surgery, pharmacotherapy, and psychological and familial support must be considered together as part of obesity assessment. Before beginning a weight-loss program, patients should be evaluated for number and severity of cardiovascular risk factors. These conditions may require that treatment be initiated along with weight-loss strategies.

*See also:* **Adolescents**: Nutritional Requirements. **Body Composition**. **Children**: Nutritional Requirements. **Dietary Intake Measurement**: Methodology; Validation. **Obesity**: Fat Distribution; Childhood Obesity; Complications; Prevention; Treatment.

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# Fat Distribution

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In 1956, the French physician, Jean Vague, noted that an upper body, or masculine, fat distribution was associated with adverse health consequences. It has now been clearly demonstrated that obesity-related chronic diseases are associated with the location, as well as the amount, of adipose tissue on the body. Although the relative importance of total adiposity versus type of adiposity continues to be debated, the notion that an 'apple-shaped' (or android) body is associated with greater obesity-related health risks than a 'pear-shaped' (or gynoid) body is well accepted (**Figure 1**). Imaging techniques such as computed tomography (CT) allow measurement of visceral adipose tissue and layers of subcutaneous fat. Anthropometric studies do not provide precise measures of fat depots but nevertheless have provided clues to the causes and consequences of differences in fat distribution. Guidelines are being developed for the use of anthropometric assessments of fat distribution in clinical and public health settings.

## Measurement of Fat Distribution

Fat patterning, the distribution of fat, is measured using either imaging or anthropometric

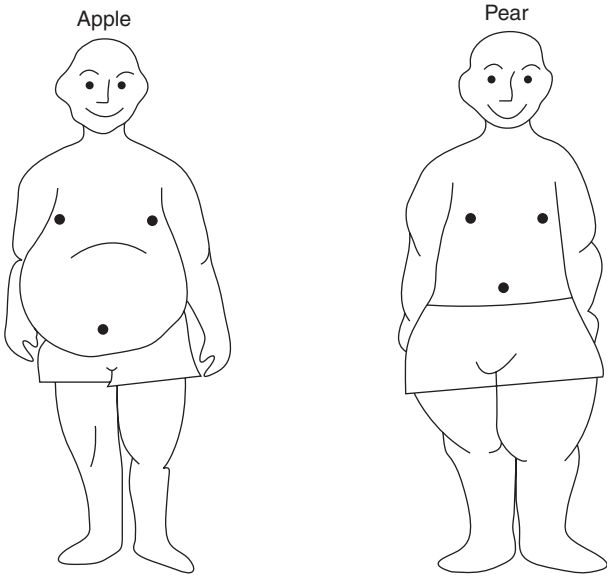


**Figure 1** The apple (android) and pear (gynoid) body shapes.

techniques. Measurement has focused on assessment and differentiation of subcutaneous and intraabdominal (visceral) depots; however, recently measurement of fat residing in muscle has become of interest. Imaging techniques have the advantage of providing separate measurements of fat in these three different depots, but they remain too expensive for use in most clinical and community settings. Anthropometric measurements cannot provide a direct assessment of the amount of fat in different depots, but they can provide variables that correlate with assessments from imagining techniques and are quick, inexpensive, and noninvasive.

### Imaging Techniques

Computed tomography and magnetic resonance imaging (MRI) are considered the most precise methods for measuring body fat distribution. MRI has the advantage of not exposing subjects to radiation. Dual energy X-ray is primarily used to measure bone mineral content and total body fat. This technique can measure total abdominal fat, but it cannot differentiate between visceral and subcutaneous fat.

**Figure 2** shows two different cross-sectional images of the abdomen obtained by MRI. These images are constructed from $256 \times 256$ pixels, which vary from white to black with different shades of gray. Each pixel represents $2.4\,mm^2$. The fat regions are depicted as the lighter portions of the images. The subcutaneous fat area delineates the perimeter of the abdomen, whereas the visceral area is contained within the subcutaneous area. **Figure 2A** represents a cross section of an abdomen with a relatively small subcutaneous fat area in comparison with an enlarged visceral fat area. **Figure 2B** shows a subject with a small visceral fat-to-subcutaneous fat depot ratio.

