One hypothesis suggests the ability to store excess calories as fat in adipose tissue was useful during prehistoric times as a protection against food shortages. However, this ability later became a handicap in industrialized societies. The industrial revolution allowed food to be available consistently and reduced the physical effort needed to obtain, process, and prepare it. Increased control of infectious diseases, modern transportation, and increasingly sedentary patterns of work and recreation contributed to the presence of the current high level of obesity in the United States.

Moderate obesity was considered a sign of health in this country until the present century, and many societies still consider it healthy. The positive attributes of body fat were extolled by early fertility symbols (e.g., the Venus of Willendorf from prehistoric times), by the ancient Greeks, and by Shakespeare. On the other hand, Dickens described the association of extreme obesity with poor health in the character of Mr. Pickwick, who suffered from the cardiorespiratory syndrome that now bears his name. These concerns eventually led life insurance companies to develop and use rate structures based on tables of body weight-for-height (Rittenbaugh 1982).

Widespread interest in studying body fat (adipose tissue) was stimulated by the publication in 1940 of "Adipose Tissue: A Neglected Subject" (Wells 1940). A subsequent prophetic observation correlated the distribution and size of body fat cells with the development of atherosclerosis: fat cells concentrated in the trunk region were more likely to be linked to heart disease than were those on the arms or legs (Bjurulf 1959). In 1965, the American Physiological Society published a large compendium of information on adipose tissue that is still a good source of information (Renold and Cahill 1965).

The first data using weight tables to predict life expectancy were collected by the life insurance industry in 1913 (Association of Life Insurance Medical Directors and Actuarial Society of America 1913), but tables of ideal or desirable weight did not appear until the Metropolitan Life Insurance Company published its first tables of ideal or desirable weight in the 1940's (Simopoulos and Van Itallie 1984). Concurrently, modern epidemiologic techniques began to identify obesity as a risk factor for some of the major diseases of modern times: cardiovascular disease, diabetes, hypertension, and cancer. The most recent epidemiologic data on obesity as a health hazard were the subject of a national conference (NIH 1985) and are reviewed in this chapter.

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Significance for Public Health

Obesity is one of the most prevalent diet-related problems in the United States. It affects about 34 million adults ages 20 to 74 (NCHS 1987), with the highest rates observed among the poor and minority groups (Van Itallie 1985; Van Itallie and Abraham 1985).

Population data for heights and weights (and, therefore, obesity) among Americans derive from three national surveys: the National Health Examination Survey (NHES I) of 1960–62 (DHEW 1964), the first National Health and Nutrition Examination Survey (NHANES I) of 1971–74 (DHEW 1973), and NHANES II of 1976–80 (McDowell et al. 1981; NCHS 1987). In addition, data are available from the Framingham Heart Study (Hubert et al. 1983) and from the American Cancer Society Study of 1959–62 (Lew and Garfinkel 1979).

The 1971–74 NHANES found that 23.7 percent of men and 26.0 percent of women ages 20 to 74 were overweight. These figures were based on a definition of overweight that included persons whose weights met or exceeded the 85th percentile of the body mass index, or BMI (weight in kilograms divided by height in meters squared for men, and for women, divided by height to the power 1.5) for individuals ages 20 to 29. The BMI is discussed in more detail below. Because body weights increase with age, population rates of overweight that have been reported for adults ages 25 to 74 (and age-adjusted) are higher—26.0 percent of men and 29.4 percent of women (NCHS 1986).

NHANES II reported 25.6 percent of Americans ages 20 to 74 as overweight. Age-adjusted rates for blacks (36.6 percent) exceeded those for whites (24.6 percent), and those for females of all races (26.7 percent) exceeded those for males (24.4 percent). Although the rate for black males (26.3 percent) was only slightly higher than that for white males (24.4 percent), the rate for black females of 45.1 percent was highest of all— almost twice that of the 24.6 percent for white females (NCHS 1987). Rates of obesity in the United States appear to be higher than those observed in England, Canada, or Australia, at least among certain age groups (Bray 1985; Millar and Stephens 1987). The reasons for these differences have not been established.

The peak overweight rates for men occur from ages 35 to 64, whereas rates for women continue to increase throughout the ages in which they are

measured. The percent of adults classified as overweight increases from a low of 5.5 percent among black males ages 20 to 24 to a high of 61.2 percent among black women ages 45 to 54 (NCHS 1987).

NHANES II data also identified individuals at or above the 95th percentile BMI for a reference population ages 20 to 29. By this criterion, 9.3 percent of all Americans were classified as severely overweight, with the rate for blacks (15.5 percent) almost twice that for whites (8.8 percent). Here too, rates for females (10.6 percent) were higher than for males (8.0), with a much higher prevalence among black females than among white females (19.7 and 9.6 percent, respectively). The highest prevalence of severe overweight was the 26.3 percent observed among black females ages 35 to 44; the lowest was the 3.4 percent rate seen in white females ages 20 to 24 (NCHS 1987). When adjustments were made for differences in age distribution among populations between surveys, the age-adjusted proportions of men and women severely overweight (95th percentile by BMI) in 1976–80 significantly exceeded those in 1961–62 (Van Itallie and Abraham 1985).

The influence of poverty on the prevalence of overweight in women has been established. For example, in the 45 to 54 age range, 54.1 percent of poor women were overweight compared with 32.5 percent in the general female population (Van Itallie 1985).

Severe overweight increases the risk for high blood cholesterol, high blood pressure, and diabetes and, hence, for diseases for which these conditions are risk factors (see chapters on diabetes, coronary heart disease, high blood pressure, neurologic disorders, and kidney diseases). It also increases the risk for gallbladder disease (see chapter on gastrointestinal diseases) and for some types of cancer (see chapter on cancer). Its psychosocial consequences are significant (see chapter on behavior). The great prevalence of obesity and its physical and mental health consequences suggest that its prevention should be a high public health priority.

Perhaps for these and other reasons, many Americans try to lose excess weight. According to data from the 1985 National Health Interview Survey, 27 percent of males and 46 percent of females were trying to lose weight by reducing caloric intake, increasing physical activity, or both (Stephenson et al. 1987). The health consequences of these activities are also of public health concern and are reviewed in this chapter.

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Key Scientific Issues

- Definition of Obesity
- Health Consequences of Obesity
- Causes of Obesity
- Treatment of Obesity

Definition of Obesity

An ideal, health-oriented definition of obesity would be based on the degree of excess body fat at which health risks to individuals begin to increase. No such definition currently exists. Instead, the most commonly used methods estimate body fat as a percentage of total body weight (underwater weighing), establish an index of body fat level (skinfold thickness or waist-to-hip circumference measurements), compare weight-for-height measurements (height and weight tables), or compute an index of body weight as a function of height (BMI) in reference to population standards.

