Review Article

Primary Care

Guidelines for Healthy Weight

Walter C. Willett, M.D., Dr.P.H.,
William H. Dietz, M.D., Ph.D.,
and Graham A. Colditz, M.D., Dr.P.H.

EXCESS body fat is a cause of cardiovascular diseases, several important cancers, and numerous other medical conditions[1] and is a growing problem in many countries. In the United States, for example, the age-adjusted prevalence of obesity increased by approximately 30 percent from 1980 to 1994.[2] In this review we consider the assessment of body fat and the definition of a healthy body weight for an adult. We also discuss how clinicians can use this information in caring for patients. Because overt obesity has undisputed adverse consequences for health, our focus is on lesser degrees of adiposity, the consequences of which have been more controversial.

ASSESSMENT OF BODY FAT

The measurement of body fat, which is composed mainly of adipose in the form of triglyceride, represents a challenge to researchers and clinicians. The main stores of fat are subcutaneous and intraabdominal, and considerable amounts of fat can also reside within muscles, particularly in elderly persons. Because fat is diffuse and inaccessible, it is impossible to measure total adipose directly. Traditionally, the gold standard for estimating body fat has been hydrodensitometry (underwater weighing), which is based on the principle that fat tissue is less dense than muscle and bone. Dual-energy x-ray absorptiometry is now replacing densitometry as a standard because of its high precision and its simplicity for the subject. Both of these methods are used primarily for research purposes and are not available for routine clinical care, but they can be used to validate other methods of measuring body fat.

In clinical practice and in large epidemiologic studies, body fat is most commonly estimated by using a formula that combines weight and height. The underlying assumption is that most of the variation in weight for persons of the same height is due to fat mass. The formula used most frequently in epidemiologic studies is the body-mass index, also called the Quetelet index, which is the weight in kilograms divided by the square of the height in meters. In the 19th century, Quetelet observed empirically that in adults this index is minimally correlated with height and thus provides an appropriate measure of weight adjusted for height.[3] Among middle-aged adults, body-mass index is strongly correlated with fat mass measured densitometrically and adjusted for height (r is approximately 0.9 for both men and women); other indexes based on weight and height do not appear to be superior.[4,5]

Measurements of weight and height, even those reported by subjects themselves,[5] are highly accurate and do not contribute importantly to errors in assessing body-mass index. The principal limitation of the body-mass index as a measure of body fat is that it does not distinguish fat mass from lean mass. Although direct evidence is lacking, body-mass index is probably a less valid measure of body fatness in older adults in whom differential loss of lean mass contributes increasingly to variation in weight. The body-mass index has proved useful for epidemiologic research, but many physicians and patients find it difficult to interpret. Therefore, the values are often back-translated to weights for specified heights and presented in tables that give weight guidelines (Table 1).

Other methods used in assessing fatness are body circumferences (most commonly waist and hip), skinfold thicknesses, and bioimpedance. The measurement of body circumference has received attention because of the interest in excess visceral (intraabdominal) fat — independent of total adiposity — as a potential risk factor for chronic diseases. Waist circumference and the ratio of waist circumference to hip circumference have both been used for this purpose, but neither provides a precise estimate of visceral fat.[6,7] Furthermore, their ability to predict disease may result, in part, from the information they provide on overall fatness, not just visceral fat. There is usually no doubt that a large or increasing abdominal circumference is due to excess fat, after ascites has been ruled out.

Measurements of skin-fold thickness can provide a reasonable assessment of body fat, especially if tak-

From the Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston (W.C.W., G.A.C.); the Departments of Epidemiology (W.C.W., G.A.C.) and Nutrition (W.C.W.), Harvard School of Public Health, Boston; and the National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Atlanta (W.H.D.). Address reprint requests to Dr. Willett at 655 Huntington Ave., Boston, MA 02115.

TABLE 1. U.S. GUIDELINES FOR WEIGHT FOR MEN AND WOMEN, 1995.*

HEIGHT†	WEIGHT‡
	pounds
4′10″	91–119
4′11″	94–124
5′0″	97–128
5′1″	101–132
5′2″	104–137
5′3″	107–141
5′4″	111–146
5′5″	114–150
5′6″	118–155
5′7″	121–160
5′8″	125–164
5′9″	129–169
5′10″	132–174
5′11″	136–179
6′0″	140–184
6′1″	144–189
6′2″	148–195
6′3″	152–200
6′4″	156–205
6′5″	160–211
6′6″	164–216

*Data are from the Department of Agriculture and Department of Health and Human Services.[1] These guidelines represent a body-mass index of 19 to 25. The body-mass index can be calculated as [weight (lb) × 703] ÷ [height (in.)^2]. To convert values for height to centimeters, multiply the height in inches by 2.54. To convert values for weight to kilograms, multiply by 0.45. Weight gains of more than 10 lb after 21 years of age should be avoided, even if weight remains within the healthy range.

