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APPROVAL BY PROJECT DIRECTOR

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DEFINITIONS USED IN THE REPORT

APN: Advanced Practice Nurse

BMI: Body Mass Index is calculated by dividing the body weight in kilograms by the square of height in meters (BMI= kg/m²)

Overweight: A BMI in excess of 25 indicates overweight

Obesity: A BMI above 30 is considered obese

Waist circumference: the smallest circumference below the rib cage and above the umbilicus

Hip circumference: the circumference hips is measured at the largest circumference at the posterior extension of the buttocks

Waist-hip ratio: the circumference of the waist divided by the circumference of the hips
ABSTRACT

This literature review highlights the current literature on the theories of causes, contributing factors, assessment and diagnosis, comorbid conditions associated with, and current treatments for overweight and obesity. This paper will outline current tools used to assess and identify those who are or are at risk for overweight and obesity. The current gold standard for assessing and diagnosing overweight and obesity is the body mass index (BMI). Current treatment options include lifestyle modification, behavior therapy, pharmacologic treatment, and surgery. The theoretical underpinning for counseling patients about weight issues is the Health Belief Model, will be discussed in Chapter 2.

Obesity is a growing epidemic that affects women more often than men. The prevalence of overweight and obesity among women in the United States has reached the point where it is more common than a normal body weight. The combined prevalence of overweight and obesity in women over the age of twenty years is about 51%. Studies have shown that many overweight and obese patients are not counseled to lose weight and are not counseled about the health consequences of obesity and overweight.

The recommendation made by the author in this project is that all healthcare providers should assess all women for overweight and obesity using the BMI, and offer appropriate counseling as needed at each patient encounter, regardless of the presenting problem.
CHAPTER 1

In this chapter, the author outlines the purpose of this project and a statement of the problem. This chapter will briefly discuss the background of obesity, and the implications it has to nursing, women, and society.

Introduction

Obesity is the number one nutritional disorder in the developed world. According to the most recent National Health and Nutrition Examination Survey (NHANES III, 1988-1994), between one-third and one-half of US men and women 20 years and older are overweight, and nearly one-fourth are clinically obese. It is often a lifelong problem that preferentially affects women (Dickerson, 2001).

Many overweight and obese patients report that they have not been counseled to lose weight by their primary care provider. Although the diagnosis at times is clear just by looking at the patient, there are useful tools available to clinicians to assess body weight in terms of risk of co-morbid conditions associated with obesity, such as heart disease, hypertension, hyperlipidemia, diabetes, cholelithiasis, cancer, depression, reproductive disorders, joint and bone pain, among many others. Obesity has also been shown to lead to premature death. Many more women are affected by obesity than men, and women tend to have more health complications associated with obesity than men, as will be outlined in this project.

There are many treatment options for those who are overweight or obese. The most obvious of the treatment options are to reduce the caloric intake to below that of the
As this report will show, there is evidence to suggest that there are genetic influences that make people become overweight. So simply eating less may be difficult or impossible for some patients. None of these methods guarantee sustained weight loss, owing to the nature of the disease. Obesity is a chronic condition requiring chronic assessment and treatment.

Purpose Statement

The purpose of this project was to review the current literature on overweight and obesity, with particular emphasis on the impact it has on women’s health issues. This project examines current recommendations for screening for, preventing, and treating overweight individuals. Current therapeutic regimens for overweight and obese clients are offered. The importance of healthcare providers screening for overweight, obesity, and related co-morbid conditions is stressed in this paper.

Statement of the Problem

Many recommendations have been made about the frequency and quality of weight screening, yet as this report will show, all too often practitioners do not routinely address weight issues with their clients. If health practitioners are to impact the health of their clients, they cannot ignore this rapidly growing epidemic. There are many health disparities that are caused by, related to, or exacerbated by overweight and obesity, as will be discussed in Chapter 2.
Background

The U.S. Preventative Services Task Force has issued a recommendation that clinicians screen all adults for obesity and offer obese patients intensive counseling and behavioral interventions to promote sustained weight loss or refer them to other clinicians for these services (1989). The American Academy of Family Physicians, the American Heart Association, the Institute of Medicine, the American Academy of Pediatrics, the Bright Futures Guidelines, and the American Medical Association (AMA) Guidelines for Adolescent Preventative Services all recommend measurement of height and weight as part of a periodic health examination for patients. Periodic height and weight measurement are inexpensive, rapid, reliable, and require minimal training to perform. They may also be useful for the detection of medical conditions causing unintended weight loss or weight gain, such as cancer or thyroid disorders.

The AMA urges physicians to assess their patients for overweight and obesity during routine medical examinations and discuss with at-risk patients the health consequences of further weight gain. If treatment is indicated, physicians should encourage and facilitate weight maintenance or reduction efforts in their patients and refer them to a physician with a special interest and expertise in the clinical management of obesity (Lyznicki, Young, Riggs & Davis, 2001).

Despite these recommendations, a study conducted by Nawaz, Adams and Katz (1999), showed that only 29% of overweight patients were counseled by a physician to lose weight. The study showed that there was no relation to gender, race, education, or
income among the patients who were counseled and those who were not. Patients who were overweight and had diabetes or hyperlipidemia were more likely to be counseled than those with no additional risk factors. Overall, 10%, 31%, 35%, and 43% of overweight subjects with 0, 1, 2 and three additional risk factors, respectively, were counseled to lose weight. Overall, 71% of the overweight adults in this survey were not counseled to lose weight, while 5% reported inappropriate advice to either gain or maintain weight.

In this study, physicians counseled overweight patients more consistently when metabolic derangements or complications attributable to obesity were evident (heart disease, diabetes, hyperlipidemia) than when seemingly unrelated risk factors such as smoking or sedentary lifestyle were concurrent. This pattern suggests an emphasis on secondary and tertiary prevention and a relative neglect of the primary prevention of obesity and its sequelae.

What these findings show is that clinicians often do not address the issue with their obese patients. A change is needed in the training of physicians and other health care providers to incorporate nutrition education and tools for counseling, with a greater emphasis on prevention. The purpose of screening for obesity is to assist the obese individual to lose or at least maintain weight and thereby prevent complications of obesity. To benefit from the detection and treatment of obesity, however, patients must be motivated to lose weight, must have access to an efficacious method of reducing body weight, and must maintain the resulting weight loss.
Significance to Nursing

Overweight and obesity are health disparities that not only affect patients, but also health care providers as well. From a nursing standpoint, nursing duties are more difficult and sometimes impossible with overweight and obese clients. For example, instruments used for health screening typically used by nursing staff are all too often insufficient to meet the needs of overweight patients. Scales many times do not read weights over 300 pounds, thereby making weight measurements unreliable. Blood pressure cuffs do not fit properly, thereby making it impossible to obtain accurate readings. Excess adipose tissue makes it difficult to administer medications intramuscularly and subcutaneously, and also makes it difficult to access an IV, thereby limiting or affecting the delivery of medications and life-saving therapies. Office chairs and exam tables are often too small to allow comfortable positioning for assessment.

Nurses in advanced practice positions may find routine assessment skills difficult. Inspection of skin and bony structures is difficult due to the excess amount of adipose tissue and loss of landmarks due to excess fat. Auscultation and palpation are also difficult because of the large amounts of adipose tissue, and thorough evaluation of heart, lung, and bowel sounds is impaired. Palpation of internal organs and bony structures may be inadequate due to limited mobility and increased adipose tissue. All of these impact diagnosis. Clinicians may find that diagnostic reasoning is greatly impaired due to limitations placed on them and the patient because of large body size, increased adiposity, and limited mobility of overweight patients.
Many assessment tools such as vaginal speculums, radiography, CT scanning, MRIs and sonography are often difficult or impossible to perform and interpret due to excessive adiposity and larger body sizes. If the epidemic of obesity is not properly addressed and treated, then these difficulties in providing adequate care will have a negative impact on health screening and care delivery of overweight patients. Medical and surgical procedures are more risky for overweight patients, limiting the availability of safe and effective diagnostic and treatment options.

Many nurses and other healthcare providers have injured themselves by trying to lift or assist obese patients. That results in a decrease in the number of available nursing staff, which further exacerbates the nation’s problem of too few nurses able to deliver healthcare. More and more of these patients will end up in hospitals, with fewer nursing staff to provide care.

Manufacturers of hospital beds have now developed bariatric beds that have built-in scales, and easily transferable mattresses to aid in easier transfer of patients. There are now machines that can be used to lift patients. The equipment developed to reduce the risk of personal injury are largely unavailable to most nursing staff and can be very expensive. There have been many changes in the delivery of healthcare for obese patients, but there are still far more health care issues that have not been able to be solved in recent years. For example, there is still a great need for diagnostic testing that will accommodate to larger body sizes.
Many issues surrounding the current health care delivery system have also made it difficult for primary care providers to adequately assess and treat obesity and its comorbid conditions. Lack of payment by most health-insurance and managed-care plans for overweight or obesity treatment programs, and lack of sufficient time for dedicated patient education and counseling on weight loss and maintenance make it difficult for practitioners to work with patients on weight management. In hospitals, increased time and assistance is needed to carry out simple duties with obese clients. Many times it takes the help of a team of staff to move or lift patients, and to carry out basic tasks with patients who cannot move themselves.