Much of the scientific disagreement about the level of body fat associated with increased health risks to individuals or to populations can be attributed to the difficulties in measuring body fat content and in defining overweight and obesity. Even underwater weighing, which measures body fat as a percent of total body weight and which is too cumbersome and expensive to use in population studies, can produce inaccurate results (Barnes 1987). The easily available measurements of heights, weights, and skinfold thicknesses, which are more frequently used in epidemiologic studies for assessing obesity and its effects, can be even less accurate.

Reference Body Weight Standards

An ideal body weight-for-height based on minimal health risks has not yet been defined for either individuals or populations. Instead, weight-for-height is usually compared with standards based on survey populations. The accuracy of such determinations, therefore, depends on the size and type of the reference population, as well as on the precision and appropriateness of the measures used.

Height and Weight Tables. The reference standards most commonly used to define obesity are those based on actuarial data from the Metropolitan

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Life Insurance Company (MLIC), in which "desirable" or "ideal" weight is the weight-for-height of insured persons with the longest lifespans. The original 1949 tables were revised in 1959 (MLIC 1959) based on the Build and Blood Pressure Study of 1959 (Society of Actuaries 1960), and again in 1983 (MLIC 1983) based on the Build Study of 1979 (Society of Actuaries and Association of Life Insurance Medical Directors of America 1980).

Tables of heights and body weights derived from several studies are given in Table 6-1. These tables present ranges of weight-for-height associated with the lowest mortality rates. The midpoint of the range is usually used as the standard for ideal or desirable weight. As seen in Table 6-1, Metropolitan desirable weights of 1983 are higher than those for 1959 in nearly all height categories but especially in those that are shorter. Both sets of data present as "desirable" body weights that are significantly lower than average weights measured in population surveys.

Relative Weight. Relative weight refers to actual weight as a percent of the desirable weight defined in the tables. A relative weight of 100 would thus be a desirable weight. As commonly used, a relative weight of 120 percent of that desirable is considered overweight, and relative weights of 140 percent or more are considered severely overweight (NIH 1985; Foster and Burton 1985). The desirable weights were slightly more liberal in the 1983 Metropolitan tables than in the 1959 tables, particularly for persons of short stature; weights for the shortest men in the 1983 table were heavier by 12 lb and for the shortest women by 14 lb.

These tables are handicapped by major flaws in the data from the studies on which they are based. For example, the tables present heights and weights with shoes and clothing but whether heights and weights were obtained with or without clothes and shoes was not consistent. The data for the Metropolitan tables were drawn from persons who could afford life insurance and were predominantly white and from middle-class males of young and middle age; thus, they were not necessarily representative of other groups. For these and other reasons, some experts believe that these tables are limited in value (Knapp 1983).

Body Mass Index. Ratios of weight to height estimate total body mass rather than fat mass, but they correlate highly with amount of body fat (Revicki and Israel 1986). The most commonly used ratio is known as Quetelet's index, or the BMI, and is usually defined as body weight in kilograms divided by the square of the height in meters (wt/ht^2). A simple nomogram to facilitate calculation of the BMI is given in Figure 6-1. In

population studies such as NHANES, this index is modified to reflect body composition differences between men and women.

A great advantage of such an index is the capability to evaluate and compare not only individuals but populations or subgroups within populations. Thus, various experts have recommended that the degree of obesity be defined by the BMI and have established reference standards for its use. These standards were derived from the same studies used to construct the Metropolitan tables. The BMI equivalent to a relative weight of 100 was 22 kg/m² for men and 21.5 kg/m² for women (Simopoulos and Van Itallie 1984). Table 6-2 shows the equivalent body mass indices derived from the three reference populations in common use: the 1959 and 1983 Metropolitan tables of desirable weights (MLIC 1959, 1983) and the 1976–80 NHANES (NCHS 1987). The indices in Table 6-2 are translated from the 85th and the 95th percentile weight distribution of NHANES II and from the 120 percent and 140 percent overweight cutoff points of the Metropolitan tables of 1959 and 1983. These indices are in rather close agreement. While some experts suggest that the body mass indices based on the 1959 Metropolitan tables, 26.4 for men and 25.8 for women, be considered the upper limits of normal weight (NIH 1985), others favor the higher levels derived from the more rigorous national probability sample of NHANES II (NCHS 1987).

The term overweight rather than obesity is used in this discussion because, although the BMI correlates highly with body fat, it does not distinguish between body fat and lean body tissue. While excess fat tissue is generally assumed to account for the additional weight and, therefore, the additional mortality among overweight individuals, excess weight can also include lean body mass (which weighs more than fatty tissue) (Van Itallie 1985; Forbes and Welle 1983) or a larger body frame size (Simopoulos and Van Itallie 1984). Such problems limit the use of the BMI as a standard for health risk (Garn, Leonard, and Hawthorne 1986). Its clinical significance, discussed below, has also been questioned (Callaway 1984).

Body Composition. Careful measurements of skinfold thickness made with calipers generally correlate well with body fat content. The sums of the scapular and triceps skinfolds that correspond to the 85th percentile of the NHANES II population were 45.5 mm in men and 70.1 mm in women (Van Itallie 1985). The lean body mass (or fat-free weight) can be calculated as the difference between total body weight and the weight of adipose tissue estimated from skinfold thickness or other methods. Elbow breadth and wrist circumference may be useful indicators of body frame size (Simopoulos and Van Itallie 1984).

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Table 6-1
Comparison of Metropolitan Desirable Weights With Average Weights
From U.S. Cohort Studies

Height (Without Shoes), cm (ft in)	Metropolitan Tables[a] (Medium Frame), Weight in Kilograms (Pounds) (Without Clothing) 1959[a]	1983	Average Weight for Age 40–49 y, kg (lb) Insured Lives Build and Blood Pressure Study 1959[b] (1935–1953)[e]	Build Study 1979[b] (1950–1971)[e]	American Cancer Society Study 1979[c] (1959)[e]	Health and Nutrition Examination Survey (HANES I, 1979)[d] (1971–1974)[e]
			Men			
156 (5 1)	50–55 (111–122)	57–62 (126–136)	60 (133)	61 (135)	—	—
159 (5 2)	52–57 (114–126)	58–63 (128–138)	62 (137)	63 (139)	67 (148)	66 (145)[f]
162 (5 3)	53–59 (117–129)	59–64 (130–140)	64 (141)	65 (144)	68 (149)	68 (150)[f]
164 (5 4)	54–60 (120–132)	60–65 (132–143)	66 (145)	68 (149)	69 (153)	73 (162)
167 (5 5)	56–62 (123–136)	61–66 (134–146)	68 (149)	69 (153)	71 (156)	72 (159)
169 (5 6)	58–64 (127–140)	62–68 (137–149)	70 (154)	72 (158)	73 (160)	75 (166)
172 (5 7)	59–66 (131–145)	64–69 (140–152)	72 (158)	73 (162)	74 (163)	78 (173)
174 (5 8)	61–68 (135–149)	65–70 (143–155)	73 (162)	76 (167)	77 (169)	79 (174)
177 (5 9)	63–69 (139–153)	66–72 (146–158)	76 (167)	78 (171)	78 (173)	79 (175)
179 (5 10)	65–72 (143–158)	68–73 (149–161)	78 (171)	80 (176)	80 (177)	83 (184)
182 (5 11)	67–74 (147–163)	69–75 (152–165)	80 (176)	82 (181)	83 (182)	85 (188)
185 (6 0)	68–76 (151–168)	70–77 (155–169)	82 (180)	85 (187)	85 (187)	88 (194)
187 (6 1)	70–78 (155–173)	72–78 (159–173)	84 (185)	87 (192)	87 (192)	92 (203)
190 (6 2)	73–81 (160–178)	73–80 (162–177)	86 (190)	90 (198)	90 (198)	92 (203)[f]
192 (6 3)	75–83 (165–183)	75–83 (166–182)	89 (196)	92 (203)	92 (203)	—