†The subject does not wear shoes when height is measured.

‡The subject is unclothed when weight is measured.

en at multiple sites, and they provide additional information on the location of the fat. However, these measurements are subject to considerable variation between observers, do not provide information on abdominal and intramuscular fat, and in general are not superior to simple measurements of weight and height.[4,5] Thus, measurements of skin-fold thickness are most useful to specialists in body composition and for research.

The measurement of bioimpedance is based on the principle that lean mass, because it is primarily an electrolyte solution, conducts current better than fat mass. Thus, a measurement of the resistance to a weak current (impedance) applied across the extremities, when combined with height and weight and an empirically derived equation, provides an estimate of body fat. Devices to measure bioimpedance are simple and moderately priced and impose a minimal burden on patients, but they do not measure fat better or predict biologic outcomes more accurately than simple anthropometric measurements.

Because many of the adverse effects of excess body fat on cardiovascular risk factors and disease may result from increased resistance to insulin, biochemical measurements that reflect insulin resistance may be useful as indicators of fat mass. Such measurements could include fasting levels of insulin and triglycerides and levels of high-density lipoprotein cholesterol. Plasma leptin levels are strongly correlated with fat mass,[8] but these measures have not been evaluated adequately for the assessment of adiposity.

DEFINITION OF HEALTHY WEIGHT

Traditionally, the criterion for setting weight guidelines has been the range of weights that correspond to the lowest mortality. Using mortality as an end point is simple, reliable, and intuitively appealing. However, basing guidelines on total mortality rates is fraught with methodologic problems.

Reverse causation is the most serious problem associated with using total mortality as an outcome; people frequently lose weight as a result of an illness that is ultimately fatal, a situation that creates the appearance of higher mortality among those with lower weights. Conditions that cause weight loss may remain undiagnosed for several months or years, as could be the case for occult neoplasms, chronic lung or cardiac disease, alcoholism, or depression. Several strategies can be used to minimize the effect of reverse causation. Subjects with diagnoses that might affect weight and subjects who report recent weight loss, such as during the previous five years, can be excluded from a prospective study. Deaths that occur during the first several years of follow-up — possibly as a result of conditions that caused lower weights at base line — can also be excluded. Although valuable, these strategies do not provide a perfect solution to the problem of reverse causation, because some chronic conditions may cause weight loss many years before death occurs. If the period for excluding deaths or the total follow-up is too long, many subjects will have substantial changes in weight that are not the result of underlying disease, and weight status during the later years of the study will be misclassified. Thus, some compromise must be reached: at least the first several years of follow-up should be excluded, and follow-up should probably not continue for more than 10 or 15 years without some updating of weight status. Analyses that vary the censoring period and follow-up time can be used to determine how sensitive the findings are to these decisions.

A second major concern is that confounding factors may distort the association between body weight and mortality. Smoking is particularly important, because smokers tend to weigh less and to have much higher mortality rates than nonsmokers. As with ill-

ness, smoking will make leaner persons appear to be at elevated risk. Many large studies, including those conducted by the Metropolitan Life Insurance Company that were used for many years to set weight standards,[9] did not include data on smoking and thus overstated desirable weights. Even if data on smoking are available, simple statistical adjustments for smoking are not entirely satisfactory, because nuances such as depth of inhalation and genetic susceptibility, which cannot be accounted for, could influence the effect of smoking on both weight and mortality.

The most satisfactory way to deal with smoking is to restrict the analysis to subjects who have never smoked. Unfortunately, many studies have been too small to have adequate statistical power when the analysis is limited to those who have never smoked, in part because death rates are lower in this group. Other factors that could confound the association between body weight and mortality are the presence or absence of alcoholism, the composition of the diet, and physical activity. Adjustment for physical activity may not be appropriate, because activity is an important determinant of body weight.

A third problem in some earlier analyses of studies of weight and mortality is that the physiologic effects of excess fatness, such as hypertension, diabetes, and dyslipidemia, were controlled for statistically, thus artificially removing some of the effects of being overweight.