Other barriers on the part of the health care provider may prevent optimal assessment and treatment of overweight and obese patients. Provider characteristics include lack of recognition of obesity as a chronic condition that is difficult to treat, and one that requires continuous and long-term management, and that also has high recidivism rates. Negative and unsympathetic perceptions that obesity represents a lack of patient discipline, self-control or will power, rather than a chronic disease, may prevent the provider from adequately assessing and treating the patient. Another barrier involves inadequate training for primary care providers in the medical management of obesity, which ultimately leads to inadequate assessment and management of overweight or obese patients. Patient interest and readiness to change ultimately determine whether or not any management strategy will result in weight loss (Lyznicki et al., 2001).
Until recently, obesity has been largely unrecognized by physicians as a condition that requires medical intervention, and patients have been regarded as having “no will-power” or as gluttons (Klauer & Aronne, 2001). Research shows that healthcare providers, like others in society, describe overweight individuals as repulsive, disgusting, weak, and lacking self-discipline (Rogge, Greewald & Golden, 2004). Each clinician needs to develop self-awareness of the overt and covert messages conveyed to obese patients about their weight, their weight loss efforts, and especially their weight control failures. Nurse practitioners are in a wonderful position to educate patients regarding diet and exercise, as well as help with the maintenance of appropriate weight and identification of those at risk for overweight and obesity (Holcomb, 2004).

Significance to Women

Overweight and obesity are a significant health disparity that disproportionately affects women. In the U.S. today, the healthcare provider who treats women, from adolescence through the post reproductive and geriatric years, sees a population particularly vulnerable to the ramifications of obesity and excessive weight gain. The prevalence of overweight and obesity among women in the United States has reached the point where it is more common than a normal body weight (Klauer & Aronne, 2002). The combined prevalence of overweight and obesity in women over the age of twenty years is about 51% (Lyznicki et al., 2001).
Significance to Society

The Centers for Disease Control and Prevention estimates that the total economic cost of obesity in the United States is about $117 billion per year, including more than $50 billion in avoidable medical costs (Klauer & Aronne, 2002). It is estimated that obesity results annually in the loss of 39 million workdays. The cost of obesity to U.S. companies amounts to more than $13 billion annually in decreased productivity and increased medical fees (Couzelis, 2005).

Obese people experience chronic diseases more frequently than non-obese individuals. As a result, obese people pay an average of $10,000 more during their lifetime for medical care related to chronic diseases than their non-obese counterparts. A team of Stanford University researchers has calculated that every excess pound of body fat on US citizens drains approximately $25 from the American health care system annually (Peterson, 2005).

Lower salaries are yet another way in which obesity can negatively impact an individual. Economists have noted that there is a negative relationship between weight and wages. However, the cause and nature of this relationship is not well understood. For women, wages are lower for those with higher BMI and body weight (Couzelis, 2005).

What these statistics do not convey is the fact that everyone, not just obese people, will be affected by the epidemic. The obesity epidemic will increase health care costs, and employers and government agencies will pass on at least some of these
increases to consumers. Increased health care costs could influence people to only seek out symptomatic care while neglecting to stay current with indicated preventative services and failure to control disease risk factors, which can only result in a spiral of unnecessary illnesses, health care costs and disability. These economic and health factors will increase poverty and disability rates (Kottke, Wu & Hoffman, 2003).

Summary

The rates of overweight and obesity are rising to epidemic proportions, especially among women. There are many health risks associated with increased weight. Excess body weight makes it difficult for healthcare providers to deliver adequate healthcare for many reasons. Routine health maintenance is ignored by many overweight persons for various reasons. Patients may be embarrassed or ashamed, or they may feel judged or ridiculed. They may have difficulty fitting into gowns, on exam tables, positioning themselves for pelvic or rectal exams, or even standing on a scale or having their blood pressure taken. For whatever reason, lack of routine healthcare maintenance leads to increasing incidence of health disparities and decreased overall well-being in this population.

Health professionals, particularly nurse practitioners, have an important role in promoting preventative measures and encouraging positive lifestyle behaviors, as well as identifying and treating obesity-related co-morbidities. They also have a role in counseling patients about safe and effective weight loss and weight management programs.
We know, by reviewing the literature, that overweight and obesity are not assessed as frequently as recommended. We also know that obviously overweight or obese patients are not always counseled about the health implications associated with their weight. According to the guidelines set by many health organizations, adults should be screened for overweight and obesity periodically, and offered appropriate counseling and referrals as needed.

All patients, but women in particular, should be screened for overweight and obesity at every patient encounter, since the literature reveals that more women are more often obese than overweight, and the incidence of obesity is higher in women than in men.
Chapter 2

Introduction

In this chapter, the theoretical framework that is used in this project as an explanation of why weight assessment and counseling is important in all female clients is outlined. Current guidelines for defining obesity will be discussed. There will also be an overview of the current literature concerning theories about obesity development, co-morbid conditions associated with obesity, associated medical conditions, as well as current successful therapies for treating obesity in adults.

Theoretical Framework

The framework for explaining health behavior is the Health Belief Model (HBM). The HBM addresses a person's perceptions of the threat of a health problem and the accompanying appraisal of a recommended behavior for preventing or managing a problem (Robison & Kish, 2001). Its focus is on the behavior related to the prevention of disease, and seeks to explain why people do or do not engage in preventative health action in response to a specific disease threat. The following are factors that determine whether or not a person undertakes the recommended health actions: the person's perceptions of their own susceptibility or perceived threat of the disease; the degree of severity of consequences perceived; the health action's potential benefits in preventing susceptibility; and the physical, psychological, financial and other barriers related to the behavior. Demographic, social-physiological, and structural variables are included in the theory as potential modifying factors that influence both individual perceptions and the
perceived benefits of the preventative actions.

APNs using this model will plan care with motivation as their central focus. The APN must educate patients about potential health threats, persuade them to take preventative action by heightening perceived susceptibility of a health problem, enhance perceived benefits of health actions, offer assistance in identifying barriers, provide reassurance and incentives, and assistance in making preventative health care choices. "The most promising application for this model is to serve as a guide for APNs to develop messages that are likely to persuade individuals to make healthy decisions" (Robinson & Kish, 2001, p.574).

This theoretical framework is relevant to the obesity epidemic because much of our society equates overweight and obesity to solely a cosmetic issue. Patients may not fully understand the health implications of being overweight, making it imperative that the subject of weight be out in the open and not ignored in the health care setting. Prevention is difficult unless the patient understands and internalizes his or her own personal risk for the co-morbid conditions associated with obesity. That is why every health care practitioner who encounters women who are, or are at risk for becoming overweight or obese should provide a thorough assessment for obesity and its co-morbid conditions, and offer individualized counseling and treatment to either prevent or reverse the progression of overweight and obesity.
Review of the Literature

Guidelines for defining overweight and obesity are useful for practitioners when screening patients for excessive adiposity and when prescribing treatment for overweight and obese patients (Gallagher, Heymsfield, Heo, Jebb, Murgatroyd & Sakamoto, 2000).

Assessing Body Fat

Ideal Body Weight

Body weight alone is not a very sensitive indicator for defining obesity (Atkinson, 1993). Factors such as age, sex, height, lean muscle mass and body fat percentage are all unaccounted for when using weight alone to measure healthy body weight on an individual level. Thus, weight alone should be avoided as a standard for assessing a person’s relative risk for overweight and obesity. Ideal body weight is an unscientific measure, and can provide only an estimate of appropriate weight. A simple way to calculate ideal body weight for women is as follows: 100 lbs for the first 60 in (5 ft) + 5 lbs for every additional inch (Davis & Sherer, 1994, p.292).

Percentage of Excess Body Weight

Height-for-weight charts (Appendix A) are not predictive of medical conditions associated with increased weight. They can best be used as a screening tool in a nutritional assessment. Deviations from the ideal body weight (IBW) indicate the degree of depletion or overweight. Percentage of excess body weight is calculated by: \( \text{Percentage of IBW} = \frac{\text{actual weight} - \text{IBW}}{\text{IBW}} \times 100 \). This number is the percentage of IBW. Obesity, by this measurement, is defined as 20% above ideal. These tables reflect a population consisting
of mainly white, middle class Americans, and may not yield an accurate assessment of other populations (Atkinson, 1993). Also, the 85th percentile is the upper limit for desirable weight. As Americans continue to become fatter, the 85th percentile increases as the weight of the population increases, making the 85th percentile higher and higher with each survey. Therefore, using percentage of excess body weight is not an accurate indicator of desirable, healthy weight status.

**Body Fat Mass**

The most accurate assessment of obesity is body fat content. Methods used to measure body fat mass include measuring body circumference, skin fold measurements, bioelectric impedance, underwater weighing, computed tomography, nuclear magnetic resonance imaging, or dual x-ray absorptiometry. These assays are largely unavailable to most practitioners and are prohibitively expensive (Atkinson, 1993). Therefore these methods are not suggested for routine screening purposes.

