The Obesity Epidemic, Part 1: Understanding the Origins

A review of the underlying physical, psychological, and social factors.

OVERVIEW: The obesity epidemic remains a significant health problem in the United States and worldwide, with multiple associated physical and societal costs. To contribute to obesity's treatment and prevention, nurses must be conversant in a wide range of theoretical and clinical perspectives on the problem. This article, the first in a two-part series, defines the terms used in the treatment of obesity and outlines pathophysiologic, psychological, and social factors that influence weight control. Part 2, which will appear in next month's issue, presents a theoretical framework that can be used to guide nursing assessment of both patient and family, thereby informing intervention.

Keywords: body mass index, environment, genetics, obesity, overweight, weight control

Recent data from the National Center for Health Statistics estimate that in the United States more than 35% of adults and 16% of children ages two to 18 are obese.[1,2] Obesity disproportionately affects racial and ethnic minorities as well as people at lower income and educational levels, though it is prevalent among men and women in every segment of society.[3,4] Obese children and adults are at risk for type 2 diabetes, cardiovascular disease, musculoskeletal dysfunction, and certain types of cancer.[5-7] The Centers for Medicare and Medicaid Services estimates the annual national health care expenditure on obesity to be about $147 billion, with per capita spending on obese people averaging $1,429 more than spending on individuals of normal weight.[8]

This two-part series provides an overview of the factors contributing to the rise in obesity rates and the implications for nurses. Here in part 1, we'll discuss the relevant definitions; the physiology, pathophysiology, and psychology underlying weight changes; and the social context in which obesity has risen to epidemic proportions. Next month's installment presents a theoretical framework to guide nursing assessment and intervention.

DEFINITIONS

Body mass index (BMI) is universally accepted as the standard for defining overweight and obesity. BMI is determined by dividing weight in kilograms by height in meters squared (kg/m2); an online BMI calculator is available on the National Heart, Lung, and Blood Institute Web site (http://1.usa.gov/1ooHYzU). BMI is used to assess adiposity in adults, regardless of sex or age. BMIs of 25 and above are associated with increasing risk of developing obesity-related health problems (see Table 1[6]).[6,9]

While an absolute BMI may capture underweight and overweight in adults, age- and sex-specific percentiles provide a more accurate picture of a healthy BMI for children.[10] During childhood, there are several periods in which sex, growth, and maturation

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patterns trigger muscular gains rather than adiposity, accounting for much BMI variation. The Centers for Disease Control and Prevention (CDC) has thus developed BMI charts and calculators for determining a child's BMI percentile, based on age and sex (http://nccd.cdc.gov/dnpabmi). A BMI that's at or above the CDC's 95th percentile for age and sex represents childhood obesity (see Table 2[9]).[10,11]

PHYSIOLOGY OF WEIGHT CONTROL

Obesity occurs over time when more kilocalories are consumed than are expended, creating a positive energy balance. Research indicates that high food intake is the greatest factor fueling the obesity epidemic.[12] Short-term energy balance is based on the hour-to-hour need for homeostasis, which depends on energy stored in the liver (glycogen), adipose tissue (fat), and blood (glucose) and on the interaction between these storage units and the central nervous system (CNS), mediated through the following hormones[13,14]:

- cholecystokinin and glucagon-like peptide-1 from the small intestine
- the adipose hormone leptin, which can induce satiety
- the hormone ghrelin, which is secreted primarily by the stomach and acts as a potent appetite stimulator
- insulin and several other hormones from the pancreas

All work together to stimulate appetite and satiation through a complex system of neural pathways and regulatory modulators in the brain's hypothalamus. In humans, appetite is also influenced by habit, social factors, and stress.[15,16]

Leptin and insulin are secreted in proportion to body fat.[14] Increases in the body's store of adipose tissue generally cause levels to rise. As levels rise, these hormones signal neurons in the hypothalamus that the body has excess fat, and the brain responds by directing the body to reduce food intake; as levels fall, the brain instructs the body to increase consumption.[14] Accordingly, humans with leptin deficiency have an insatiable appetite and tend to be obese.[17] Even if leptin levels are normal, however, over the long term, signals to the CNS may "drop out." As with insulin resistance, leptin resistance is associated with obesity.[13] To date, research attempting to control weight by altering leptin levels has had limited success.