The leanest group in a population is a mix of smokers, persons who have lost weight as a result of underlying disease, and persons who have maintained a lean weight by balancing physical activity and caloric intake. Thus, in analyses adjusted only for age, the relation between body weight and mortality is typically U-shaped, with increased death rates among both the leanest and the heaviest persons. However, because of the potential for bias, these results should not be accepted as evidence that low weight is harmful. Also, in analyses controlled only for age, the range of weights associated with the lowest mortality tends to increase with age. However, this finding may reflect the burden of chronic illness that accumulates with age, which would result in the low-weight groups gaining a greater percentage of persons at higher risk of dying. Such observations, based on data biased by reverse causation and inadequate control for smoking, were the foundation for the 1990 U.S. guidelines for weight, in which the range of healthy weights increased with age from a body-mass-index range of 19 to 25 before 35 years of age to 21 to 27 thereafter.[10] These guidelines implied that a weight gain of 4.5 to 6.8 kg (10 to 15 lb) at 35 years of age was desirable and that about 5 percent of middle-aged men and 17 percent of middle-aged women in the United States risked having poor health by being underweight.[11]

Unfortunately, few studies have tried simultaneously to minimize reverse causation, account for cigarette smoking, and not adjust statistically for the physiologic effects of excess body fat. A review of the 25 major studies published up to 1986[12] found that not one study met these three criteria. In a recent meta-analysis,[13] only two studies (both of men)[14,15] analyzed data on persons who had never smoked and also attempted to account for reverse causation. One of these studies[14] included only 13 deaths; in the other, mortality rates among nonsmokers increased linearly with greater weight.[15] Among women in the Nurses' Health Study, the typical U-shaped relation between body-mass index and mortality was found in the overall age-adjusted analysis, but the relation became a simple positive one after reverse causation was accounted for and the analysis was limited to persons who had never smoked.[16] In the most powerful analysis to date, Stevens et al.[17] studied, over a 10-year period, mortality rates among men and women in the large American Cancer Society cohort who had never smoked. After early deaths were eliminated, mortality increased linearly with increasing body-mass index from very lean to clearly obese at all ages up to 75 years; the association was weaker at older ages. For persons younger than 75, total mortality rates were 8 to 35 percent higher among those with body-mass indexes of 25 to 26.9 and 18 to 40 percent higher among those with body-mass indexes of 27.0 to 28.9 than among persons with body-mass indexes of 19 to 21.9 (Fig. 1).

Although total mortality should be one criterion for weight guidelines, the incidence of disease should also be considered. Artifacts resulting from reverse causation are much less of a problem in studies of the incidence of disease than in studies of death. Also, conditions such as coronary heart disease, stroke, diabetes, cancer, and osteoarthritis contribute greatly to suffering even if they do not result in death. Furthermore, guidelines should be based on something more than statistical associations between weight and death. For leanness to cause excess mortality, it should cause either an increased incidence of one or more common diseases or a higher case fatality rate. The lack of evidence for such effects or of a biologic mechanism to explain an elevated risk of death among lean persons should have aroused concern in the early studies that the excess mortality in that group may have been due to artifact.

The relation between body-mass index and the incidence of several common conditions caused by excess body fat — specifically, type 2 diabetes, hypertension, coronary heart disease, and cholelithiasis — in women and men is presented in Figure 2. The relations in the range of body-mass indexes less than 30 appear monotonic and approximately linear. In women with a body-mass index of 26, the risk of coronary heart disease was about twice the risk in women with a body-mass index of less than 21; the risk in

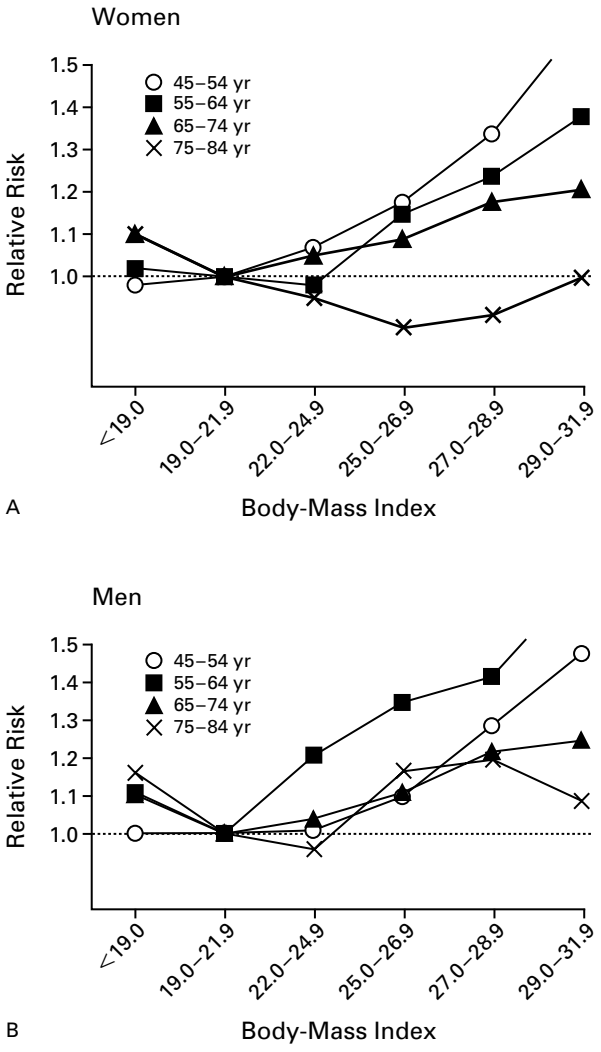

Figure 1. Age-Specific Relation between Body-Mass Index and the Risk of Death among Women (Panel A) and Men (Panel B) 45 to 75 Years of Age.

