate of initial weight loss is rapid but then slowly declines. The low point is reached after approximately six months. Weight regain then begins and continues gradually until weight stabilizes near

baseline levels" (p. 229). Other strategies employed include teaching/learning stress management and problem-solving techniques (Goodrick & Foreyt, 1991; NHLBI, 2000; Poston & Foreyt, 2000; Poston, Hyder, O'Byrne, & Foreyt, 2000).

The cognitive approach focuses on changing individual beliefs and thoughts that are self-perpetuated and negative. The role of the healthcare professional in counseling the obese individual is to guide and teach individuals to modify their behavior (Foreyt & Poston, 1998a), to become aware of their self-perceptions and their obesity, and to learn and accept what is considered a reasonable weight loss (Poston & Foreyt, 2000).

Cognitive-behavioral treatment for obese individuals subsumes five strategies used to modify eating behaviors (Foreyt & Poston, 1998b). These strategies include (a) self-monitoring (that includes keeping a food and exercise diary, recording any weight changes, and identifying conditions that stimulate an individual to overeat), (b) controlling environmental stimuli that triggers overeating and decreased exercise, (c) using cognitive restructuring to help individuals correct negative thoughts, (d) assisting individuals to learn stress management techniques (such as relaxation), and (e) encouraging social support from family or peers. Two other strategies include emphasizing physical activity and preventing relapse (Foreyt & Poston, 1998a), setting reasonable eating and exercising goals, and "behavioral contracting" (Poston, Hyder, O'Byrne, & Foreyt, 2000, p. 1901).

Physical exercise.

Physical exercise is an important component of weight loss; however, exercise alone will not produce weight loss. Exercise must be combined with a low-calorie diet for weight loss to occur (Hecker, Kris-Etherton, Zhao, Coval, & St. Jeor, 1999; NHLBI, 2000). Exercise also helps to reduce the development of type II diabetes and cardiovascular disease. Clients must be encouraged to decrease sedentary activities and to get up and move. Exercise also has a positive effect on negative moods and stress (Pendleton, Goodrick, Poston, Reeves, & Forcyt, 2002).

Dietary treatments.

Approximately 24% of females and 8% of men are currently on a diet (Hill, 2002a). The United States has the highest prevalence of dieting than any other country in the world (Hill). There are many dieting programs in the United States (e.g., Jenny Craig®, the Atkins Diet®, Weight Watchers®, the Food Pyramid®, etc.) that market their methods for weight loss to the public.

One diet method used to lose weight quickly is the Very Low Calorie Diet (VLCD), usually consisting of an intake of <500 calories per day that includes supplemental protein. The National Task Force on the Prevention and Treatment of Obesity (1996) suggested that medically supervised VLCD programs should be used only for clients whose BMI >30, and to include behavioral modification and exercise (Garner & Wooley, 1991; Stunkard, 1996). These authors stated that the main problem with VLCD's was the rapid weight re-gain that occured once treatment was finished. Reasons people re-gained lost weight may be due to genetic factors, that the

body has a specific set point to maintain a certain weight. Another explanation is that the people return to previously-learned eating behaviors.

Measuring Obesity

Measuring body mass index (BMI) is the most frequently used method to measure obesity (Nies, Cook, & Hepworth, 1999; NHLBI, 1998; NHLBI, 2000) as it provides a "more accurate measure of total body fat compared with the assessment of body weight alone" (p. 1). BMI was cited frequently in the literature as the preferred method to use to distinguish obese subjects from normal and underweight subjects.

Waist circumference is also an appropriate method to assess risk factors for developing comorbid conditions (Despres, 1999), as increased waist circumference over time indicates increased fat deposits and a higher likelihood of developing cardiovascular disease. Significant abdominal fat is a predictor for both primary and comorbid disease conditions (NHLBI, 2000). Men with a waist measurement larger than 40 inches and women with a waist measurement larger than 35 inches are at a higher risk for developing hypertension, cardiovascular disease, dyslipidemia, and type II diabetes.

Gender Issues in Depression and Obesity

In some individuals, obesity may compromise psychological well being (Kawachi, 1999; Pi-Sunyer, 1993). Many obese individuals suffer from repeated dietary failures, experience difficulties with employment, and general ignorance of the potential physiologic and psychological ramifications of obesity (Brownell & Wadden, 1992; Lissner, 1993; Puhl & Brownell, 2002). In addition, many obese

patients who present for weight counseling and/or dietary management are not assessed for the presence and/or absence of depression (Anderson & Wadden, 1999; Carpenter, Hasin, Allison, & Faith, 2000; United States Preventive Task Force, 2002). Some individuals can be overweight without being obese (such as body builders); however, individuals who are overweight are most likely obese (St. Jeor, Silverstein, & Shane, 1996).

Early research on obese and morbidly obese people focused on personality disorders. Hopkinson and Bland (1982) found among 73 obese females scheduled for gastric bypass surgery, 19.2% (n = 14) of the women presented with depression that required treatment. Hafner, Watts, and Rogers (1987) studied women scheduled for gastric bypass surgery, and found that 118 out of 142 women scored significantly higher on a measure of depression. In a study by Prather and Williamson (1988) on female obese, bulimic, and binge eaters, subjects were assessed for personality disorders using the Minnesota Multiphasic Personality Inventory (MMPI) (Hathaway & McKinley, 1940) and the Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Morck, & Erbaugh, 1961). The obese subjects scored from mild to severe on the depression section of the BDI in comparison to bingers and bulimics, who scored low on depression.

Many factors contribute to overweight and obesity that are gender related. Different stages in development are hypothesized to contribute to overweight and obesity, such as puberty, when "female fat tissues are a significant extragonadal source of estrogen and may affect hormonal feedback mechanisms controlling

ovulation and the menstrual cycle" (St. Jeor, Silverstein, & Shane, 1996, p. 369). Other contributory factors are pregnancy, chronic dieting, and perhaps menopause, although specific causal factors are unknown at this time (Krenkel & St. Jeor, 1995). Kordik and Reitz (1999) stated that among women, measuring body mass index (BMI) does not take into account the placement of fat around the hips, upper body, and abdomen, therefore making it "difficult to set a definitive BMI level threshold to define obesity" (p. 181). Lissner (1993) stated that individuals with upper body adiposity (an android somatotype) may be at an increased health risk as opposed to individuals with adiposity around the hips and thighs (a gynoid somatotype).

Kessler et al. (1994) found that women "have higher prevalences than men of both lifetime and 12-month comorbidity of three or more disorders" (p. 13) especially during the ages between 25-34 years. Obese women who experience a myocardial infarction "are more likely than men to have a history of major depressive disorder and more prone to post myocardial infarction depression" (Schwartzman & Glaus, 2000, p. 53). In addition, women report comorbid physiological conditions such as headaches, irritable bowel syndrome, fibromyalgia and chronic fatigue syndrome, and higher rates of anxiety and eating disorders that result in weight gain. Women also have a slower response to antidepressant therapy, and a greater sensitivity to medication side effects (Kornstein & McEnany, 2000). Nolen-Hoeksema (1987) found that women ruminated and became inactive when they were depressed, whereas men exhibited physical behaviors (e.g., playing sports) that diverted their attention away from their depressive symptoms. Women also tend to overeat when

they experience unpleasant feelings such as stress, boredom, anxiety, and depression (Grilo, Shiffman, & Wing, 1993; Heatherton, Polivy, & Herman, 1991). Ruminating and inactivity may be a reason why depressed women gain weight and depressed men do not, as inactivity causes weight gain if caloric intake increases or remains the same while the amount of exercise decreases. Siegel, Yancey, and McCarthy (2000) examined obesity and depression among African-American women (N=429) recruited for a health promotion program, and found that lower levels of depression (as measured using the CES-D; Radloff, 1977) occurred among women who were less overweight and had a higher sense of ethnic identity.

There is some evidence that women have a more difficult time losing weight than men, and depressed women may present with weight gain and increased appetite as their primary symptoms (Ernst & Angst, 1992; Fava, 2000; Frank, Carpenter, & Kupfer, 1988; Kornstein, 1997; Kornstein & McEnany, 2000; Schatzberg, Cole, & DeBattista, 1997; Young, Scheftner, Fawcett, & Klerman, 1990). Losing weight and keeping weight off is difficult (Sarlio-Lahteenkorva, Rissanen, & Kaprio, 2000), and women may have more trouble with losing weight and maintaining weight loss than men (Wing, 1993). This suggests that a sub-population of women who cannot lose weight may be depressed. Ross (1994) postulated a causative relationship between women, obesity, and depression. Among women, "being overweight is positively associated with dieting; dieting is positively associated with depression; and dieting explains much of the positive association between being overweight and depression" (p. 72). In addition, due to the high rate of dietary failures over time, individuals tend

to become depressed when they regained their weight and view themselves as failures (Garner & Wooley, 1991). Siegel, Yancey, and McCarthy (2000) are the only investigators to date that have examined women, depression, and obesity. They found in a sample of African-American women (N=429) who were recruited for a health promotion program, that lower levels of depression (as measured by the CES-D; Radloff, 1977) occurred among African-American women who were less overweight and had a higher sense of ethnic identity. Carpenter, Hasin, Allison, and Faith (2000) found that overweight individuals who suffered from major depression had a greater BMI, and obese women were 37% more likely to be diagnosed with depression.

In contrast, some research conducted during the 1980's and 1990's did not support a relationship between women, obesity, and depression. Wadden and Stunkard (1985) found that most studies focused on psychosocial problems in obese women, not obese men, and they found that obese individuals did not report a greater degree of psychological disturbance as compared to normal weight individuals. They stated that "these are important findings because they refute the long-standing belief that overweight persons suffer from serious emotional disturbances" (p. 1064). Wadden and Stunkard also stated that, in general, there was no increased psychopathology in obese persons with the exception of individuals who were morbidly obese (using the MMPI; Hathaway & McKinley, 1940), defined as >75% overweight. Nutzinger, Cayiroglu, Sachs, and Zapotoczky (1985) found no increased incidence of depression among obese individuals; however; if individuals previously experienced depression, their level of depression worsened. A major problem with

this study was that their sample size was small (N=23), and the investigators generalized results and treatment based on their findings from 23 subjects.

Recently, Carpenter, Hasin, Allison, and Faith (2000) remarked about the inconsistency among study findings and discussed methodological problems in previous research, such as the use of different instruments to measure depression. They also mentioned the lack of including obese individuals not seeking therapy or treatment as problematic, as there may be a subpopulation of obese individuals that are perfectly happy with their obese condition. This may contribute to selection bias among obese individuals who seek treatment. In addition, very few investigators discussed effect size, power of the statistical test, nor threats to design, and fewer reported Cronbach's alpha when reporting test reliability. Admittedly, investigators revealed inconsistencies when comparing different measures of depression.

In general, obesity creates psychological problems in some people but not all, and occurs in relative degrees of severity (Friedman & Brownell, 1995). Brownell and Wadden (1992) suggested that psychological factors are usually ignored when treating obese individuals, and that people are at risk for developing psychological problems who have repeatedly failed with weight loss (Garner & Wooley, 1991). Both Anderson and Wadden (1999) and FitzGibbon and Kirschenbaum (1990) recommended screening all obese individuals for depression.

The RENO Diet-Heart Study examined gender, weight, and age differences in relation to developing cardiovascular disease risk factors in a cohort of 508 subjects (St. Jeor, 1997). The RENO study was a naturalistic, observational, cohort study that

examined how weight changes over time, and how these changes affect the development of risk factors for cardiovascular disease. The groups were divided into male versus female, and normal weight versus obese, stratified into five different decades. The investigators found that in general, obese women dieted, enrolled in weight loss programs, and used more diet pills and weight loss products than men. In contrast, both normal and obese males exercised more than any other group. Normal weight females participated in more physical activity, more nutrition education, and cognitive restructuring. More detailed information about the structure of the RENO Diet-Heart Study is found in Chapter 3 (St. Jeor, 1997).

Pharmacologic therapy combined with diet and exercise appears to help some obese individuals lose weight at least initially (National Task Force on the Prevention and Treatment of Obesity, 1996); however, no studies were located about how depression affects weight loss. If the reason for repeated dietary failures is due to chronic depression, continuing and differing weight loss treatments will not help the person lose weight as these treatments do not address underlying depressive symptoms.

Many investigators emphasized the importance of looking at causal relationships between depression and obesity (Blehar & Oren, 1995; Carpenter, Hasin, Allison, & Faith, 2000; Friedman & Brownell, 1995; Khaodhiar, McCowen, & Blackburn, 1999; Klesges, Klem, & Klesges, 1992) to develop an integrative model that explains relationships between depression and obesity. Friedman and Brownell (1995) presented results from their meta-analysis and concluded "it is reasonable to

hypothesize that women and severely obese individuals are particularly at risk for suffering from their obesity because societal pressures focus more on women, specifically young women, and severely obese individuals" (p. 13). They also speculated that obese women suffer more than obese men, but "this may be an artifact of women being more likely to seek treatment, and seeking treatment rather than gender may be linked with psychopathology" (p. 13). They also suggested that future work should examine the possibility of "causal links between risk factors and psychopathology" (p. 15).

Summary and Statement of Proposed Research

There is evidence that women suffer from depression more than men, have earlier onsets of depression, and may gain weight as a symptom of depression. Women also tend to become vegetative and overeat when depressed, and have higher cardiovascular morbidity and mortality rates than men. The literature is replete with theories on the heterogeneous causal factors of both obesity and depression, yet few investigators have examined the effect of depression on weight loss in overweight or obese women who cannot lose weight. There is convincing evidence that a subpopulation of women seeking treatment for overweight or obesity may suffer from underlying depression, and, because of this depression, may fail at weight loss. In addition, the National Institution of Health recommends and supports screening all clients for depression in primary care. The purpose of this study was to investigate if baseline measures of depression, gender, weight group, and age in overweight and obese women would predict weight change over a five year period. Data were

analyzed for males as a comparison group. A secondary purpose for this study was to examine if a higher level of depressive symptoms positively correlated with body mass index (BMI) over a five year period, using archival data in a secondary data analysis from the RENO Diet-Heart study (St. Jeor, 1997) to explore relationships between obesity and depression in women.

The Influence of Depression on Overweight and Obese Women: A Secondary Analysis of the RENO Diet-Heart Study

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Abstract

Twice as many women suffer from depression than men, and may gain weight as a symptom of depression; additionally, more women are obese than men. Few studies have examined relationships between depressive symptoms and weight gain in a community sample of adults. The investigators hypothesized that higher levels of depressive symptoms (measured by the CES-D), gender, weight group (normal, overweight, and obese), and age would predict weight change over time in obese and overweight women. A secondary data analysis using five years of archival data from the RENO Diet-Heart study was performed to examine relationships between obesity and depression in women. A hierarchical linear mixed model was used to determine if depressive symptoms predicted weight change over time in overweight and obese women, and men from the same study were used as a comparison group. Although potential subjects were excluded if they reported any history of depression or taking antidepressants, the hierarchical linear mixed model overall test revealed that increased depressive symptom scores were associated with significantly increased weight gain over time for obese females and males, $F(3,3000)=3.91$, $p=.0085$. In addition, time was significant for gender, $F(2,3000)=25.07$, $p<.0001$) and age,

$F(1,3000)=43.26$, $p<.0001$. Results indicated that in this community sample of adults, when depressive symptom scores increased in obese females and males, weight gain also increased, even though CES-D scores remained within normal limits. Overweight screening and treatment would be revised to include interventions for depressive symptoms. Further research is needed to explore the relationships between depressive scores and weight gain in an entire community setting as increased depressive symptoms may account for weight gain or the inability to lose weight.

Key Words: depression, depressive symptoms, overweight, and obesity.

Introduction

The prevalence of overweight and obesity continues to rise, affecting almost two-thirds of the population in the United States (Flegal, Carroll, Ogden, & Johnson, 2002). Furthermore, more women are obese than men (33% versus 28%, respectively) (National Center for Health Statistics, 2002). Women who experience difficulty in losing weight may experience depressive symptoms, as increased appetite and weight gain are two of the many symptoms of depression (American Psychiatric Association, 2000; Ernst & Angst, 1992; Fava, 2000; Frank, Carpenter, & Kupfer, 1988; Kornstein, 1997; Kornstein & McEnany, 2000; Schatzberg, Cole, & DeBattista, 1997; Young, Scheftner, Fawcett, & Klerman, 1990). Many obese patients who seek help for weight counseling and dietary management are not assessed for depressive symptoms or depression (Anderson & Wadden, 1999; Carpenter, Hasin, Allison, & Faith, 2000; United States Preventive Task Force, 2002). Anderson & Wadden (1999) and FitzGibbon and Kirschenbaum (1990) advocated screening obese individuals for depression. Based on the reviewed literature, individuals seeking treatment for overweight or obesity may suffer from underlying depressive symptoms, and depressive symptoms experienced over time may lead to weight gain. If a relationship exists between untreated depressive symptoms and weight gain, treatment guidelines for overweight or obese women could be impacted to consider obesity and depression as related co-morbidities, and could contribute to existing knowledge about women, depressive symptoms, and obesity.

The National Institute for Mental Health stated that 12% of women (compared to 7% of men) suffer from depression, and at least 20% of the total female population has experienced a treatable depressive episode. Women report more symptoms of depression than men, score higher on instruments that measure depression (Frank, Carpenter, & Kupfer; Nolen-Hoeksema, 1987), have longer periods of symptoms, and experience chronic re-occurrences of depressive episodes (Amenson & Lewisohn, 1981; Ernst & Angst, 1992; Kessler et al., 1994; Nolen-Hoeksema, 1987). Gender does not cause depression by itself, but combined with social role problems women face (e.g., discrimination, single parenting), lower socio-economic status (SES), psychological problems, environmental and developmental factors, a family history of depression, dieting failures, loss of self-esteem, and reproductive/hormonal issues may increase women's vulnerability to develop depression (Blehar & Oren, 1995; Foreyt, Brunner, Goodrick, Cutter, Brownell, & St. Jeor, 1995; Nolen-Hoeskema, 1987; Weiser, Frishman, Michaelson, & Abdeen, 1997).

Experts and researchers agree that obesity is a chronic disorder that requires long-term therapy (Devlin, Yanovski, & Wilson, 2000; Friedman & Brownell, 1995; Kornstein, 1997; National Task Force on the Prevention and Treatment of Obesity, 1996; Poston & Foreyt, 1999; Poston & Foreyt, 2000; Stunkard, 1996). Many gender-related factors contribute to overweight and obesity in women such as pregnancy, chronic dieting, and perhaps menopause, although delineating specific causal factors are unknown at this time (Krenkel & St. Jeor, 1995). Losing weight and keeping weight off is difficult (Sarlio-Lahteenkorva, Rissanen, & Kaprio, 2000), and women

may have more trouble with losing weight and maintaining weight loss than men (Wing, 1993).