CT scans have shown that approximately 12% of fat in normal weight subjects is among and inside muscles. Some researchers advocate considering this fat in a separate compartment, which, from a metabolic standpoint, is more closely related to visceral fat. Some researchers have suggested that subcutaneous fat be separated into deep and superficial layers separated by the 'fascia superficialis.'

### Anthropometric Techniques

Anthropometric indices used to measure fat patterning include skinfold thicknesses, circumferences, sagittal diameter, and ratios such as waist-to-hip, waist-to-thigh, waist-to-height, and subscapular-to-triceps skinfolds. Skinfold thicknesses and skinfold ratios have not been found to be very well correlated with metabolic measurements or with visceral fat and are not recommended for use as indicators of fat patterning. Numerous equations using combinations of anthropometric measurements to predict the amount of visceral fat have not offered substantial improvement over the simpler measurements, and an accurate equation has yet to be developed.

Waist circumference (WC) alone and waist-to-hip ratio (WHR) are the most popular anthropometric methods used to measure fat distribution in both clinical and community settings. Both measures are correlated with visceral fat, with a correlation coefficient ($r$) generally ranging from 0.5 to 0.8. It is problematic that there is no uniform method of defining the location at which the waist and hip measurement should be assessed (**Table 1**). Waist

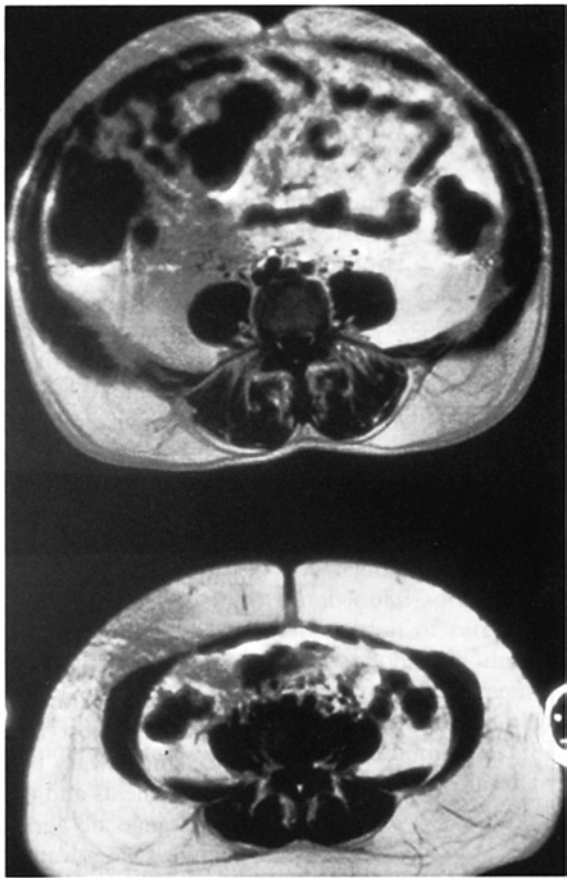


**Figure 2** Cross-sectional images of the abdomen obtained by MRI. (A) Small subcutaneous fat area and enlarged visceral fat area. (B) Small visceral fat area in comparison with subcutaneous fat depot.