Data are from the American Cancer Society Cohort[17] and June Stevens, University of North Carolina.

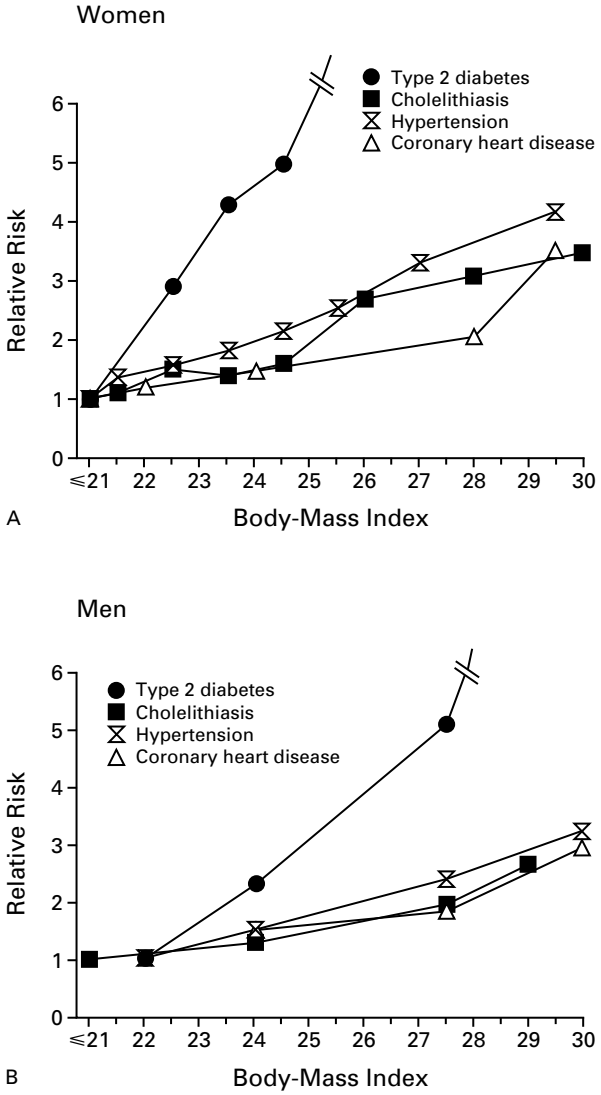

Figure 2. Relation between Body-Mass Index up to 30 and the Relative Risk of Type 2 Diabetes, Hypertension, Coronary Heart Disease, and Cholelithiasis.

Panel A shows these relations for women in the Nurses' Health Study, initially 30 to 55 years of age, who were followed for up to 18 years.[18-21] Panel B shows the same relations for men in the Health Professionals Follow-up Study, initially 40 to 65 years of age, who were followed for up to 10 years.[22,23]

men with a body-mass index of 26 was about 1.5 times the risk in men with a body-mass index of less than 21. For the same comparison, the risk of diabetes was four times as high in the men and eight times as high in the women. The risk of hypertension and the risk of cholelithiasis were two to three times as high among both men and women with a body-mass index of 26, as compared with the leanest group. With a body-mass index of 29 or higher, these risks were greatly increased. Other conditions whose incidence is increased by excess body fat are postmenopausal breast cancer[24,25]; cancers of the endometrium, colon, and kidney[25]; stroke[26]; osteoarthritis[27]; and infertility.[28] Excess body fat appears to be protective against very few conditions: the incidence (but not the associated mortality) of premenopausal breast cancer is slightly lower among heavier women,[24] and the rates of hip fracture are inversely related to body weight.[29] Hip fractures contribute only slightly to total mortality rates, however.

ISSUES PERTAINING TO THE CREATION OF GUIDELINES FOR WEIGHT

The selection of a cutoff point on a continuum involves the balancing of sensitivity and specificity. For example, as shown in Figures 1 and 2, the risk of serious conditions and death increases even at a body-mass index of 23. Therefore, a maximally sensitive guideline to identify persons at risk from excess body fat would use an upper limit for body-mass index of 22 or 23. However, many persons would be considered to have false positive results according to this criterion, because they would not subsequently have the conditions associated with being overweight. Conversely, if the upper limit were set at a body-mass index of 27, many persons with a lower body-mass index would not be classified as overweight but would later have conditions that result from excess body fat (false negative results). Balancing sensitivity and specificity is difficult, because excess body fat is clearly associated with multiple risks, but the costs of being inappropriately labeled as overweight are difficult to quantify. Thus, weight guidelines inevitably represent a somewhat arbitrary compromise. Since the first Dietary Guidelines for Americans were published in 1980, the language used to describe a healthy weight and range of weights has varied slightly.[1,10,30,31] Throughout this period, the lower boundary for body-mass index has been approximately 19, and the upper boundary, with the exception of the 1990 guidelines, has been approximately 25.