The RENO Diet-Heart Study (St. Jeor, 1997) was a naturalistic, observational cohort study designed to examine relationships between energy, nutrition, and obesity to cardiovascular disease in healthy adult females and males from 1985 to 1990 (Mifflin, St. Jeor, Hill, Scott, Daugherty, & Koh, 1990). All variables of interest (depressive symptom scores, gender, body weight, age, and body mass index) were available in the RENO study. Five years of complete data were examined in the current investigation, as funding for the RENO Diet-Heart study was extinguished before eight years transpired.

The purpose of this study was to investigate if baseline measures of depressive symptoms, gender, weight group, and age in obese and overweight women predicted weight change over five years. Archival data in a secondary analysis from the RENO Diet-Heart study (St. Jeor, 1997) were examined to explore relationships between obesity and depression in women. The investigators hypothesized that overweight and obese females with higher depression scores would gain more weight than normal weight females. Males were used as a comparison group to examine any gender differences.

Method

In the RENO Diet-Heart study, potential subjects were screened extensively to obtain a homogenous healthy sample. Potential subjects were excluded if they reported hospitalization for depression, a diagnosis of depression, loss of work due to

depression, any history of depression, or had taken antidepressants within the last five years including over-the-counter mood enhancers (St. Jeor & Dyer, 1997). Thirty females and 6 males were excluded from participating (St. Jeor, Simpson, & Daugherty, 1997, p. 26). A stratified sampling technique (2x2x5) was used where gender (male and female) and weight group classifications (normal or obese) were stratified into five age categories by decades, from age 20 through age 60 or over. The purpose of using stratification was to enroll 25 male and 25 female subjects who were either of normal weight or obese for each age decade. Subject weights were measured at baseline and every six months over five years (10 times). In addition, subject body mass index (BMI) and depressive symptoms (CES-D) were measured at baseline and annually (5 times).

The resulting sample of 508 subjects included working-class men, women, and college students who were in good reported health and lived in Reno, Nevada from 1985 through 1990. Eighteen female and 15 male subjects were over the age of 65. Overall, the majority of subjects worked in some type of professional role (there was no elaboration on what occupations comprised "professional"), were married, White (not Hispanic), and their monthly income ranged from $2,001 to $4,000 after taxes. The majority of subjects had completed formal education for twelve or more years. One hundred eighty five subjects achieved post-graduate course work beyond their initial college degree. The average age for all subjects (N=508) was 45 years (SD=14.13), and ages ranged from 19 to 77 years. At baseline, subjects' mean weight was 175 pounds (SD=36.44), mean height was 67 inches (SD=3.78), and mean BMI

was 27 (*SD*=4.58). According to current BMI indices that distinguish normal weight (a BMI <25), from overweight (a BMI from 25-29.9), and obese (a BMI >30), the average subject was overweight.

Weight groups were developed based on current BMI indices. The majority of females (*n*=113) were of normal weight, followed by overweight females (*n*=70) and obese females (*n*=68). The majority of males were overweight (*n*=113), followed by normal weight (*n*=74) and obese males (*n*=61).

Depressive Symptoms Measure

The Center for Epidemiologic Studies on Depression scale (CES-D) (Radloff, 1977) assesses depressive symptoms that one experiences during the previous week. It is a self- report scale and was designed for use in a community population. The CES-D was used annually to screen for depressive symptoms among the subjects in the RENO Diet-Heart study. Radloff (1977) described how the CES-D is sensitive to depressive symptoms, but should not be used to make a diagnosis of depression. A cutoff score of ≥17 was used to delineate subjects with increased depressive symptoms (Radloff, 1977). Internal consistency reliability indices (Cronbach's alpha) for the CES-D were calculated for each year of this study as follows: α=.86 at baseline, α=.88 at year 1, α=.89 at year 2, α=.90 at year 3, and α=.89 at year 4. These results were consistent with other studies reviewed for this investigation (Ross, 1994; Siegel, Yancey, & McCarthy, 2000).

Data Analysis

For the present investigation, the investigators hypothesized that increased depressive symptoms (CES-D $\geq$17) would result in weight gain in overweight and obese females over five years (excluding baseline measures) as compared to normal weight females and all males, controlling for age, gender, and CES-D score by weight group. Variables for inclusion were annual CES-D scores, body mass indices, age at study entry, and measures of body weight (every six months). The weight group variable was re-defined according to weight current guidelines for body mass index (Nies, Cook, & Hepworth, 1999; NHLBI, 2000). A normal weight BMI ranged up to 24.9, overweight ranged from a BMI of 25 to 29.9, and a BMI >30 was considered obese.

All CES-D scores were skewed to the right as expected. Score transformations were necessary to normalize the raw scores (Aneshensel, Frerichs, and Clark ,1981; Aneshensel, Frerichs, and Huba (1984). Cohen, Cohen, West, & Aiken, 2003). A natural logarithmic transformation was computed to standardize all raw CES-D+1 scores. Score standardization was necessary to place scores in a more normal distribution instead of a skewed distribution (Maxwell & Delaney, 2000) and to simplify the relationship between predictor and criterion variables (Cohen, Cohen, West, & Aiken, 2003).

The Hierarchical Linear Mixed Model

A hierarchical linear mixed model was used as a data analytic procedure to examine how individuals changed over time (Verbeke & Molenberghs, 2001; Wu,

1996). One advantage of using a hierarchical linear mixed model over traditional analysis of variance was that for each subject a minimum of three data points were needed to build a regression equation, as opposed to excluding subjects with missing data that violates an assumption of an analysis of variance method. Another advantage to using a hierarchical linear mixed model was that investigators could examine both individual and group changes over time (Cohen, Cohen, West, & Aiken, 2003; Hox, 2002; Kenny, Bolger, & Kashy, 2002; Wu, 1996).

This first step in the creating a hierarchical linear mixed model focused on creating a regression equation for each subject, notated as

$$Y_{ij} = a_i + b_i X_j + e_{ij}$$

where the dependent variable (Y) denoted the predicted weight gain for a subject 'i' in group 'j'over five years (X_j denoted times subjects were weighed every six months, denoted $j=1,2,3...9$). The a_i intercept described each subject's baseline weight where their weight intercepted the Y axis. The b_i regression coefficient reflected the subject's slope of the effects of time on weight gain. The error term (e_{ij}, or residual), reflected the error for each subject when predicting Y from X (Kenny, Bolger, & Kashy, 2002).

Second level equations (among subjects) examined the effects of the predictor variables, (a) age upon entering the study, (b) gender, and (c) a baseline depression score by the subject's weight category (either normal weight, overweight, or obese), and their effect on two estimated parameters, the intercepts (a_i) and slopes (b_i).

Recalling that the second level *a* intercept and *b* coefficients are now considered dependent variables, the first level 2 equation (for the intercept) is notated as

$$a_i = \text{entage}_i B_1 + \text{Female}_i B_2 + \text{Male}_i B_3 + \text{wtcat}_1 * \ln(\text{CES-D}_i + 1) B_4$$
$$+ \text{wtcat}_2 * \ln(\text{CES-D}_i + 1) B_5 + \text{wtcat}_3 * \ln(\text{CES-D}_i + 1) B_6 + d_{oi}$$

where 'a_i' is a function of the predictors of age multiplied by the *B* weight for age; gender, the *B* weight for either female or male; plus the interaction between weight category (either normal, overweight, or obese) multiplied by the *B* weight of the transformed natural logarithm CES-D score (plus 1). An error term, d_{oi}, is generated for the intercept (*a*). In addition, 'time' is not involved in predicting the intercept. Although the intercept was important, the purpose of this study focused on weight gain over time (a change in the slope, or estimate, or *B*) as a function of age, gender, and CES-D scores by weight group, notated as

$$b_i = \text{entage}_i B_7 + \text{Female}_i B_8 + \text{Male}_i B_9 + \text{wtcat}_1 * \ln(\text{CES-D}_i + 1)_i B_{10}$$
$$+ \text{wtcat}_2 * \ln(\text{CES-D}_i + 1) B_{11} + \text{wtcat}_3 * \ln(\text{CES-D}_i + 1) B_{12} + d_{1i}$$

where b_i denotes the slope or rate of change for the individual '*i*', as a function of age (their current age multiplied by the *B* weight for time*age interaction), gender (the *B* weight for the individual's gender multiplied by the *B* weight for the gender*time interaction), and their weight category *b* weight (normal weight, overweight, or obese) * by their transformed CES-D score (+1) * by time interaction. Again, an error term, d_{1i}, is generated for the slope (*b*).

The third level "omnibus" procedure (Raudenbush & Bryk, 2002), also called the "cross level interaction" (Cohen, Cohen, West, & Aiken, 2003) or the mixed

model, examines the interaction between the fixed effects of gender, age, and CES-D score by weight group, with and without 'time' as a covariate. Results are reported similar to an analysis of variance output using the terms 'degrees of freedom', an *F* value, and a significance level that denotes if predictor variables significantly differed from one another.

When interpreting results of a hierarchical linear mixed model, the third level (or overall/omnibus) test is examined first to determine the overall significance of the predictor variables. 'Time' was added to each of the level three predictors of gender, age, and transformed CES-D +1 score for a total of six predictors to examine if the interaction between time, weight group, and transformed CES-D score predicted weight change over time. If an omnibus test for the predictor variables in question is significant, the second level results must be examined to find out where the difference occurred, similar to evaluating results from an analysis of variance output.

Results

Obese females scored higher on the CES-D than all other gender-classified weight groups; however, all annual mean CES-D scores remained under 17 (Table 1). The lowest mean CES-D scores occurred in the normal weight groups. Scores increased for the overweight groups, and were the highest for the obese weight groups.

*Insert Table 1 here *

Results from the overall hierarchical linear model were not significant for the effect of depressive symptoms by weight group over time interaction. Jacobson

(1981) stated that extreme scores could invalidate obtained results. One suggestion was to delete the extreme scores. Using a Type 3 sum of squares model, standardized residuals and unstandardized predictor variables were plotted in a scatterplot. For a subject to be eliminated from the final analysis as an outlier, their transformed score must exceed three standard deviations (*SD*) above and below the mean. Three standard deviations above and below the mean captured 99.7% of the subjects (Waltz, Strickland, & Lenz, 1991). After six iterations, 20 subjects were classified as outliers, and eliminated from the final analysis. After excluding the extreme scores, results gain over time, $F(3,3000)=3.91$, $p=.0085$ (Table 2).

Insert Table 2 Here

In the second level, the predictor interaction between CES-D score, time, and obese weight group was also significant, $B_{12}=0.1830$, $t(488)=2.34$, $p=.0194$, but was not significant for either the normal or overweight groups. To examine if gender differences occurred, the second level output was examined. The *B* weights were significantly different from zero for both females, $B_7=1.67$, $t(488)=6.82$, $p<.0001$, and males, $B_8=1.65$, $t(488)=6.90$, $p<.0001$. The time by age interaction slope also differed significantly from zero in a negative sense, $B_9= -0.03$ $p<.0001$, that subjects 'lost time' as they aged instead of gaining years in a numerical sense. The intercept variables are shown also in Table 3.

Insert Table 3 Here

Estimated weight gain over time was accomplished by substituting data into the equation for the *B* coefficients associated with each predictor variable For

example, if we wanted to predict weight gain for a thirty year old obese female with a CES-D score of 20, we would apply the regression equation for slopes,

$$b_i = \text{entage}_i b_7 + \text{Female}_i b_8 + \text{Male}_i b_9 + \text{wtcat}_1 * \ln(\text{CES-D}_i + 1)_i b_{10}$$

$$+ \text{wtcat}_2 * \ln(\text{CES-D}_i + 1) b_{11} + \text{wtcat}_3 * \ln(\text{CES-D}_i + 1) b_{12} + e_{1i}$$

Substituting numbers generated from the output,

$b_i = (-.83) + (1.67) + (0.44) = 1.28$ pounds this obese woman could gain every six months, and approximately 12.76 pounds over five years without interventions for depressive symptoms as shown in Table 4. For obese females with a score of 30 on the CES-D, one could predict a weight gain of 14.65 pounds in five years. For an obese male with a CES-D score of 10, one could predict a weight gain of 12.58 pounds at five years, and with a score of *30*, predict a weight gain of *14.48* pounds at five years.

Insert Table 4 Here

Figures 1 (females) and 2 (males) graphically depict how weight increased among obese females and males as their CES-D scores increased, and how weight gain differed between obese females and males from normal weight and overweight subjects.

Insert Figure 1 Here

Insert Figure 2 Here

Discussion

The growing prevalence of depression and obesity in American society is forecasted to continue despite national campaigns to combat the problem (Carpenter, Hasin, Allison, and Faith, 2000). Currently, obesity rates have increased to 30.5%

among adults, and more women were obese (33%) than men (28%) (Center for Disease Control, 2002; Flegal, Carroll, Ogden, & Johnson, 2002). All subjects in the RENO Diet-Heart study were screened to assure they were a healthy sample. Despite excluding some potential subjects due to self-reported history of depression, the effect of depressive symptoms on weight gain was still detected in both obese female and male subjects.

Data supported that risk for gaining weight related to increased depressive symptoms was greatest for obese female and male subjects than for normal weight and overweight subjects after controlling for age. The purpose of this study was to investigate if baseline measures of depression, gender, weight group, and age in overweight and obese women predicted weight change over five years as compared to men.

Many investigators have suggested that depression be included in the obesity model (Blehar & Oren, 1995; Carpenter, Hasin, Allisonk, & Faith, 2000; Friedman & Brownell, 1995; Khaodhiar, McCowen, & Blackburn, 1999; Klesges, Klem, & Klesges, 1992) as depression (or depressive symptoms) may contribute to weight gain. Data from the RENO Diet-Heart study supported that obese individuals gained the most weight as their CES-D scores increased.The investigators recognized that the effects of depressive symptoms on weight gain may be underestimated in the RENO study due to the exclusion criteria of a history of depression. Also, despite overall CES-D scores within normal limits and with little variability, as increased depressive symptoms increased among the obese, so did their weight. Additional research is

warranted to evaluate whether increased depressive symptoms affect weight gain in a community population that does not exclude depressed individuals, and to examine potential gender differences.

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Table 1

Mean CES-D Scores with Standard Deviations Over Five Years Stratified by Gender and Weight Group

	Baseline	Year 1	Year 2	Year 3	Year 4
	X(SD)	*X(SD)*	*X(SD)*	*X(SD)*	*X(SD)*
Males	4.97(5.30)	5.92(5.52)	5.24(6.24)	5.55(6.50)	4.79(6.0)
Normal wt	5.07(4.82)	5.07(4.82)	5.08(6.36)	5.05(5.96)	4.74(6.74)
Overwt	5.88(6.7)	5.56(5.4)	5.67(6.19)	5.47(6.89)	4.85(6.31)
Obese	7.8(8)	8.7(8.6)	5.67(6.19)	5.47(6.89)	6.45(6.7)
Females	5.51(6.24)	7.26(7.95)	6.83(7.30)	7.28(8.34)	6.87(7.67)
Normal wt	5.05(5.51)	6.59(6.87)	5.95(6.51)	5.77(5.93)	5.92(6.84)
Overwt	4.73(4.67)	4.73(4.67)	7.48(7.51)	7.89(8.51)	7.7(8.54)
Obese	8.03(8.15)	10.7(9.98)	8.04(8.53)	10.43(11.47)	8.5(8.37)

Table 2

Results of the Final Data Analysis Excluding Outliers: The Mixed Model (Third Level)

Effect	Numerator degrees of freedom	Denominator degrees of freedom	*F* value	*p* level of significance
Gender	2	3000	1189.50	<.0001*
Age	1	3000	3.52	0.0606
Transformed CES-D * weight group	3	3000	167.19	<.0001*
Time * gender	2	3000	25.07	<.0001*
Time * age	1	3000	43.26	<.0001
Transformed CES-D * time * weight group	3	3000	3.91	0.0085*

Note. * indicates significance at $p<.05$. The model was adjusted for age, gender, and weight group by CES-D score.

Table 3

Results of the Final Data Analysis Excluding Outliers: The Second Level

Effect	Gender	Weight Group	Estimate (*b*); (CI±)	Standard error	Degrees of freedom	*t* value	*p*
Gender	1= female		159.70 (±8)	4.08	3000	39.10	<.0001
	2=male		194.32 (±7.89)	4.02	3000	48.26	<.0001
Age			-0.13 (±.14)	0.07	3000	-1.88	0.061
NLCESD* Weight group		0= normal	-13.03 (±2.63)	1.34	3000	-9.73	<.0001
		1=over-weight	2.16 (±2.56)	1.31	3000	1.65	0.09
		2= obese	17.31 (±2.51)	1.2786	3000	13.54	<.0001
Time* gender	1= female		1.67 (±.47)	0.24	3000	6.82	<.0001
	2=male		1.65 (±.451)	0.23	3000	6.90	<.0001
Age*time			-0.028	.004	3000	-6.58	<.0001

		(±.008)				
NLCESD*	0=	0.041	0.080	3000	-0.52	0.60
Weight	normal	(±.16)				
group*	1=over-	0.089	0.079	3000	-1.12	0.26
time	weight	(±.15)				
	2=	0.18	0.078	3000	2.34	0.019
	obese	(±.15)				

Note. "NLCESD" represents the transformed CES-D score. The model was adjusted for age, gender, and weight group by CES-D score.