**Body Mass Index**

Healthcare providers should screen for obesity using the body mass index. Those with a BMI between 25 and 29.9 are considered overweight and those with a BMI above 30 are considered obese. The U.S. Preventative Services Task Force found good evidence that body mass index (BMI) is reliable and valid for identifying adults at increased risk for mortality and morbidity due to overweight and obesity (Calonge, 2004). The BMI is a desirable measurement of the tendency towards obesity (Atkinson, 1993). BMI is easy to determine, correlates fairly well with body fat and consequent
morbidity and mortality. BMI is therefore thought to be the most desirable clinical measure of obesity short of measuring body composition.

In adults, overweight and obesity are defined as BMI levels at which adverse health risks increase (Lyznicki et al., 2001). The BMI is an accurate and cost effective alternative to more expensive and invasive tools to evaluate body fatness. The BMI is a desirable measurement of the tendency towards obesity, and helps to identify those at risk for overweight and obesity.

*Waist Circumference*

The distribution of body fat has significant implications for health. Central, or visceral adiposity increases the risk for cardiovascular and other diseases, independent of obesity. Individuals with this characteristic are at the highest risk for developing type 2 diabetes, metabolic syndrome, and subsequent cardiovascular complications, including retinopathy, nephropathy, neuropathy, macular degeneration, and cardiovascular disease (Ribisl, 2004). Risk for such disease progression is caused by excess visceral adipose tissue; simply being overweight is not the culprit. It is not the total amount of body fat that creates this problem, but the location of the body fat. The Gothenberg, Sweden, longitudinal study showed that increased waist size was positively correlated with an increased incidence of myocardial infarction, angina, and stroke independent of age and BMI (Klauer & Aronne, 2002).

Clinicians may use the waist circumference as a measure of central adiposity. According to the US Preventative Task Force, men with a waist circumference greater
than 40 inches and women with a waist circumference greater than 35 inches are at increased risk for cardiovascular disease (2003).

**Waist-Hip Ratio**

An elevated waist/hip circumference ratio, which may indicate central adiposity, has been shown to correlate to with the presence of hypercholesterolemia, diabetes and coronary artery disease, independent of BMI, and may predict the complications of obesity in adults better than BMI does.

In addition, determination of the waist/hip ratio may be useful for assessing some adults, particularly those whose weight or BMI is borderline for classification as overweight and who have personal or family history medical histories placing them at increased health risk. The waist/hip ratio may be a better predictor of the sequelae associated with adult obesity then BMI, can also be measured in the clinical setting. The reliability of the waist/hip ratio is comparable to that of the BMI. A wait/hip ratio greater than 1.0 in men and 0.8 in women has been shown to predict complications from obesity, independent of BMI, although the waist/hip ratio has not been evaluated in all ethnic groups. See Appendix D for waist/hip ratio tool.

**Pediatric Screening**

Defining overweight in the pediatric population is difficult, because a child’s BMI changes dramatically with age during childhood and adolescents. Therefore defining overweight in children and adolescents is done so by determining “BMI-for-age” in children aged 2-20 years old (Dalton & Watts, 2002). Children at or above the 85th to
95th BMI percentile are defined as “at risk of becoming overweight”; children at or above the 95th percentile are defined as “overweight”.

Defining overweight or obesity is difficult using the percentile method, since as the average weight of children continues to increase, then so does the percentile. So although, compared to their peers, a child may be at a “normal” weight by the charts, they are heavier and at increased health risk. The “normal” values increase as society is getting progressively overweight, making it difficult to assess this population adequately by the standard percentile method.

Theories and Causes of Obesity

Storage of excess calories as fat must ultimately result from a net positive energy balance (energy intake is greater than energy expenditure) over time (Rosenbaum & Liebel, 1998). The rise of global obesity is thought to be a by-product of environmental and behavioral changes linked to economic development, modernization, and urbanization. Paradoxically, obesity often coexists with a substantial level of malnutrition (Peterson, 2005).

Increasing evidence suggests that obesity is not a simple problem of will power or self-control, but a complex disorder involving appetite regulation and energy metabolism that is associated with a variety of co-morbid conditions. Although its etiology is not clearly established, genetic, metabolic, biochemical, cultural and psychosocial factors contribute to obesity (Lyznicki et al., 2001). In most cases, the
increasing prevalence of overweight and obesity reflects changes in society and behaviors over the past 20-30 years.

Evolution of Obesity

Humans have evolved as a species from hominids that were well-equipped to survive and reproduce in environments that yield an unsteady supply of readily available foods. Survival and reproduction were dependent on energy stores of the individual and the species. For evolutionary reasons, human physiology is predisposed to conserve and store weight, not to shed excess amounts. However, in the modern industrial environment that provides easy access to calorically-dense foods and encourages a sedentary lifestyle, the metabolic consequences of these genes are maladaptive (Rosenbaum & Liebel, 1998). The prevalence of childhood obesity has increased by more than 30% over the past decade. The rapid increase in the prevalence of obesity emphasizes the role of environmental factors, because genetic changes could not occur at this rate.

This increasing prevalence of obesity in the United States apparently represents the interaction of genes with an environment that encourages a sedentary lifestyle and consumption of calories. The current relative adiposity is a product of the interaction between genetic predisposition with regard to the storage of body fat and an environment (low physical activity, high availability of calorically-dense foods) that is increasingly permissive to the expression of that genetic tendency. Although there are clearly strong genetic influences on susceptibility to obesity, large changes in the prevalence of obesity over such a short time must reflect major changes in non-genetic factors, providing tacit
evidence that some instances or aspects of obesity must be responsive to, or preventable by, manipulation of the environment (eg, diet, physical activity).

In most humans, body fatness is a continuous quantitative trait reflecting the interaction of development and environment with genotype (Rosenbaum & Liebel, 1998). Studies in twins, adoptees, and families indicate that as much as 80% of the variance in BMI is attributable to genetic factors. Genetic influences on body weight are as potent as those on height. Heritability of adipose tissue distribution, physical activity, resting metabolic rate, changes in energy expenditure that occur in response to over-feeding, certain aspect of feeding behavior, food preferences, lipoprotein lipase activity, maximal insulin-stimulated acyglyceride synthesis, and basal rates of lypolysis are estimated to be as high as 40% (Rosenbaum & Liebel, 1998, p. 529).

According to Rosenbaum & Liebel (1998), there is substantial evidence that body weight is regulated by complex signaling systems that provide afferent signals, including glucostatic, lipostatic, and aminostatic signals to the CNS about the nutritional state of the organism, which are translated into efferent signals that affect energy intake and expenditure (p. 529).

Pathophysiology of Obesity

The following information concerning the pathophysiology of obesity was found in Kumar, Abbas & Fausto (2005), unless otherwise noted.

Energy intake from food and energy expenditure from cellular metabolism and exercise are precisely matched over long intervals in healthy adults resulting in stable
body fat stores. Energy is continuously expended, and the rate of expenditure varies among persons. The brain and the liver are efficient at controlling nutrient levels based on need. Following ingestion of food, nutrient levels move from the gut into tissues for immediate use or storage. Decreases of plasma fuels below levels to meet tissue requirements are rare in normal, free-feeding individuals. Under homeostatic conditions, the supply of energy in the blood does not decrease to below threshold levels and cause the brain to trigger eating. Even though ample energy is generally readily available, animals and humans still initiate meals.

Prior to initiation of a meal, there is a small decrease in plasma glucose of about 12%. The brain initiates a decline in plasma glucose by eliciting a small increase in plasma insulin via the vagus nerve to the pancreas, which precedes the pre-meal decline in glucose. Small physiologic fluctuations of glucose are hypothesized to provide important signals to the brain to elicit meals. There is also evidence that the liver responds to small fluctuations of fatty acids and their metabolites by sending signals to the brain via the vagus nerve, which in turn stimulates food ingestion.

Other events also predict the onset of meals, such as an increase in body temperature, past experiences of meal initiation, such as the time of day, social factors, and others. According to this evidence, individuals do not initiate meals because there is a lack of available energy, but rather the individual eats when it is accustomed to eating. The timing of meals is dictated by the individual’s lifestyle, convenience, and
opportunity. The timing and frequency of meals are driven more by lifestyle than by immediate need.

Most adult mammals, including humans, maintain a relatively constant level of adiposity over long intervals, and this occurs in spite of the variability of daily energy intake, expenditure and meal patterns.

**Hormonal Regulation of Appetite**

The central regulation of caloric intake and energy expenditure that contribute to energy balance involves interactions between the peripheral hormonal and neuromodulatory factors and neural pathways. Peripheral signals of hunger and satiety are interpreted in the hypothalamus and distributed to the periphery by the sympathetic nervous system. A positive energy balance or satiety is mediated by increased intestinal distention and other mechanical-chemical changes that induce neural impulses carried by the afferent vagus nerve and by augmentation in the circulating concentrations of glucose, leptin, cholecystokinin, glucagons-like peptide-1, and peptide YY.