In setting the 1995 guidelines for weight,[1] the Dietary Guidelines Advisory Committee concluded that mortality clearly increased significantly with a body-mass index above 25,[16,17,32] whereas the incidence of diabetes, hypertension, and coronary heart disease began to increase well below a body-mass index of 25.[18-20,22,33] Because of the importance of total mortality, and because designating a cutoff point below 25 for the classification of overweight would label more than 50 percent of U.S. adults overweight, the committee concluded that a body-mass index of 25 represented a reasonable upper limit of healthy weight. This cutoff point is consistent with that recommended by a steering committee of the American Institute of Nutrition[33] and an expert committee of the World Health Organization.[34] The International Obesity Task Force provided a more detailed classification of values for the body-mass index: healthy weight, 18.5 to 24.9; overweight, 25.0 to 29.9; class I obesity, 30.0 to 34.9; class II obesity, 35 to 39.9; and class III obesity, 40.0 or higher.[35]

The cutoff point of 25 in the 1995 U.S. guidelines for weight is well supported by data, but the lower limit of 19 in the current recommendations (or 18.5 in the WHO guidelines) has far less empirical justification. The relatively few persons with a body-mass index of 19 or lower in the United States include not only healthy, active persons who have maintained a low body-mass index for many years, but also heavy smokers and those who have lost weight as a result of illness.[36] Morbidity and mortality among nonsmoking persons with stable body-mass indexes of 18 or lower, after the exclusion of those with recent weight loss or ill health, have rarely been studied.[12] Among nonsmoking women in the Nurses' Health Study who had stable weights and had body-mass indexes as low as 17, there was no excess mortality.[16] A point exists at which low body-mass index becomes a cause of ill health, but available data suggest that using a body-mass index of 19 or lower to identify persons at risk for morbidity and mortality will be highly nonspecific. Because of the lack of information about body-mass indexes below the range of healthy weight, the U.S. Dietary Guidelines Advisory Committee decided not to label body-mass indexes lower than 19 as healthy or unhealthy. However, as clinicians have long known, a low body-mass index resulting from unexplained weight loss demands a thorough investigation for underlying causes.[37]

WEIGHT GAIN WITH AGE

A major limitation of standard weight guidelines is that a person initially at the low end of the range can gain as much as 15 or 20 kg (33 or 44 lb) and still remain within the recommended range. Much smaller gains in weight during adulthood, however, are associated with significantly increased risks of many chronic diseases (Fig. 3). For example, as compared with women and men in the Nurses' Health Study[18-21] and the Health Professionals Follow-up Study[22] who maintained their weight within 2 kg (4 lb) of their weight at 18 to 20 years of age, those who gained 5.0 to 9.9 kg (11 to 22 lb) had risks of coronary heart disease, hypertension, cholelithiasis, and type 2 diabetes that were 1.5 to 3 times as high. These increases in risk were greater with larger gains in weight.

Changes in weight that have taken place since young adulthood are useful for assessing risks associated with body fat because they take into account individual differences in frame size and lean mass that are difficult to measure. In addition, change in weight provides a single, readily interpretable number that is known, at least approximately, to most persons. Most persons in the United States are not overweight when growth ends, at about 18 years of age for women and 20 years for men[11]; most excess body fat accrues in the subsequent decades. Except for the few persons involved in muscle building, substantial gains are largely fat. The absence of weight gain, particularly among men older than 50, however, does not indicate that fat has not increased. Beyond this age, muscle mass is, to varying degrees, replaced by fat, much of it within the abdomen. This phenomenon may be manifested by an increasing waist circumference.