**Table 1** Anatomical locations used to measure waist and hip circumferences

| |
|---|
| **Waist circumference** |
| One-third between the xyphoid process and umbilicus |
| Narrowest part of torso |
| Midway between xyphoid process and umbilicus |
| Midway between lower rib and iliac crest |
| One inch (~2.5 cm) above umbilicus |
| Level of umbilicus |
| Level of iliac crest |
| Immediately below the lowest rib |
| Immediately above the iliac crest |
| **Hip circumference** |
| Largest horizontal circumference around the buttocks |
| Level of iliac crest |
| Maximal circumference between superior border of iliac crest and thigh region 4 cm below superior iliac crest |

circumferences measured at four sites (immediately below the lowest rib, at the narrowest point, midpoint between the lowest rib and the iliac crest, and immediately above the iliac crest) have been compared and found to differ from each other. Other work has shown that the highest correlations with risk factors were obtained when WHR was calculated as the waist measured at the point midway between the lower rib margin and iliac crest (approximately 1 inch (~2.5 cm) above the umbilicus) or when the waist was measured at the umbilicus and hips measured at the widest point of the buttocks. Although two different waist measurements have been demonstrated to perform equally well, the bony landmark measurement (the point midway between the lower rib margin and iliac crest) may be preferred since the umbilicus may shift position when an individual gains or loses weight. The World Health Organization (WHO) has recommended measuring the waist at the midpoint between the lowest rib and the iliac crest, whereas immediately above the iliac crest is the site recommended by the National Institutes of Health (NIH).

Sagittal diameter, the height of the abdomen measured with the subject in the supine position, can be measured anthropometrically or by imaging. Figure 3 shows a technique for the anthropometric measurement of sagittal diameter using a caliper. Measurement is usually taken at the largest supine anteroposterior diameter between the xyphoid process and umbilicus. Some studies have found sagittal diameter to be a better indicator of visceral fat than WHR. Correlations between sagittal diameter and amount of visceral fat range from $r = 0.51$ to $r = 0.87$, with higher correlations occurring when sagittal diameter is measured using imaging techniques. In general, correlations tend to be higher in men than women.

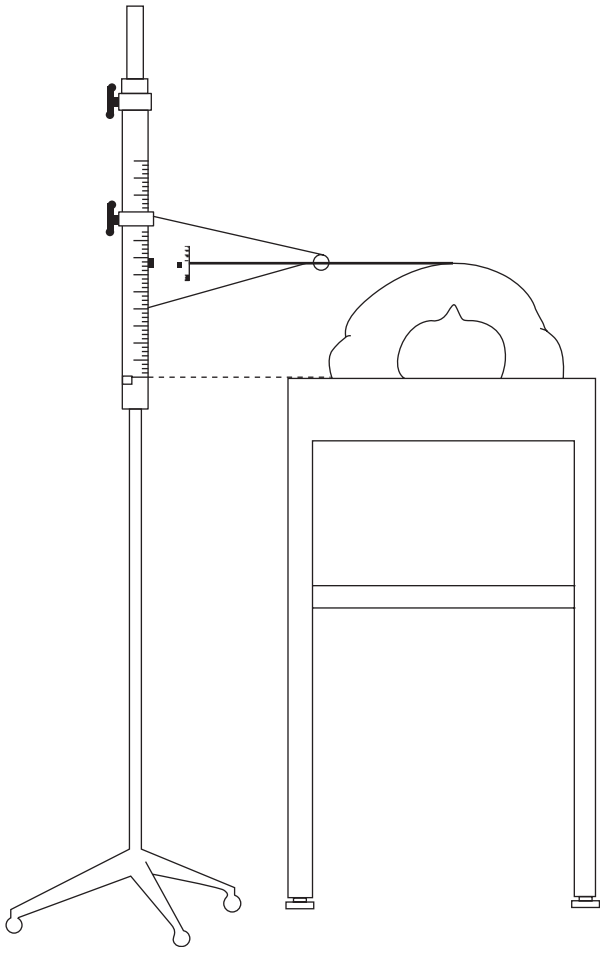

**Figure 3** Sagittal diameter measured anthropometrically using calipers.

## Metabolic Characteristics of Visceral and Subcutaneous Fat

The main function of adipose tissue is to store and break down fat based on energy excess or need, respectively. The uptake of fat is regulated by the enzyme lipoprotein lipase (LPL). This enzyme hydrolyzes triacylglycerols into free fatty acids, which can then be transported into the adipocyte and reesterified for storage. Greater LPL activity is associated with greater accumulation of fat. In premenopausal women, its activity is higher in the gluteal–femoral adipose areas than in the abdominal areas. The opposite is true in men, in whom LPL activity is the same or higher in the abdominal adipose areas than in the gluteal–femoral regions.