PSYCHOLOGY OF OBESITY

French and colleagues reviewed eating behaviors associated with increased calorie consumption and body weight.[18] These included:

Table 1. BMI in Adults[6]

BMI	Classification
< 18.5	Underweight
18.5–24.9	Normal weight
25–29.9	Overweight
30–34.9	Class I obesity (low risk)
35–39.9	Class II obesity (moderate risk)
≥ 40	Class III obesity (high risk)

- heightened food responsiveness and enjoyment of eating
- eating in the absence of hunger
- a powerful reinforcement value associated with food
- eating disinhibition
- impulsive eating

All have been studied as potential "onset factors that influence craving, appetite, motivation to eat, hedonic responses to food, or food reward" and can lead to overeating. Genetics may well be involved in these processes, though no specific link has been identified.[18]

Cohen and Babey describe the automatic or unconscious food choices people make as "heuristic," meaning that they rely on familiar experiences and simple procedures to arrive at "adequate, though often imperfect" solutions.[15] Heuristic eating, therefore, is learned food behavior that is unregulated by deliberative thought and decision making. While "relying on heuristic cues" to determine food intake may be less taxing than making conscious choices, such behavior tends to result in food selections that are less than optimal. For example, parents who work late and leave work feeling enervated may be more likely to pick up burgers and fries for themselves and their children than to cook a healthful meal. Habitual, nonreflective eating choices are heuristic and difficult to change. Given the ubiquity and relative convenience and affordability of most fast food, such behavior tends to support large portion sizes and unhealthy food choices. Research into heuristic eating behavior suggesting a strong unit bias ("a sense that a single entity . . . is the appropriate amount") in human food choices has led to the gradual expansion of portion sizes in many fast-food restaurants and elsewhere.[19]

Hedonism, or pleasure-seeking behavior, is another psychological construct used to explain eating behavior. Stroebe and colleagues maintain that hedonistic eating is an extremely strong urge that often unconsciously undermines dieting, weakens restraint, and leads to disinhibited eating.[20] In hedonistic eating, the drive to eat overrides the homeostatic sense of satiety. This behavior can be driven by extrinsic stimuli, such as palatability and smell, or by intrinsic factors, such as brain rewards similar to those that occur in the context of drug addiction.[21]

Stressful eating—when college students studying for exams order takeout at 3 AM or when depressed people eat for comfort, for example—is also known to contribute to obesity.[16] Dallman found that about 80% of people who experience stress change their eating habits: 40% reduce their intake and 40% increase their intake.[16] Those who are overweight or obese tend to increase their intake when stressed, and to prefer high-fat or sweet foods.[22] As measured by the glucocorticoid cortisol, high reactivity, both chronic and stress induced, has been related to higher food intake.[23]

PATHOLOGY OF OBESITY

Since the 1970s, obesity prevalence has increased at an alarming rate.[24] The propensity to become obese, however, is inconsistent across populations, suggesting that biological, behavioral, sociocultural, and environmental pressures account for individual differences.