Leptin is a signal secreted mainly from fat cells that controls food intake and energy homeostasis. The concentration of leptin in the blood is highly correlated with total body fat mass. Excess body fat that results in increased leptin production may actually be a correction for primary or secondary impairment of leptin-induced signal transduction in the hypothalamus. The decrease in body fat that occurs with diet-induced weight loss causes leptin concentrations to decrease and triggers responses that aim to conserve body fat.
In addition to stimulating secretion of anorexigenic peptides, leptin decreases expression of orexigenic neuropeptide Y (NPY) and agouti-related protein (AgRP). Activation of the melanocortin 4 receptor (MC4R) gene (a receptor site in the “satiety center”) within the hypothalamus not only suppresses food intake, it also increases energy utilization. Thus, loss of function mutations in the proopmelanocortin (POMC) or MC4R lead to hyperphagia and obesity in both experimental animals and humans.

When the leptin feedback system is disturbed, such as in animals or humans that either make no leptin or have no leptin receptor sites, there is a chronic bias to overeat and gain weight. These individuals are hyperphagic and extremely obese, and administration of leptin reverses this syndrome.

However, leptin alone cannot regulate this energy homeostasis alone. Both insulin and leptin satisfy the criteria to be adiposity signals. The hypothalamus relies upon information from adipose stores in the form of insulin and leptin, as well as information on immediately available energy from the liver and hindbrain, to help control food intake and energy expenditure. Insulin-deficient animals are hyperphagic and administration of insulin eliminates their hyperphagia, which suggests the importance of insulin as an adiposity signal.

Ghrelin stimulates hunger. Circulating ghrelin concentrations increase preprandially and decrease postprandially. Ghrelin increases food intake through the stimulation of ghrelin receptors on hypothalamic NPY expressing neurons and AgRP expressing neurons. Ghrelin suppresses the effects of leptin. Increased ghrelin secretion
and activation of ghrelin receptor sites increases expression of NPY, and AgRP. Ghrelin increases food intake by inhibiting MC4R, and decreases energy expenditure by lowering the catabolism of fat. Ghrelin also inhibits expression of POMC and other anorexigenic factors. Leptin (an anorexigenic, or appetite-suppressing factor) and ghrelin (an orexigenic, or appetite-stimulating peptide) act in a mutually antagonistic manner through their respective receptors in the hypothalamus and brainstem to regulate caloric intake and energy expenditure.

Peptide YY (PYY) signals satiety. PYY is secreted postprandially by endocrine L cells lining the distal small bowel and colon. PYY is secreted in proportion to the calories ingested. The initial release of PYY occurs shortly after food intake. The release of PYY is stimulated by nutrients, particularly carbohydrates and lipids, within the lumen of the small intestine and the colon. PYY inhibits food intake through inhibition of gut motility, causing a sense of satiety and by way of vagal influences on the hindbrain.

In a study conducted by Batterham et al. (2003), a single infusion of PYY, as compared to an infusion of saline, reduces appetite and food consumption by approximately 30% at an all-you-can-eat buffet lunch provided two hours after the infusion. In obese subjects, the endogenous postprandial PYY response was diminished as compared with that in lean subjects, even though the obese subjects consumed a greater amount of calories (Korner & Leibel, 2003).
Peptides released in the intestinal tract following nutrient ingestion such as PYY and cholecystokinin may inhibit food intake by decreasing gut motility, afferent signaling through vagal impulses, and inhibition in ghrelin secretion, as well as by direct central actions.

The amount of food intake is regulated by signals generated as food is eaten. Cholecystokinin (CCK) is secreted in the intestine in response to ingested food and stretch receptors in the gut. CCK acts to reduce meal size. According to Clegg and Woods (2004) when CCK is administered to animals or humans prior to a meal, meal size is reduced dose-dependently (p. 971). The availability of meal-generated, pre-absorptive negative feedback signals such as CCK to reduce meal size depends upon estrogen levels. Estrogen increases the satiating action of CCK. Hence, females generally eat less food, assuming normal circulating plasma estrogen concentrations.

Genetics of Obesity

Obesity is a disorder with a multifactorial etiology. According to the literature, only rarely does is result from a single gene disorder (Kumar et al., 2005, p. 464). In recent years, many “obesity” genes have been identified; genes that encode molecular components of the neuroendocrine system that regulates energy balance. Leptin, as described above, is the product of the OB gene. Animal studies have shown that mutations in this gene (OB/OB) result in and absence of leptin secretion, which leads to obesity. Administration of exogenous leptin reverses the obesity. Animals with
mutations in the leptin receptor (\textit{db/db}) do not, however respond to exogenous leptin administration.

Leptin receptors are found in various sites within the brain, and those most critical for the regulation of leptin are found in the hypothalamus. In this site, there are two major types of neurons: one called orexigenic receptors that produce appetite-stimulating neurotransmitters (NPY and AgRP). Leptin reduces the expression of these genes. The other set of leptin-sensitive neurons are POMC and cocaine and amphetamine-related transcript (CART). These neurons transcribe two anorexigenic neuropeptides. These neuropeptides are alpha-melanocyte-stimulating hormone (\textit{\&}-MSH) and CART. The POMC/CART neurons are also known as first-order neurons. The neurotransmitters produced by them (NPY, AgRP and \textit{\&}-MSH) then interact through their own specific receptors with second-order neurons that trigger the efferent systems with peripheral actions. When these neurons are activated by leptin signals, they exert a catabolic effect in the body. These neurons are the initial targets of leptin action.

In the anabolic pathway, the first-order NPY/AgRP neurons make monosynaptic connections to second-order neurons, which express orexigenic peptides melanin-concentrating hormone (MCH) and orexins A and B. This binding transmits feeding signals. These signals are attenuated when leptin is in excess and are activated by low levels of leptin.

In the catabolic pathway, \textit{\&}-MSH produced by the POMC/CART neurons bind to a second set of second-order neurons that express the MC4R gene. Catabolic output from
the MC4R neurons is relayed to the periphery via the endocrine and autonomic systems. This reduces feeding and increases energy expenditure. The energy-consuming actions of MC4R neurons are mediated in part by thyrotropin-releasing hormone (TRH). TRH activates the thyroxine axis in the anterior pituitary. TRH not only increases thermogenesis through the secretion of thyroxine, but it is also an appetite suppressant. A subset of MC4R neurons projects to sympathetic motor output areas. Fibers from these areas innervate brown adipose tissue, rich in beta-adrenergic receptors. When these receptors are stimulated, they cause fatty acid hydrolysis and also uncouple energy production from storage. Thus, fats are literally burned, and the energy produced is dissipated as heat.

There are six single gene defects that give rise to human obesity, and all of them involve proteins in the leptin-melanocortin pathway. Four of these are autosomal recessive and affect the leptin receptor, POMC. In all of these cases, there is profound hyperphagia and childhood-onset massive obesity. While these forms are rare, those caused by mutations in the MC4R gene are quite common. In a recent study cited by the authors (Kumar, Abbas & Fausto, 2005, p. 464), 5-8% of a cohort of 500 obese individuals had functionally important mutations in the MC4R gene. In these patients, despite abundant fat stores and leptin, energy consumption cannot be stimulated. The sixth monogenic form of human obesity results from a mutation in a transcription factor (SIM1) that is essential for the formation of second-order leptin neurons.
Estrogenic Influences

Gonadal hormones also influence food intake and energy expenditure through mechanisms that are not yet fully understood. Estrogen is recognized to help regulate food intake. Food intake reportedly varies in women across the menstrual cycle, with changes in food intake directly related to the cycling of estrogen in women according to a study cited by Clegg & Woods (2004). There is a reduction in food consumption around ovulation, and food intake in the follicular phase is less than that during the luteal phase of the cycle.

Over the 10-day to 18-day duration of these phases of the menstrual cycle, this difference in intake is sufficient to affect energy balance and adiposity. There are some reports of an increase in energy expenditure during the luteal phase in women, but this increase is small and unlikely to compensate for the changes in intake. In women who displayed intermittent anovulatory cycles, food diaries reflected changes in intake present during cycles in which ovulation occurred, but not during cycles where ovulation did not occur (Clegg & Woods, p.969). After puberty in females, estrogen modulates leptin synthesis and secretion directly, and leptin fluctuations during the menstrual cycle directly correlate with estrogen levels.

Over-consumption of Calories

Americans live in an environment that promotes obesity. Food is in abundance and portion sizes have increased. US studies showed that of children between 7 and 14 years of age, only 46% met the recommended daily intake for grain, 20% for vegetables,
5% for fruit, 9% for dairy, and 26% for meat. Moreover, a large proportion of total caloric intake came from fat and added sugar, accounting for more than 46% of the total calories (Luke, Philpot, Brett, Cruz, Lun, Prasad & Zetaruk, 2004, p.265). Another trend in nutrition that have attributed to the overconsumption in calories are the increased consumption of soda and juice. Only 2% of school-aged children currently meet the number of servings suggested in the Food Guide Pyramid (Luke et al., 2004). These poor dietary habits follow these children into adolescence and adulthood, leading to overweight and obese adults.

**Decreased Physical Activity**

While caloric consumption has steadily increased, daily physical activity has significantly declined for several reasons. First, there has been an increased reliance on motor vehicles for transportation. In addition, more workers now have sedentary jobs because of the continual decline in manufacturing and other physically demanding types of labor. The proliferation of modern technology, such as video games and computers, the increase in number of hours people watch television, and our propensity for convenience all contribute to our sedentary lifestyles (Spence-Jones, 2003).