146 (4 9)	43–48 (94–106)	48–54 (106–118)	54 (120)	52 (115)	—	58 (127)[f]
149 (4 10)	44–49 (97–109)	48–54 (106–120)	56 (123)	54 (118)	52 (115)	59 (131)[f]
151 (4 11)	45–51 (100–112)	50–56 (110–123)	57 (126)	54 (120)	55 (121)	62 (136)
154 (5 0)	47–52 (103–115)	51–57 (112–126)	59 (129)	56 (124)	57 (126)	64 (141)
156 (5 1)	48–54 (106–118)	52–59 (115–129)	60 (132)	57 (126)	58 (128)	63 (138)
159 (5 2)	49–55 (109–122)	54–60 (118–132)	62 (136)	59 (130)	60 (132)	64 (141)
162 (5 3)	51–57 (112–126)	55–61 (121–135)	63 (139)	60 (133)	62 (136)	67 (148)
164 (5 4)	53–59 (116–131)	56–63 (124–138)	65 (143)	62 (136)	63 (139)	68 (151)
167 (5 5)	54–61 (120–135)	58–64 (127–141)	67 (147)	64 (140)	64 (142)	71 (156)
169 (5 6)	56–63 (124–139)	59–65 (130–144)	68 (151)	65 (144)	66 (146)	71 (156)
172 (5 7)	58–65 (128–143)	60–67 (133–147)	70 (155)	67 (147)	68 (150)	72 (158)
174 (5 8)	60–67 (132–147)	62–68 (136–150)	73 (160)	70 (152)	71 (156)	78 (172)
177 (5 9)	62–68 (136–151)	63–69 (139–153)	75 (165)	70 (155)	73 (161)	—
179 (5 10)	64–70 (140–155)	64–71 (142–156)	77 (170)	72 (159)	75 (165)	—

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aNot age specific: 1959 tables recommended for ages 25 and older; 1983 tables for ages 25 to 59 years.

bWithout shoes or clothing.

cValues are means for age groups 40 to 44 and 45 to 49. Self-reported heights without shoes and weights with indoor clothing.

dValues are means for age groups 35 to 44 and 45 to 54. Measured without shoes; clothing ranged from 0.20 to 0.62 lb (not deducted from weights shown).

eYears when measurements taken.

fEstimated values obtained from linear regression equations.

Source: Manson, J.E.; Stampfer, M.J.; Hennekens, C.H.; and Willett, W.C. 1987. Body weight and longevity. A reassessment. *Journal of the American Medical Association* 257:353–58. Copyright 1987, American Medical Association, reprinted with permission.
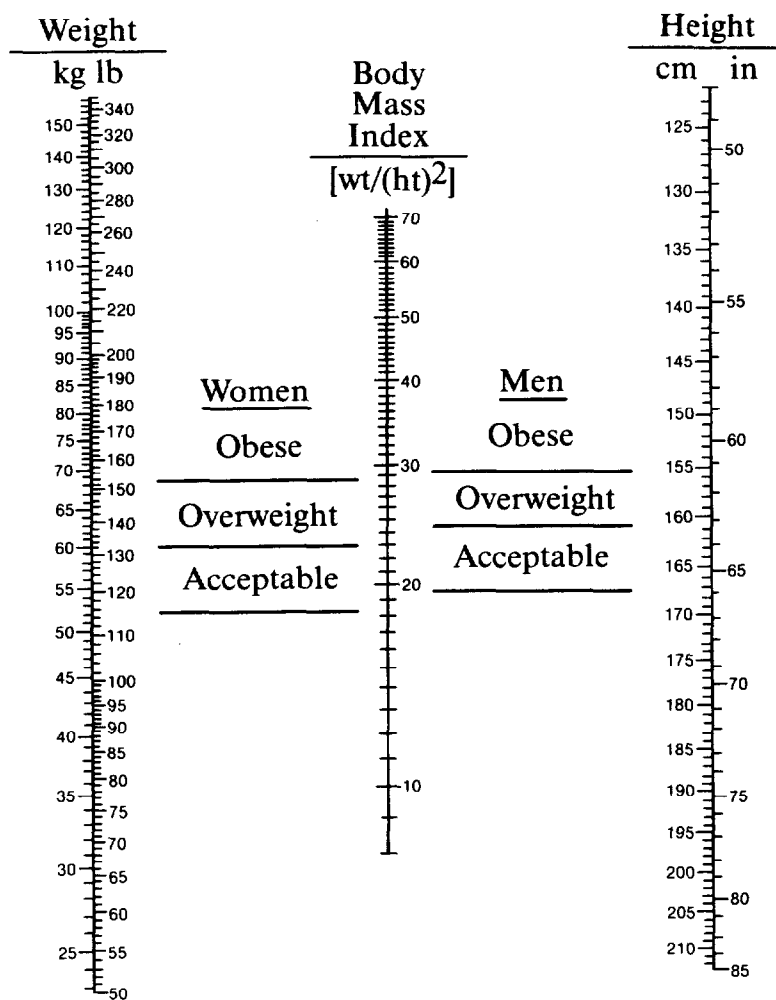
Obesity

Figure 6-1. A nomogram for determining body mass index (BMI). To use this nomogram, place a ruler or other straight edge between the column for height and the column for weight connecting an individual's numbers for those two variables. Read the BMI in kg/m² where the straight line crosses the middle lines when the height and weight are connected. Overweight: BMI of 25-30 kg/m²; obesity: BMI above 30 kg/m². Heights and weights are without shoes or clothes.

Source: Bray, G.A. 1985. Obesity: definition, diagnoses and disadvantages. *The Medical Journal of Australia* 142:S2-S8. Copyright 1985, *The Medical Journal of Australia*, reprinted with permission.