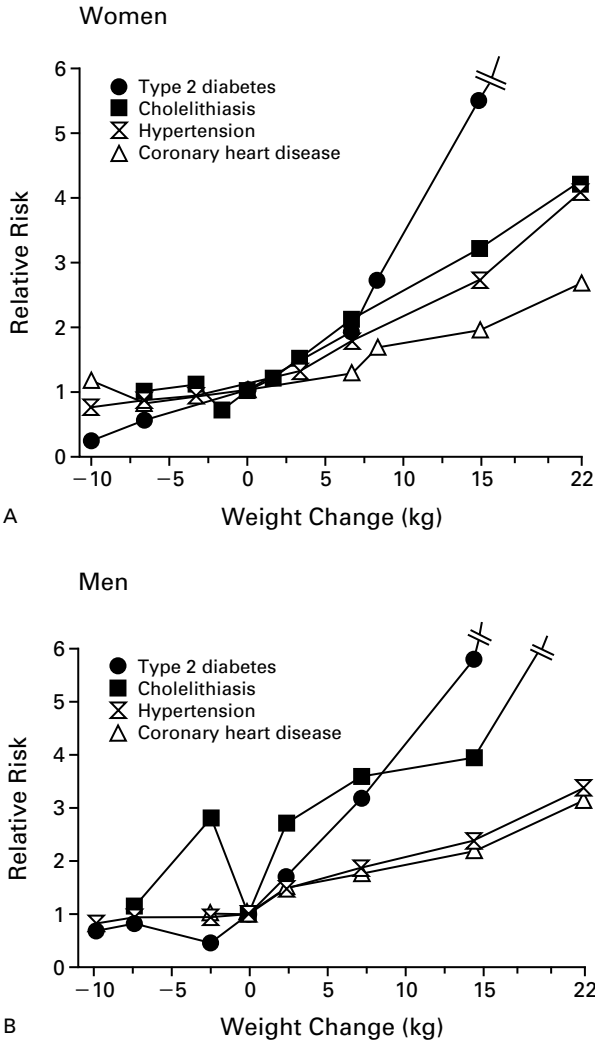

Figure 3. Relation between the Change in Weight and the Relative Risk of Type 2 Diabetes, Hypertension, Coronary Heart Disease, and Cholelithiasis.

Panel A shows these relations for change of weight from 18 years of age among women in the Nurses' Health Study, initially 30 to 55 years of age, who were followed for up to 18 years.[18-21] Panel B shows the same relations for change of weight from 20 years of age among men in the Health Professionals Follow-up Study, initially 40 to 65 years of age, who were followed for up to 10 years.[22]

CONSIDERATIONS OF SEX AND AGE

Should weight guidelines differ for men and women? Should guidelines change with age? At identical levels of body-mass index, women will, on average, have more body fat.[38] However, as shown in Figures 2 and 3, morbidity appears to increase with increasing body-mass indexes in a similar fashion for men and women, as does mortality.[17] Small differences according to sex may exist in these relations, but separate guidelines do not appear to be justified.

The relation between age and body weight is complex. For example, in the large American Cancer Society cohort,[17] the relation between body-mass index and total mortality or mortality from cardiovascular causes was approximately linear for all age groups up to 75 years, although the relative risks declined with age (Fig. 1). That the relative risks decline with age does not necessarily mean that excess body fat becomes less serious in older persons. Because mortality rises dramatically with age, the absolute excess risk associated with a higher body-mass index increases rather than decreases with age. Furthermore, because changes in body composition with age may make body-mass index a less valid indicator of body fatness in older people, the lower relative risks may reflect a lower validity of body-mass index as a measure of body fat. In a cohort of men,[22] body-mass index was a strong predictor of coronary heart disease among those younger than 65 but was minimally predictive for older men. However, waist circumference was weakly predictive of coronary disease among men younger than 65 but was strongly predictive for older men, which suggests that body fat is important at all ages but that the optimal means of assessing fat changes with age.

CRITERIA FOR RECOMMENDATIONS ON BODY CIRCUMFERENCE

In 1990, the Dietary Guidelines for Americans were accompanied for the first time by recommendations for the ratio of waist circumference to hip circumference; the ratio was not to exceed 0.95 for men and 0.80 for women.[10] In 1995, however, the guidelines stated that concern was warranted if the ratio exceeded 1.0. Waist circumference and the ratio of waist circumference to hip circumference are similarly correlated with measures of risk factors for coronary heart disease, such as high blood pressure or blood lipid levels,[39] and neither method has been consistently superior in predicting the risk of disease. Thus, because of its greater simplicity, waist circumference may be most useful in clinical practice.

As with the body-mass index, choosing a point on the waist-circumference continuum as a guideline involves a trade-off of sensitivity for specificity. For example, an expert panel on overweight and obesity in adults has recently suggested that increased risks exist if waist circumference is greater than 102 cm (40 in.) in men and 89 cm (35 in.) in women.[40] With waist circumferences smaller than these cutoff points, however, the relative risks of type 2 diabetes can be 3 to 5,[23,41] which suggests that these limits are insensitive to important degrees of abdominal adiposity. Han et al. found that in men, a waist circumference of 94 to 102 cm (37 to 40 in.) was associated with a relative risk of 2.2 of having one or more cardiovascular risk

factors, and in women a circumference of 80 to 88 cm (32 to 35 in.) was associated with a relative risk of 1.6.[39] Ideally, guidelines for waist circumference should be adjusted for overall body size, in particular for height, but this adds complexity with minimal gains in predictiveness.[23]