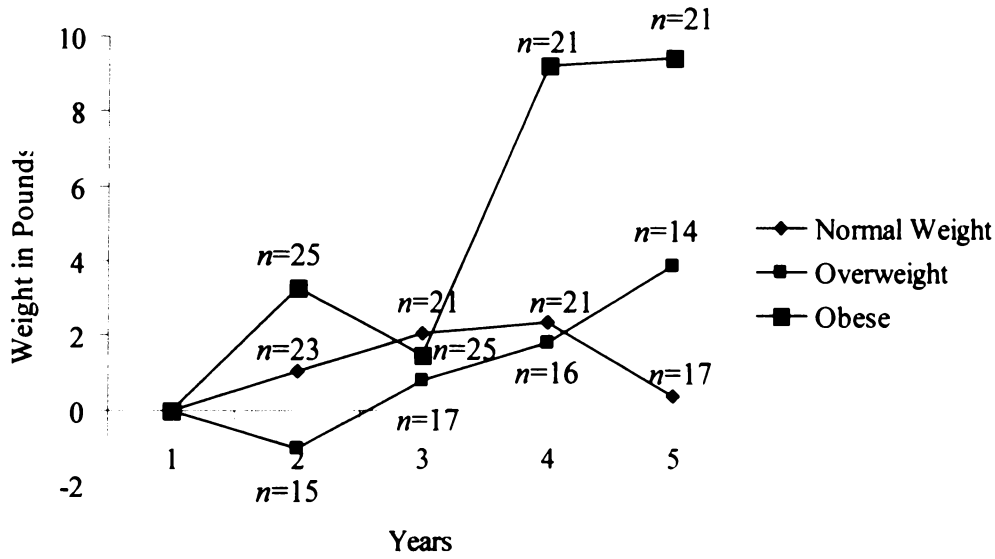
Table 4

Predicted Weight Changes over 5 Years Among 30-Year Old Subjects by CES-D Scores by Weight Group

Transformed CESD score	Normal Weight Females (pounds)	Normal Weight Males (pounds)	Over-Weight Females (pounds)	Over-Weight Males (pounds)	Obese Females (pounds)	Obese Males (pounds)
CESD=10	7.39	7.21	6.25	6.07	12.76	12.58
CESD=20	7.12	6.94	5.68	5.50	13.94	13.76
CESD=30	6.96	6.78	5.33	5.15	14.65	14.48

Figure 1

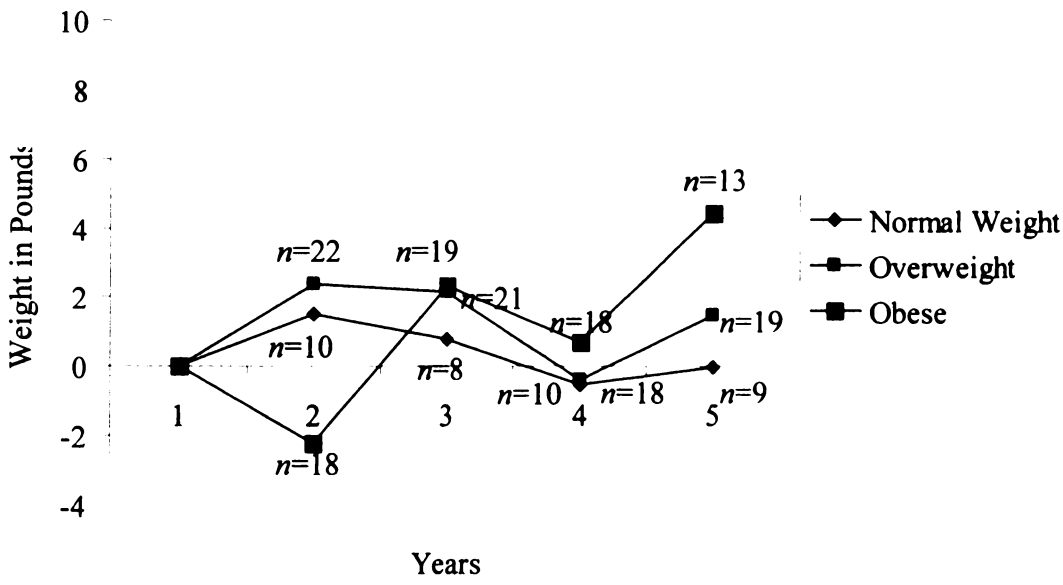
Weight Changes in Pounds for Females with CESD Scores ≥17 Over Five Years



Note. Baseline *n* for normal weight females = 24; baseline *n* for overweight females = 17; and baseline *n* for obese females = 25.

Figure 2

Weight Changes in Pounds for Males with CESD Scores ≥17 Over Five Years



Note. Baseline *n* for normal weight males = 12; baseline *n* for overweight males = 26; and baseline *n* for obese males = 20.

CHAPTER 3

Methods

There is evidence that individuals seeking treatment for overweight or obesity may suffer from underlying depressive symptoms, and because of their depressive symptoms, may fail at weight loss. Data from the RENO Diet-Heart Study (St. Jeor, 1997) were used in a secondary data analysis to examine the relationships among body weight, depressive symptoms, gender, initial weight groups and age over five years. Information from the original RENO Diet-Heart study will be presented in this chapter, along with demographics from the original study, inclusion and exclusion criteria, and statistical testing.

Purpose

The purpose of this study was to investigate if baseline measures of depression, gender, weight group, and age in overweight and obese women predicted weight change over five years as compared to men. A secondary purpose of this study was to examine if depressive symptoms positively correlated with weight change over five years. This study, a secondary analysis of archival data from the RENO Diet-Heart study (St. Jeor, 1997), focused on women but used men as a comparison group.

Overview of the Research Design

The design for the original RENO Diet-Heart study (St. Jeor, 1997) was an eight year, prospective, observational cohort study among subjects in Reno, Nevada. The study purpose was "to study weight maintenance behavior, weight trends and fluctuations, and behavioral differences between healthy normal weight...and mild to

moderately obese adults retrospectively by history and prospectively over five years" (St. Jeor & Dyer, 1997, p. 4). Specifically, the study examined "the effects of weight-fluctuations and weight changes on cardiovascular disease risk factors in both normal weight and obese healthy subjects" (Pattishall & Hayes, 1997, p. 30). Complete data were available for the first five years, from 1985 through 1990, due to a change in grant funding.

A cohort study is a type of study that follows groups of individuals, known as a cohort, over time (longitudinally). The RENO Diet-Heart study (St. Jeor, 1997) followed a cohort of subjects to examine the development and progression of cardiovascular risk factors and disease among adults. One advantage of a cohort study was that the design limited selection bias (such as enrolling only depressed individuals, or only obese individuals; Hennekens & Buring, 1987).

Limitations of the Research Design

The major limitation in the RENO Diet-Heart study was that potential subjects who, upon screening, were excluded from participating if they acknowledged that they had been diagnosed with depression, suffered a depressive episode, missed work due to feeling 'depressed', or took an antidepressant medication within the last five years. Results were biased toward a sample that was essentially not depressed. Consequently, the sample was not a 'true' reflection of the prevalence of depression among Reno residents. Findings were limited to residents of Reno, Nevada, and could not be generalized to other populations in different areas around the country.

St. Jeor and Dyer (1977) stated that weight change was most likely due to "a multitude of complex, interrelated behavioral, environmental, psychological, nutritional, and psychological factors...but questions still remain regarding how best to measure these factors as they naturally occur or are affected by dieting" (p. 6). The investigator postulated that testing variables in this secondary analysis study (e.g., CES-D scores, weight, gender, and weight groups) may provide evidence that overweight and obese depressed women may gain weight as a result of increased depressive symptoms.

Assumptions

Assumptions for the original RENO Diet-Heart study were that all variables were normally distributed within the population.

Definitions

For the current study, 'body mass index' was defined as weight in kilograms (or kg) divided by height in meters squared (m^2) (National Institutes of Health, National Heart, Lung, and Blood Institute, 2000).

'Weight groups' *were* based on body mass index, and reflect whether an individual is of normal weight (a BMI <24.9), overweight (a BMI of 25-29.9), or obese (a BMI >30).

'Dropouts' were classified for this study as subjects who missed 3 to 4 out of 5 annual assessment appointments. Characteristics of dropouts were examined to explore how dropouts differed from subjects who remained in the study.

Description of the Secondary Database

The RENO Diet-Heart study contained variables that contributed to the current investigation (Table 3). Obesity was defined in the original study (using a 1985 guideline) as "a mean BMI of 24.4 for women and 26.5 for men...interpretations for the obese may be on the conservative side using these data" (Foreyt & St. Jeor, 1997, p. 47). The sample included working class men, women, and students who were age 18 to over 60 years of age and who were in good self-reported health. Subjects were recruited for the study by word of mouth, newspaper advertisements, and radio. There were complete data for years 1 through 5 that were analyzed in the current investigation.

Data Collection Procedures

Subjects were interviewed and enrolled in the RENO Diet-Heart Study at the University study site. They signed informed consents allowing investigators to obtain subjective and objective information biannually. All subjective data were obtained by subject self-report using questionnaires, and objective data (e.g., weight, blood samples, blood pressure) were collected by team investigators at baseline and biannually for five years. Subjects were given free gifts annually (e.g., thermal cups) for their participation, as they were not paid.

The protocol for testing subjects included eight measurement categories that included:

"1. Baseline and yearly update, health, weight, nutrition and activity questionnaires.

2. Structured interviews.

3. Body measurements.

4. Nutrition and dietary intake.

5. Sleep, activity, and exercise assessments.

6. Behavior and health questionnaires.

7. Laboratory analyses.

8. Cardiovascular examination" (St. Jeor & Dyer, 1997, p. 7).

After pre-screening took place, subjects attended a one-hour orientation meeting where they signed an informed consent, and had their height, weight, and % ideal body weight recorded, set up future appointments, and received questionnaires to fill out at home. The questionnaires took approximately two hours to fill out. The first visit took approximately two hours, where the investigators reviewed the questionnaires for completeness and recorded a 24 hour diet and activity recall questionnaire. A body graph was obtained, along with skinfold measurements, body circumferences, blood pressures, pulse, and respirations along with height and weight. At the end of the first visit, subjects were instructed to maintain records for one week on activity and eating, as well as providing a urine sample. The second visit took one hour, and included a review of records by the investigators, urine collection, a fasting blood sugar, metabolic measurements, bioelectrical impedance, along with weight, blood pressure, and pulse. The third visit took approximately 45 minutes, for the investigator to review lab results with the subject. A cardiovascular exam was performed, along with an electrocardiogram. After the third visit, subjects scheduled future return visits at four and eight months for the first year, then annually every six

months for five years. Subjects were instructed to schedule their annual visits within a two week timeframe around their anniversary visit (month, date, and year). All variables in the total RENO Diet-Heart study are presented in Table 3.

Sources of Data for Secondary Analysis

For the present investigation, specific variables were selected for analysis. These variables were annual CES-D scores and body mass indices, and measures of body weight (every six months). The Center for Epidemiologic Studies on Depression (CES-D; Radloff, 1977) was given annually to all subjects as one measure of depressive symptoms, and was designed for use in a community population.

A new variable, weight group, was created that reflected current guidelines for body mass index (Nies, Cook, & Hepworth, 1999; NHLBI, 1998; NHLBI, 2000) where a normal weight BMI ranged up to24.9, overweight ranged from a BMI of 25 to 29.9, and a BMI >30 was considered obese.

Setting and Sample

All data collection took place at the University of Reno, Nevada Health Sciences Center. The sample included working class men, women, and students who were in good self-reported health, recruited for the study by word of mouth, newspaper advertisements, and radio. A stratified sampling technique (2x2x5) was used to select subjects. Gender (male and female) and weight group classifications (normal or obese, with obese having a BMI >27kg/m^2) were stratified into age categories by decades that ranged from 20-29 years of age up to 60+ years. The purpose for using stratification was to enroll 25 male and female subjects, either of

normal weight or obese, for each age decade. The number of subjects who completed all five years of data collection was (a) 96% at year two, (b) 92% at year three, and (c) 90% at years four and five. Hennekens and Buring (1987) stated that study validity is challenged when attrition reaches 40%.

Researchers recruited 253 female subjects and 255 male subjects, for a total of N=508. Out of the total n for each gender, 112 females and 122 males were classified as obese. The mean age for women was 44.6 years ($SD\pm$14.0 years) and 44 years for men ($SD\pm$14.3 years). The mean body mass index (BMI) for women was 26, and the mean BMI for men was 27. The majority of subjects was Caucasian (93%), white collar (51%), married (72%), and had at least one year of college education (89%). Self-reports of mental health was found to be rated "good to excellent" (St. Jeor & Dyer, p. 7) in 95% of the subjects.

Subject Inclusion/Exclusion Criteria

Subjects were originally screened for the RENO Diet-Heart study and met certain inclusion criteria. Inclusion criteria for the study required that subjects had to agree to be measured at certain times during the year, to remain within the Reno area for five years, and be willing to complete multiple self-reports, questionnaires, and labwork (Dyer, 1997). Potential subjects had to be between the ages of 20 and 75 years, between 80% and 180% of ideal body weight "as determined from the midpoint of weight for height of medium frame from the 1959 Metropolitan Height and Weight Tables for Desirable Weights at the initiation of the study in 1985" (Scott, 1997).

Subjects were excluded if they experienced depression within the last five years, were diagnosed with depression, missed work due to depression, or took antidepressants. Other exclusion criteria were that subjects could not be pregnant nor lactating; have no history of cardiac disease nor taking cardiac medications; and presently "working or retired from at least a half-time position or student at least half-time or married to someone who is employed " (St. Jeor, Simpson, & Daugherty, 1997, p. 21).

Sample Variables

There were several variables under investigation in this study (Table 3). Descriptive information is presented on certain variables for gender, profession, marital status, ethnicity, socio-economic status, and education (Table 4), weight (measured in pounds every 6 months over 5 years) (Table 5), weight, height, and body mass index by gender (Table 6) and weight group (Table 7), and by gender and weight group assignment (Tables 8 and 9). In addition, CES-D scores (measured annually over 5 years) are presented, stratified by gender (Table 10), and gender by weight group (Table 11). A score equal or greater than 17 on the Center for Epidemiological Studies-Depression scale (CES-D; Radloff, 1977) indicates a higher level of depressive symptoms.

A new variable was created that reflected current guidelines of body mass indices. A normal body mass index (BMI) is less than 24.9, an overweight BMI ranges from 25.0-29.9, and an obese body mass index is above 30 (Nies, Cook, & Hepworth, 1999; NHLBI, 1998; NHLBI, 2000). These weight groupings differ from

the RENO Diet-Heart study, where weight was dichotomized into normal weight subjects who had a BMI of <26.9, and obese subjects who had a BMI >27. The major outcome for this study was to predict if overweight and obese females gained weight over time due to increased depressive symptoms, as compared to normal weight females and males.

Instrumentation

The Center for Epidemiologic Studies on Depression (CES-D; Radloff, 1977) was given annually to all subjects as one measure of depressive symptoms. The CES-D measures depressive symptoms one would experience during the previous week, and was designed for use in a community population. In addition, the CES-D was designed to be administered by both experts and those without mental health expertise. The CES-D does not differentiate between types of depression nor should it be used to diagnose depression (Miller & Harrington, 1997; Radloff, 1977). The 20-item scale was developed from other established depression scales, and has demonstrated reliability and validity (Table 2).

Each question on the CES-D (Appendix B) has four possible answers: (a) 0 = rarely or none of the time, (b) 1=some or a little of the time (1-2 days per week), (c) 2=occasionally or moderately (3-4 days per week), and (d) 4=most or all of the time (4-7 days per week). Scores range from 0 - 60, and the cutoff score for depression is 17. Four questions on the CES-D (4, 8, 12, and 15) "were scaled in reverse, 3 for "rarely or none of the time", and 0 for "most or all of the time", and [were] reverse scored before being included in the total score" (Miller & Harrington, 1997, p. 457).

This scale takes approximately ten minutes to complete. Coefficient alphas range from α=0.85 in community samples to approximately α=0.90 in psychiatric samples Individuals who score 17 and above should be referred to a professional practitioner (Yonkers & Samson, 2000; Radloff, 2000). Body weights were consistently measured "using a standard balance beam scale that was calibrated frequently...to the nearest quarter pound (Scott & Zahrt, 1997, p. 57).

Results based on the original data indicated that overall, mean depression scores did not differ between men and women (Miller & Harrington, 1997). They found that obese subjects had higher mean CES-D scores compared to normal weight subjects (X = 6.6, SD = 7.2; X = 5.2, SD = 5.6). Although obese subjects scored somewhat higher, the difference was not substantial as the mean scores were well below the threshold that indicated subject intervention was needed.

Database Development

Permission was given to this investigator by the original primary researcher (Appendix C). Data were given to the current investigator on a CD-ROM. The original database was screened three times, compared to the typed data dictionary and the published book for missing variables and data (St. Jeor, 1997).

An a priori power analysis was conducted by the investigators in the RENO Diet-Heart Study (St. Jeor & Dyer, 1997) to determine the sample size necessary for their study. The investigators stated that no previous studies were published that examined weight trends over time to assist in estimating an effect size. A decision was made to recruit 500 subjects, "25 normal and 25 obese subjects of each sex in

each decade of age from 20 to over 60" (p. 13) with a planned attrition rate of 20%, with a resulting N=400 at the end of five years. For a total N=400, "the power was 0.85 for detecting correlations of 0.15 between variables based on a two-sided test with a Type I error of 0.05" (p.13).

Hypotheses

The two hypotheses posed for this secondary data analysis were:

1. Depressive symptoms (CES-D $\geq$17) at baseline will predict weight gain in overweight and obese females at five years (excluding baseline measures) as compared to normal weight females and all males, controlling for age, gender, and CES-D score by weight group.

2. Controlling for gender, depressive symptoms (CES-D scores) would positively correlate with body mass index (BMI) annually over five years.

Based on the literature, the investigator hypothesized that overweight and obese, depressed females would gain more weight at five years than overweight or obese males, or normal weight females and males. If a relationship exists between untreated depressive symptoms and weight gain, treatment guidelines for obese women could be impacted to consider obesity and depression as related co-morbidities, and could contribute to existing knowledge about women, depression, and obesity.

Data Analysis

Data will be presented and stratified by gender, and gender by weight group. Descriptive analyses will be performed using the Statistical Package for the Social

Sciences (SPSS) for Windows, version 11.5, and for the inferential statistics, SAS (v.8.0.2; Windows version 5.1.2600) will be used. Descriptive statistics will be computed for each variable to check for distributional qualities such as normality. Baseline characteristics of the cohort will be examined and stratified by gender, such as CES-D scores, age, education, and socioeconomic status. In addition, data are analyzed for subjects who dropped out of the RENO study. Confidence intervals for the estimates (*b*'s) are presented. To calculate confidence intervals, one adds or subtracts the estimate (*b*) from 1.96*standard error for the *b* (Cohen, Cohen, West, & Aiken, 2003). Standard errors are analogous to standard deviations used to describe score dispersion among subjects; however, the standard error of the estimate is the square root of the variance of the residuals around the regression line (Pedhazur, 1997). Researchers want to determine if their results are representative of the theoretical sampling distribution. If the standard error is relatively large, the less precision one has in trying to estimate the population. In addition, if a confidence interval crosses over zero, then the investigator would fail-to-reject the null hypothesis, the hypothesis of no effect. Pedhazur, along with Cohen, Cohen, West, and Aiken, advocated using confidence intervals instead of null hypothesis testing for the above reasons. For this study, both confidence intervals will be presented along with traditional probability (*p*) levels.