Table 6-2
**Body Mass Index (kg/m²) Used to Define Desirable Weight and
Overweight According to Three Different
"Ideal" Reference Populations**

Study[a]	"Ideal" Reference Population	BMI for "Ideal" Reference Population					
		Mean		Overweight		Severe Overweight	
		Men	Women	Men	Women	Men	Women
NHANES II	20- to 29-year-olds	24.3	23.1	27.8	27.3	31.1	32.3
Metropolitan 1959	Desirable weight insured	22.0	21.5	26.4	25.8	30.8	30.1
Metropolitan 1983	Desirable weight insured	22.7	22.4	27.2	26.9	31.8	31.4

[a]The NHANES II data define overweight as the 85th percentile or more of the distribution of BMI for men and women ages 20 to 29 and severely overweight as the 95th percentile for that reference population (NCHS 1987). The Metropolitan 1959 data are taken from the 1959 Metropolitan Life Insurance tables. Weights and heights were adjusted to approximate those without shoes or clothing (Simopoulos and Van Itallie 1984; NIH 1985). The Metropolitan 1983 data are taken from the 1983 Metropolitan tables. Height and weight were with shoes and light clothes (Van Itallie 1985; NIH 1985). For both the 1959 and the 1983 Metropolitan data, the weights for midpoint of medium-frame persons were used, and for both studies overweight and severe overweight were defined as 20 percent and 40 percent, respectively, over desirable weight.

Types of Obesity

Hyperplastic vs. Hypertrophic. Obesity is associated with too many adipose cells (hyperplastic obesity), adipose cells that are too large (hypertrophic obesity), or both (Hirsch and Batcheloer 1976). Studies on differences and changes in fat cell sizes and numbers have led to the hypothesis that while changes in the size of adipose cells may occur at any age, the number of adult cells may be fixed and determined by weight gain during certain periods of childhood development; fat cell number is established by late adolescence (Knittle et al. 1979) and, once established, does not decline. These suggestions have been interpreted to imply that the potential for sustained weight reduction in adulthood may depend, in part, on the number of adipose cells present. That is, because people with excessive numbers of fat cells may expect only limited success in weight reduction, prevention of adult obesity should begin with limits on excessive weight gain during childhood (Hirsch and Batcheloer 1976).

This hypothesis has received limited support. Although adult-onset obesity is more commonly due to an increase in the size of a normal number

of adipose cells than to an increase in their number (Krotkiewski et al. 1983), adipose cell number also increases in response to excessive weight gain at any time throughout adult life (Sjostrom 1980). Some studies have reported better treatment outcome among hypertrophic than hyperplastic obese subjects (Bjorntorp et al. 1975), but others have not observed any such difference (Strain et al. 1984), and the clinical significance of differences in fat cell size and number remains to be determined.

Upper Body vs. Lower Body. Women generally have been observed to have more subcutaneous fat than men, but men appear to suffer a greater cardiovascular risk from a given degree of fat than women (Bjorntorp 1983). The distribution of body fat may be an indicator of this difference. More men than women accumulate large fat cells in the abdominal region. This distribution around the abdomen with increased waist-to-hip ratio, referred to as upper body obesity, is associated with increased cardiovascular risk factors such as hypertriglyceridemia and impaired glucose tolerance (Krotkiewski et al. 1983). Lower body obesity is more typical of women, who tend to accumulate fat in the hips, gluteal regions, and extremities, a distribution that does not appear to be associated with increased cardiovascular risk factors (Krotkiewski et al. 1983), perhaps because fat on the hips is not well mobilized (Rebuffe-Scrive et al. 1985). Adipose cells are insulin-resistant in the abdominal region but usually are insulin-sensitive in the gluteal region (Maugh 1982). Studies of siblings ranging in relationship from identical twins to adoptive suggest that these differences in adipose cells may be genetically determined (Bouchard et al. 1985). Accordingly, women with upper body obesity with a waist-to-hip ratio similar to that of men are, like men, at increased risk for hypertriglyceridemia, hyperinsulinemia, diabetes, and cardiovascular disease (Krotkiewski et al. 1983).

Regardless of gender, a high waist-to-hip ratio predicts an increased risk for cardiovascular disease and diabetes. Measurements made in 1967 of waist-to-hip circumference of 792 54-year-old men in Gothenberg were positively correlated 13 years later with deaths from stroke and ischemic heart disease (Larsson et al. 1984). Other indices of body fatness such as the BMI, the sum of three skinfolds, or either waist or hip circumference alone showed no such correlation. A 12-year study of 1,462 women found the waist-to-hip ratio to be a better predictor of myocardial infarction, angina pectoris, stroke, and death than any other anthropometric measurement obtained (Lapidus et al. 1984). In both of these studies, persons whose waist-to-hip ratio was in the top quintile of distribution (greater than 1.0 for men or 0.8 for women) suffered the greatest incidence of cardiovascular disease.

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Childhood Obesity. Obesity and extreme obesity in children have been defined in several studies by a triceps or scapular skinfold thickness greater than or equal to the 85th or 95th percentile, respectively, of children in the same age group and sex between the baseline and a later year of measurement in national surveys (Dietz 1986). Increased weight-for-height above the 85th percentile for a defined population has also been used and can be deduced from standard growth charts (see chapter on maternal and child nutrition). Obesity in children can cause psychosocial dysfunction, orthopedic problems, abnormal glucose tolerance, hypertension, and elevated cholesterol and triglycerides that may persist into adulthood (Dietz 1986; Freedman et al. 1985). It also can increase the risk of obesity in adulthood (Garn 1985a, 1985b; Shear et al. 1988; Sorensen and Sonne-Holm 1988). According to a comparison of children studied in the NHES Cycle II and Cycle III between 1963 and 1970 and children studied later in NHANES I and II from 1971 to 1980, obesity and extreme obesity in children appear to be increasing by as much as 54 and 98 percent, respectively, in 6- to 11-year-olds and by 39 and 64 percent, respectively, in 12- to 17-year-olds (Gortmaker et al. 1987).

Health Consequences of Obesity

Excess Mortality

Numerous studies have examined the effects of excessive body weight on mortality. The best known studies include the Build and Blood Pressure Studies of 1959 and 1979 (Society of Actuaries 1960; Society of Actuaries and Association of Life Insurance Medical Directors of America 1980) and the American Cancer Society Study (Lew and Garfinkel 1979; Lew 1985). Several smaller studies include the Manitoba Study (Rabkin, Mathewson, and Hsu 1977), the Chicago People's Gas Company (Dyer et al. 1975), the Seven Countries Study (Keys 1980), the Longshoremen Study (Borhani, Hechter, and Breslow 1963), and the Provident Mutual Life Study (Blair and Haines 1966). This subject has been reviewed extensively (Simopoulos and Van Itallie 1984; NIH 1985; Manson et al. 1987).