USING WEIGHT GUIDELINES

A primary use of weight guidelines is to provide direction for healthy persons. Thus, periodic measurements of height and weight are recommended for all patients.[42] Even small gains in weight within the range of healthy weights can carry health risks, and they indicate an imbalance between energy intake and activity that portends larger increases in the future. Thus, physicians should counsel their adult patients to make small but permanent adjustments in physical activity and eating patterns if even a small weight gain (e.g., 4 to 5 kg, or 10 lb) occurs after adult height is attained or if they are approaching the limit of the range for healthy weight. Attention to increases in waist circumference by 5 cm (2 in.) or more is also appropriate, even if weight has remained stable or within the range of healthy weights. Past weight guidelines have emphasized the development of complications, such as hypertension or diabetes, as additional indications for weight reduction. However, these conditions should be prevented by avoiding weight gain and overweight, because conditions such as diabetes and vascular complications may not be reversible. Furthermore, these conditions may make exercise and weight control more difficult.

For patients who are already overweight, weight guidelines should not be used to define goals for weight reduction, because for seriously overweight persons the range of healthy weights is often practically unachievable. However, reductions of even 5 to 10 percent can substantially improve blood pressure, serum lipid levels, and glucose tolerance[43] and reduce the incidence of diabetes[19] and hypertension.[20,40]

Only limited data support the notion that intentional weight loss will reduce total mortality rates,[44] but this issue is particularly difficult to evaluate because intentional and disease-related weight losses are difficult to distinguish. Nevertheless, the clear improvements in physiologic end points as well as the reduced incidence of diabetes[19] and hypertension[20] provide justification for weight loss as an objective. Among women in the Nurses' Health Study, those who lost 11 kg (24 lb) or more had a risk of type 2 diabetes that was more than 75 percent lower than that of nurses with unchanged weight.[19] For most overweight persons, the initial goal is to prevent further weight gain and then to achieve moderate reductions in weight.

In summary, the rapidly rising prevalence of obesity in the United States and most other countries will add substantially to future morbidity and mortality and will increase health costs. Preventing weight gain and overweight among persons with healthy weights and avoiding further weight gain among those already overweight are important public health goals. The road to prevention must begin with an increased awareness of even small weight gains and the counseling of patients to modify their diet and activity patterns appropriately.

REFERENCES

1. Department of Agriculture, Department of Health and Human Services. Nutrition and your health: dietary guidelines for Americans. 4th ed. Home and garden bulletin no. 232. Washington, D.C.: Government Printing Office, 1995.
2. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys, 1960 to 1991. JAMA 1994;272:205-11.
3. Quetelet A. Physique sociale: ou, essai sur le développement des facultés de l'homme. Brussels, Belgium: C. Muquardt, 1869.
4. Spiegelman D, Israel RG, Bouchard C, Willett WC. Absolute fat mass, percent body fat, and body-fat distribution: which is the real determinant of blood pressure and serum glucose? Am J Clin Nutr 1992;55:1033-44.
5. Willett WC. Nutritional epidemiology. 2nd ed. New York: Oxford University Press, 1998.
6. Schreiner PJ, Terry JG, Evans GW, Hinson WH, Crouse JR III, Heiss G. Sex-specific associations of magnetic resonance imaging-derived intra-abdominal and subcutaneous fat areas with conventional anthropometric indices: the Atherosclerosis Risk in Communities Study. Am J Epidemiol 1996;144:335-45.
7. Seidell JC, Oosterlee A, Thijssen MA, et al. Assessment of intra-abdominal and subcutaneous abdominal fat: relation between anthropometry and computed tomography. Am J Clin Nutr 1987;45:7-13.
8. Considine RV, Sinha MK, Heiman ML, et al. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. N Engl J Med 1996;334:292-5.
9. New weight standards for men and women. Stat Bull Metrop Insur Co 1959;40(November-December):1-11.
10. Department of Agriculture, Department of Health and Human Services. Nutrition and your health: dietary guidelines for Americans. 3rd ed. Home and garden bulletin no. 232. Washington, D.C.: Government Printing Office, 1990.
11. Kuczmarski RJ, Carroll MD, Flegal KM, Troiano RP. Varying body mass index cutoff points to describe overweight prevalence among U.S. adults: NHANES III (1988 to 1994). Obes Res 1997;5:542-8.
12. Manson JE, Stampfer MJ, Hennekens CH, Willett WC. Body weight and longevity: a reassessment. JAMA 1987;257:353-8.
13. Troiano RP, Frongillo EA Jr, Sobal J, Levitsky DA. The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. Int J Obes Relat Metab Disord 1996;20:63-75.
14. Tuomilehto J, Salonen JT, Marti B, et al. Body weight and risk of myocardial infarction and death in the adult population of eastern Finland. BMJ 1987;295:623-7.
15. Garrison RJ, Castelli WP. Weight and thirty-year mortality of men in the Framingham Study. Ann Intern Med 1985;103:1006-9.
16. Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. N Engl J Med 1995;333:677-85.
17. Stevens J, Jianwen C, Pamuk ER, Williamson DF, Thun MJ, Wood JL. The effect of age on the association between body-mass index and mortality. N Engl J Med 1998;338:1-7.
18. Willett WC, Manson JE, Stampfer MJ, et al. Weight, weight change, and coronary heart disease in women: risk within the 'normal' weight range. JAMA 1995;273:461-5.
19. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med 1995;122:481-6.
20. Huang Z, Willett WC, Manson JE, et al. Body weight, weight change, and risk for hypertension in women. Ann Intern Med 1998;128:81-8.
21. Maclure KM, Hayes KC, Colditz GA, Stampfer MJ, Speizer FE, Willett WC. Weight, diet, and risk of symptomatic gallstones in middle-aged women. N Engl J Med 1989;321:563-9.
22. Rimm EB, Stampfer MJ, Giovannucci E, et al. Body size and fat distribution as predictors of coronary heart disease among middle-aged and older US men. Am J Epidemiol 1995;141:1117-27.
23. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Obesity,