Previous research indicated that CES-D scores often are skewed to the right (Aneshensel, Frerichs, & Clark, 1981; Aneshensel, Frerichs, & Huba, 1984), as the estimated prevalence of depression is around 10% (American Psychological

Association, 2002). As expected, the majority of scores were clustered (highly skewed to the left) which indicated that in general, the RENO subjects were not depressed. Aneshensel, Frerichs, and Clark (1981) and Aneshensel, Frerichs, and Huba (1984) recommended performing a score transformation using a natural logarithm+1 for all scores including the cutoff score which standardized the CES-D scores.

Statistical Analysis-The Hierarchical Linear Model

Wu (1996) encouraged hierarchical linear modeling (a type of multi level modeling) as an alternative data analytic procedure to examine how individuals grow over time, and to examine the effect of predictor variables at different levels. In the first level, a regression equation is developed for each individual (Wu, Clopper, & Wooldridge, 1999), and the parameters (intercepts and regression coefficients) are estimated from data. In the second level, the intercept (*a*) and regression coefficient (*b*) assume the role of dependent variables as a function of the predictor variables. In this level, the investigator determines if they significantly differ from zero using the *t*-test, the test for a regression coefficient (Pedhazur, 1997; Wu, 1995). The third level (or mixed model) examines the overall interaction between the predictor variables and whether they differ from one another. Results are generated into a type of analysis of variance output, with degrees of freedom for both the numerator and denominator, and an *F* value is reported with a significance level.

Advantages of using the linear mixed model.

The advantage of the hierarchical model over traditional analysis of variance was that subjects needed only three data points to build their regression equation, as opposed to an analysis of variance assumption that excludes subjects with missing data (Kenny, Bolger, & Kashy, 2002; Wu, 1996). Another advantage to using a hierarchical linear mixed model was that investigators could examine both individual and intraindividual changes over time as a function of individual characteristics (or covariates). Since the purpose of this study was to investigate if baseline measures of depression, gender, weight group, and age in overweight and obese women would predict weight change over a five year period. hierarchical linear modeling was appropriate to use.

The linear mixed model approach is not constrained by 'compound symmetry', a type of sphericity (Maxwell & Delaney, 2000), where variances and covariances in a covariance matrix are equal. This is an assumption for analysis of variance for repeated measures that is not required for hierarchical linear modeling (Wu, 1997). In addition, the linear mixed model is flexible in that subjects can be measured at roughly the same time during follow-up assessments (e.g., weight, CES-D scores) are gathered at the same time points for all subjects" (Wu, 1996, p. 77).

Hox (2002) emphasized the advantages of hierarchical linear modeling that were (a) every subject has a growth curve, using the *b* to generate the model, (b) measurement times will vary, and some individuals will have missing data, but this does not present a problem, (c) one can model covariances, (d) when using restricted

Maximum Likelihood procedure (as in this study), both the *F* and *t* tests can be obtained by the regression output, (e) hierarchical linear modeling allows for higher levels of examination among higher-level groups, and (f) 'time' can be either allowed to vary or to be a constant that allows modeling for both the individual and the group(s).

The hierarchical linear model level 1 equation.

This first step in the model focused on creating a regression equation for each subject, notated as

$$Y_{ij} = a_i + b_i X_j + e_{ij} \quad (1)$$

where the dependent variable (Y) denoted the weight for a subject 'i' in group 'j'over five years (X_j denoted times subjects were weighed every six months, notated as j=1,2,3...9). The a_i intercept described each subject's interception to the Y axis. The b_i regression coefficient reflected the individual changes in weight every six months the subject was weighed. The error term (e_{ij}, or residual), reflected the error for each subject when predicting *Y* from *X* (Kenny, Bolger, & Kashy, 2002).

The level 2 equations.

Second level equations (among subjects) examined the effects of the predictor variables, (a) age upon entering the study, (b) gender, and (c) a baseline depression score by the subject's weight category (either normal weight, overweight, or obese), and their effect on two parameters, the intercepts (a_i) and slopes (b_i). The *t*-test of

significance determines if the estimate significantly differs from zero while holding the other predictor variables constant (Pedhazur, 1997). In the traditional output of multiple regression, an R^2 change denotes the amount of additional variance a predictor contributes to the overall equation. Recalling that the second level *a* intercept and *b* coefficients are now considered dependent variables, the first level 2 equation (for the intercept) is notated as

$$a_i = \text{entage}_i\beta_1 + \text{Female}_i\beta_2 + \text{Male}_i\beta_3 + \text{wtcat}_1*\ln(\text{CES-D}_i + 1)\beta_4 + \text{wtcat}_2*\ln(\text{CES-D}_i+1)\beta_5 + \text{wtcat}_3*\ln(\text{CES-D}_i + 1)\beta_6 + d_{oi} \quad (2)$$

where 'a_i' is a function of the predictors of age multiplied by the β weight for age; gender, the β weight for either female or male; plus the interaction between weight category (either normal, overweight, or obese) multiplied by the β weight of the transformed natural logarithm CES-D score (plus 1). An error term (*d*) is generated for the intercept (*a*). Although the intercept was important, the purpose of this study focused on weight gain over time (a change in the slope, or estimate, or β) as a function of age, gender, and CES-D scores by weight group, notated as

$$b_i = \text{entage}_i\beta_7 + \text{Female}_i\beta_8 + \text{Male}_i\beta_9 + \text{wtcat}_1*\ln(\text{CES-D}_i+1)_i\beta_{10} + \text{wtcat}_2*\ln(\text{CES-D}_i+1)\beta_{11} + \text{wtcat}_3*\ln(\text{CES-D}_i+1)\beta_{12} + d_{1i} \quad (3)$$

where β_i denotes the slope or rate of change for the individual '*i*', as a function of age (their current age multiplied by the β weight for time*age interaction), gender (the β weight for the individual's gender multiplied by the β weight for the gender*time interaction), and their weight category β weight (normal weight, overweight, or obese) * by their transformed CES-D score (+1) * by time interaction.

The mixed procedure, or level 3 analysis.

The third "omnibus" procedure (Raudenbush & Bryk, 2002), also called the "cross level interaction" (Cohen, Cohen, West, & Aiken, 2003) or the mixed model, examines the interaction between the fixed effects of gender, age, and CES-D score by weight group, with and without 'time' as a covariate. Results will be reported similar to an analysis of variance output using the terms 'degrees of freedom', an *F* value, and a significance level that denotes if predictor variables significantly differed from one another.

When interpreting results of a hierarchical linear mixed model, the third level (or overall/omnibus) test is examined first to determine the overall significance of the predictor variables. 'Time' was added to each of the level three predictors of gender, age, and transformed CES-D +1 score for a total of six predictors to examine if the interaction between time, weight group, and transformed CES-D score predicted weight change over time. If an omnibus test for the predictor variables in question is significant, the second level results must be examined to find out where the difference occurred, similar to evaluating results from an analysis of variance output.

The β represents the amount of change in Y for every unit of change in X, holding constant the other predictor variables. To interpret the substantive contribution of each predictor variable, the b is the appropriate coefficient to use as b remains stable under the effects of the covariates as they differ in populations or settings. However, for the ease of reading, the β will be used to denote the b coefficient.

Regression diagnostics.

A series of regression diagnostics were completed for 'weight' vs. 'time' for individuals to identify outliers associated with the slope (β) in equation 3. Outlier scores can increase or decrease the mean, standard deviation, correlation or regression coefficient, and the t-ratio (Jacobsen, 1981). Jacobsen described several methods to deal with outlier scores, including elimination or plotting the residuals. Pedhazur (1997) stated that the simplest method for analyzing residuals (or error in predicting Y from X) was to plot "standardized residuals against corresponding X's or predicted Y's (in raw or standardized form)" (p. 36). The equation for a standardized score (z score) on the residuals was obtained by subtracting the raw score from the mean (in the numerator), divided by the standard deviation (denominator).

Estimation methods: The maximum likelihood and restricted maximum likelihood models.

The specific estimation method used in this analysis was the restricted maximum likelihood model (REML), subsumed under the maximum likelihood model. In the full maximum likelihood model, "both the regression coefficients and

the variance components are included" (p. 38), whereas the restricted maximum model estimates the regression coefficients (*b*) in the second level of the model, estimating "the variance components after removing the fixed effects from the model" (Hox, 2002, p. 38).

Maximum likelihood is an estimation procedure for how one estimates the regression coefficients (β). The distribution for the -2loglikelihood is the chi square (Pedhazur, 1997) that is used to determine significance between the reduced and full models. Investigators want to know what are the most likely β's (estimates, or parameters) generated from our data that assesses how the model 'fits' the data. The lower the -2loglikelihood (or higher likelihood), the better the fit. As an analog consider when a regression equation contains a large amount of predictor variables, this results in a decreased R^2 in traditional regression terms ('R^2' denotes "the proportion of variance of the dependent variable accounted for by the independent variables" (Pedhazur, 1997, p. 103). When too many predictors are vying for their share of variance with the dependent variable, this can inflate the R^2 due to chance (Garson, 2003). What the adjusted R^2 procedure does is to decrease the R^2 as predictors are added to the equation, the same as the -2loglikelihood.

One solves the dilemma of the effect of multiple predictor variables in a hierarchical mixed model with a Bayesian Inference Criterion (BIC; Cohen, Cohen, West, & Aiken, 2003) which is similar to an adjusted R^2. The BIC takes into consideration "the number of predictors. The BIC may be negative or positive in value; the more negative the value of the BIC, the better the fit" (p. 509). One should

compare the BIC in both the full and reduced model and choose the BIC with the lesser number.

Procedure for Hypothesis Testing.

In the current investigation, the probability level for rejecting the null hypothesis was set at $p \leq .05$, denoting the probability of obtaining a result greater than the tabled value would be obtained by chance. Clinical significance will be emphasized. When appropriate, Bonferroni adjustments will be made to insure an overall Type I error rate. For the second hierarchical linear model procedure, the output will be presented similar to output generated from a multiple regression procedure. Knapp (1994) encouraged presenting the number and name of independent variables, the group sample size, and both standardized and unstandardized regression coefficients, along with confidence intervals for the regression coefficients. Confidence intervals will be presented along with the traditional p values denoting significance.

Assumptions for Statistical Tests.

Assumptions for a linear mixed model are that the dependent variable (in this case, weight) is a linear function of gender, weight group, and time. Bryk and Raudenbush (1992) identified additional assumptions that included error terms that are generated in equation 1 are independent from equation 2, and errors in both levels 1 and 2 are independent from each other.

Ethical Considerations

Informed consent for the original RENO Diet-Heart study was obtained from each participant as well as from the University of Nevada (St. Jeor, 1997). The current study qualifies for an expedited review according to the University of Kansas Guidelines for Human Subjects (Appendix A). All results will be reported in aggregate.

Procedures to Protect Confidentiality

The database contains no subject names, only numbers. Printed results will be kept in a locked cabinet for two years, then destroyed. An original copy of the dissertation, tables, figures, and results will be kept by the investigator on paper and on the computer.

Summary

To reiterate, there is empirical evidence that women suffer from depression more than men, and currently, more women are obese. The effect of depression on weight change has not been investigated in the literature. Therefore, the purpose of this study using data in a secondary data analysis from the RENO Diet-Heart study (St. Jeor, 1997), was to investigate if baseline measures of depression, gender, weight group, and age in obese and overweight females would predict weight change over five years, compared to normal weight females. Males were used as a comparison group to explore any gender differences. A secondary purpose for this study was to examine if depressive symptoms are positively correlated with BMI over five years.

Chapter 4

Results and Conclusions

Overview

The purpose of this study was to investigate if baseline measures of depression, gender, weight group, and age in overweight and obese women predicted weight change over five years as compared to men. A secondary purpose of this study was to examine if depressive symptoms were positively correlated with weight change over five years. This study was a secondary analysis of archival data from the RENO Diet-Heart study (St. Jeor, 1997).

Hypotheses

The two hypotheses posed for this secondary data analysis were:

1. Depressive symptoms (CES-D $\geq$17) at baseline will predict weight gain in overweight and obese females at five years (excluding baseline measures) as compared to normal weight females and all males, controlling for age, gender, and CES-D score by weight group.

2. Controlling for gender, depressive symptoms (CES-D scores) would positively correlate with body mass index (BMI) annually over five years.

In this chapter, demographics, physiologic measures (body weight and body mass indices), and CES-D mean scores and standard deviations are presented for subject age and weight group. Results for each research question are presented for (a) all subjects, (b) grouped by gender, (c) grouped by weight group (normal weight, overweight, and obese), and (d) grouped by gender and weight group.

Limitations of the Research Design

In the RENO Diet-Heart study, potential subjects were excluded from participating who, upon self-report, acknowledged either a diagnosis of depression, suffered a depressive episode, missed work due to feeling 'depressed', or took an antidepressant medication within the last five years. By excluding individuals who reported a history of depression, generalizing results of this study will be limited as they will reflect a healthy, non-depressed sample.

Subjects resided in Reno, Nevada, which limits generalizing to other populations around the country; however, the data may provide new information to predict if obese women who score above the cutoff point on a depression measure actually gain weight over time. Another limitation was that data were collected almost twenty years ago and reflected a white, middle-class population (St. Jeor, Simpson, & Daugherty, 1997). On the other hand, the RENO sample could serve to function as a healthy population to which future comparisons can be made.

Subjects

The average age for all subjects (N=508) was 45 years (SD=14.13), and ages ranged from 19 to 77 years. Eighteen female and 15 male subjects were over the age of 65. Table 4 provides baseline demographic characteristics for subjects grouped by gender. Overall, the majority of subjects worked in some type of professional role, followed by clerical workers, homemakers, physical laborers, technical workers, and other work not specified (there was no elaboration on what occupations comprised "professional"). The majority of subjects were married, White (not Hispanic), and

their monthly income ranged from $2,001 to $4,000 after taxes. The majority of subjects had completed formal education for twelve or more years. One hundred eighty five subjects achieved post-graduate course work beyond their initial college degree.

Physiologic Measures

At baseline, all subjects' average weight was 175 pounds (*SD*=36.44), average height was 67 inches (*SD*=3.78), and average BMI was 27 (*SD*=4.58). According to current BMI indices that distinguish normal weight (a BMI <25), from overweight (a BMI from 25-29.9), and obese (a BMI >30), the average subject was overweight. Details are presented in Table 6 for overall weights and body mass indices. Obese subjects were in the minority for both females (27.1%) and males (24.6%). The greatest proportion of females (*n*=113) were normal weight (45%), while the greatest proportion of males (*n*=113) were overweight (45.56%). Tables 7 through 9 provide information stratified by weight groups.

Weight changes over time.

Average weight changes over time were examined between (a) genders, (b) between genders and weight groups, and (c) between genders, weight groups, and CES-D scores ≤16 or ≥17. Weight gain was defined as an increase in weight, and weight loss was defined as a decrease in weight. Overall, females as a group gained an average of 6.06 pounds from baseline to year 5, and males as a group gained an average of 8.47 pounds from baseline.; however, all annual mean CES-D scores remained under 17. The lowest mean CES-D scores occurred in the normal weight

groups. Scores increased for the overweight groups, and were the highest for the obese groups.

The Y-axis on each scale in Figures 1 to 9 were purposely set to range from -2 to 10 to provide a consistent scale to pictorially represent annual weight changes between the different groups under analysis. Weight gain from baseline to five years was highest among normal weight females who gained 3.93 pounds on average, while overweight women gained 3.21 pounds (Figure 1). Obese females gained the least amount, 1.73 pounds. For males, the average weight gain occurred among the obese who gained an average of 6.35 pounds over five years (Figure 2).

Normal weight females gained an average of 3.93 pounds (Figure 3), while normal weight males gained an average of 5.04 pounds over five years. Overweight females gained an average of 3.21 pounds (Figure 4), while overweight males gained an average of 2.27 pounds over five years. Obese females gained 1.73 pounds (Figure 5) while obese males only gained 6.34 pounds at five years.

For subjects who had CES-D scores at or above 17, normal weight females gained 0.38 pounds (Figure 6), while normal weight males lost 0.01 pounds over five years (Figure 7). Overweight females gained 3.88 pounds (Figure 6), while overweight males gained 1.46 pounds (Figure 7). Obese females gained 9.44 pounds (Figure 6), while obese males gained 4.45 pounds (Figure 7). Obese females scored higher on mean CES-D scores than all other gender-classified weight groups (Table 11).

For subjects who had CES-D scores at or below 16, normal weight females gained 3.28 pounds (Figure 8), while normal weight males gained 5.39 pounds (Figure 9) over five years. Overweight females gained 3.29 pounds (Figure 8) while overweight males gained 2.68 pounds (Figure 9) over five years. Obese females gained 0.55 pounds (Figure 8) while obese males gained 5.86 pounds over five years (Figure 9).

Depressive Symptoms and Screening

During the screening process for the RENO Diet-Heart study, potential subjects were screened for depression by self-report, interview, and if they answered "very often" to screening question # 13, "How often are you depressed?" (choices were never, seldom, often, or very often) (St. Jeor, Simpson, & Daugherty, 1997, p. 26; S. T. St. Jeor, personal communication, June 5, 2003). Thirty females and 6 males (out of N=504, or roughly 7%) were excluded because they reported previous hospitalizations due to depression, missed days from work due to depression, or taking anti-depressants during the previous five years.

The CES-D is sensitive to depressive symptoms, but is not a definitive diagnosis for depression as "the concordance between an elevated CES-D and actual clinical depression are two distinctly different things" (W. S. C. Poston, personal communication, May 20, 2003; Radloff, 1977). Overall frequencies of CES-D scores were examined at or over the cutoff score of 17 for all five years. Increased depressive symptoms were found in 40 subjects at baseline, 48 subjects at year 1, 32 subjects at year 2, 45 subjects at year 3, and 37 subjects at year 4.

Results of the CES-D Reliability Analysis

Cronbach's alpha (α) is a measure of internal consistency reliability denoting how reliable or consistent an instrument performs when measuring a concept, in this case, depressive symptoms (Ferketich, 1990). Cronbach's alpha is the most commonly used measure of internal consistency reliability (Knapp, 1991), and is dependent upon the length of the questionnaire. Questionnaires with more items will produce higher alphas. In addition, increased variance among scores will results in a higher alpha. Cronbach's alpha should be reported every time an instrument is used (Waltz, Strickland, & Lenz, 1991). Reliability indices on the CES-D for this study were calculated for each year in the current study: α=.86 at baseline, α=.88 at year 1, α=.89 at year 2, α=.90 at year 3, and α=.89 at year 4. Results obtained in this study were consistent with other studies, as presented in Table 2.