In most of these studies, mortality increased with increasing weight. This effect can be expressed as the mortality ratio, defined as the actual number of deaths as a percent of expected deaths for the population as a whole. The mortality ratio has been shown to increase with degree of obesity, and with its duration, from 110 among persons 5 to 15 percent overweight to 227 among those 55 to 65 percent overweight (NIH 1985). Extreme (morbid) obesity, defined in this instance as either 100 percent or 100 lb over

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desirable weight (Kral 1985), has been reported in one small series to be associated with a mortality ratio of 1,200 percent (NIH 1985).

Although experts generally agree that excessive body weight is associated with increased mortality (NIH 1985), the body weight associated with the lowest mortality still lacks precise definition. Several important methodologic issues contribute to this uncertainty. These include the failure to control for cigarette smoking and inappropriate control for biologic effects of overweight such as hypertension, hyperlipidemia, and hyperglycemia, for weight loss due to subclinical disease (Manson et al. 1987), for weight history, for body fatness, or for duration of the condition (Simopoulos and Van Itallie 1984).

Several studies, for example, have reported a higher mortality at the lower as well as the higher range of fatness. This J- or U-shaped curve was found in the Build Study of 1979 (Society of Actuaries and Association of Life Insurance Medical Directors of America 1980; Van Itallie and Abraham 1985), the Framingham Heart Study (Hubert et al. 1983; Garrison and Castelli 1985), the American Cancer Society Study (Lew and Garfinkel 1979), and the Hawaiian Japanese Study (Rhoads and Kagan 1983). The increased mortality at lower weights was related to several types of cancer (Lew and Garfinkel 1979). The J-shaped curve was markedly reduced but still observable when adjusted for smoking history (Garrison and Castelli 1985), as well as for serum cholesterol level, blood glucose level, and systolic blood pressure (Harris et al. 1988).

In the Build and Blood Pressure Study of 1959 (Society of Actuaries 1960), optimal weight for all ages, judged by survival, was close to that of 20- to 29-year-olds. Older cohorts were heavier, and the increase in relative weight with increasing age was associated with increased mortality. Data from the Framingham Heart Study confirm that being overweight increases health risks for older people (Harris et al. 1988). Implicit in this trend is the undesirable effect of weight gain beyond desirable weight after maturity and the suggestion that weight gain acquired during adult life might be more detrimental than the degree of overweight. In fact, one study has reported the highest rates for cardiovascular and renal disease among overweight adults who were below average weight as children but who developed late-onset obesity (Abraham, Collins, and Nordsieck 1971). However, in the Build Study of 1979, the effect of overweight on mortality was more marked in obese men issued insurance policies between ages 15 and 39 than for equally obese men issued insurance policies between ages 40 and 69. A problem with both the 1959 and 1979 studies was the rather

short average duration of time that policyholders were observed—6.6 years for the latter study. In the Build Study of 1979, the adverse effects of underweight became evident in a relatively shorter time than the adverse effects of overweight (Society of Actuaries and Association of Life Insurance Medical Directors of America 1980).

The studies that do not show increased risk from obesity, such as the Seven Countries Study and the Longshoremen Study, have limitations because they were too short in duration, too small in sample size, or did not consider the adverse effects of smoking or of preexisting illness (Simopoulos and Van Itallie 1984; Van Itallie and Abraham 1985; Manson et al. 1987). Under these circumstances, the adverse effects of overweight would be obscured and of underweight exaggerated. Prospective data from the Framingham Heart Study, in which a cohort was followed for 26 years, clearly show the adverse effect on mortality of as little as 10 percent overweight, using the 1959 Metropolitan tables (Hubert et al. 1983). Bearing in mind data limitations, minimal mortality appears to occur at a weight 10 percent below the U.S. averages as shown in Table 6-1 (Manson et al. 1987).

Other Health Consequences

Relative body weights above 100 to 109 percent of desirable are associated with increased mortality as well as morbidity from heart disease, cancer, diabetes, digestive diseases, and cardiovascular disease. The higher the relative weight, the greater the risk for these conditions (Lew and Garfinkel 1979). From the NHANES II data, rates of hypertension and diabetes were nearly tripled for persons 20 percent or more overweight, and hypercholesterolemia was 50 percent more common (Van Itallie 1985). Obesity is associated with reduced levels of high density lipoproteins but with elevated levels of triglycerides and atherogenic lipoproteins (Albrink et al. 1980). The large American Cancer Society Study found mortality from various causes to increase according to the degree of overweight. Data from this study are shown in Table 6-3. Of all obesity-related diseases, noninsulin-dependent diabetes is most clearly and strongly associated with obesity. The details of these relationships are discussed in the chapter on diabetes.

Many other serious conditions such as gallstones, sleep apnea, osteoarthritis, and other disabling disorders of locomotion bear a direct relationship to obesity, although causality is not necessarily proved by these associations.

Table 6-3
Mortality Ratios for All Ages Combined in Relation to the Death Rate of Those 90 to 109 Percent of Average Weight

	Sex	<080	080–089	090–109	110–119	120–129	130–139	140+
				Weight Index				
Total deaths	M	1.25	1.05	1.00	1.15	1.27	1.46	1.87
	F	1.19	0.96	1.00	1.17	1.29	1.46	1.89
Coronary	M	0.88	0.90	1.00	1.23	1.32	1.55	1.95
heart disease	F	1.01	0.89	1.00	1.23	1.39	1.54	2.07
Cancer,	M	1.33	1.13	1.00	1.02	1.09	1.14	1.33
all sites	F	0.96	0.92	1.00	1.10	1.19	1.23	1.55
Diabetes	M	0.88	0.84	1.00	1.65	2.56	3.51	5.19
	F	0.65	0.61	1.00	1.92	3.34	3.78	7.90
Digestive	M	1.39	1.28	1.00	1.45	1.88	2.89	3.99
diseases	F	1.58	0.92	1.00	1.66	1.61	2.19	2.29
Cerebral	M	1.21	1.09	1.00	1.15	1.17	1.54	2.27
vascular disease	F	1.33	0.98	1.00	1.09	1.16	1.40	1.52

Causes of Obesity

The causes of obesity are incompletely understood, so that effective treatment is difficult. Obesity is the net result of an excess of energy consumption over expenditure. Factors that must be considered as contributing to causation are: (1) heredity, (2) primary overeating, (3) altered metabolism of adipose tissue, (4) defective or decreased thermogenesis (the process by which calories are converted into heat), (5) decreased physical activity without an appropriate reduction in food intake, and (6) certain prescribed medications. These potential causes can interact with one another. Of the six factors, individuals may have some control of overeating and underactivity.

Genetic Causes

Some studies of incidence of obesity among population groups, and among individuals within population groups, suggest that the tendency for obesity is inherited. On the other hand, separating genetic from cultural and environmental contributions is difficult—a fact brought out by studies of American Indians and blacks (Van Itallie and Abraham 1985; Van Itallie

1985), two populations that display higher levels of obesity than other groups. To explore the question of environment versus heredity, investigators have examined the weight characteristics of twins and adopted siblings. In addition, studies of reduced energy expenditure in overweight infants and adults, as reviewed later in this chapter, have provided evidence in support of a genetic basis for obesity.