The breakdown of fat (lipolysis) is regulated by the enzyme hormone-sensitive lipase (HSL). This

enzyme releases free fatty acids, which are then released into the bloodstream and taken up by tissues, with the exception of the brain and red blood cells, for energy use or storage. The rate of basal lipolysis is higher in gluteal–femoral fat tissue than in abdominal tissue in both men and women. This may be due to greater cell size in that region. In the abdominal area, basal lipolysis is higher in subcutaneous fat than in visceral fat. However, when stimulated hormonally, rates of lipolysis may differ between men and women. Lipolytic rates have been shown to be higher in the visceral compared to the subcutaneous region in men, whereas the opposite trend is seen in women.

### Regulators of Lipolysis and Fat Storage

The processes of lipolysis and fat storage are regulated by hormonal factors, which either enhance or suppress the activities of HSL and LPL. Through the action of glucocorticoid receptors, glucocorticoids enhance LPL activity and promote abdominal deposition of fat. The density of glucocorticoid receptors is greater in the visceral abdominal depot than in the subcutaneous abdominal depot. Therefore, an increase in glucocorticoid secretion is associated with increases in abdominal fat deposition compared to other fat depots.

Insulin favors fat storage by increasing LPL and decreasing HSL activity. Insulin has stronger antilypolytic effects in adipose located in the abdominal region compared to the femoral regions in both men and women. Paradoxically, insulin binding is stronger in the gluteal–femoral region than the abdominal region. Therefore, it has been hypothesized that insulin regulates lipolysis at the postreceptor level.

Catecholamines regulate lipolysis through $\alpha_2$- and $\beta$-adrenoreceptors. The $\beta$-adrenoreceptors increase lipolysis, whereas the $\alpha_2$-adrenoreceptor inhibits it. Although both the $\alpha_2$- and the $\beta$-adrenoreceptors coexist in adipose tissue, they are regionally specific such that there may be an excess of one type of receptor relative to the other in various adipose regions. The lipolytic effect of catecholamines is 10–20 times greater in the abdominal region than in the gluteal–femoral region, as marked by a twofold increase in the number of $\beta$-adrenoreceptors in both sexes. The lipolytic action of catecholamines is more pronounced intraabdominally than in the abdominal subcutaneous tissue. Sex differences are displayed with the $\alpha$-adrenoreceptor. Although the number of receptors is similar in both sexes, the sensitivity of the receptors is reduced by a factor of 10–15 in the abdominal compared with the gluteal–femoral region.

Sex hormones, such as estrogen, testosterone, and progesterone, also affect the balance of fat accumulation/mobilization, although their effects vary in men and women and the mechanisms are not clearly understood. Studies show that estrogen decreases LPL expression and activity in adipose tissue. It has been shown that testosterone stimulates lipolysis by increasing the number of $\beta$-adrenoreceptors. Estrogen and progesterone, on the other hand, stimulate fat storage and inhibit lipolysis, preferentially in the gluteal–femoral area compared to the abdominal area.

An increased androgenic profile is associated with upper body fat accumulation in women, but studies on men are conflicting. Significant inverse associations between fat distribution and testosterone have been found in population studies on men. Reduced visceral fat has also been observed when testosterone treatment was administered to men. These findings challenge the hypothesis that an androgenic hormone profile contributes to a more 'male type' of fat pattern and the associated metabolic sequelae.