Histograms

Histograms for all annual CES-D scores for both males and females revealed that raw scores were highly skewed to the right with the greatest percentage of scores to the left of the mean (Figure 10). Therefore, the investigator determined that score transformations were necessary to standardize the raw scores (Cohen, Cohen, West, & Aiken, 2003). Other investigators who transformed CES-D scores were Aneshensel, Frerichs, and Clark (1981), and Aneshensel, Frerichs, and Huba (1984).

CES-D Score Transformations

A natural logarithmic transformation (Table 12) was computed to normalize all raw CES-D+1 scores (Figure 11). This was necessary to place scores in a more

normal distribution instead of a skewed-to-the-right distribution (Magnusson, 1967; Maxwell & Delaney, 2000) to simplify the relationship between predictor and criterion variables (Cohen, Cohen, West, & Aiken, 2003). This procedure was accomplished by adding '1' as a constant to each score, then calculating the natural logarithm using a calculator (Cohen, Cohen, West, and Aiken). '1' was added because the natural log of '0' is undefined, and a subject could score a '0' on the CES-D. To calculate a new cutoff score from the original score of 17, the constant '1' was added, totaling 18. The natural logarithm (ln) was calculated for 18 which resulted in a new cutoff score of 2.89. As depicted in Figure 11, the transformed CES-D scores now are dispersed more symmetrically than in Figure 10.

First Analysis Mixed Model Results

To reiterate the two hypotheses posed for this study:

1. Depressive symptoms (CES-D $\geq$17) at baseline will predict weight gain in overweight and obese females at five years (excluding baseline measures) as compared to normal weight females and all males, controlling for age, gender, and CES-D score by weight group.

2. Controlling for gender, depressive symptoms (CES-D scores) would positively correlate with body mass index (BMI) annually over five years.

A hierarchical linear mixed model was used to analyze data to examine if weight gain in obese women at five years could be predicted as a function of gender, weight group, and age by CES-D scores. 'Time' was added to the second and third levels to examine if weight significantly changed over time (in the third level) as a

function of the interaction between time and weight group by CES-D score. In the second level, the effect of 'time' was examined for the individual predictors (covariates) of gender, age, and CES-D score by weight group (normal, overweight, or obese). When interpreting results of a hierarchical linear mixed model, the third level (or overall) test is examined first to determine the overall significance of the predictor variables, and if they differed from one another. If significance is found, the second level test of the slopes denotes which of the predictor variables significantly differed from zero (e.g., gender=females and males; weight groups=normal weight, overweight, and obese; and whether 'time' contributed to each predictor variable).

The third level of the model was examined first for the effect of 'time' on gender, age, and CES-D scores by weight group over time interaction. Results are presented in Table 14. For this first analysis, no subjects were excluded. The overall mixed test was not significant for the time*transformed CES-D score+1 *weight group interaction, $F(3,3112) = 1.95, p=0.1193$. However, the estimated parameters for time by gender and time by age were significant, $F(2,3112)=20.74, p<.0001$, and $F(1,3112)=37.08$, respectively. CES-D scores were influenced by weight groups, $F(3,3112)=176.14, p<.0001$. Age upon study entry was not significant.

The second level tests.

The second level tests of the regression coefficients (*b*) or parameter estimates, are presented in Table 14. The slope and intercept (now considered dependent variables), are a function of the covariates of age, gender, and depression by weight group. In addition, each slope and intercept produced its own random

effect which allows for the correlation between measurements to be in the linear mixed model. Stated differently, each subject had an error/residual on their estimated slope (b), and within each subject, their weights were correlated over time.

Isolating the effect of depression on the intercept (a) and excluding 'time' as a predictor variable, the effect of the transformed CES-D score (notated as lnCESD+1) was significant for normal weight subjects, $b = -13.64$, $t(508) = -10.13$, $p < .0001$; and obese subjects, $b = 17.095$, $t(488) = 13.66$, $= p < .0001$), in that their slopes significantly differed from zero; however, results for the overweight group were not significant. The effects for gender on the intercept (a) were also significant, and differed between females, $b = 160.95$, $t(508) = 39.69$, $p < .0001$, and for males, $b = 195.47$, $t(508) = 48.45$, $p < .0001$.

When 'time' was included as a predictor variable for the slopes (b_i) in the second level, the time-gender interaction was significant, as every six months, both females and males increased their weight; $b = 1.9095$, $p < .0001$, and $b = 1.7720$, $p < .0001$. For the time by age interaction, the slope differed significantly from zero in a negative sense, $b = -0.031$, $p < .0001$, which indicated that the older one became, the less one would weigh (0.03 pounds), counter to the finding of increased weight over time. Finally, the interaction between time, transformed CES-D score, and weight groups were analyzed. There were no significant differences which meant that depressive symptoms did not significantly affect weight gain over time.

Regression Diagnostics

After the initial results were obtained, a series of regression diagnostics were run to identify outliers associated with the slope (*b*) in the second part of the second level equation. Investigators are most interested in change over time, denoted by the *b*. Pedhazur (1997) stated that the simplest method for analyzing residuals (or error in predicting *Y* from *X*) was to plot "standardized residuals against corresponding *X*'s or predicted *Y*'s (in raw or standardized form)" (p. 36). The equation for a standardized score (z score) on the residuals was obtained by subtracting the raw residual from the zero (in the numerator), divided by the standard deviation (denominator).

Using the General Linear Model (SPSS, 11.5), diagnostics were run on the second level 2 equation (the slopes, or *b*) since the primary purpose was to examine average weight change per subject. Using least squares, a slope was created for every subject. A Type 3 sum of squares model was used to plot standardized residuals and unstandardized predictor variables in a scatterplot. For a subject to be eliminated from the final analysis as an outlier, their transformed score must exceed three standard deviations (*SD*) above and below the mean. Three standard deviations above and below the mean captured 99.7% of the subjects (Waltz, Strickland, & Lenz, 1991). After six iterations, 20 subjects were classified as outliers, and eliminated from the final analysis, and examined for common characteristics, presented in Table 15 through 17.

The majority of outliers were classified as 'obese' for females (n=7 out of 14), and males (n=6, or 100%). The majority of outliers who were female held clerical

jobs, took classes beyond their college degree, and rated their mental health good to excellent. Only one female took a mood enhancing drug, recorded at year five. Among males, the majority held a professional job, had some college education, and rated their mental health from good to excellent. There were very few disease processes that could have influenced outlier depression scores. Only two females had diabetes, and three stated they had depression at year five, but none of the three had any comorbid disease processes.

Dropouts

'Dropouts' were defined for this study as subjects who missed 3 to 4 out of 5 annual assessments. There were no cohort studies reviewed during the literature to provide a benchmark for the attrition rate. Mean CES-D scores and weights were stratified by gender and weight group. Results were tabled and compared to subjects who remained in the RENO study (Table 18). There were 24 females and 25 males who dropped out of the RENO study, approximately 10% for each gender. Overall, the majority of dropouts who had higher CES-D scores were overweight males (n=12) and obese females (n=10). When stratified by age decade, the majority of dropouts were twenty year old obese (5 out of 9) females (9 out of 17). Results of this study found that younger obese females gained the most weight as their depression scores increased. The investigator speculated that increased depressive symptoms and failure to lose weight may have been reasons for attrition among this group of females.

The Final Analysis

In the first data analysis (with outliers included), "time" was a significant predictor for age and gender (p<.0001), but not the interaction of CES-D scores by time and by weight group (p=.1193). However, after removing the outlier values and re-analyzing the data, the mixed test revealed that higher CESD scores among obese females significantly predicted weight gain over time, $F(3,3000)$=3.91, p=.0085 (Table *19)* as well as for obese males. When examining gender differences, gender significantly impacted all obese subjects but not overweight subjects. Females also gained slightly more weight, approximately 1.67 (b = 1.67, $t(488)$ = 6.82, p <.001) pounds every six months more than men, who gained approximately 1.65 pounds every six months (b = 1.65, $t(488)$ = 6.90, p <.0001). Once the outliers were removed from the final analysis, the investigator expected that there would be no effect for depressive symptoms on weight gain over time as evidenced in the first analysis. However, excluding the outliers produced a significant estimate that over time, depressive symptoms resulted in weight gain in both obese women and men. Outlier obese females weights ranged from 178.6 to 206.29 pounds, and outlier male weights ranged from 241 to 242.6 pounds.

Degrees of freedom changed when depression was removed from the analysis in the restricted model. The Neg2loglikelihood for the full model was 27488.3, and for the reduced model (minus depression) was 27915.5, a difference of 427.2 which was significant when examining a chi-square table with 6 degrees of freedom. When significance was found in the third level mixed model, the investigator examined the

second level model to determine where differences occurred, presented in Table 20. Using the second level regression equation for a significant change in slopes, estimates for weight gain were tabulated. Surprisingly, obese subjects in their twenties gained the most weight as their depressive symptoms scores increased (Table 21). Although the CES-D is used only to screen for depressive symptoms, higher baseline scores were related to weight gain in the obese.

Results from the second level test allowed the investigator to predict weight gain over five years for all subjects by age. Using the estimated population parameters in an equation generated for the change in slope (b) we could predict weight gain for an obese, thirty year old woman with a CES-D score of 35. The regression equation would be written as:

$$\beta_i = \text{entage}_i\beta_7 + \text{Female}_i\beta_8 + \text{Male}_i\beta_9 + \text{wtcat}_1 * \ln(\text{CES-D}_i+1)_i\beta_{10} + \text{wtcat}_2 * \ln(\text{CES-D}_i+1)\beta_{11} + \text{wtcat}_3 * \ln(\text{CES-D}_i+1)\beta_{12} + e_{1i} \quad (2)$$

Substituting numbers generated from the output,

β_i=30(-0.02775) + 1.67 + (3.58)*0.1830 = 1.49 pounds gained every six months without intervention for depression. For a 30 year old male with a CES-D score of 35, β_i=30(-0.02775) + 1.65 + (3.58)*0.1830 = 1.47 pounds gained every six months.

Regression equations for the change in slope (b) were calculated for each decade (20-60) by gender and weight group to estimate how much weight gain (or loss) subjects incurred over five years. Based on the RENO data, obese subjects in

their twenties gained the most weight as their CES-D scores increased. For a twenty year-old obese female with a score of 10 on the CES-D, one could predict a weight gain of 15.53 pounds in five years, and with a score of 30, predict a weight gain of 17.43 pounds (Table 21). For a twenty-year old obese male with a CES-D score of 10, one could predict a weight gain of 15.35 pounds at five years, and with a score of 30, predict a weight gain of 17.25 pounds at five years. This age group produced the largest weight gain of any other decade.

Among the 30 year old subjects, both males and females who were normal and overweight subjects lost weight while obese subjects gained weight as their CES-D scores increased (Table 22). For obese females with a score of 10 on the CES-D, one could predict a weight gain of 12.76 pounds in five years, and with a score of 30, predict a weight gain of 14.65 pounds. For an obese male with a CES-D score of 10, one could predict a weight gain of 12.58 pounds at five years, and with a score of 30, predict a weight gain of 14.48 pounds at five years.

Among the 40 year old subjects, both males and females who were normal weight and overweight subjects lost weight while obese subjects gained weight as their CES-D scores increased (Table 23). For an obese female with a CES-D score of 10, one could predict a weight gain of 9.98 pounds at five years, and with a CES-D score of 30, a weight gain of 9.98 pounds at year 5. For an obese male with a CES-D score of 10, one could predict a weight gain of 9.8 pounds at five years, and with a CES-D score of 30, predict a weight gain of 11.70 pounds at five years.

Among the 50 year old subjects, both males and females who were normal and overweight subjects lost weight while obese subjects gained weight as their CES-D scores increased (Table 24). For obese females with a score of 10 on the CES-D, one could predict a weight gain of 7.21 pounds in five years, and with a score of 30, predict a weight gain of 9.10 pounds. For an obese male with a CES-D score of 10, one could predict a weight gain of 7.03 pounds at five years, and with a score of 30, predict a weight gain of 8.93 pounds at five years.

Again, among the 60 year old subjects, both females and males who were normal and overweight, lost weight while obese subjects gained weight as their CES-D scores increased (Table 25). For obese females with a score of 10 on the CES-D, one could predict a weight gain of 0.44 pounds in five years, and with a score of 30, predict a weight gain of 0.63 pounds. For an obese male with a CES-D score of 10, one could predict a weight gain of 4.25 pounds at five years, and with a score of 30, predict a weight gain of 6.15 pounds at five years. In this age decade, obese males gained more than obese females. In other words, depressive symptoms significantly contributed to weight gain among obese females and males.

Correlation Matrices

The second research question for this study was to examine if ln(CESD+1) scores positively correlated with BMI annually over five years (Table 26). A Bonferroni approach was used to control for Type I error rate (Green, Salkind, & Akey, 2000), dividing the traditional $p<.05$ by the number of comparisons (ten)

resulting in a new probability level of $p<.005$ denoting significance. For females, only years 1 and 3 revealed significant correlations, $r(230) = .206, p = .002$, and $r(216) = .26, p = .000$. There were no significant correlations between ln(CES-D+1) scores among males. For gender by weight groups, there was only one significant correlation which occurred in obese females between ln(CESD+1) scores and BMI at year 2, $r(60) = .372, p = .003$.

Discussion

The purpose of this study was to investigate if baseline measures of depression, gender, weight group, and age in overweight and obese women would predict weight change over a five year period. Data were analyzed for males as a comparison group. A secondary purpose for this study was to examine if a higher level of depressive symptoms positively correlated with body mass index (BMI) over a five year period. This study was a secondary analysis of archival data from the RENO Diet-Heart study (St. Jeor, 1997). To reiterate, the investigators hypothesized that depressive symptoms (CES-D $\geq$17) at baseline would predict weight gain in overweight and obese females at five years (excluding baseline measures) as compared to normal weight females and all males, controlling for age, gender, and CES-D score by weight group. Results confirmed that regardless of gender, both obese women and men who scored higher on the CES-D gained more weight over time than the other weight groups as a function of age, gender, and weight group. Based on the data, the differences in weight gain between males and females were small, but only the obese subjects gained weight over time.

In relation to depressive symptoms, normal weight subjects scored lower on the CES-D than overweight subjects, and obese subjects scored the highest, even though their scores remained well below the cutoff score of 17. These results were similar to results found in the original RENO Diet-Heart study although weight group assignation was based on current guidelines for body mass indices. There was minimal variance among the scores; therefore, our findings may underestimate the effect of CES-D scores on weight changes.

There were few published studies that examined the relationship between depressive symptoms and weight gain. In one of the few studies that examined relationships among body mass index, depression, suicide attempts and ideations, Carpenter, Hasin, Allison, and Faith (2000) found that obesity placed females at a "37% higher probability of being diagnosed with depression" (p. 254) than males in a population-based study that examined if relative body weight was associated with depression (and suicide) between Whites and African Americans. In their study of over 40,000 people during 1992, they identified several limitations that included the possibility of unreliable self-reported height and weights, and reporting biases from culturally-sensitive, trained census staff (who collected the data by interview method). The investigators did not use a precise measure of percent body fat, only body mass index based on self-reported height and weight. They also did not examine causation, and they used a different assessment tool (the AUDADIS; Grant & Hasin, 1992) that is based on DSM-IV criteria to diagnose depression, not depressive symptoms as was emphasized in this study (St. Jeor, 1997). However, data from the

RENO Diet-Heart study revealed that among obese individuals, as depressive symptoms increased, so did weight gain. Carpenter's (et al.) study was conducted two years later, and findings were similar from a national perspective.

Siegel, Yancey, and McCarthy (2000) examined depressive symptoms among 429 African American women using the CES-D (Radloff, 1977). They found that African American women who were overweight, better educated, and had a strong sense of ethnic identity were the least depressed, with a mean CES-D score of 9.45. The researchers found that women who self-reported they were in poor health had higher depressive scores and tended to be overweight.

Other investigators have suggested over time, obese females may suffer from a higher level of depressive symptoms or depression (Anderson & Wadden, 1999; Carpenter, Hasin, Allison, & Faith, 2000; United States Preventive Task Force, 2002). As early as 1982, Hopkinson and Bland found that almost 20% of obese females scheduled for gastric bypass suffered from depression. Hafner, Watts, and Rogers found similar results in their study (1987), as well as Prather and Williamson (1988). The secondary data analysis supported that obese females scored higher on the CES-D than all other gender-classified weight groups, and the lowest CES-D scores occurred in the normal weight groups (Table 10). Study findings concurred with Carpenter (et al.) that both depression and obesity are prevalent in our society, and given the trends over time, the prevalence of obesity and depression are predicted to increase.

Many investigators have suggested that depression be included in the obesity model (Blehar & Oren, 1995; Carpenter, Hasin, Allisonk, & Faith, 2000; Friedman & Brownell, 1995; Khaodhiar, McCowen, & Blackburn, 1999; Klesges, Klem, & Klesges, 1992) as depression (or depressive symptoms) may contribute to weight gain. Data from the RENO Diet-Heart study supported that obese individuals gained the most weight as their CES-D scores increased.

In addition, no qualitative studies were found that examined the phenomena of obesity and depressive symptoms among genders. Exploring gender differences using qualitative methods would add depth to understanding the effect of depressive symptoms among obese individuals.

Conclusions

Subjects in the RENO Diet-Heart study were healthy people. Although potential candidates were screened and some were eliminated from participation due to a self-reported history of depression, the effect of depressive symptoms on weight gain was detected not only in women but also in men from data that is 20 years old. Currently, obesity rates have increased to 30.5% among adults (Flegal, Carroll, Ogden, & Johnson, 2002), and the National Center for Health Statistics reported in 2002 that more women were obese (33%) than men (28%). The amount of difference in weight gain was small between females and males, but over time may compromise individual health and psychological well-being especially among the obese.

Data support that the risk for developing weight gain due to increased depressive symptoms affects the obese population, after controlling for age, gender, and weight

group by CES-D score. In addition, the risk for weight gain appears to be the greatest in obese individuals in their twenties. The effect of depressive symptoms on weight gain over time was not significant until extreme scores were removed from the analysis. After excluding the extreme scores, results denoted that higher levels of depressive symptoms caused weight gain over time among obese individuals, including the covariates of age, weight group by CES-D interaction, and gender. Although the difference in weight gain between men and women was small, a moderate significant correlation occurred between obese women and BMI at year 1 that could be explained by a 2.77 increase in the year 1 CES-D mean score from baseline among obese females.