One study measured the body fatness of 871 biologic and adopted siblings of French descent, including 87 pairs of monozygotic twins, and found that the amount and distribution of fat were related to the closeness of the genetic relationship. Similarities were not observed between adopted siblings. The genetic effect was particularly noted in measurements of skinfold thicknesses and in lean body mass. At the same time, a substantial environmental, or cultural, effect was also observed (Bouchard et al. 1985). Aerobic capacity in response to submaximal exercise also appeared to be at least partially genetically determined because similarities were greater among twins than among siblings or adopted siblings (Bouchard et al. 1984). Although these findings do not entirely rule out effects of early learned practices, they do make it tempting to speculate that for genetic reasons, some persons are less able to benefit from exercise and are thus more susceptible to obesity than others.

Another recent study provided even broader evidence for the genetic determination of obesity. In a study of 4,071 pairs of twins, investigators estimated that 80 percent of the contribution to obesity could be explained by genetics (Stunkard, Foch, and Hrubec 1986). Evidence for a smaller but still substantial genetic contribution was provided by investigations on 540 Danish adoptees (Stunkard et al. 1986). In this latter study, the BMI of the adoptees correlated strongly with that of their biologic parents but not at all with that of their adoptive parents, an observation that suggests that in this Danish population, early family environment had apparently little influence in determining the degree of fatness.

Overeating

Overeating is clearly a prominent contributor to obesity. Yet, obese persons do not necessarily consume more calories for their weight than lean individuals. Nutrient intake, established by dietary interview of a probability sample of 6,219 male and nonpregnant female adults selected from NHANES I, did not correlate with the degree of obesity (Braitman, Adlin, and Stanton 1985). Although underreporting of food intake by the obese could not be ruled out, the authors suggested that factors other than overeating, such as decreased levels of physical activity, be given increased consideration in the etiology of obesity.

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Additional factors include complex controls of feeding behaviors at the biologic and behavioral levels. At the biologic level, feeding behavior is regulated in part by the brain hypothalamus (Dallman and Bray 1986) and its interactions with brain neurotransmitters, nervous impulses from the intestinal tract or from higher brain centers, circulating nutrients absorbed from the intestinal tract, and certain hormones. While anatomic or biochemical lesions in the brain can cause obesity, more frequent and likely causes of overweight are improper signals coming to the brain, particularly those from the adipose tissue and the intestinal tract (Stricker 1984).

Feeding behavior occurs in response to hunger and to appetite induced by the presence of food. Satiety and the resulting cessation of eating occur in response to certain hormones, nervous impulses, and absorbed nutrients signaling the brain (Dallman and Bray 1986). Whether specific foods affect feeding and satiety and, consequently, over- or undereating is uncertain. Laboratory animals overeat and become obese if offered highly palatable foods, especially those high in fat and sugar and low in fiber. The same may be true of some humans (Herman and Polivy 1984). Foods low in fat and high in naturally occurring fiber appear to induce satiety in humans at lower levels of caloric intake than do high-fat, low-fiber foods (Heaton et al. 1983; Duncan, Bacon, and Weinsier 1983).

Psychologic and behavioral factors also influence eating behavior, although their precise role has been difficult to define. Suggestions that obese persons were more dependent on external cues than internal signals of physiologic hunger (Schachter 1971) or were more restrained (and, therefore, overcompensated) in their eating behavior (Herman and Polivy 1984) have not proved as useful in diagnosis and treatment as was once thought (Stunkard and Messick 1985). The effects of peer pressure (Herman 1978) are also not well established.

Altered Adipose Cell Metabolism

Obesity might result from a metabolic error in energy balance in which an unusually high proportion of available dietary calories is directed to adipose tissue for storage. In humans and in many animals, adipose tissue is not as active in fat synthesis as other tissues but instead depends largely on circulating triglycerides for its fat content. These, in turn, originate from chylomicrons derived from absorbed dietary fat or from very low density lipoproteins synthesized by the liver from excess nutrients not immediately needed as fuel.

Lipoprotein Lipase. The removal of circulating triglyceride and its deposition in adipose tissue depends on lipoprotein lipase, an enzyme in adipose tissue. In studies of genetically obese strains of experimental animals, the observed increase in fat mass at the expense of lean body mass, even with caloric restriction, has been attributed to an excess of adipose tissue lipoprotein lipase (Greenwood and Vasselli 1981). Too much of this enzyme appeared to "pull" triglyceride into the adipose tissue, cause deprivation of other tissues (such as muscle), and lead to overeating. Thus, the overproduction of adipose tissue lipoprotein lipase might account for human obesity. Increased synthesis of lipoprotein lipase has been observed in the adipose tissue of obese persons who are losing weight, which could explain the rapid regain of weight lost with dieting (Schwartz and Brunzell 1981). However, other observers have reported no increase of adipose tissue lipoprotein lipase activity in response to caloric restriction. Instead, they report a decline in the enzyme with reduced calories and an increase with return to higher caloric intake (Rebuffe-Scrive, Basdevant, and Guy-Grand 1983). Until these inconsistencies can be resolved, the role of lipoprotein lipase in human obesity remains uncertain.

Hormones. In times of caloric need, as in fasting or during exercise, lipoprotein lipase is inactivated and adipose tissue is mobilized. Many hormones stimulate lipolysis (breakdown of lipid), but epinephrine released from the adrenal medulla and norepinephrine released from sympathetic nerve endings are probably the chief activators of lipolysis during exercise and stress, respectively. A genetic component to epinephrine-stimulated lipolysis has been identified (Despres et al. 1982), which suggests that an inherited impairment of epinephrine response during exercise could account for the observed failure of obese individuals to lose weight with exercise (Ribeiro et al. 1984).

Insulin is another hormone that modifies energy balance and may contribute to obesity (Dallman and Bray 1986). Hyperinsulinemia and insulin resistance, for example, can occur in all types of obesity and may be a link between hypertension, obesity, and glucose intolerance (Modan et al. 1985). Plasma insulin, which increases in response to feeding, promotes uptake of circulating triglycerides by adipose tissue and inhibits fat mobilization, thereby favoring storage of excess calories as fat. These reactions are reversed during fasting, when insulin levels are low. As discussed in the diabetes and high blood pressure chapters, weight loss often is followed by a correction of these abnormalities; when weight is regained, the problems return.

Whether human obesity is caused by a primary abnormality in adipose tissue or in the hormones that regulate its deposition is unknown at this time. However, adipocyte precursors obtained from massively obese subjects release a factor that, when added to rat adipose cells in tissue culture, causes cells to multiply (Lau, Roncari, and Hollenberg 1987). This finding suggests that metabolic effects may indeed play a role.