According to the US Surgeon General, approximately 25% of American adults are completely sedentary (Lyznicki, 2001), and approximately 70% of US adults are not regularly physically active and fail to meet the minimal modest amount of exercise associated with disease prevention (at least 30 minutes of continuous or accumulated moderate intensity physical activity five days per week). Contributing further to children
and adolescents’ inactivity is a decline in physical education in schools and recess (McInnis, 2003). For example, in 1997, only 27% of high school students participated in physical education classes. Again, poor exercise habits tend to follow the child into adolescence and adulthood.

Societal Influences

Fashion designers and clothing manufacturers, as well as advertising agencies, promote and ideal, often emaciated, body image (Rogge, Greewald & Golden, 2004). Women are disproportionately stigmatized by the disease, given society’s premium of female physical attractiveness (Klauer & Aronne, 2002).

Also, an emerging economic explanation (for obesity) is that the increase in BMI among people in the US may be attributable to technological advances that have reduced job strenuousness and increased consumption of mass-produced foods. The overall decrease in occupational physical activity, as well as other changes in the ways we use our time, have contributed to the trend toward a higher BMI. Advancing food technology may have accelerated the rate of BMI increase by making it easier for people in the US to consume more high-calorie, prepackaged and snack foods. Although technologic advances have enriched our lives and made things much easier, it has become almost impossible to get a decent amount of exercise.

Family Influences

Risk factors associated with childhood and adolescent overweight and obesity include high birth weight, maternal diabetes, and a family history of obesity. If one
parent is obese, there is a three-fold increase for the child to become obese in adulthood. If both parents are obese, the risk is ten times greater. Before age 3, parental weight is more of a risk factor for developing obesity than the child’s actual weight. Low income, low education, absence of family meals, and sedentary behavior are also linked with the development of overweight and obesity in children (Shepard, 2004).

Co-morbid Conditions Associated with Obesity

Obesity is regarded as the most preventable causes of morbidity and mortality, primarily because of the links to hypertension, coronary artery disease, stroke and diabetes (Rogge et al., 2004). Obesity is a risk factor for major causes of death, including cardiovascular disease, numerous types of cancer, and diabetes. It is also linked with markedly diminished life expectancy (McTigue, Hemphill, Lux, Sutton, Bunton & Lohr, 2003). Studies suggest that people who are more than 20% overweight have prevalences of hyperlipidemia, hypertension, and diabetes that are between 1.5 and 3.5 times higher than those people whose weight is normal (Mulrow, 1998). Other complications associated with obesity include osteoarthritis, joint pain, gall bladder disease, sleep apnea, respiratory impairment, diminished mobility and psychosocial distress. See Appendix E for Medical Complications Associated with Obesity

Premature Mortality

The Centers for Disease Control estimates that obesity and related lifestyle issues caused 300,000 deaths in 2002 (Klauer & Aronne, 2002, p.1080). In a study to predict years lost due to obesity, the results showed that as compared to a group of normal
weight participants, the overweight group had 4 to 5 more deaths per 100 people, and the obese group had 10-11 more deaths than the normal weight group per 100 people. This represents a 115% (women) and 81% (men) increased risk for death in the obese group. The decreases in life expectancy were reflected in increased probabilities of premature death (defined as death before the age of 70 years). The probability of death increased with each higher category of BMI group (i.e. normal weight, overweight, and obese). Among 40-year-old nonsmokers without previously diagnosed cardiovascular disease, overweight was associated with a 3-year decrease in life expectancy and obesity was associated with a 7-year decrease in life expectancy for women and a 6-year life expectancy decrease in men (Peeters, Barendregt, Willenkens, Mackenbach, Mamun & Bonneaux, 2003). More than 400,000 deaths and one in five cancer deaths can be attributed to obesity annually (Peterson, 2005, p. 44).

Heart Disease

The American Heart Association has cited obesity as a major modifiable risk factor for coronary heart disease. Compared with their lean counterparts, obese women have an increased mortality risk that rises in proportion to the degree of obesity (Klauer & Aronne, 2002). The risk of developing coronary heart disease is increased threefold in women with a BMI greater then 29 compared to BMI less than 21.

Obese persons are likely to have hypertriglyceridemia and a low HDL cholesterol values, and these factors may increase the risk of coronary artery disease. The association between obesity and heart disease is not straightforward, and the linkage may
be related to the associated hypertension and diabetes rather than to weight. Observational studies have established a clear association between overweight and hypercholesterolemia and suggest an independent relationship between overweight and coronary artery disease (Klauer & Aronne, 2002).

The mechanisms underlying these associations are complex and are likely to be interrelated. Obesity for instance, is associated with insulin resistance and hyperinsulinemia, important features on type 2 diabetes mellitus. It has been speculated that excess insulin, in turn may play a role in the retention of sodium, expansion of blood volume, production of excess norepinephrine, and smooth muscle proliferation that are the hallmarks of hypertension. The prevalence of diabetes and hypertension is three times higher in overweight adults than in those of normal weight.

Cholelithiasis

Cholelithiasis is six times more common in obese than in lean subjects (Kumar et al., 2005). The mechanism is mainly an increase in total body cholesterol, increased cholesterol turnover, and augmented biliary excretion of cholesterol in the bile, which in turn predisposes to the formation of cholesterol-rich gallstones.

Osteoarthritis and Degenerative Joint Disease

Obesity has been identified as the main preventable risk factor for developing osteoarthritis (Powell, Teichtahl, Wluka & Cicuttini, 2004). It has been speculated that obesity increases subchondral bony stiffness, making bones less adept at coping with impact loads.
People who are overweight have a higher prevalence of osteoarthritis of the knee than those who are not. The risk for osteoarthritis increases by 35% for every 5 kg of excess weight. The relation of osteoarthritis of the knee to obesity is stronger in women than in men for reasons that are unknown. Also, studies have shown that obese women are at a higher risk of osteoarthritis of the hand than women who are thinner. This suggests that the effect of obesity on osteoarthritis is mediated not only by excess loading on the joints, but also by metabolic or inflammatory factors that may accompany obesity. These metabolic factors may have deleterious effects on the joint (Felson, 2004).

Arthritis, which typically appears in older persons, is attributed in large to the cumulative effects of wear and tear on the joints. It is reasonable to assume that the greater the body burden of fat, the greater the trauma to the joints with passage of time.

*Respiratory Impairment*

Physiological studies have documented that obesity decreases chest wall compliance and increases airway resistance and the work of breathing. Respiratory studies in obese individuals have shown a decrease in forced vital capacity and forced expiratory volume at one second compared with normal weight controls (Castro & Avina, 2002). Hypoventilation syndrome is a constellation of respiratory abnormalities in very obese persons. Hypersomnolence, both at night and during the day, is characteristic, and is often associated with apneic pauses during sleep, polycythemia, and eventual right-sided heart failure.
Cancer

According to a recent American Cancer Society report, one in five cancer deaths is linked to obesity (Peterson, 2005). It is noteworthy that obese women, who are at a higher risk of breast and endometrial cancer, undergo screening for breast and cervical cancer less frequently than nonobese women. Obese patients may choose to forego early or preventative healthcare so as to avoid oppressive encounters with clinicians (Rogge, Greewald & Golden, 2004).

Obesity predisposes individuals to a wide variety of diseases, and of special concern for women’s health is the association of obesity with breast and endometrial cancers and disorders of reproduction (Klauer & Aronne, 2002). The link with uterine and breast cancer is believed to be due to the increase in estrogen produced by adipose tissue. The link between obesity and breast cancer in postmenopausal women may be related to the amount of visceral fat present. Obesity has also been associated with certain cancers, including those of the colon, rectum, gallbladder, biliary tract, breast, cervix, endometrium, and ovary.

Psychosocial

Obesity is associated with body image dissatisfaction across gender and ethnic groups. Women are more dissatisfied with their body weight and shape than men (Klauer & Aronne, 2002).

“Historically, being overweight has been associated with gluttony and sloth, which represent overindulgent behaviors. This connection between obesity
and 2 of the 7 deadly sins equates obesity with sinfulness and connotates a lack of moral rectitude by the obese person. Obesity represents the outward manifestation of self-indulgence and spiritual imperfection, exemplifying the biblical admonition “the spirit is willing but the flesh is weak (Matthew 26:41)” (Rogge et al., 2004, p. 305).

Obese young people, between the ages of 16 and 24 were less likely to be married and had lower household incomes than did those who were not overweight. These findings were more pronounced among women than among men (Rogge et al., 2004).

Several studies have indicated that obesity is even more prevalent among those seeking primary care services than it is among the general population because of the increased morbidity brought on by obesity (Ahmed, Lemkau & Birt, 2002). Unfortunately, obese patients often feel unwelcome in medical settings, where they encounter negative attitudes, discriminatory behavior, and a challenging physical environment (Ahmed et al., 2002). These negative experiences may explain why obese patients are more likely to delay seeking clinical breast exams, gynecological exams and Pap smears, delays which account for some of the increased health risks associated with obesity. If the obese are to receive adequate preventative services and adequate diagnosis and treatment of co-morbid conditions, we must change the way we care for them.