The controversy over the effect of sex hormones on fat distribution is complicated by the metabolism of sex hormones. Sex hormone-binding globulin (SHBG) binds circulating testosterone and estrogen. Decreased SHBG concentration may be associated with an android shape. Therefore, studies need to distinguish between total circulating and unbound sex hormones and SHBG.

### Sequelae of Altered Metabolism in Visceral Fat

Intraabdominal adipose tissue has metabolic characteristics that are different from those of adipose tissue from other sites. These differences seem to be most pronounced in the regions that are drained by the portal circulation. These 'portal adipose tissues' have a sensitive system for the mobilization of free fatty acids due to a preponderance of $\beta$-adrenergic receptors and little $\alpha$-adrenergic inhibition.

The hypothesis has been advanced that the heightened responsiveness of intraabdominal fat to lipolytic agents results in increased lipolysis with venous drainage of the released free fatty acids directly to the liver. These fatty acids may contribute to increases in triacylglycerol synthesis and hyperinsulinemia secondary to decrements in insulin degradation. Hyperinsulinemia could produce insulin resistance and eventually type 2 diabetes in susceptible individuals. However, the hypothesis that increased release of free fatty acids from intraabdominal adipose tissue leads to insulin resistance through effects on the liver lacks supporting evidence *in vivo*.

The proposed mechanism of action of fat patterning on metabolic syndrome is linked to

hyperinsulinemia. Hyperinsulinemia may lead to increased blood pressure through increased sympathetic stimulation of the vessels, heart, and kidneys. In addition, insulin resistance combined with a relative increase in androgenic activity may lead to an unfavorable lipid profile. In addition to the effects of free fatty acids on insulin and glucose, an increased visceral depot decreases the activity of LPL. This causes an increase in very low-density lipoprotein (VLDL) secretion and a decrease in its catabolism. The production of high-density lipoprotein (HDL) therefore decreases, the transfer of lipids (i.e., VLDL to LDL and HDL) increases, and an enrichment of triacylglycerols results.

In obesity and type 2 diabetes, there is an increased content of lipids within and around muscle fibers. Researchers have suggested that the accumulation of triaclyglycerols within the skeletal muscle may play an important role in insulin resistance. In obese individuals with elevated amounts of visceral adipose tissue, there is a strong correlation between visceral adipose tissue and insulin resistance independent of subcutaneous (abdominal and nonabdominal) adipose tissue and cardiovascular fitness. It has been suggested that the discrepancies in the literature regarding the independent effect of visceral or subcutaneous adipose tissue on insulin resistance are due to the large variations of abdominal obesity within the study populations.

Leptin is a hormone that is produced in the adipose cells and can act on the hunger center in the hypothalamus to reduce hunger and appetite and thereby lower food intake. Plasma leptin levels are correlated with body fat. Researchers have discovered a leptin receptor gene that is responsible for obesity due to the mutation or absence of the gene. This condition is extremely rare in humans. In general, in obese humans the leptin levels are elevated (hyperleptinemia).

There is a progressive increase in plasma levels during puberty in girls due to the increase in body fat during this period and in response to the effect of estrogens. Circulating leptin levels tend to decrease in response to testosterone in boys, thus resulting in higher plasma leptin levels in women compared to men. Leptin levels are also affected by insulin and glucocorticoids.

## Correlates and Possible Determinants of Fat Distribution

A large number of studies have examined correlations between fat distribution and genetic, behavioral, and physiological variables. Many factors, including heredity, overall fatness, gender, age, smoking, alcohol consumption, physical activity, and ethnicity, are associated with either an android or a gynoid shape. The underlying reasons for the observed associations between these variables and fat patterning remain to be elucidated. Correlates of fat distribution are important to understand since they may confound relationships between fat patterning and physiological outcomes or morbidity or mortality outcomes. There is evidence that body shape and amount of visceral fat are partially determined by genetics. After eliminating effects of age and overall fatness, studies have shown that heritable factors can account for as much as 20–50% of the variability in waist-to-hip ratio.