Genetics of obesity development. The rise in obesity among adults and children worldwide over the past 30 years cannot be attributed to human genetic changes or mutations.[10,25] Nevertheless, it is clear that genetics predispose some people to obesity when food is abundant.[26] The precise relationship between genetics, eating, and weight is not yet known. A review of studies involving dizygotic and monozygotic twins indicates that genetic factors account for 32% to 74% of a person's likelihood of becoming obese; in studies of parent-offspring pairs and siblings, genetic factors account for 19% to 24%.[27]

Speakman and colleagues speculate that more than 1,000 gene combinations may be involved in the regulation of energy expenditure during physical activity, food choices, consumption, absorption, and metabolism.[28] Nevertheless, McCaffrey and colleagues found that a variation in a single gene (the *FTO* gene) was significantly associated with more daily meals, snacks, and servings of fats and sweets.[29] Upon reviewing studies of magnetic resonance imaging and positron emission tomography scans of brain activity in response to food cues, Carnell suggested that food-related brain behavior could be influenced by familial and genetic factors.[30]

Biology of obesity development. After decades of investigation, scientists can only hypothesize about the causative mechanisms for obesity. According to one hypothesis, a "thrifty" gene (or genes), which prevents starvation during times of food scarcity, increases the deposition of fat in times of abundance.[31] In some people, this thrifty phenotype seems to suppress thermogenesis during periods of catch-up growth.[32] Other investigators discount the thrifty gene hypothesis because it is inconsistent with the findings of the

genome-wide association studies for BMI, which suggest that the increased prevalence in obesity more likely results from many genes with a very small effect or from a few alleles with a large effect.[26]

A second obesity theory postulates that body weight is genetically determined to a certain set point.[28] If adipose tissue increases above the body's genetically determined set point, the brain triggers an increase in energy expenditure and a decrease in food intake, hence returning the body to its predetermined size.[28] While weight gain can raise the steady-state set point, with weight loss it readjusts at a lower level, after which a lower food intake and greater energy expenditure are required to maintain the lower weight. According to scientists, this explains why most people regain their lost weight.[33]

Another theory is the genetic "drift" theory, which maintains that body weight is controlled by genetically programmed upper and lower boundaries. Over millions of years, evolution has pushed the higher limit of the weight boundary upward. This "drift" was caused by a slow genetic adaptation fueled by the advent of fire and weapons that negated the need for humans to actively hunt and run from predators.[28] Critics of this theory assert that a genetic drift could not be responsible for the worldwide rise in obesity rates over the past two generations because human genes could not change substantially within that period.[34]

SOCIOCULTURAL AND ENVIRONMENTAL ISSUES

Most experts place the blame for the high global prevalence of obesity on overeating and reduced physical activity caused by cultural and environmental factors.[25] Such sociocultural changes as an increased reliance on automobiles and other modern conveniences have reduced the need to be physically active. Computers and television have promoted a sedentary lifestyle for adults and children, whose opportunities for physical activity have been further diminished because of academic pressures, overscheduling, and safety concerns for out-of-doors play. Suburban living has produced long commutes to work, and technologic advances have resulted in more people working in occupations that require no physical activity. Overweight and obesity thrive in such a social context.

The 'built environment.' Environmental factors such as buildings, parks, and walkways are often termed the "built environment." Traffic patterns, road design, zoning, city planning, business practices, and community finances either enhance or inhibit physical activity.[25, 35, 36] Rural areas may have fewer suitable sites for physical activity. Urban environments often have a dearth of safe recreational facilities, affordable grocery stores, or reasonably priced and available fresh produce.[35]

Food supply. Over the past 50 years, in the United States and worldwide, the food supply has become highly processed, affordable, and effectively marketed. Many obesity experts blame processed and fast foods, marketed through aggressive advertising and sold at low cost, for the rise in obesity.[24] The term "obesogenic" is used to describe today's sociocultural environment, which promotes overeating.

While obesity rates in the United States have remained high overall, there are signs that the increase is starting to slow.