**Reproductive Disorders**

Significant associations are seen in reproductive endocrinology between excess body fat and irregular menstrual cycles, reduced spontaneous and induced fertility,
increased risk for miscarriage and hormone-sensitive carcinomas. Distinct changes in circulating sex hormones appear to underline these abnormalities (Pasquali, Pelusi, Genghini, Cacciari & Gambineri, 2003). One study cited by Pasquali et al. (2003) found that 43% of women affected by various menstrual disorders, infertility and frequent miscarriages were either overweight or obese. It is also known that the presence of anovulatory cycles, oligoamenorrhea and hirsutism, either separately or in association, were significantly higher in obese than in normal-weight women.

Obesity may interfere with many neuroendocrine and ovarian functions, thereby reducing both ovulatory and fertility rates in otherwise healthy women. Obesity is associated with increased risk of hyperandrogenism and anovulation in women of reproductive age as supported by the strong association between obesity and the polycystic ovarian syndrome (PCOS). Approximately 50% of women with PCOS are overweight (Pasquali et al., 2003). Obesity may be associated with several alterations in the balance of sex hormones. Such alterations involve both androgens and estrogens, and their carrier protein, sex hormone-binding globulin (SHBG). Changes in SHBG concentrations lead to an alteration of estrogen and androgen delivery to target tissues. Obesity also affects the metabolism of the androgens not bound to SHBG, leading to increased levels in the blood.

Obesity is a condition of insulin resistance and compensatory hyperinsulinemia. Target tissues, such as the muscles, liver, and adipose tissue become resistant to insulin over time. But the ovaries remain responsive to insulin through interaction with its own
receptor. Excess insulin has been shown to stimulate steroidogenesis and excessive androgen production from the theca cell system (Pasquali et al., 2003). In addition, by inhibiting SHBG synthesis by the liver, excess insulin further increases the delivery of free androgens to target tissues. The excess in local ovarian androgen production induced by excess circulating insulin may also cause premature follicular atresia, which leads to anovulation. Insulin resistance and hyperinsulinemia, which develop together in the presence of obesity, may play a dominant role in the development of hyperandrogenism in women with PCOS.

The role of adipose tissue is crucial in controlling the balance of sex hormone availability in the target non-fat tissues. Adipose tissue is able to store various lipid soluble steroids, such as androgens. Most sex hormones appear to be preferentially concentrated in the adipose tissue rather than in the blood. As a consequence, since the amounts of fat in obesity are larger than their intravascular space, and the steroid tissue concentration is much higher than in plasma, the steroid pool in obese individuals is greater than that found in normal-weight individuals (Pasquali et al., 2003). Obesity can also be considered a condition of increased estrogen production. Reduced SHBG concentrations may in turn lead to an increased exposure of target tissues to free estrogens.

As discussed earlier, obesity may represent a condition of leptin resistance. Leptin acts directly on the ovary, in particular on the follicular cells, including the granulosa, thecal and interstitial cells. Leptin may exert a direct inhibitory effect on
ovarian function, by inhibiting both granulosa and thecal cell steriodogenesis, probably through the antagonism of stimulatory factors (Pasquali et al., 2003). High leptin concentrations in the ovary may interfere with the development of dominant follicles and oocyte maturation.

Ghrelin levels are negatively correlated with androgen levels, indicating that the gonads may be important targets of ghrelin action. Since ghrelin concentrations are negatively correlated with insulin resistance, it can be speculated that this peptide in some way represents a link between hyperandrogenism and the insulin system in conditions such as obesity and PCOS. It therefore appears that ghrelin, like leptin, may represent a further endocrine factor that is related not only to energy balance and metabolism, but also to gonadal function.

*Adverse Pregnancy Outcomes*

Excess weight gain in pregnancy can be difficult to shed postpartum, and is an additional contributing factor in parous women. Many women tend to become overweight or obese during pregnancy, and many are already obese when they become pregnant. In general terms, obesity increases morbidity for both the mother and the fetus. Overweight pregnant women have higher incidences of hypertension, pre-eclampsia, toxemia, gestational diabetes, urinary infections, cesarean delivery and subsequent wound infections, endometritis, and increased hospitalization. Overweight pregnant women also have an increased risk of venous thromboembolic disease and respiratory complications.
Obese women have a 70% increase in postpartum hemorrhage (Castro & Avina, 2002). Surgical procedures compound these risks.

Fetal effects include increased incidence of neural tube defects, preterm delivery, and fetal mortality. Other complications include large for gestational age neonates, macrosomia, failure to progress, and shoulder dystocia. Infants of obese mothers are at an increased risk themselves for childhood obesity and its associated morbidity.

*Childhood Obesity*

Immediate consequences of childhood obesity include a prevalence of atherosclerotic plaques, hypertension, and an adverse lipid profile, with poor self-image that limits participation in physical activity (Shepard, 2004). Tracking is such that many obese children become obese adults, and in consequence, the long-term risk of cardiovascular and all-cause deaths are increased. Although many children who are overweight seem clinically healthy, a growing number of those who are overweight are showing evidence of Type 2 diabetes mellitus, hyperlipidemia, and hypertension, along with a growing prevalence of atherosclerotic lesions in the aorta and coronary blood vessels. BMI readings are consistently associated with the extent of such lesions between the ages of 2 and 39 years of age (Shepard, 2004, p. 163). Other cardiac complications include left ventricular hypertrophy and metabolic syndrome. Even if an adolescent's BMI is greater than or equal to the 75th percentile, well below the current definition of overweight, there is a risk of dying from cardiovascular disease as an adult (Holcomb, 2004).
Other problems that overweight pediatric patience face are orthopedic problems, such as slipped capital femoral epiphysis, and tibia vera. Other associated health disorders include asthma and sleep apnea, gallstones, and hormonal disturbances. Poor self-esteem may result from teasing at school, leading to isolation and more eating if the child is bored and/or depressed.

Successful Interventions in the Treatment of Obesity

The prevention of obesity is easier than its cure. A combination of increased lifestyle activities, less sedentary behavior, and dietary modification seems the most effective approach. Weight loss should be supported by the use of behavioral modification techniques and changes in the urban environment that encourage an active lifestyle (Shepard, 2004). Weight loss can be achieved through dietary intervention and behavior modification, while weight maintenance is facilitated through increased physical activity (Klauer & Aronne, 2002).

According to the U.S. Preventative Services Task Force, counseling is more effective in helping people lose weight if combined with behavioral interventions that assist them in developing skills, motivation, and support systems. Primary care clinicians have an important role in diagnosing and in either providing intensive counseling and behavioral interventions or referring patients to receive these services. “Physician counseling is effective in changing certain health risk behaviors, and there is evidence that patients are more likely to lose weight when counseled by a physician” (Nawaz, Adams & Katz, 1999, p.764).
The health benefits of modest weight loss include: decreased blood glucose and insulin levels; decreased blood pressure; decreased LDL and triglyceride levels; increased HDL levels; decreased severity of sleep apnea; reduction in symptoms associated with degenerative joint disease; and improvement in gynecologic conditions (Klauer & Aronne, 2002). Intentional weight loss of 0.5-9.0 kg in overweight women with disorders related to obesity was associated with a 20% reduction in all-cause mortality (Mulrow, 1998).

Basic treatment of overweight and obese patients requires a comprehensive approach involving diet and nutrition, regular physical activity, and behavioral change, with an emphasis on long-term weight management rather than a short-term extreme weight reduction. The clinician should obtain a history, perform a physical examination, and conduct a laboratory work-up with attention to the complications of obesity.

Medications should be reviewed and consideration given to changing those that cause weight gain. These include antidepressants, antiepileptics, antipsychotics, lithium, glucocorticoids, progestational hormones, antihistamines, sulfonylureas, insulin, thiazolidinediones, and beta-blockers, among others (Klauer & Aronne, 2002). The patient should be thoroughly examined for evidence of an endocrine disorder that may be the cause for obesity.

Endocrine problems that are associated with the development of overweight and obesity include hypothyroidism, Cushing’s disease, and primary hyperinsulinemia (Shepard, 2004). Eating disorders, particularly bulimia and binge-eating disorder should
be considered. Other differential diagnoses must include: hyperinsulinemia, polycystic ovarian syndrome, hypothalamic state, and growth hormone deficiency.

Women who should not be treated for overweight or obesity include women who are pregnant or nursing or plan to become pregnant, women who have a history of anorexia nervosa, and women with terminal or unstable medical or psychiatric illnesses. Women who have osteoporosis or cholelithiasis must be warned that these conditions may be aggravated by weight loss.

Advising a patient on weight reduction involves balancing and weighing the risks of obesity against the risks and benefits of weight loss, and setting that in context of treatment possibilities with those risks and benefits (Mulrow, 1998).

**Diet**

Fundamental to treatment of obesity is reduction in the number of calories consumed. An overall decrease in the number of calories is necessary for weight loss to occur, with emphasis on consumption of raw fruits and vegetables, protein, fiber, and should be sufficient in nutrients and vitamins. Decreasing intake of processed foods, sugars, salts, fats, oils, and nutritionally-dense foods should be encouraged. The specific diet is best developed in consultation with a registered dietician.