Fat distribution becomes more central or android as overall fatness increases. The correlation between overall fatness and fat patterning indices ranges from $r = 0.5$ to 0.9, depending on which measure of fat patterning is used. The more obese an individual, the more difficult it is to measure the waist and hip circumferences and the higher the measurement error.

Fat distribution has long been known to vary by gender, with men more android (apple shaped) than women, who are more gynoid (pear shaped). Men have a higher WHR and significantly more intra-abdominal adipose tissue than women. During weight gain in normal weight men, fat is preferentially deposited abdominally in the subcutaneous and visceral regions—proportionately more in the upper compared to the lower abdomen. In men, little fat is deposited in the gluteal–femoral regions until they become obese. Women have a higher percentage of body fat and higher proportion of fat in the gluteal–femoral regions than men. The gender differences are sufficiently large that recommended cutpoints for indices of fat distribution must be gender specific.

Aging is accompanied by changes in both weight and fat distribution. The largest increase in body weight occurs between young adulthood and middle adulthood. Independent of weight gain, abdominal fat increases with aging. This increase tends to be most pronounced between young adulthood and middle age in men and between middle age and old age in women (related to menopausal status).

Although cigarette smokers tend to be leaner than nonsmokers, they have more central adiposity (as indicated by larger waist circumference and WHRs) compared to nonsmokers, after the effects of age and body mass index (BMI) are eliminated. Furthermore, WHR increases progressively with an increase in the number of cigarettes smoked daily. The WHR increases with increasing 24-h cotinine excretion, indicating that central fat accumulation is dependent on the dose of smoke inhaled. Some studies have found a more androgenic hormone profile

in cigarette smokers, although this finding has been inconsistent. Increased cortisol secretion, an endocrine response to stressors associated with upper body fat deposition, may explain some of the association between smoking, alcohol consumption, and fat distribution.

Although alcohol consumption has been postulated to be correlated with fat distribution, studies have been inconclusive. There is evidence that beer and spirits are associated with higher levels of WHR, whereas wine is not. Not only frequency but also intensity of alcohol consumption may be important. One study showed that frequency of alcohol consumption was inversely associated, but intensity was positively associated, with abdominal adiposity (measured by sagittal diameter), even after the effects of age, education, physical activity, smoking, and grams of alcohol had been controlled. After combining the effects of frequency and intensity, the high frequency (daily) but low intensity (<1 drink/day) group had the lowest sagittal diameter, whereas the low frequency (<weekly) but high intensity (>3 drinks/day) group had the largest sagittal diameter.

Physical activity is inversely correlated with fat distribution in both men and women. Negative associations exist between WHR and various sports and exercise indices after controlling for the effects of BMI, smoking, and education. There is evidence that activity may be associated with a preferential mobilization of abdominal fat. Endurance training has been shown to increase aerobic fitness and decrease body mass and fat mass. Resistance training results in an increase in fat-free mass and muscle strength.

Fat distribution varies by ethnicity. African Americans have less visceral fat than whites at the same BMI, whereas Asians have a larger percentage of visceral fat. At the same BMI, African Americans have greater bone density and muscle mass than whites. Asians have smaller body frames, less muscle mass, and a larger percentage of fat mass at the same BMI as African Americans and whites. A study comparing migrant and British-born South Asian women to a general population of women in Scotland found that after controlling for the effect of age, migrant South Asians had larger waist circumference and WHR. However, after also controlling for physical activity, cigarette smoking, alcohol consumption, and parity, only WHR remained different between the two groups.