New evidence suggests that obesogenic foods that contain high fructose corn syrup or combine the tastes of fat and sugar have potentially addictive properties.[37-40] Some commentators have described such findings as preparing the way for similar public health campaigns and litigation as those occasioned by the U.S. Department of Health and Human Services' release of its 1988 report, *The Health Consequences of Smoking*.[41,42] After the concept of nicotine addiction was well established and widely publicized, tobacco companies were found liable for damages because of the high cost of health care (largely provided through Medicaid) for smokers. Since then, policies prohibiting smoking in public areas have been enacted and legislation prohibiting the sale of tobacco to those under age 18 has been strictly enforced. The history of successful smoking reduction measures may offer insight into the most effective ways of addressing obesity prevention. Past experience suggests that similar strategies, including litigation by states attorneys general, may be equally effective in reducing the consumption of unhealthy foods.[43]

Table 2. BMI-for-Age Percentile[9]

Percentile	Classification
< 5th	Underweight
5th to < 85th	Healthy weight
85th to < 95th	Overweight
≥ 95th	Obese

RESOURCES

Centers for Disease Control and Prevention
"Overweight and Obesity"
Contains links to BMI calculators, obesity data and statistics, and strategies to combat obesity, among others.
www.cdc.gov/obesity

"National Diabetes Prevention Program"
Contains information about the diabetes services provided by a partnership of public and private organizations.
www.cdc.gov/diabetes/prevention

National Heart, Lung, and Blood Institute
U.S. Department of Health and Human Services
"What Are Overweight and Obesity?"
http://1.usa.gov/1hddmDT

"Clinical Trials"
Contains information on all federally funded obesity research.
http://1.usa.gov/1ndWAmf

National Institute for Children's Health Quality
"Obesity: Developing Solutions for a Complex Problem"
Information on how the nonprofit has been working to lower obesity rates across the country.
http://obesity.nichq.org

Obesity Action Coalition
A national nonprofit dedicated to helping people with obesity to achieve better health through education, advocacy, and support.
www.obesityaction.org

Physical Activity Readiness Questionnaire for Everyone (PAR-Q+)
Online health questionnaire developed by a collaboration of health and fitness organizations to help identify people who may require a fitness appraisal before becoming more physically active.
http://bit.ly/1phHWzk

Robert Wood Johnson Foundation
The State of Obesity: Better Policies for a Healthier America, coproduced with the Trust for America's Health.
http://bit.ly/1nJ4TqS

Rudd Center for Food Policy and Obesity
A nonprofit organization devoted to improving children's eating behaviors.
www.yaleruddcenter.org

The Vegetarian Resource Group
Includes nutritional advice and links to low-fat vegetarian and vegan recipes and resources.
www.vrg.org

COMMUNITY CONSIDERATIONS

Some communities have made small inroads into tackling some of the factors associated with increased obesity rates.

Schools, where children spend the majority of their days, have been the focus of obesity treatment and prevention for many years.[44] The National Association of School Nurses has developed a position paper, *Overweight and Obesity in Youth in Schools—the Role of the School Nurse*, which is available at http://bit.ly/1yHzLCl.

School health advisory committees offer numerous opportunities to plan and promote obesity prevention and treatment, and the CDC has guidelines for promoting healthful eating and physical activity in schools.[45] The first step is to assess community needs, interests, and ideas for change. Use a coordinated approach to develop, implement, and evaluate the school's healthful eating and physical activity policies. It's important to ensure that quality meals and healthful foods are available in school vending machines and other venues and to implement comprehensive physical activity, nutrition, and healthful behavior programs. Mental and social health services and employee wellness programming for staff should also be part of any school plan.[45]

Obesity treatment in schools has been the subject of a systematic review. Lavalle and colleagues evaluated the effects on BMI of school interventions that incorporated physical activity, nutrition, or education in 43 studies: 37 included students regardless of baseline weight and six included only overweight students.[46] Twenty-eight (65%) of the studies focused on two or more of these health aspects, five (12%) incorporated education alone, and 10 (23%) incorporated physical activity alone. The meta-analysis revealed that the pooled effect for all children was a statistically significant change in BMI of –0.17 kg/m^2 (95% confidence interval [CI], –0.26 to –0.08, $P < 0.001$).[46] For overweight and obese children, the interventions produced a statistically significant change in BMI of –0.35 kg/m^2 (95% CI, –0.58 to –0.12, $P = 0.003$). Although the programs' absolute benefit of a –0.17-kg/m^2 change in BMI (a loss of 0.375 lbs.) would not be clinically significant for an individual, it demonstrates that intervention programs such as these may impart tangible health benefits across a population.