Low calorie diets with 1,000-1,500 kcal per day have been shown to provide approximately an 8% weight reduction in obese individuals over 3-12 months when compliance is good (Klauer & Aronne, 2002). Very low calorie diets, including liquid formula diets, provide 400-800 kcal per day, and should be reserved for obese individuals
when immediate weight reduction is necessary for medical reasons, such as prior to surgery or if respiratory function or quality of life is impaired.

Low fat diets (20-30% of calories) have not been shown to significantly reduce weight without a reduction of calories. However, low-fat diets coupled with caloric restriction are a useful and healthy way to lose weight (Klauer & Aronne, 2002).

The American Dietetic Association and American Health Association recommend a diet of 30% fat (with no more than 10% saturated fat), 15-20% protein, and 55-60% carbohydrate based on the USDA Food Pyramid and the National Cholesterol Education Program Step 1 and Step 2 diets. If calories are reduced to 1,200 kcal per day, a wide variety of nutrients can be incorporated while still allowing for weight loss. With a balanced, reduced-calorie diet, weight loss should be at a rate of about one to two pounds per week (Klauer & Aronne, 2002).

Calculating daily caloric requirements can assist the health care provider in counseling patients about caloric needs to maintain weight. One way to determine how many calories a women should consume each day is calculating the patients basal metabolic rate (BMR), or the energy required for involuntary physiologic functions to maintain life, including respiration, circulation, and maintenance of muscle tone and body temperature. The BMR accounts for 65-70% of the body’s energy requirement. It is calculated by $10 \times \text{ideal weight (lbs)} = \text{kcal needed for BMR daily}$.

A more efficient way to calculate caloric needs is by calculating the resting energy expenditure. Resting energy expenditure is the estimated kilocaloric requirements
for the basal metabolic rate plus additional kilocalories needed for thermogenesis, voluntary activities, and any increased need from catabolic or anabolic processes. The first step is to estimate the recommended individual caloric requirement (kcal per day) by calculating the resting energy expenditure (REE). For adult women the formula for calculating REE is: \( \text{REE} = 10 \times \text{weight (kg)} + 6.25 \times \text{height (cm)} - 5 \times \text{age (years)} - 161 \). Then, multiply the REE by an activity factor (AF) of 1.5 for women who engage in light activity or 1.6 for women who engage in higher activity to estimate caloric need.

A useful application of this formula is \( \text{REE} \times \text{AF} = \text{estimated caloric need (kcal)} \) to maintain current weight (Lyznicki et al., 2001, p.2187). It can be inferred then, that by either increasing the energy expenditure, decreasing the caloric need values, or both, weight loss should occur.

**Exercise**

Daily physical exercise is basic to health. The benefits of regular exercise are well-documented and include positive effects on mind, bone, lipid profile, endothelial function, risk for cancer, glucose tolerance and insulin sensitivity, and quality of life. The Nurse’s Health Study documented a lower incidence of cardiovascular disease, including both coronary heart disease and stroke (Klauer & Aronne, 2002, p.1084). Even a moderate-intensity exercise such as walking is associated with a lower risk of disease. The recommendation for aerobic activity is thirty minutes on most, if not all, days of the week. This is the minimum recommendation; those wishing to lose weight should aim to exceed this (Klauer & Aronne).
The patient should be advised to gradually increase her energy expenditure through changes to her daily routine (e.g., climbing stairs rather than riding the elevator, parking farther from a destination if safe) and the incorporation of regular exercise likely to be continued over the long-term.

*Behavior Modification*

Behavior therapy is a useful adjunct to diet and physical activity. The clinician should assess patient motivation and readiness to implement the weight management plan and take steps to motivate the patient for treatment. Behavior strategies to promote diet and exercise should be used routinely, as they are helpful in achieving weight loss and maintenance.

All weight reduction programs should incorporate some form of behavioral modification. The best programs aim to help the patient identify the cause of the weight gain and thereby gain better control over situations that cause overeating. Goals for treatment include identification of situations that trigger eating, improving exercise habits, food shopping with awareness, and recognition of hunger versus craving. Behavior modification alone provides weight losses of ten percent (Klauer & Aronne, 2002, p.1087). Key techniques include self-monitoring (such as keeping a food diary), stimulus control (mindful eating), reinforcement, and relapse prevention (social support).

Weight loss and maintenance therapy should involve a combination of low-calorie diets, increased physical activity and behavior therapy. The combination of a reduced
calorie diet and increased physical activity has been shown to produce weight loss, decrease abdominal fat, and increase cardio respiratory fitness (Lyznicki et al., 2001).

**Pharmacologic Therapy**

After attempting weight loss with the first line treatment of behavior modification, dietary restriction, and increased physical activity, medication can be considered for some patients. Pharmacotherapy can increase compliance and increase the amount of weight loss above that achieved with diet and exercise. Pharmacotherapy may be used as part of a comprehensive weight loss program for patients with a BMI equal to or greater than 30 with no obesity-related risk factors or diseases, and for patients with a BMI of 27 or greater with accompanying risk factors or diseases (Lyznicki et al. 2001).

Orlistat is a pancreatic lipase inhibitor that works in the gut by preventing the absorption of 30% of dietary fat. It has also been shown to lower total and LDL cholesterol more than would be expected by weight loss alone. Orlistat-treated patients with a 10% weight loss lowered their LDL by 14%, with only a 4% reduction in the placebo group. In a two-year trial, an average of 7.8% weight loss was observed in orlistat users, as compared with only a 4.6% reduction in the placebo group. Thirty-three percent of the orlistat group lost more than 10% of their body weight, whereas only 14.6% in the placebo group did so (Klauer & Aronne, 2002).

Side effects of orlistat include flatus, oily spotting, abdominal pain, fecal incontinence, and a slight reduction in the level of fat-soluble vitamins. These side effects relate to the amount of fat that is ingested, and with a higher-fat diet, there are
more symptoms. These symptoms may serve as a deterrent to the consumption of excess dietary fat. Also, supplementation with a nightly dose of psyllium minimizes these effects.

Sibutramine is another weight-loss medication that acts centrally in the brain to suppress the reuptake of norepinephrine and serotonin, which increase satiety during meals. Thus patients feel fuller sooner, and hence eat less. Forty-three percent of patients on a program of diet, exercise, and sibutramine 10 mg were able to maintain more than 80% of their weight loss for two years, versus only sixteen percent with diet and exercise alone (Klauer & Aronne, 2002, p.1087).

Side effects associated with sibutramine are mild, and usually resolve in a few weeks. They include dry mouth, insomnia, headache, constipation, and restlessness. Some patients experience a rise in blood pressure and heart rate, and it is therefore necessary to monitor blood pressure in patients who take this drug.

Other medications associated with weight loss include metformin in patients with non-insulin-dependent diabetes. Metformin promotes weight loss by enhancing satiety. In combination with orlistat, metformin has been shown to offer significant weight reduction and improvement in lipid profiles in patients with type 2 diabetes.

Topiramate, approved as adjunctive therapy in epilepsy, has been found to reduce body weight. It may be effective in reducing psychiatric symptoms of affective disorder, bipolar disorder, and binge eating disorder (Klauer & Aronne, 2002).
Bupropion is an antidepressant that does not cause weight gain, as other antidepressants do, such as monoamine oxidase inhibitors and selective serotonin re-uptake inhibitors. Patients treated with bupropion demonstrated a weight loss of 4.9% after eight weeks of use, and of 12% after twenty-four weeks of treatment (Klauer & Aronne, 2002).

*Surgery*

Surgical interventions can be considered for patients with a BMI greater than 40 who fail other methods of treatment, particularly if serious obesity-related complications are present. Surgery may also be considered for patients with a BMI between 35-40 who have severe life-threatening complications such as sleep apnea or cardiomyopathy. With surgical intervention, sixty to eighty percent of excess weight is lost. After surgery, patients may need lifelong supplementation with vitamins and minerals, may experience vomiting or diarrhea after meals, and 20% of patients ultimately regain all lost weight (Klauer & Aronne, 2002, p.1088).

Vertical banded gastroplasty, or gastric stapling, constructs a 30-mL pouch with a restricted outlet constructed along the lesser curvature of the stomach. A Silastic ring or band of Marlex mesh restricts the outlet size, and four rows of staples reinforce the free wall to prevent breakdown. It is effective, as 70% of patients maintain a loss of 20% of total body weight at five-year follow-up (Klauer & Aronne, 2002).

Gastric bypass is the gold standard in anti-obesity surgery. It involves constructing a proximal gastric pouch whose outlet is a limb of small bowel of varying
lengths, as in a Roux-en-Y gastrojejunostomy. The procedure produces malabsorption of food and the dumping syndrome. Because of malabsorption of vitamins and minerals, these patients need careful nutritional instruction and follow-up, as well as behavioral training. More weight is lost with this procedure than with gastroplasty, but the risk of complications is greater. These include a 1% mortality risk, and perioperative complications include wound infection, leaks from staple line breakdown, stomal stenosis, marginal ulcers, deep vein thrombophlebitis, and other pulmonary problems, which occur in about 10% of cases (Klauer & Aronne, 2002).