One advantage from using the hierarchical linear model was that predicted weight change could be estimated for all subjects, using the formula from the second level regression equation for slopes. Obese subjects consistently gained weight as their CES-D scores increased, and the greatest increase in weight was projected for the 20 year-old obese group. Screening for depressive symptoms is important not only for younger, obese clients, but for all obese clients to evaluate whether depressive symptoms may have an effect on their weight gain. Using the data to predict weight gain over time (Table 18), obese females in their twenties (who had a low CES-D score of 10) were predicted to gain 18.67 pounds over five years, and obese males (who had a CES-D score of 10) would gain 18.49 pounds. Although the CES-D is not definitive for diagnosing depression, using it as a screening tool may identify obese individuals who evidence depressive symptoms as 'at risk' for gaining

more weight. This involves changing treatment options that may include interventions for depressive symptoms.

Implications for Nursing Practice

Nurse health practitioners in many settings should screen individuals for depression, especially obese women and men. Data supports that obese women and men with CES-D scores as low as 10 gain weight over time. Implementing a treatment regimen may need to include further investigation into whether depressive symptoms are related to weight gain. Revising standard treatments for obesity may include referral to a mental health practitioner, and re-designing traditional obesity treatments to include psychological or pharmacologic interventions.

Recommendations for Future Research

Results presented in this study support previous research findings that obese individuals with increased depressive symptoms tend to gain weight over time. Although subjects were generally in good health, and excluded if they reported any diagnosis of depression or took antidepressants/mood altering drugs, the effect of depression on weight gain among the obese was present. Community studies that include depressed individuals as part of the community population are necessary to examine the effects of depressive symptoms on weight gain among obese individuals.

These results also suggest that obesity treatment should consider screening all clients for depressive symptoms, but to specifically target obese clients in their twenties. Even younger obese clients who present with lower CES-D scores may require more attention by practitioners, as the risk for prospectively developing

depressive symptoms and subsequent weight gain may be present. Depression appears to add another factor to the multiple causes of obesity, and, in turn, obesity may be another contributing factor in developing depression. Whether obesity influences depression or depression influences obesity is unknown; however, by examining whether depression is causative factor in the current models of obesity may help alleviate the risk for weight gain among obese individuals.

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Table 1

Instruments Used to Measure Depression

Instrument and author	Purpose of instrument	Sample tested, alpha reliability coefficient (α)
Beck Depression Inventory (BDI; Beck & Steer, 2000)	A screening tool to assess depression in clients with diagnosed depression. 21 items, takes 5 to 10 minutes to complete the self-report. Scores range from 0 to 3 (mild to severe) for each question. A score of 0-9 denotes minimal depression, 10-16 denotes mild depression, 17-29 denotes moderate depression, and 30-63 denotes severe depression.	Psychiatric populations, α =0.76-0.95. Student populations, α=0.82-0.92. Major depressive disorder single episode, α=0.80 Major depressive disorder, repeated episodes, α=0.86

*Center for Epidemiologic Studies - Depression (CES-D; Radloff, 1977).	Screening tool that measures depressive symptoms in community populations. 20 items, takes 5 minutes to complete. Administered by self-report. Scores ≥17 indicates depression (scores range from 0-60).	~ α=0.85 in community samples, ~ α=0.90 in psychiatric samples.
*Hamilton Rating Scale for Depression (HDRS; Hamilton, 2000).	Interviewer-administered screening tool that measures depressive symptoms. 17 items, and takes 15-20 minutes to complete. Scores > 23 indicates severe depression; 19-22, severe; 14-18, moderate depression; 8-13, mild depression; and ≤ 7, normal.	α ~ 0.80; higher using structured interviews.
Structured Clinical Interview for Depression (SCID; Spitzer, Williams, Gibbon & First, 1992).	Based on DSM criteria to make DSM diagnoses. Interview completed by an experienced clinician trained in the use of the SCID. Takes 1.5 hours to complete. Is organized into DSM category modules including psychotic, mood, substance abuse, anxiety, somatoform, eating, adjustment, and personality disorders.	Not available

Zung Self-Rating Depression Scale (SDS). Zung, 2000.	Rates the severity of depressive symptoms in individuals diagnosed with depression. Used in both community and private practice populations. 20 items, takes between 5 and 30 minutes to fill out (depressed individuals take longer). Administered by self-report. Does not ask about weight gain. Likert scale ranges from 1 (a little of the time) to 4 (most of the time). Scores below 50 are normal; below 60 indicate mild depression; below 70 indicate moderate depression; and above 70 indicate severe depression.	α=0.79

Note.* indicates used in this study.

Table 2

Coefficient Alphas in Studies Using the CES-D (Radloff, 1977)

Authors	Year	*N*	Coefficient Alpha (α)
Radloff	1977	4996	α=.85 for the general population
Eaton & Kessler	1981	2867	Reported Radloff's original α
Aneshensel, Frerichs, & Huba	1984	774	α=.90
Reis & Herz	1986	116	α=.88 (from their study? Unclear).
Ross	1994	2020	α=.88
McCrone, Dennis, Tomoyasu, & Carroll	2000	135	Reported Radloff's original α
Siegel, Yancey, & McCarthy	2000	429	α=.68
Ellis-Stoll, Aaronson, Gajewski, Hise, Pallikathayil, Popkess-Vawter, Poston, Foreyt, & St. Jeor (unpublished dissertation)	2003	varied	α=.86 at baseline, α=.88 at year 1, α=.89 at year 2, α=.90 at year 3, α=.89 at year 4.

Table 3

RENO Diet-Heart Study Variables of Interest

Variable Name	Values measured at baseline unless otherwise Indicated
Age	
Gender	1=female, 2=male
Education	What is the highest level you have completed in school? 1=less than high school 2=started but didn't finish high school 3=high school diploma, GED, or equivalent 4=some college credit, but no degree 5=college degree 6=course work beyond college degree
Marital Status	1=single, never married 2=married 3=living with significant other'

	4=engaged
	5=separated
	6=divorced
	7=widowed
Socio-economic status	Approximate monthly income after taxes:
	1=$0-$500
	2=$501-$1,000
	3=$1,001-$2,000
	4=$2,001-$4,000
	5=$4,001 or more.
Racial/ethnic background	1=Asian or Pacific Islander
	2=Black, not Hispanic
	3=Chicano, Latino, Hispanic
	4=Native American, Native Alaskan, Indian
	5=White, not Hispanic
	6=other
Weight	In pounds every six months
Body Mass Index	Using the formula kg/m^2, or weight in kilograms divided

	by
	height in meters, squared.
	Every six months.
CES-D Scores	Each item, totaled for a
	summed score
	Annually.
Weight group (based on current BMI	Recoded for this study.
standards)	0=normal weight (a BMI
	<24.90)
	1=overweight (a BMI ranging
	from 25-29.9)
	2=obese (a BMI over 30).
	Every six months.
Ever diagnosed with depression?	1=yes, 2=no. Baseline and five
	years.
Taking prescribed antidepressants	Selection '7' under Medications
Taking OTC mood enhancers/tranquilizers	Section '7' under Medications

Table 4

Demographics of Subjects in the RENO Diet-Heart Study (N=499).

	Males (*n*=248)	Females (*n*=251)
Professional	65.9%	35.6%
Married	72.9%	70.4%
Ethnic	White; 94.9%	White; 92.1%
Income	$2,000-4,000 per month; 46.3%	$2,000-4,000 per month; 39.1%
Years educated 12+	96.5%	94.9%
Post-college hours	48.2%	42.7%
College graduates	13.3%	15.4%

Note. Largest percentages are reported. Missing data on seven male subjects and two female subjects.

Table 5

Weight in Pounds Every 6 Months for Women and Men

Women	N	Mean Weight	SD	Minimum	Max
Baseline	253	156.17	31.09	101.5	266
6 months	253	155	30.93	101.25	265.5
Year 2	240	157.3	30.85	97.5	274.5
18 months	239	155.7	30.76	97	270.5
Year 3	223	157.4	31.1	103.75	293
30 months	223	155.92	30.92	104.25	292.25
Year 4	217	157.47	32.43	98.5	293
42 months	215	156.24	32.27	97.75	287.75
Year 5	211	158.53	32.74	104.25	301.5
Men					
Baseline	255	192.76	31.99	127.5	316.5
6 months	254	192.53	31.88	127	314.25
Year 2	244	193.33	32.53	131	326
18 months	241	191.93	32.29	128.75	320
Year 3	233	195.74	33.32	128.5	335.5
30 months	231	194.18	33.10	128.5	331.5
Year 4	225	194.22	33.17	124	339
42 months	223	192.82	32.44	123.5	336.25
Year 5	222	196.21	34.57	126.25	336.25

Table 6

Baseline Weight, Height, and Body Mass Index for All Subjects (N=499)

Gender	Weight in pounds *X (SD)*	Height in inches *X (SD)*	Body Mass Index *X (SD)*
Male (*n*=248)	192.76 (31.99)	70.20 (2.65)	27.55 (4.12)
Female (*n*=251)	156.17 (31.10)	64.62 (2.46)	26.33 (4.93)

Note. Missing data on nine subjects.

Table 7

Baseline Mean Weight and BMI for All Subjects by Weight Group (N=499)

Weight Group	Weight	Body Mass Index
	X (*SD*)	*X* (*SD*)
Normal Weight (*n*=187)	142.57 (21.02)	22.35 (1.70)
Overweight (*n*=183)	180.01 (20.80)	27.24 (1.36)
Obese (*n*=129)	211.84 (31.27)	33.09 (2.56)

Note. Missing data from nine subjects.

Table 8

Baseline Mean Weight and BMI for Females by Weight Group (n=251)

Weight Group	Weight in pounds *X (SD)*	BMI *X (SD)*
Normal weight (*n*=113; 45%)	129.96 (12.50)	21.87 (1.70)
Overweight (*n*=70; 27.9%)	162.30 (14.51)	27.12 (1.32)
Obese (*n*=68; 27.1%)	193.57 (22.94)	32.92 (2.51)

Note. Missing data from two subjects.

Table 9

Baseline Mean Weight and Body Mass Index for Males by Weight Group (n=248)

Weight Group	Weight	Body Mass Index
	X (SD)	*X (SD)*
Normal weight	161.82 (16.28)	23.08 (1.42)
(*n*=74; 29.8%)		
Overweight	191 (15.98)	27.32 (1.38)
(*n*=113; 45.6%)		
Obese	232.21 (26.41)	33.29 (2.62)
(*n*=61; 24.6%)		

Note. Missing data from seven subjects.

Table 10

Mean CES-D Scores, Standard Deviations, and Ranges by Gender

	Baseline	Year 1	Year 2	Year 3	Year 4
Females	5.82 (6.32);	7.91 (8.16);	6.92 (7.34);	7.54 (8.50);	7.10 (7.90);
	0-32	0-48	0-38	0-49	0-45
Males	6.02 (6.59);	6.15 (6.42);	6.02 (7.01);	5.84 (6.80);	5.06 (6.80);
	0-39	0-44	0-44	0-46	0-33

Table 11

Mean CES-D Scores with Standard Deviations Over Five Years Stratified by Gender and Weight Group

	Baseline	Year 1	Year 2	Year 3	Year 4
	n; X(SD); range	*n; X(SD); range*	*n; X(SD); range*	*n; X(SD); range*	*n; X(SD); range*
Males					
Normal wt	*n*=74; 5.07(4.82); 0-14	*n*=70; 5.07(4.82); 0-22	*n*=63; 5.08(6.36); 0-29	*n*=64; 5.05(5.96); 0-26	*n*=62; 4.74(6.74); 0-28
Overwt	*n*=112; 5.88(6.70); 0-33	*n*=108; 5.56(5.40); 0-33	*n*=100; 5.67(6.19); 0-29	*n*=96; 5.47(6.89); 0-46	*n*=89; 4.85(6.31); 0-33
Obese	*n*=61; 7.8(8); 0-30	*n*=56; 8.7(8.6); 0-44	*n*=100; 5.67(6.19); 0-44	*n*=96; 5.47(6.89); 0-38	*n*=40; 6.45(6.7); 0-22
Females					
Normal wt	*n*=113; 5.05(5.51); 0-29	*n*=106; 6.59(6.87); 0-39	*n*=106; 5.95(6.51); 0-27	*n*=104; 5.77(5.93); 0-28	*n*=93; 5.92(6.84); 0-31
Overwt	*n*=70; 4.73(4.67);	*n*=70; 4.73(4.67);	*n*=66; 7.48(7.51);	*n*=62; 7.89(8.51);	*n*=56; 7.7(8.54);

	0-17	0-41	0-35	0-35	0-45
Obese	*n*=68;	*n*=61;	*n*=56;	*n*=54;	*n*=50;
	8.03(8.15);	10.7(9.98);	8.04(8.53);	10.43(11.47);	8.5(8.37);
	0-32	0-48	0-38	0-35	0-33

Table 12

Transformed CES-D Scores

Normal Weight Females		Normal Weight Males	
CES-D score (+1)	Transformed CES-D Score	CES-D score (+1)	Transformed CES-D score
1	0	1	0
5+1	1.791759469	5+1	1.79175947
10+1	2.397895273	10+1	2.39789527
15+1	2.772588722	15+1	2.77258872
20+1	3.044522438	20+1	3.04452244
25+1	3.258096538	25+1	3.25809654
30+1	3.433987204	30+1	3.4339872
Overweight Females		Overweight Males	
1	0	1	0
5+1	1.791759469	5+1	1.791759469
10+1	2.397895273	10+1	2.397895273
15+1	2.772588722	15+1	2.772588722
20+1	3.044522438	20+1	3.044522438
25+1	3.258096538	25+1	3.258096538
30+1	3.433987204	30+1	3.433987204
Obese Females		Obese Males	

1	0	1	0
5+1	1.791759469	5+1	1.791759469
10+1	2.397895273	10+1	2.397895273
15+1	2.772588722	15+1	2.772588722
20+1	3.044522438	20+1	3.044522438
25+1	3.258096538	25+1	3.258096538
30+1	3.433987204	30+1	3.433987204

Table 13

Results of the First Analysis – The Third level Mixed Model Results

Effect	Numerator degrees of freedom	Denominator degrees of freedom	*F* value	*p* level
Gender	2	3112	1193.16	<.0001*
Age on study entry	1	3112	3.7	0.055 NS
lnCESD+1 score * weight group	3	3112	176.14	<.0001*
Time*gender	2	3112	20.74	<.0001*
Age on study entry * time	1	3112	37.08	<.0001
Baseline lnCESD+1 score*time* weight group	3	3112	1.95	0.11193 NS

Note. '*' denotes significance at $p \leq .05$. The model was adjusted for age, gender, and weight group by CES-D score.

Table 14

Results of the First Analysis – The Second Level Mixed Model Results

Effect	Gender	Recoded Weight group	Estimate (*b*)	Standard error	Degrees of freedom	*t* value	*p* value
Gender	1=female		160.95	4.06	3112	39.69	<.0001
	2=male		195.47	4.04	3112	48.45	<.0001
Age			-0.14	0.07	3112	-1.92	0.055
NLCESD*		0=normal	-13.64	1.35	3112	-0.13	<.0001
Weight		1=over-	1.63	1.32	3112	1.24	0.22
group		weight					
		2=obese	17.10	1.25	3112	13.66	<.0001
Time*	1=female		1.91	0.299	3112	6.39	<.0001
Gender	2=male		1.77	0.295	3112	6.01	<.0001
Age*time			-0.03	0.005	3112	-6.09	<.0001
NLCESD*		0=normal	-0.04	.098	3112	-0.37	0.71
Weight		1=over-	-0.06	.098	3112	-0.62	0.54
group*		weight	0.16				
Time		2=obese		.016	3112	1.62	0.09

Note. The model adjusts for age, gender, and weight group by CES-D score.

Table 15

Annual Weights by Genders among Outliers

Gender	Baseline weight; *X*(*SD*); *n*	Year 1 weight; *X*(*SD*); *n*	Year 2 weight; *X*(*SD*); *n*	Year 3 weight; *X*(*SD*); *n*	Year 4 weight; *X*(*SD*); *n*
Females	178.64	184.36	196.19	200.78	206.29
(*n*=14)	(30.94)	(33.64)	(30.61)	(41.10)	(47.96)
	n=14	*n*=14	*n*=13	*n*=10	*n*=7
Males	241.42	232.46	230.92	226.45	242.60
(*n*=6)	(38.68)	(35.88)	(45.50)	(45.95)	(57.71)
	n=6	*n*=6	*n*=6	*n*=5	*n*=5
Overall	**Baseline**	**Y1**	**Y2**	**Y3**	**Y4**
Females	**154.32**	**155.63**	**156.46**	**156.80**	**157.61**
	(29.16)	**(29.67)**	**(31.19)**	**(32.20)**	**(32.51)**
	199	**199**	**199**	**199**	**199**
Males	**192.17**	**193.24**	**194.71**	**193.85**	**195.88**
	(32.03)	**(32.49)**	**(32.80)**	**(32.85)**	**(34.01)**
	204	**204**	**204**	**204**	**204**

Table 16

Annual Body Mass Index by Gender among Outliers.

Gender	Baseline BMI; *X*(*SD*); *n*	Year 1 BMI; *X*(*SD*); *n*	Year 2 BMI; *X*(*SD*); *n*	Year 3 BMI; *X*(*SD*); *n*	Year 4 BMI; *X*(*SD*); *n*
Females	28.78	29.84	31.56	31.42	32.39
	(4.59)	(5.65)	(4.44)	(4.56)	(5.36)
	n=14	*n*=14	*n*=13	*n*=10	*n*=7
Males	35.05	33.87	33.22	32.89	34.93
	(2.44)	(2.68)	(4.05)	(5.21)	(5.89)
	n=6	*n*=6	*n*=6	*n*=5	*n*=5

Table 17

Annual CES-D Scores by Gender among Outliers.