## Fat Distribution and Disease Risk

Numerous studies have examined associations between fat patterning and mortality and morbidity. Since fat distribution is correlated with age as well other risk factors for disease, such as smoking, alcohol consumption, physical activity, and menopause in women, it is important to control for the effects of these variables in order to obtain an estimate of the independent effect of central obesity on morbidity. The impact of some of these correlates of fat distribution may be subtle and unlikely to seriously distort relationships between fat patterning and disease. However, age, the ultimate risk factor for disease and death, is sufficiently highly correlated with fat distribution to result in substantial distortion. Similarly, cigarette smoking is related adequately strongly to fat patterning and to various diseases and outcomes to make analyses that do not adjust for smoking difficult to interpret.

The large correlation between fat patterning and overall adiposity also influences the interpretation of results, making it difficult to differentiate between the two effects. Some researchers compare the size of the correlation between fat distribution (usually measured as WHR) and total adiposity (usually measured as BMI) in an attempt to show the relative importance of each. Others examine effects within tertiles (or other categories) of BMI and WHR simultaneously or test for an independent effect of WHR or BMI in multiple regression models that include both variables. In the latter type of analysis, the associations of both WHR and BMI with an outcome can be greatly reduced or even disappear because of collinearity between the two measures.

Researchers have found positive correlations between fasting glucose, insulin, blood pressure, total cholesterol, LDL cholesterol, and triaclyglycerols using imaging techniques, sagittal diameter, waist circumference, and WHR in most, but not all, studies. Visceral fat and HDL cholesterol are inversely associated. The strength of the associations varies but tends to be largest for triaclyglycerols. Associations are reduced after controlling for BMI and age.

There is strong evidence to link waist circumference and WHR with the risk of developing type 2 diabetes, even after eliminating the effects of age, smoking, BMI, and other important correlates. An individual who is obese (>150% ideal body weight) and has an elevated WHR (>0.8) may have as much as a 10-fold increased risk for developing type 2 diabetes compared with an individual who is of normal weight (<120% ideal body weight) and has a low WHR (<0.72).

Elevated WHR has been positively associated with cardiovascular disease in some population studies, although not as consistently as diabetes. Scientists have recognized that several of the cardiovascular

disease risk factors, including abdominal obesity, cluster in individuals. This cluster of risk factors is referred to as metabolic syndrome. The other risk factors in metabolic syndrome are insulin resistance/glucose intolerance, dyslipidemia (high triaclyglycerols and low HDL cholesterol), and high blood pressure.

## Applications

Waist circumference can be used to assess obesity-related health risks in public health and clinical settings. Because it consists of only one measurement instead of two, it introduces less measurement error than WHR. A large waist has been shown to reflect both generalized obesity and centralized body fat distribution, which suggests that waist circumference could replace both BMI and WHR as a simple indicator of the need for weight management. Also, waist circumference tends to be more highly correlated with visceral fat than WHR.

It has been shown that hip circumference alone is inversely associated with cardiovascular disease risk after controlling for age, BMI, smoking, and waist circumference. Therefore, some predictive information may be lost if hip circumference is not assessed. If an index of body shape, independent of total body fatness, is desired the WHR may be preferred over waist alone because it is less highly correlated with total adiposity. Waist-to-hip ratio is a widely accepted form of fat patterning assessment. It is a good predictor of disease and metabolic disorders, with an increasing WHR indicating increased risk. Cutpoints used to define elevated WHR range from 0.90–1.00 in men to 0.80–0.90 in women.

Guidelines for the use of waist circumference in combination with BMI have been issued by NIH and WHO. NIH guidelines use BMI cutoffs for an initial assessment of overweight and obesity and recommend waist circumference cutoffs as a supplementary indicator of health risk. Increased relative risk for the development of obesity-associated risk factors in most adults is predicted for adults within the BMI range of 25–35 when the waist is $\geq$102 cm (40 in.) in men and $\geq$88 cm (35 in.) in women.

## Conclusions

The relationship of body fat distribution to metabolic abnormalities and disease has now been well recognized. Individuals with a more android than gynoid body shape tend to have a more adverse metabolic profile and an increased risk for type 2 diabetes and cardiovascular disease.