Longer-term complications, which occur with both procedures, include cholecystitis and failure to lose weight. Complications unique to bypass include nutrient deficiencies, particularly vitamin B-12, folate, iron, calcium, zinc, and dumping syndrome. Of particular note is the risk of fetal damage related to maternal undernutrition and nutrient deficiencies. Therefore, women of child bearing age need to be on birth control during the weight loss phase, and once they are weight stable, they may become pregnant, but they must be carefully monitored during pregnancy to prevent such deficiencies.
CHAPTER 3
Critique of the Literature

Weight cycling, or losing and regaining weight, may have negative implications for health, although there is conflicting evidence in the literature concerning health risks associated with weight fluctuations over time. Both men and women experience weight cycling that occurs from environmental, socioeconomic, and emotional and physical causes. However, in addition to the weight cycling that seems to commonly occur throughout life, we know that women experience weight fluctuations at different stages in their reproductive lifespan, as well. More information is needed about the long-term health implications of weight cycling.

The literature reviewed for the purposes of this project offered consistent data that emphasized the magnitude of obesity and the health concerns for women who are overweight or obese. Ongoing efforts to identify the genetic factors that contribute to overweight and obesity show promise in the identification and treatment of this disease. Providers need to develop a plan on how and when to counsel their patients, how to best motivate patients to maintain healthy body weight; and especially on the prevention of recidivism rates, in women throughout the lifespan.

Gaps in the Literature

There are insufficient data on the effectiveness of physician weight loss counseling and skepticism about the success of any medical treatment of obesity. There are also insufficient data on the long-term safety and efficacy of pharmacotherapeutic
agents for obesity. There are inadequate data to determine the optimal frequency of obesity screening, and this is best left to clinical discretion (USPS).

Evidence of poor assessment and counseling for overweight and obesity has been found in studies evaluating physicians. There was no data found on the frequency and efficacy of assessment and counseling for overweight and obesity by nurse practitioners in adults. This is an important area that should be researched further, especially now that more non-physician providers are providing primary care services in outpatient settings.

Areas for Future Research

The science behind body fat regulation is vastly growing. There is still further research needed to uncover the exact role of genetics and hormones, specifically female sex hormones that influence body composition. Studies have shown that one cannot easily fool the homeostatic mechanisms that maintain body fat. Gene deletions and knockouts of known mediators of body fat have shown little to no promise of advancing the scientology of body fatness. There is no single cause of all human obesity, so we must explore prevention and treatment approaches that encompass many aspect, such as behavioral, socio-cultural, socioeconomic, environmental, physiologic, and genetic factors.

A clear link needs to be made between female sex hormones and obesity. We know that there are cyclical changes in food consumption and energy expenditure that accompany the menstrual cycle, pregnancy, and lactation. We also know that estrogen influences the effects of gut hormones, such that women tend to eat less when estrogen is
present. This information contradicts the data that more and more women are surpassing overweight and becoming obese, more so than men.

Further research is needed on the specific genes outlined in this paper in order to establish a more clear role in the development of obesity, and to direct research towards finding therapies that are available to the public that prove to be safe, effective and economical. Long-term treatment of obesity is extremely difficult, despite the many therapeutic interventions available. Research should be geared towards finding a long-term treatment for obesity as well prevention of obesity in adults, but even more importantly, in children.

More research is needed to directly link obesity, and only obesity, as a single cause in the health disparities listed in this paper. It is difficult, and almost impossible, to separate obesity from many of the diseases discussed. In order for the public, health care providers, and insurance companies to realize the health risk and dangers associated with overweight and obesity, especially as they impact women and pregnancy, further research is needed to identify obesity as an individual causative factor in disease development and progression.

Recommendations

Primary prevention, which has been called “the most promising therapeutic modality available to combat obesity” (Buttar et al., 2003), would suggest that providers check in routinely with their patients about the quality of their diet, whether they have a consistent exercise practice that they enjoy, and their means of balancing and coping with
the increasing demands of work and life. In addition, providers can help with prevention by prescribing alternatives to weight gain-promoting medications whenever possible and exploring instances of significant adult weight gain (>10 pounds).

The author recommends that all patients, women in particular, should have weight, height, BMI, and waist circumference assessed at every patient encounter, regardless of presenting problem. This recommendation extends to all health care providers in all settings. Since seemingly healthy adult patients may under-utilize health prevention services, every opportunity to screen and educate women about the health implications of their weight status should be fully optimized in order to reduce the rates of obesity-related complications and deaths in this population.

Findings should be individualized and the patient counseled about health consequences of further weight gain, and appropriate measures taken to ensure the patient receives adequate counseling on diet, exercise, and behavior modification techniques to improve quality of life through lifestyle modifications. Collaboration among healthcare providers is needed, and this recommendation is extended to specialty providers as well as adjunct healthcare personnel, such as medical assistants, nursing staff, as well as primary care providers and specialists. Since women are disproportionately affected by obesity, every time she presents to a healthcare provider, this information should be gathered and her risks assessed, and she should receive appropriate counseling.
Summary

This report offers current data on obesity. Included are definitions of obesity, clinical tools to assess patients for overweight and obesity. There is a review of the current literature that links obesity to many of the common illnesses and chief complaints faced by practitioners at all levels. The current therapies for treating obesity are listed, and recommendations from the author for when to, and how to counsel patients is included in this paper.

Sensitive care of obese patients involves attending to their needs for comfort, safety, and self-esteem in the primary care setting. Encouraging compassionate care of obese patients will positively impact the health of this population even if a pound is never lost. This may occur through increased compliance with preventative screening, better attention to co-morbid conditions and more regular medical care. The person, not the obesity, should be the focus of treatment. All of these characteristics are part of many nurses’ world-view and theories of delivering compassionate, effective preventative services and healthcare.
APPENDIX A

*Ideal Body Weight for Women Using Weight-for-Height Chart*

<table>
<thead>
<tr>
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<th>Inches</th>
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<th>Medium frame</th>
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<td>109-121</td>
<td>118-131</td>
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<td>113-126</td>
<td>122-137</td>
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<td>1</td>
<td>106-118</td>
<td>115-129</td>
<td>125-140</td>
</tr>
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<td>2</td>
<td>108-121</td>
<td>118-132</td>
<td>128-143</td>
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<td>111-124</td>
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<td>4</td>
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<td>117-130</td>
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<td>0</td>
<td>138-151</td>
<td>148-162</td>
<td>158-179</td>
</tr>
</tbody>
</table>

(Davis & Sherer, 1994, p.1068)
APPENDIX B

Determining Body Frame for Weight-for Height Chart

The wrist is measured distal to styloid process of the radius and ulna at smallest circumference. Use height without shoes and inches for wrist size to determine frame type from this chart. (Davis & Sherer, 1994, p.1071).
APPENDIX C

Body Mass Index Chart

To determine the client’s BMI: 1) Locate the client’s height (without shoes) on the left side of the chart. 2) Using a pencil and an index card, draw a line across the graph. 3) Locate the client’s weight (without clothes) on the grid on the bottom of the chart. Draw a line up from the mark to cross the horizontal line already drawn. 4) The
lines will cross at, or very near, the curved line, which shows the client’s BMI. (Davis & Sherer, 1994, p. 1069)
Nomogram for determining the ratio of abdominal (waist) circumference to gluteal (hips) circumference. Place a straight edge between the column for waist circumference and the column for hip circumference and read the ratio from the point where this straight edge crosses the AGR or WHR line. The waist or abdominal circumference is the smallest circumference below the rib cage and above the umbilicus, and the hips, or gluteal circumference is taken at the largest circumference at the posterior extension of the buttocks. (Davis & Sherer, 1994, p. 1070)
## APPENDIX E

*Medical Complications Associated with Obesity*

<table>
<thead>
<tr>
<th>Category</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>Gallstones, pancreatitis, abdominal hernia, NAFLD (steatosis, steatohepatitis, and cirrhosis), GERD</td>
</tr>
<tr>
<td>Endocrine/Metabolic</td>
<td>Metabolic syndrome, insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus, dyslipidemia, polycystic ovarian syndrome</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Hypertension, stroke, coronary artery disease, congestive heart failure, arrhythmias, pulmonary hypertension, venous stasis, deep vein thrombosis, pulmonary embolus</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Abnormal pulmonary function, obstructive sleep apnea, obesity hypoventilation syndrome</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Osteoarthritis, gout, low back pain</td>
</tr>
<tr>
<td>Gynecologic</td>
<td>Abnormal menses, infertility</td>
</tr>
<tr>
<td>Genitourinary</td>
<td>Urinary stress incontinence</td>
</tr>
<tr>
<td>Ophthalmologic</td>
<td>Cataracts</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Idiopathic cranial hypertension (pseudomotor cerebri)</td>
</tr>
<tr>
<td>Cancer</td>
<td>Esophagus, colon, gallbladder, prostate, breast, uterus, cervix, kidney</td>
</tr>
<tr>
<td>Postoperative events</td>
<td>Atelectasis, pneumonia, deep vein thrombosis, pulmonary embolus</td>
</tr>
</tbody>
</table>

(Kumar et al., 2005, p 465)
REFERENCES