Gender	Baseline score; *X*(*SD*); *n*	Year 1 score; *X*(*SD*); *n*	Year 2 score; *X*(*SD*); *n*	Year 3 score; *X*(*SD*); *n*	Year 4 score; *X*(*SD*); *n*
Females	6.14	8.71	4.79	10	4.14
(*n*=14)	(6.97)	(5.81)	(3.94)	(9.57)	(3.89)
	n=14	*n*=14	*n*=14	*n*=10	*n*=7
Males	8.67	9.00	8.33	11.5	6.00
(*n*=6)	(7.45)	(7.51)	(6.66)	(5.74)	(4.08)
	n=6	*n*=6	*n*=6	*n*=4	*n*=4
Overall	**Baseline**	**Year 1**	**Year 2**	**Year 3**	**Year 4**
Females	**5.82(6.32)**	**7.91(8.16)**	**6.92(7.34)**	**7.54(8.50)**	**7.10(7.90)**
Males	**6.02(6.59)**	**6.15(6.42)**	**6.02(7.01)**	**5.84(6.80)**	**5.06(6.80)**

Table 18

Mean CES-D Scores for Dropouts Compared to Sample Data with Standard Deviations and Ranges Stratified by Gender and Weight Group

	Baseline scores: *n, X(SD),* range	Year 1 scores: *n, X(SD),* range	Year 2 scores *n, X(SD),* range	Year 3 scores *n, X(SD),* range	Year 4 scores *n, X(SD,* range
Males	*n*=177; 4.97(5.30), 0-21	*n*=177; 5.92(5.52), 0-22	*n*=177 5.24(6.24), 0-29	*n*=177 5.55(6.50) 0-26	*n*=177 4.79(6.0) 0-28
Normal weight	*n*=74 5.07(4.82), 0-21;	*n*=70 5.07(4.82), 0-21;	*n*=63 5.08(6.36), 0-25;	*n*=64 5.05(5.96), 0-26;	*n*=62 4.74(6.74), 0-28;
dropouts	***n=5*** ***9.22(7.58),*** ***0-21***	***n=3*** ***7.83(7.58),*** ***0-21***	***n=2*** ***17(7.21),*** ***0-25***	***n=1*** ***6(0)*** ***6***	***n=0*** - -
Over-weight	*n*=112 5.88(6.7), 0-33	*n*=108 5.56(5.4), 0-22	*n*=100 5.67(6.19), 0-29	*n*=96 5.47(6.89), 0-46	*n*=89 4.85(6.31), 0-33
dropouts	***n=13*** ***10.31(7.36),*** ***1-24***	***n=6*** ***10(8.17)*** ***1-21***	***n=2*** ***1(0)*** -	***n=1*** - -	***n=1*** - -

Obese	*n*=61	*n*=56	*n*=100	*n*=96	*n*=40
	7.8(8),	8.7(8.6),	5.67(6.19),	5.47(6.89),	6.45(6.7),
	0-39	0-44	0-44	0-38	0-22
dropouts	***n=7***	***n=5***	***n=2***	***n=2***	***n=0***
	9.86(13.89),	***6(8.6)***	***12(9.9)***	***15.5(16.26)***	-
	0-39	***0-21***	***5-19***	***4-27***	-
Females	*n*=251	*n*=190	*n*=190	*n*=190	*n*=190
	5.51(6.24)	7.26(7.95)	6.83(7.30)	7.28(8.34)	6.87(7.67)
Normal	*n*=113	*n*=106	*n*=106	*n*=104	*n*=93
weight	5.05(5.51),	6.59(6.87),	5.95(6.51),	5.77(5.93),	5.92(6.84),
	0-29	0-39	0-27	0-28	0-31
dropouts	***n=8***	***n=6***	***n=2***	***n=1***	***n=0***
	6.63(6.89),	***13(7.16),***	***11(5.66),***	***6***	-
	1-23	***3-24***	***7-15***	-	-
Over-	*n*=70	*n*=70	*n*=66	*n*=62	*n*=56
weight	4.73(4.67),	4.73(4.67),	7.48(7.51),	7.89(8.51),	7.7(8.54),
	0-17	0-41	0-35	0-35	0-45
dropouts	***n=6***	***n=3***	***n=2***	***n=2***	***n=0***
	4.5(4.14),	***3(2),***	***13(0)***	-	-
	0-11	***1-5***	***0-26***	-	-
	n=68	***n=61***	***n=56***	***n=54***	***n=50***
Obese	8.03(8.15),	10.7(9.98),	8.04(8.53),	10.43(11.5),	8.5(8.37),

	0-32	0-48	0-38	0-49	0-33
dropouts	***n=10***	*n=5*	***n=1***	***n=1***	***n=0***
	10.1(7.69),	*13.2(13.37),*	-	-	-
	1-23	*2-34*	-	-	-

Note. Dropout information is presented in italics and bold print. Values that could not be calculated are represented by a '-'.

Table 19

Results of the Final Mixed Model Excluding Outliers

Effect	Numerator degrees of freedom	Denominator degrees of freedom	*F* value	*p*
Gender	2	3000	1189.50	<.0001*
Age	1	3000	3.52	0.0606
lnCESD+1 * weight group	3	3000	167.19	<.0001*
Time * gender	2	3000	25.07	<.0001*
Time * age	1	3000	43.26	<.0001
ln CES-D+1 * time * weight group	3	3000	3.91	0.0085*

Note. * indicates significance at $p<.05$. The model adjusts for age, gender, and weight group by CES-D score.

Table 20

Results of the Final Analysis – Second Level Results Excluding Outliers

Effect	Gender	Weight Group	Estimate (*b*); (CI±)	Standard error	Degrees of freedom	*t* value	Sig.
Gender	1=female		159.70 (±8)	4.08	3000	39.10	<.0001
	2=male		194.32 (±7.89)	4.02	3000	48.26	<.0001
Age			-0.13 (±.14)	0.07	3000	-1.88	0.06
lnCESD+1 * weight group		0=normal	-13.03 (±2.63)	1.34	3000	-9.73	<.0001
		1=over-weight	2.16 (±2.56)	1.31	3000	1.65	0.09
		2=obese	17.31 (±2.51)	1.28	3000	13.54	<.0001
Time* Gender	1=female		1.67 (±.47)	0.24	3000	6.82	<.0001
	2=male		1.65 (±.45)	0.23	3000	6.90	<.0001
Age*time			-0.03	.004	3000	-6.58	<.0001

		(±.008)				
lnCESD+1	0=normal	0.04	0.08	3000	-0.52	0.60
* weight		(±.16)				
group*	1=over-	0.09	0.08	3000	-1.12	0.26
Time	weight	(±.15)				
	2=obese	0.18	0.08	3000	2.34	0.02
		(±.15)				

Note. The model adjusts for age, gender, and weight group by CES-D score.

Table 21

Actual Weight Changes Over 5 Years Among 20-Year Old Subjects for Raw CES-D Scores by Gender and Weight Group

Raw CESD score	Normal Weight Females (pounds)	Normal Weight Males (pounds)	Over-Weight Females (pounds)	Over-Weight Males (pounds)	Obese Females (pounds)	Obese Males (pounds)
10	10.16	9.98	9.02	8.85	15.53	15.35
20	9.90	9.72	8.45	8.27	16.72	16.54
30	9.74	9.56	8.11	7.93	17.43	17.25

Note. Very few subjects scored >30, reflected in this table.

Table 22

Actual Weight Changes over 5 Years Among 30-Year Old Subjects for Raw CES-D Scores by Gender and Weight Group

Raw CESD score	Normal Weight Females (pounds)	Normal Weight Males (pounds)	Over-Weight Females (pounds)	Over-Weight Males (pounds)	Obese Females (pounds)	Obese Males (pounds)
10	7.39	7.21	6.25	6.07	12.76	12.58
20	7.12	6.94	5.68	5.50	13.94	13.76
30	6.96	6.78	5.33	5.15	14.65	14.48

Note. Very few subjects scored >30, reflected in this table.

Table 23

Actual Weight Changes over 5 Years Among 40 Year Old Subjects for Raw CES-D Scores by Gender and Weight Group

Raw CESD score	Normal Weight Females (pounds)	Normal Weight Males (pounds)	Over-Weight Females (pounds)	Over-Weight Males (pounds)	Obese Females (pounds)	Obese Males (pounds)
10	4.61	4.43	3.47	3.30	9.98	9.80
20	4.35	4.17	2.90	2.72	11.17	10.99
30	4.19	4.01	2.56	2.38	11.88	11.70

Note. Very few subjects scored >30, reflected in this table.

Table 24

Actual Weight Changes over 5 Years Among 50-Year Old Subjects for Raw CES-D Scores by Gender and Weight Group

Raw CESD score	Normal Weight Females (pounds)	Normal Weight Males (pounds)	Over-Weight Females (pounds)	Over-Weight Males (pounds)	Obese Females (pounds)	Obese Males (pounds)
10	1.84	1.66	.070	0.52	7.21	7.03
20	1.57	1.39	.013	-0.04	8.39	8.21
30	1.41	1.23	-0.21	-0.39	9.10	8.93

Note. A '-' denotes a weight loss. Very few subjects scored >30, reflected in this table.

Table 25

Actual Weight Changes over 5 Years Among 60-Year Old Subjects for Raw CES-D Scores by Gender and Weight Group

Raw CESD score	Normal Weight Females (pounds)	Normal Weight Males (pounds)	Over-Weight Females (pounds)	Over-Weight Males (pounds)	Obese Females (pounds)	Obese Males (pounds)
10	-0.93	-1.11	-2.07	-2.24	0.44	4.25
20	-1.19	-1.37	-2.64	-2.82	0,56	5.44
30	-1.35	-1.53	-2.98	-3.16	0.63	6.15

Note. A '-' number denotes weight loss. Very few subjects scored >30, reflected in this table.

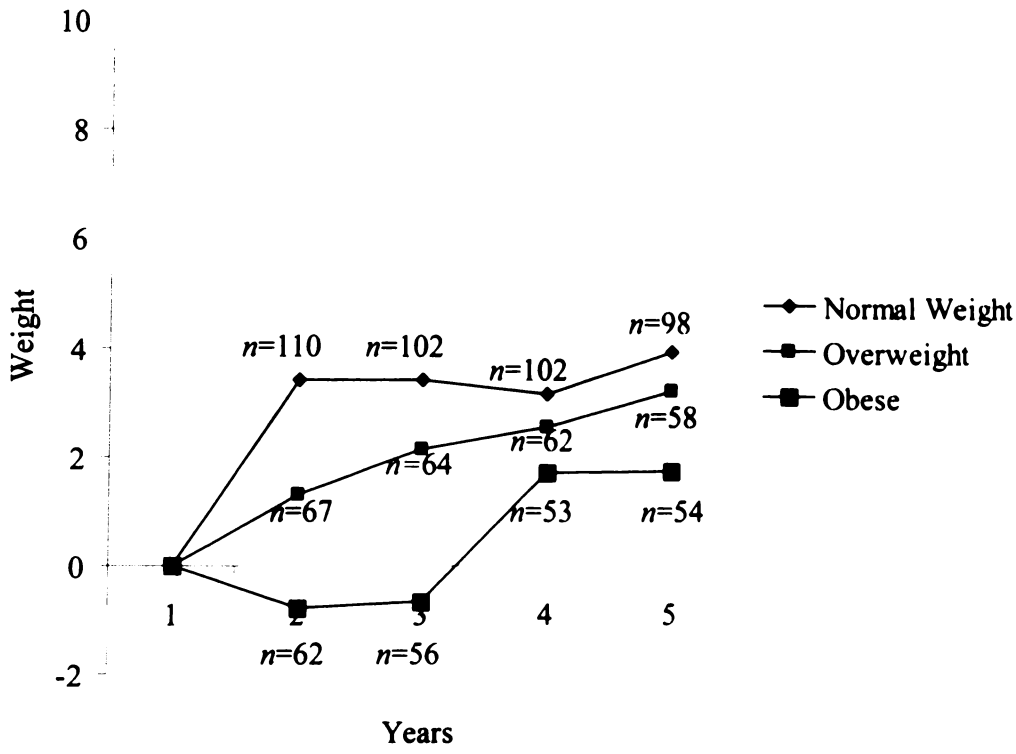
Table 26

Correlation Matrix of ln(CESD+1) scores and BMI by Gender and Weight Group

	CES-D by BMI; (*r*, *p* value, *n*)	CES-D by BMI (*r*, *p* value, *n*)	CES-D by BMI (*r*, *p* value, *n*)	CES-D by BMI (*r*, *p* value, *n*)	CES-D by BMI (*r*, *p* value, *n*)
Females	Baseline	Year 1	Year 2	Year 3	Year 4
	$r = 0.13$,	$r = 0.21$,	$r = 0.16$,	$r = 0.26$,	$r = 0.18$,
	$p = .04$,	$p = .002$,	$p = .02$,	$p = .000$,	$p = .01$,
	n=253	n=230	n=104	n=216	n=197
Males	$r = 0.12$,	$r = 0.14$,	$r = 0.15$,	$r = 0.15$,	$r = 0.11$,
	$p = .09$,	$p = .04$,	$p = .03$,	$p = .03$,	$p = .13$,
	n=254	n=239	n=219	n=212	n=192

Figure 1

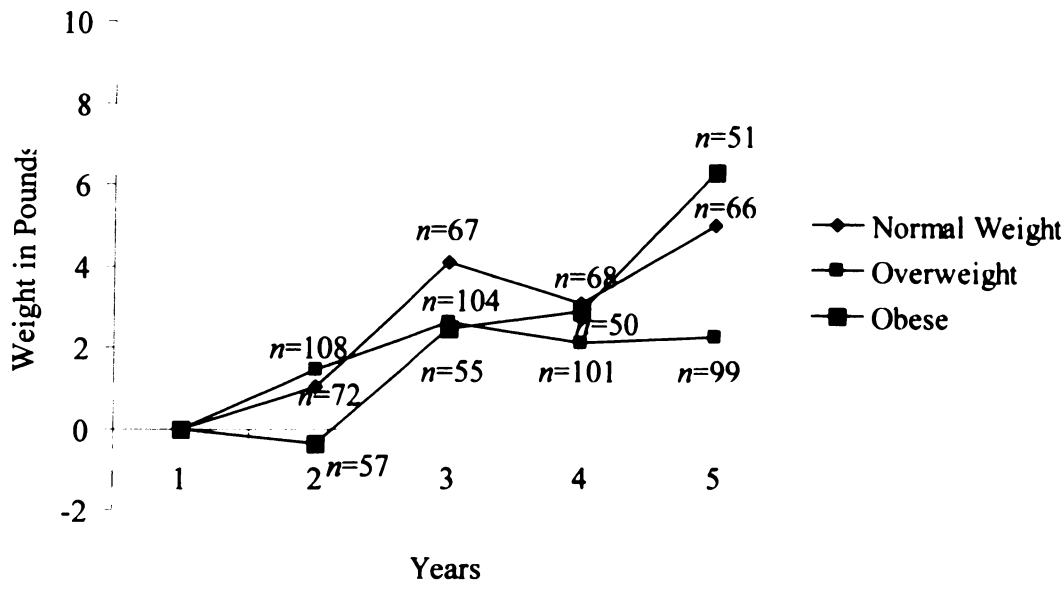
Average Weight Change in Pounds From Baseline for Females by Weight Group Over Five Years



Note. Baseline *n* for normal weight females = 113; baseline *n* for overweight females = 70; and baseline *n* for obese females = 68.

Figure 2

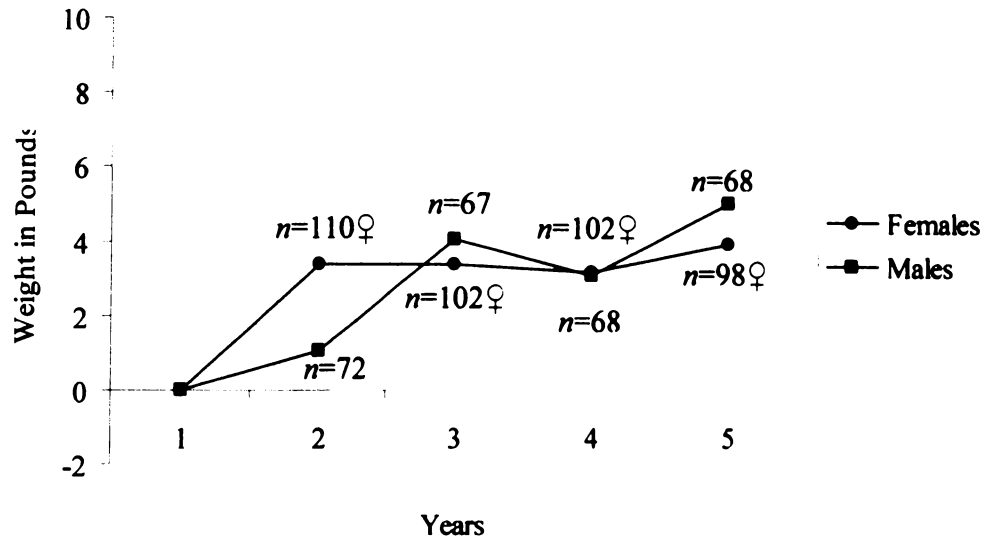
Average Weight Change in Pounds From Baseline for Males by Weight Group Over Five Years



Note. Baseline *n* for normal weight males = 74; baseline *n* for overweight males = 113; and baseline *n* for obese males = 61.

Figure 3

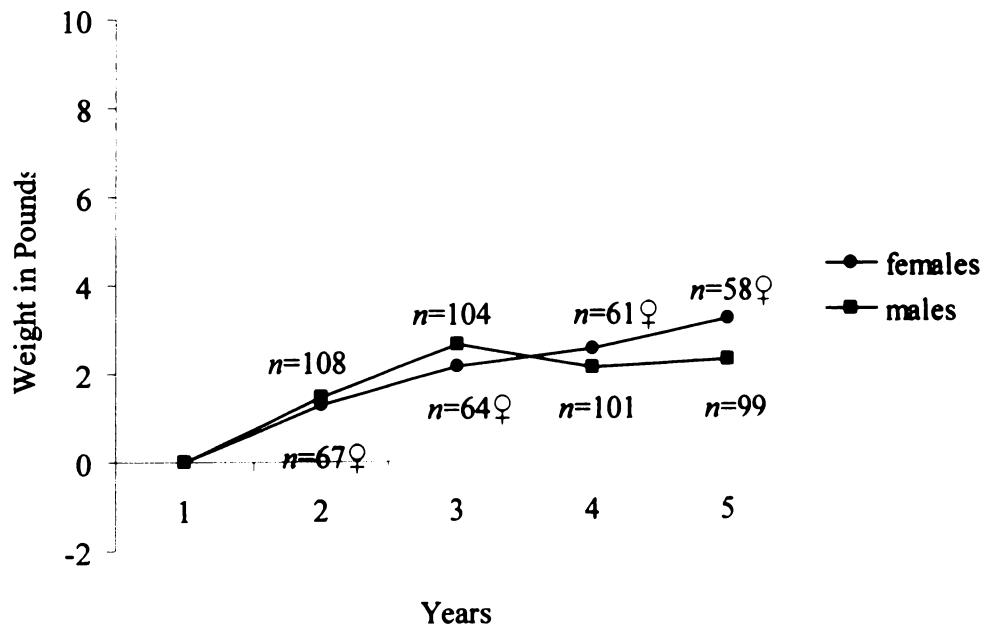
Weight Gain in Pounds for Normal Weight Females and Males Over Five Years



Note. Baseline *n* for normal weight females = 113; baseline *n* for normal weight males = 74.