fat distribution, and weight gain as risk factors for clinical diabetes in men. Diabetes Care 1994;17:961-9.
24. Huang Z, Hankinson SE, Colditz GA, et al. Dual effects of weight and weight gain on breast cancer risk. JAMA 1997;278:1407-11.
25. World Cancer Research Fund, American Institute for Cancer Research. Food, nutrition and the prevention of cancer: a global perspective. Washington, D.C.: American Institute for Cancer Research, 1997.
26. Walker SP, Rimm EB, Ascherio A, Kawachi I, Stampfer MJ, Willett WC. Body size and fat distribution as predictors of stroke among US men. Am J Epidemiol 1996;144:1143-50.
27. Felson DT. Weight and osteoarthritis. Am J Clin Nutr 1996;63:Suppl: 430S-432S.
28. Rich-Edwards JW, Goldman MB, Willett WC, et al. Adolescent body mass index and infertility caused by ovulatory disorders. Am J Obstet Gynecol 1994;171:171-7.
29. Meyer HE, Tverdal A, Falch JA. Body height, body mass index, and fatal hip fractures: 16 years' follow-up of 674,000 Norwegian women and men. Epidemiology 1995;6:299-305.
30. Department of Agriculture, Department of Health and Human Services. Nutrition and your health: dietary guidelines for Americans. Home and garden bulletin no. 232. Washington, D.C.: Government Printing Office, 1980.
31. *Idem*. Nutrition and your health: dietary guidelines for Americans. 2nd ed. Home and garden bulletin no. 232. Washington, D.C.: Government Printing Office, 1985.
32. Lee I-M, Paffenbarger RS Jr. Change in body weight and longevity. JAMA 1992;268:2045-9.
33. Kuller LH, St Jeor ST, Dwyer J, et al. Report of the American Institute of Nutrition (AIN) Steering Committee on Healthy Weight. Bethesda, Md.: American Institute of Nutrition, 1993.
34. Physical status: the use and interpretation of anthropometry: report of a WHO expert committee. WHO Tech Rep Ser 1995;854:1-452.
35. Obesity: preventing and managing the global epidemic: report of a WHO Consultation on Obesity, Geneva, June 3–5, 1997. Geneva: World Health Organization, 1998.
36. Harris TB, Ballard-Barbasch R, Madans J, Makuc DM, Feldman JJ. Overweight, weight loss, and risk of coronary heart disease in older women: the NHANES I Epidemiologic Follow-up Study. Am J Epidemiol 1993;137:1318-27.
37. Foster DW. Gain and loss in weight. In: Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL, eds. Harrison's principles of internal medicine. 13th ed. Vol. 1. New York: McGraw-Hill, 1994:221-3.
38. Gallagher D, Visser M, Sepulveda D, Pierson RN, Harris T, Heymsfield SB. How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups? Am J Epidemiol 1996;143:228-39.
39. Han TS, van Leer EM, Seidell JC, Lean MEJ. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. BMJ 1995;311:1401-5.
40. NHLBI Obesity Education Initiative Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. Bethesda, Md.: National Heart, Lung, and Blood Institute, 1998.
41. Carey VJ, Walters EE, Colditz GA, et al. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women: the Nurses' Health Study. Am J Epidemiol 1997;145:614-9.
42. Preventive Services Task Force. Guide to clinical preventive services. 2nd ed. Baltimore: Williams & Wilkins, 1996.
43. Goldstein DJ. Beneficial health effects of modest weight loss. Int J Obes Relat Metab Disord 1992;16:397-415.
44. Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C. Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40-64 years. Am J Epidemiol 1995;141: 1128-41. [Erratum, Am J Epidemiol 1995;142:369.]