Altered Thermogenesis

Set Points. The weight of most people remains remarkably stable over the adult years, despite large fluctuations in dietary intake. Experiments in animals indicate that body weight is stabilized at a specific set point that is maintained without conscious control by variations in metabolic rate in response to food intake (Keesey 1986). A possible mechanism for such stability is a change in body heat production, or thermogenesis, to compensate for changes in energy intake (James 1983). Although human body weight often appears to be regulated at a set point, its controlling mechanism has not yet been demonstrated.

Heat production is an accurate measure of energy expenditure or metabolic rate. Because measurement of actual heat production is cumbersome and expensive, metabolic rate is usually determined by indirect calorimetry, which converts measurements of oxygen consumption into calories, ideally expressed per unit of lean body mass. Lean body mass is the body compartment containing the most metabolically active tissues and accounts for most energy expenditure. Thus, accurate methods for estimating lean body mass are essential (Horton 1983) as are those for measuring the degree of physical activity.

Metabolic Rate. The measure of the energy used for running the body's essential metabolic machinery and for maintaining body temperature is the resting metabolic rate (RMR), obtained when the subject is resting comfortably several hours after the last meal or physical activity. The basal metabolic rate (BMR) is the energy the body needs when the subject is at complete rest, before arising in the morning and 12 hours or more after the last meal (Horton and Danforth 1982). The RMR is much more convenient to obtain, and although it is slightly higher than the BMR, it provides a reasonable estimation of the BMR. At stable body weights, BMR and RMR are higher in obese persons than in normal-weight persons because of the increased body size. It is uncertain, however, whether the RMR of the obese is the same (Himms-Hagen 1984; Blaza and Garrow 1983) or lower (Danforth 1983) than that of normal-weight persons when it is expressed in terms of lean body weight.

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Diet-Induced Thermogenesis. The energy generated metabolically when food is digested, called diet-induced thermogenesis, rises with increased food ingestion. Adaptive thermogenesis is the increase in RMR that occurs with increased food intake. Together, these forms of thermogenesis account for only about 10 percent of total energy expended. Failure to increase thermogenesis with overeating could result in obesity, but whether obese persons have defective thermogenesis has not been established. In the Vermont Prison Study, thin prisoners required many more calories to increase their body fat than could be calculated from the caloric value of the food ingested (Sims et al. 1973). These results suggest that in normal-weight individuals, extra calories are readily metabolized to heat when they are not needed.

In persons of normal weight, thermogenesis increases following a meal and is proportional to meal size (Rothwell and Stock 1986). The type of carbohydrate consumed may affect heat production; sucrose appears to be more thermogenic than glucose in normal-weight—but not obese—persons (Sharief and Macdonald 1982). Other macronutrients may also be important. The thermogenic effect of dietary protein, carbohydrate, and fat has been estimated to be 25, 10, and 3 percent of calories ingested, respectively (James and Trayhurn 1981). The small amount of heat lost when fat is processed may account for the importance of dietary fat as an inducer of obesity. Despite these findings, other investigators found a very small increase in total heat output with overfeeding and have concluded that any such adaptation to carbohydrate (but not fat) intake is very small (James 1983).

Effect of Weight Loss. In studies using continuous indirect calorimetry, reduced glucose-induced thermogenesis was found after obese subjects lost 9 to 33 kg (compared with controls), suggesting that defective thermogenesis is one of the factors causing relapse of obesity after weight loss (Schutz et al. 1984). In another study, energy expenditure was measured in a respiration chamber before and during weight reduction in obese subjects, and it declined as fat was lost; results were based on lean body mass (Ravussin et al. 1985). Other investigators analyzed the caloric content of measured liquid formula diets fed to obese and lean (never obese) patients during long-term hospital stays of many months' duration under metabolic ward conditions. They found that obese and lean subjects required similar amounts of energy per square meter of surface area per day. After weight loss and stabilization to a lower body weight, the obese subjects showed a 28 percent decrease in calories required to maintain body weight at the new level (Leibel and Hirsch 1984).

Other researchers, however, have identified problems with methodology or experimental design in such studies of defective thermogenesis in the obese (James 1983). For example, measurement of thermogenesis over periods of time longer than a few hours is only feasible where calorimetry chambers are available. Although people can reside in such chambers and move about freely for days at a time, activity is far from normal. Moreover, the need to express oxygen consumption in terms of lean body mass presupposes accuracy of methods of measuring body composition. Changes in heat production when dieting subjects are re-fed also complicate these studies. Although some evidence favors the hypothesis that adaptive thermogenesis occurs with weight change and is mediated by thyroid activity and the sympathetic nervous system (Danforth 1983; Landsberg and Young 1983; Horton 1983), the effect appears to be small and the role of thermogenesis in weight loss must be considered unsettled.

Lack of Exercise

Various studies have shown that physical inactivity in adult life shortens life expectancy (Paffenbarger et al. 1986), perhaps because underactivity without concomitant decrease in food intake can result in obesity. The very large difference in caloric expenditure between the most inactive and most active occupations, 2,300 to 4,400 kcal daily (Joint FAO/WHO Ad Hoc Expert Committee 1973), suggests that exercise could help manage and prevent obesity. Obesity may be a disease of inactivity, but that hypothesis has been difficult to prove (Stern 1984). Direct methods of measurement of daily physical activity are lacking, and current methodology for evaluating activity at home cannot detect whether inactivity contributes to obesity (Garrow 1978). Most studies that have compared the activity patterns of lean and obese subjects are based on questionnaires.

Epidemiologic data from the Lipid Research Clinics Prevalence Study show relatively low energy intakes to be coupled with increasing body weight, particularly in women (Dennis et al. 1985). Other epidemiologic data support this finding (Braitman, Adlin, and Stanton 1985). These and other investigators attribute the finding to decreased activity level. In another study, persons who participated in vigorous weekend activity were leaner than those who did not (Morris et al. 1980). A correlation also has been reported in young adolescents between degree of obesity and number of hours of television watched per week (Dietz and Gortmaker 1985).

Another interesting, but as yet unvalidated, aspect of the relationship between physical activity and body weight is involuntary movement— spontaneous fidgeting or moving, which varies widely from individual to

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individual. In one study, for example, that measured energy expenditure in unselected subjects, very large individual differences in energy—100 to 800 kcal/day—were attributed to differences in spontaneous activity (Ravussin et al. 1986). Among infants, energy expenditure at age 3 months was more than 20 percent lower in those who became overweight than in infants whose weight remains normal (Roberts et al. 1988). A reduced 24-hour energy expenditure in adults correlated strongly with subsequent weight gain in southwestern American Indians with a high prevalence of obesity (Ravussin et al. 1988). Much is still to be learned about the relationship among physical movement, caloric expenditure, and body weight, but the cumulative effects of physical activity over time could be important in preventing or correcting obesity (Black 1983).