Work sites. Anderson and colleagues completed a meta-analysis of workplace interventions for controlling overweight and obesity. Their review of nine randomized controlled trials found a difference of –2.8 lbs. (95% CI, –4.63 to –0.96) between the no-treatment and intervention groups.[47] Interventions that used standard weight loss programs, such as Weight Watchers, were excluded. The components of the interventions included instruction on nutrition, physical activity, and environmental or policy

changes. Some studies included behavioral counseling. Structured programs and those offering behavioral counseling in addition to information saw larger effects.[47]

Policy assessment and interventions. At the community level, health policies and legislation show the most promise for reducing future obesity levels.[48] To this end, federal legislation on more nutritious school lunches (the Healthy Hunger-Free Kids Act; http://1.usa.gov/1jipVKq) was passed by Congress in 2010, and First Lady Michelle Obama spearheaded a national initiative to fight childhood obesity the same year. The goal of her Let's Move campaign (www.letsmove.gov) is to reduce childhood obesity to 1970 levels within one generation.

The Robert Wood Johnson Foundation (RWJF) has committed $500 million to programs designed to reduce childhood obesity. RWJF projects include increasing the number of community-based recreational areas and improving community access to healthy fresh foods. The RWJF Web site (www.rwjf.org) provides weekly updates on childhood obesity measures in research, community action, and nutrition policy. For information on the efforts of this and other organizations to prevent, treat, and educate the public on obesity, see *Resources*.

The call for policies and legislation to restrict or limit direct (through sales to individuals) and indirect (through advertising) access to foods deemed unhealthful is growing in popularity. A recent study found that support for policy interventions that limit obesity-producing behaviors is high.[49]

The debate was played out recently in New York City, where in the summer of 2012, the city council enacted a law limiting the sale of soft drinks in containers larger than 16 fl. oz.[50] The law was challenged by vendors in a lawsuit against the city, and in December 2012 the courts overturned the law.[51] The New York City Department of Health and Mental Hygiene and the New York City Board of Health appealed the ruling, but on June 26, 2014, the New York State Court of Appeals upheld the lower court's ruling and did not reinstate the ban.

Although progress has been slow and some initiatives have not succeeded, community action has had some effect. While obesity rates in the United States have remained high overall, there are signs that the increase is starting to slow. Whereas, in 2005, all but one state saw adult obesity rates rise from the previous year, between 2012 and 2013, adult obesity increased in only six states.[52] Moreover, the federal government reported that the obesity rate among two-to-five-year-olds had dropped 43% over the past decade.[53] It's possible that increased awareness of the obesity epidemic, in conjunction with the promotion of increased physical activity for all ages, has created a new social mindfulness about health and well-being. For infants and children, greater social awareness appears to have contributed to increased breastfeeding rates, a reduction in the intake of sugary drinks, and a rise in physically active play. In 2009, the federal Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) instituted the first major changes since the 1970s, which mandated that

- whole milk was to be replaced with milk containing only 1% fat.
- fruit juice was to be eliminated.
- funds for fruits and vegetables would be increased.
- education about healthy nutrition and breastfeeding would be provided.

A study that compared administrative records from the New York State WIC program over a six-month period in 2008 (July through December) with the same six-month period in 2011 suggested that the policy changes had made a difference. In just three years, the percentage of women initiating breastfeeding had increased from 72.2% to 77.5%, and average rates of obesity in one-to-two-year-olds and two-to-four-year-olds had decreased from 15.1% to 14.2% and from 14.6 to 14.2%.[54] Such improvements suggest that small changes can make a big difference over a short period of time. ▼

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For 31 additional continuing nursing education activities on obesity-related topics, go to www.nursingcenter.com/ce.

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