Although much has been learned about fat distribution, there are several issues that need further exploration. The question of whether body type can be changed through behavior needs to be more fully addressed. Standardized anthropometric measurements need to be established, and the implications of differences in fat distribution among ethnic groups need to be elucidated. A better understanding of the risks associated with fat residing in muscle is needed, and this information must be integrated with what is known about visceral and subcutaneous fat. Finally, more research is needed to identify mechanisms of action. An increased abdominal depot may not necessarily be the cause of metabolic disturbances but an effect of underlying genetic and endocrine abnormalities.

*See also:* **Adipose Tissue**. **Body Composition**. **Diabetes Mellitus**: Etiology and Epidemiology; Classification and Chemical Pathology. **Exercise**: Beneficial Effects. **Hyperlipidemia**: Overview; Nutritional Management. **Lipids**: Chemistry and Classification. **Obesity**: Definition, Etiology and Assessment.

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# Childhood Obesity

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This article discusses obesity in children and adolescents with regard to prevalence, epidemiology, clinical features, and management/prevention. Obesity not associated with a recognized underlying clinical condition is the focus of this article since this represents far the majority of children with obesity. However, obesity associated with congenital or acquired medical conditions is discussed briefly. Management and prevention are also discussed.

Obesity is increasing in prevalence among children in virtually all developed countries. In the United Kingdom, 8.5% (twice the rate of 10 years ago) of 6-year-old and 15% (three times the rate of 10 years ago) of 15-year-old children are obese. Childhood obesity is also increasing in prevalence among the affluent in less well-developed countries. Since it has been estimated that in Western countries one-third of obese adults were obese in childhood, and since both adult and adolescent obesity carry significant risk of health complications, obesity in childhood is currently seen as a concern for families, communities, and nations.

## Body Composition in Childhood and Definition of Childhood Obesity

Obesity is an excess of body fat. However, the percentage of body weight that is fat varies normally throughout childhood (Table 1). The infant is born with modest amounts of fat. More than 50% of the energy in breast milk comes from fat, and young infants lay down fat very rapidly so that in the 4 or 5 months that it takes a normal infant to double birth weight, the weight of fat in the body has tripled. By 6 months of age, infants are increasing weight-bearing activity and fat deposition slows relative to lean tissue growth. From 1 year onward, there is a natural process of slimming with less fat than lean tissue deposited so that the child of 5 years often has a lower percentage body weight as fat than at any other time in life. This is followed by the 'adiposity rebound,' when fat deposition accelerates only to slow again with the onset of the pubertal growth spurt in males. In pubertal girls, very brief slimming early in the female growth spurt is followed by vigorous fat deposition particularly around the breasts and hips.

### Assessment of Overweight and Obesity in Childhood

Precise methods of estimating body fat are complicated and expensive. There are no accepted age-related 'norms' for percentage body weight as fat in childhood. For these reasons, anthropometric indices involving weight and height are widely used to estimate relative fatness. Such methods are relatively simple, noninvasive, well tolerated, and can be used in clinical practice and large population studies. However, they provide only indirect measures of fatness.

Weight can be related to height and age in various ways. Until recently, there was no consensus

**Table 1** Percentage of body weight as fat at different ages in childhood

| *Age (years)* | *% body weight as fat* | |
|---|---|---|
| Birth | 11[a] | |
| 0.3 | 25[a] | |
| 1.0 | 24[a] | |
| | *Males* | *Females* |
| 5 | 12.5[b] | 15.3[b] |
| 10 | 17.6[b] | 16.0[b] |
| 15 | 11.4[b] | 23.3[b] |

[a]Fomon SJ (1974) *Infant Nutrition. 2nd edition*, Philadelphia: WB Saunders, p. 69.

[b]Widdowson EM (1974) *Changes in body proportions and compositions during growth.* In Davis JA, Dobbing J. *Scientific Foundations of Paediatrics.* London: Wm Heinemann. pp. 152–63.