Figure 4

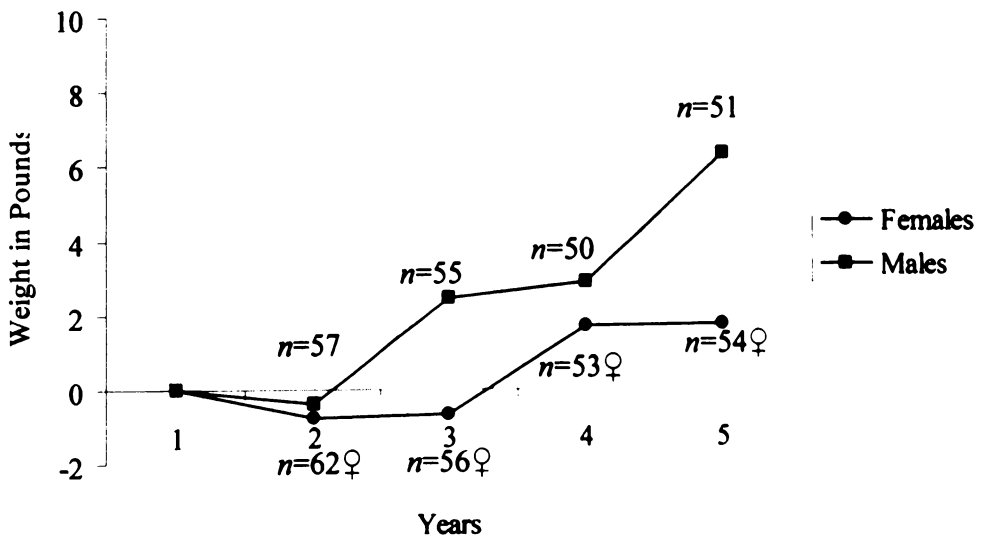
Weight Gain in Pounds for Overweight Females and Males Over Five Years



Note. Baseline *n* for overweight females = 70; baseline *n* for overweight males = 113.

Figure 5

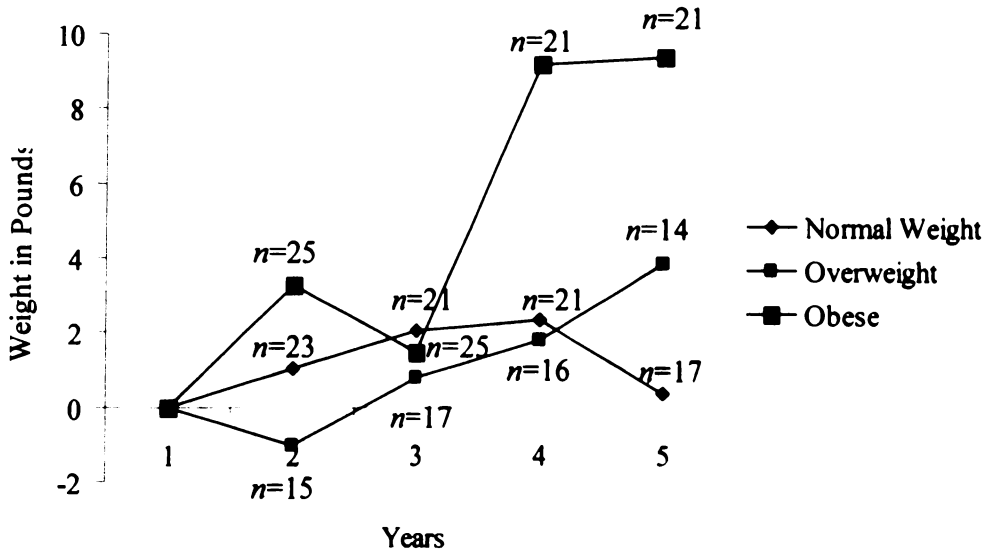
Weight Gain in Pounds for Obese Females and Males Over Five Years



Note. Baseline *n* for obese females = 68; baseline *n* for overweight males = 61.

Figure 6

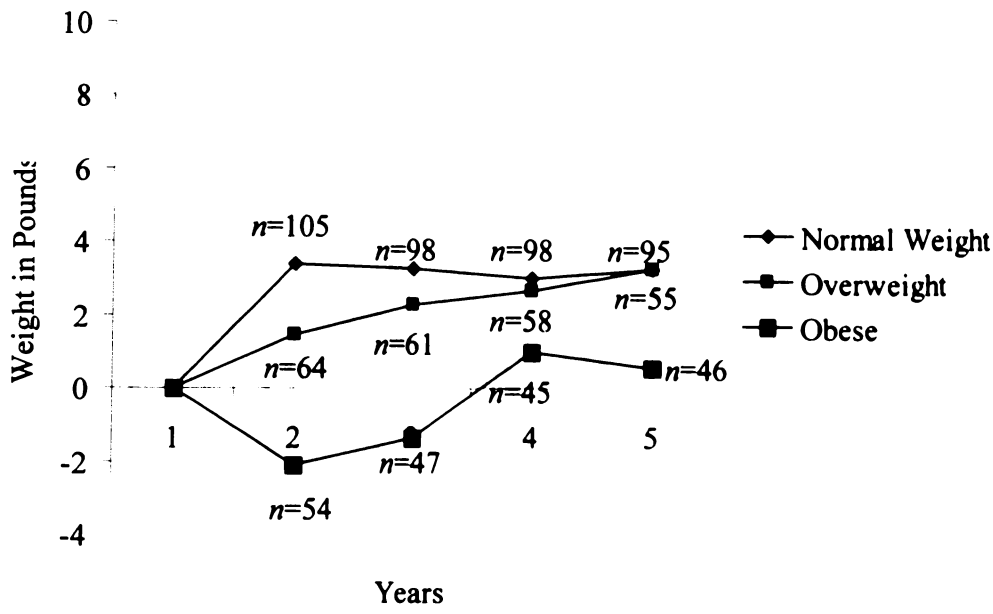
Weight Changes in Pounds for Females with CESD Scores ≥17 Over Five Years



Note. Baseline *n* for normal weight females = 24; baseline *n* for overweight females = 17; and baseline *n* for obese females = 25.

Figure 7

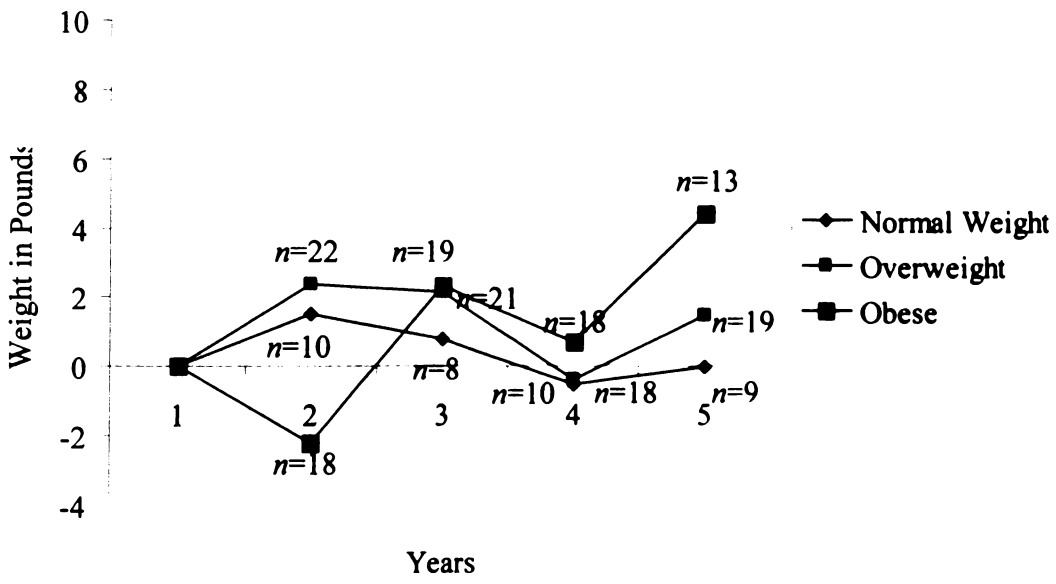
Weight Changes in Pounds for Females with CES-D Scores ≤16 Over Five Years



Note. Baseline *n* for normal weight females = 108; baseline *n* for overweight females = 67; and baseline *n* for obese females = 57.

Figure 8

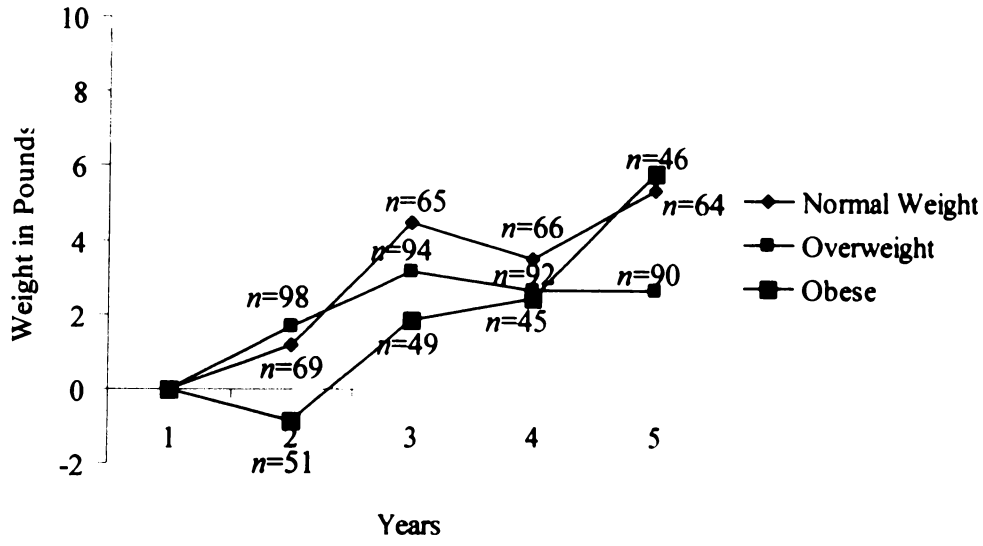
Weight Changes in Pounds for Males with CESD Scores ≥17 Over Five Years



Note. Baseline *n* for normal weight males = 12; baseline *n* for overweight males = 26; and baseline *n* for obese males = 20.

Figure 9

Weight Changes in Pounds for Males with CESD Scores ≤16 Over Five Years



Note. Baseline *n* for normal weight males = 71; baseline *n* for overweight males = 100; and baseline *n* for obese males = 53.

Figure 10

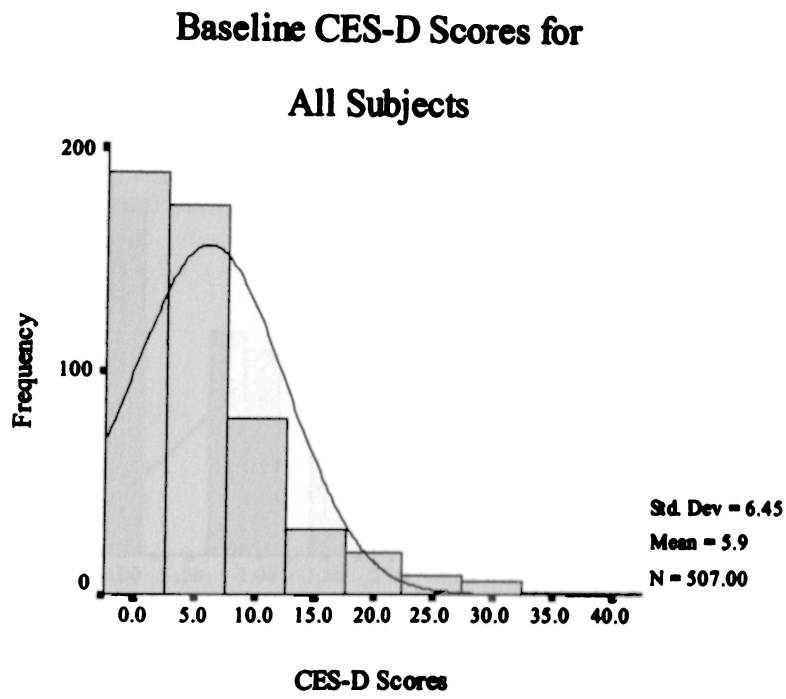
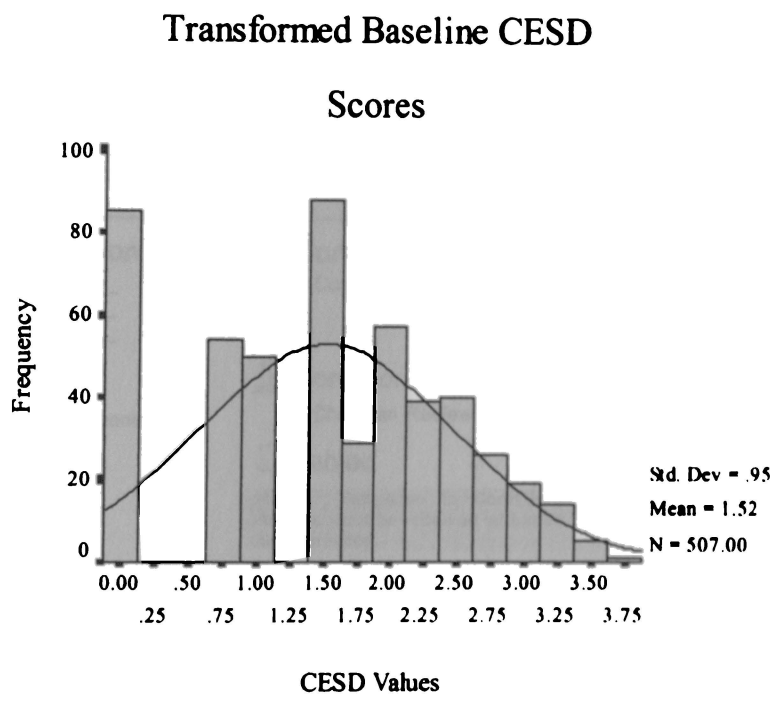

Baseline CES-D Scores for
All Subjects
200
100
0
Frequency
0.0
5.0
10.0
15.0
20.0
25.0
30.0
35.0
40.0
CES-D Scores
Std. Dev = 6.45
Mean = 5.9
N = 507.00

Figure 11


Transformed Baseline CESD Scores
Frequency
100
80
60
40
20
0
0.00
.25
.50
.75
1.00
1.25
1.50
1.75
2.00
2.25
2.50
2.75
3.00
3.25
3.50
3.75
Std. Dev = .95
Mean = 1.52
N = 507.00
CESD Values

University of Kansas
KUMC
Medical Center
HUMAN SUBJECTS COMMITTEE - REPORT OF ACTION

HSC #: 9243 TITLE: The Influence Of Depression On Overweight And Obese Women: A Secondary Data Analysis From Tl RENO Diet-Heart Study

Responsible Investigator: Sue Popkess-Vawter Ph.D.
Department: Internal Medicine
External Granting Agency: _____ Grant/Protocol #: _____

☒ Approval for Implementation
Surveillance Level ____
Exemption Class ____
Expedited Category 7

☐ Conditional Approval
(Committee Review Required)

☐ Reviewed/No Action Taken

☐ Reviewed and Recertified
☐ Implementation ☐ Non-Implementation

☒ Conditional Approval *
(Chairman Review Required)

☐ Administrative Termination

☐ Disapproved

☐ Tabled

☐ Administrative Re-instatement

☒ HIPAA

NOTE: Response to Conditional Approvals/Tabled Actions must be received within 30 days or project will be terminated.

COMMITTEE MINUTES

This is a study about the influence of depression on overweight women. Because it only involves a secondary data analysis of information collected in a prior research study, it is determined to be a minimal risk study. This study is determined to be **expeditable** under **category (7)**.

The Committee requests that the investigator respond to the following points by means of a letter:

1. Please provide a copy of the consent form that the subjects signed in the earlier study, and highlight the section in the consent form that specifically indicates that the subjects had consented to secondary data analyses such as the one that you are performing.

This project was reviewed by the full Committee and is approved with the above provisos with review by the Chairman. This action was approved unanimously. There were 15 members present and 1 absent.

CHAIRMAN'S SIGNATURE _____ **DATE OF ACTION** 5/27/2003

NOTE: Unless indicated above, signature alone does not imply approval for implementation.

*Response to Conditions Accepted: _____ 6/23/03

HSC # 9243 Popkess-Vawte

HIPAA PROVISOS - MAY 27, 2003
No HIPAA provisos.

Rachel Dalthorp
HIPAA Compliance Office Approval

Appendix B

The CES-D (Radloff, 1977)

Name:_____ Date:____/_____/_____

(day/month/year)

CES-D *Instructions: Below is a list of the ways you might have felt or behaved. Please tell me how often you have felt this way during the past week.*

Responses: A. Rarely or none of the time *(less than 1 day)*
B. Some or a little of the time *(1-2days)*
C. Occasionally or a moderate amount of time *(3-4 days)*
D. Most or all of the time *(5-7 days)*

1.	I was bothered by things that usually don't bother me.	☐	☐	☐	☐
2.	I did not feel like eating, my appetite was poor.	☐	☐	☐	☐
3.	I felt that I could not shake off the blues even with the help of my family or friends.	☐	☐	☐	☐
4.	**I felt that I was just as good as other people.**	☐	☐	☐	☐
5.	I had trouble keeping my mind on what I was doing.	☐	☐	☐	☐
6.	I felt depressed.	☐	☐	☐	☐
7.	I felt that everything I did was an effort.	☐	☐	☐	☐
8.	**I felt hopeful about the future.**	☐	☐	☐	☐
9.	I thought my life had been a failure.	☐	☐	☐	☐
10.	I felt fearful.	☐	☐	☐	☐
11.	My sleep was restless.	☐	☐	☐	☐
12.	**I was happy.**	☐	☐	☐	☐
13.	I talked less than usual.	☐	☐	☐	☐
14.	I felt lonely.	☐	☐	☐	☐
15.	People were unfriendly.	☐	☐	☐	☐
16.	**I enjoyed life.**	☐	☐	☐	☐
17.	I had crying spells.	☐	☐	☐	☐
18.	I felt sad.	☐	☐	☐	☐
19.	I felt that people dislike me.	☐	☐	☐	☐

20. I could not "get going". □ □ □ □

Scoring for the CES-D Scale:

Total score ranges from 0-60. Scores are higher in depression.

Patients with scores greater than 15 are likely to be depressed.

Score A=0, B-1, C=2, D-3 for all questions except 4, 8, 12, and 16.

Score A=3, B=2, C=1. D=0 for questions 4, 8, 12, and 16.