Prescribed Medications

Many drugs prescribed for clinical conditions other than obesity may cause weight gain. Such drugs include propranolol, clonidine, and related medications prescribed for hypertension and other cardiovascular diseases that may change metabolic rates and decrease levels of energy expenditure. Adrenal steroids such as prednisone, prescribed as anti-inflammatory agents, cause hypertrophic obesity. Some people experience weight gain with tranquilizers such as amitriptyline and diazepam, certain antihistamines such as cyproheptadine, and birth control pills. These and other issues related to medications are reviewed in the chapter on drug-nutrient interactions.

Treatment of Obesity

To lose weight, one must decrease caloric intake, increase caloric expenditure, or do both. Thus, the chief approaches to weight reduction involve behavior change related to diet and exercise, drugs to decrease hunger or increase satiety, and surgical or mechanical intervention designed to reduce food intake. To date, none of these methods has proved to be entirely effective, and none is without risk.

Weight loss reduces health risks in the obese. In theory, it should be accomplished easily, but in practice, traditional diet therapy has not been very successful; people who lose weight tend to gain it back (Stunkard 1986). Thus, a combination of diet and exercise seems the most sensible approach to treatment (Stricker 1984). Because obesity is a condition requiring continuous attention, any behavior changes required to maintain weight loss must be lifelong.

Behavior Modification

Behavior changes to induce weight loss were based initially on the theory that faulty eating behavior caused obesity and could be corrected by a program of record keeping, stimulus control, and reinforcement of appropriate behaviors. The early programs focused on the importance of eating behavior and introduced specified behavioral procedures to change eating patterns. Weight losses, however, were modest, and behavior therapy is now used as part of broader programs that include nutrition education, exercise, and cognitive restructuring (Stunkard 1987). Such programs form the basis of many commercial efforts that treat a substantial number of mildly obese people in this country (Brownell 1986). Current approaches are trying to increase the amount and duration of weight loss by combining various behavior modification techniques with low-calorie diets (Wadden, Stunkard, and Brownell 1983). The results have not been evaluated from a long-term perspective.

Drugs and Surgical Methods

Thus far, no drug therapy has induced long-term weight loss. Drugs are aimed at decreasing hunger or increasing satiety, usually by mimicking certain neurotransmitters or hormones that play a physiologic role in feeding behavior (Sullivan and Triscari 1985). One problem is that weight lost with drug therapy is more often regained than with other methods, suggesting that appetite-suppressant drugs lower the set point level at which body weight is regulated and only secondarily suppress appetite (Stunkard 1982). Regardless, weight loss would be expected to occur only during the period of drug therapy, suggesting the need for chronic treatment.

Surgical intervention appears appropriate only for selected persons with massive obesity or with its severe complications who have not responded to more conservative treatment. Jejunoileal bypass has been largely abandoned because of unfavorable side effects. Other procedures such as gastric balloons and gastric surgery, which reportedly cause substantial weight loss—although rarely to ideal weight—in morbidly obese persons (Kral 1985), require further study before their long-term effects are known.

Dangers of Dieting

Extremely low-calorie diets, 300 or 400 kcal per day, have resulted in deaths due, at least in part, to the effects of dietary deficiencies on the heart muscle (Van Itallie and Abraham 1985). Short-term studies indicate that the same problems are less likely to occur with diets higher in calories (range of

800 kcal) and with better quality protein (Wadden, Stunkard, and Brownell 1983). Nevertheless, such higher calorie diets still require careful medical supervision.

Another possible risk of dieting is the effect of repeated attempts on long-term prospects for weight less. One study of rats has suggested that cyclic weight reduction may increase the difficulty of losing weight. Obese rats subjected to two repeated episodes of weight reduction and weight gain lost less weight during the second cycle and regained it more easily and with a lower food intake (Brownell et al. 1986). Whether similar effects occur in humans is as yet uncertain.

Fad weight loss regimens of unscientific merit have been estimated to cost consumers $5 billion annually (Herbert 1981). While such diets attract many people's attention they may be dangerous, especially when they provide less than the full complement of essential nutrients (Dwyer 1985). This danger is discussed in greater detail in the chapter on dietary fads and frauds.

Depression brought on by dieting is a serious condition affecting many obese persons (Stunkard and Rush 1974). Preoccupation with being thin may lead to two related and often serious eating disorders, anorexia nervosa and bulimia, that occur most commonly in adolescent girls (Herzog and Copeland 1985). Anorexia nervosa is characterized by an extreme fear of becoming obese, with severe diet restriction and weight loss sometimes to the point of cachexia and death. Bulimia is a syndrome of secretive binge eating followed by self-induced vomiting or purging. These conditions are discussed in detail in the chapter on behavior.

Implications for Public Health Policy

Dietary Guidance

General Public

Excess weight or overweight occurs when too few calories are expended and too many consumed for individual metabolic requirements. The extraordinarily high prevalence of obesity in the United States—one-fourth of American adults are overweight and nearly one-tenth are severely overweight—coupled with its role as a risk factor for diabetes, hypertension, coronary artery disease and stroke, gallbladder disease, and some types of cancer, suggests that a reduction in the average weight of the general

population would improve the Nation's health. Americans, in general, would benefit from a lifestyle that includes more physical activity and a diet containing fewer calories.

Because fat contains more than twice the caloric value per gram of either protein or carbohydrate, the general public would benefit from reduced fat intake. In addition, it may be difficult to meet essential vitamin and mineral requirements on low-calorie diets. Because sugar and alcohol provide calories from carbohydrate but no other nutrients, individuals seeking to attain and maintain desirable body weight should use these substances sparingly.

Evidence indicates that exercise burns calories, increases the proportion of lean to fat body mass, and, therefore, raises the metabolic rate. Therefore, increased levels of physical activity are important for attaining desirable body weights among the general population.

Special Populations

Qualified health professionals should evaluate overweight persons for the presence of chronic disease risk factors—especially elevated blood cholesterol, blood glucose, or blood pressure. Such evaluation is important for individuals whose excess body fat is distributed mainly on the abdomen. This pattern is more typical for men than for women, and it increases risks for diabetes, high blood pressure, hyperlipidemia, and heart attacks.

Health professionals should work with obese persons to restrict caloric intake and to increase caloric expenditure. Such advice should also be provided to overweight persons, with or without other significant risk factors, to help reduce their risk for heart disease, stroke, some kinds of cancers, and many other diseases and to prevent or reduce psychosocial complications of obesity. Professional guidance is recommended because many popular means to reduce weight may themselves pose risks to health and because unsupervised efforts to control obesity usually fail over the long term. Although excess body fat is difficult to lose, current research suggests that long-term individual or group programs that facilitate behavioral changes in diet and exercise are most likely to be effective. The intensity of these programs and the precise goal for weight loss should depend on the patient's degree and distribution of overweight, weight history, chronic disease risk factors, health status, and personal choices.

Current evidence is insufficient to recommend similar programs for overweight children. Obesity in infancy and childhood increases the risk for