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Obesity: criteria and classification*

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Obesity is defined as an excess accumulation of body fat. To measure fat in the body accurately is difficult, and no method is easily available for routine clinical use. Traditionally, overweight and obesity have been evaluated by anthropometric measurement of weight-for-height. More recently, BMI has been used. The normal range is 19–24.9 kg/m², overweight is 25–29.9 kg/m², and obesity ≥ 30 kg/m². Not only is the total amount of fat an individual carries important, but also where the fat is distributed in the body. Fat in a central or upper body (android) distribution is most related to health risk. The most accurate way to measure central obesity is by magnetic resonance imaging or computer-assisted tomography scanning, but this approach is too expensive for routine use. Simple anthropometric measurements can be used, such as waist circumference. A waist circumference of greater than 1020 mm in men and 880 mm in women is a risk factor for insulin resistance, diabetes mellitus and cardiovascular disease. There is a clear genetic predisposition for obesity. The genetic contribution to obesity is between 25 and 40 % of the individual differences in BMI. For the overwhelming majority of individuals, the genetic predisposition will not be defined by one gene, but by multiple genes. Eventually, classification of obesity may be done by genetic means, but this approach will require more knowledge.

Obesity: Body fat: Genetic predisposition

Obesity is defined as an excess accumulation of body fat. In the young adult normal levels of body fat are considered to be 12–20 % body weight in males and 20–30 % body weight in females, while levels of >25 % body weight in males and >33 % body weight in females can be considered obese (Table 1; Bray 1998). However, as individuals age there is an accretion of fat at the expense of lean body mass, so that at older ages the percentage of the body that is fat is considerably higher, even in individuals who do not gain

weight. Fig. 1 shows values for patients of differing ages studied in our body composition laboratory (Gallagher *et al.* 1996).

Anthropometry

To measure fat in the body is difficult and expensive, and no accurate method is easily available for routine clinical use. Traditionally, overweight and obesity have been evaluated by anthropometric measurement of weight and height. Weight-for-height has been compared with a standard population of young adults (up to 85th percentile being considered normal; Najjar & Rowland, 1987), or to life tables from the insurance industry (Metropolitan Life Insurance Company, 1983). While these values are helpful for assessing population groups, they are much less useful

Table 1. Body fat levels of men and women (from Bray, 1998)

	Body fat %		
	Normal	Overweight	Obese
Men	15–22	> 22	> 25
Women	18–32	> 32	> 35

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for assessing an individual, since the variability around the mean is very high.

Other anthropometric measures that have been used are skinfold thickness at various points in the body. These measures have been placed in published equations and converted to quantity of body fat (Durnin & Womersley, 1974; Lohman, 1981). The measurement of skinfold thickness becomes more inaccurate as obesity increases, and also requires training and experience. As a result, it is not a popular or generally a very accurate measure.

Most recently, the BMI has been utilized for an assessment of fatness. BMI is the weight (kg) divided by the height squared (m^2 ; Quetelet, 1871; Garrow & Webster, 1985). By agreement of a number of voluntary and governmental health agencies, the normal range has been set at 19–24.9 kg/m^2 , overweight at 25–29.9 kg/m^2 , and obese at $\geq 30 kg/m^2$ (World Health Organization, 1997; National Institutes of Health, 1998). In those subjects with a BMI $> 30 kg/m^2$ a subclassification is recommended, as shown in Table 2. BMI, however, is also an imperfect measure of fatness. It is better for assessing a group of individuals than for following a particular individual clinically. The correlation of BMI with percentage body fat is quite good (Zumoff *et al.* 1990; Gallagher *et al.* 1996). However, there can be very muscular individuals, such as athletes, who may have a higher than normal BMI and yet have normal (or low) percentage body fat (Segal *et al.* 1987). In addition, BMI is less accurate for estimating body fatness in very short individuals, $< 1.52 m$ in height. BMI, however, is a very useful epidemiological tool, and increasing BMI has been well correlated with increasing morbidity and mortality in numerous epidemiological studies (Lindsted

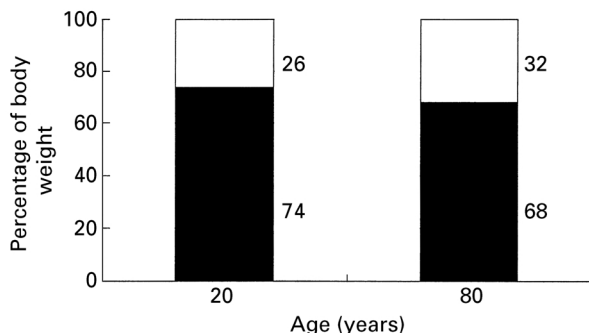


Fig. 1. Average body composition for a female subject aged 20 years and a female subject aged 80 years (BMI 23 kg/m^2), derived from data on 394 women of age 20→80 years measured by underwater weighing. (□), Fat; (■), lean body mass. (Adapted from Gallagher *et al.* 1996.)

Table 2. National Institutes of Health (1998) classification of overweight and obesity

	BMI (kg/m^2)
Normal range	18.5–24.9
Overweight	25.0–24.9
Grade I obesity	30.0–34.9
Grade II obesity	35.0–39.9
Grade III obesity	≥ 40.0

et al. 1991; Manson *et al.* 1995; Troiano *et al.* 1996; Allison *et al.* 1999b). A characteristic formula is shown in Table 3.

Other methods of assessing body composition

Other methods of assessing body composition are listed in Table 4, with the level of accuracy. There are two instrumental methods for assessing fatness available that are more accessible to clinicians. These are bioelectrical impedance and dual-energy X-ray absorptiometry. Bioelectrical impedance measures the impedance of the body using an electrode on one leg and another electrode on the arm or the other leg. The method actually measures body water, which is then used in an equation to estimate fat-free mass, and subtracting this value from weight gives body fat (Segal *et al.* 1985). Since this method measures water, it is inaccurate in individuals with oedema or electrolyte disturbances. Changes in ambient temperature and in the placement of the electrodes can also change the results obtained. There are a number of different instruments available for measuring fatness by bioelectrical impedance, and some instruments are much superior to others in accuracy and reliability. Finally, specific standards derived in a given population need to be used when a measurement is made in an individual from that population.

Dual-energy X-ray absorptiometry is done by passing two differing very-low-energy X-ray beams through the

Table 3. Calculation of percentage body fat from BMI (from Gallagher *et al.* 1996)

Subjects	<i>n</i>	Equation	R^2	SE*	Statistical significance: <i>P</i>
Men	214	$1.402 \times BMI + 0.177$ $\times age - 22.519$	0.52	5.54	< 0.001
Women	290	$1.592 \times BMI + 0.096$ $\times age - 11.666$	0.56	5.75	< 0.001

*Standard error of model.

Table 4. Methods of estimation of body fat and its distribution

Method	Accuracy	Measures regional fat
Height and weight	High	No
Skinfolds	Low	Yes
Circumferences	Moderate	Yes
Ultrasound 40	Moderate	Yes
Density		
Immersion	High	No
Plethysmograph	High	No
Heavy water		
3H -labelled	High	No
2H_2O , or heavy oxygen	High	No
K isotope (^{40}K)	High	No
Total body electrical conductivity	High	No
Bioelectric impedance	High	No
Fat-soluble gas	High	No
Absorptiometry (dual-energy X-ray absorptiometry, dual-photon absorptiometry)	High	Yes
Computed tomography	High	Yes
Magnetic resonance imaging	High	Yes
Neutron activation	High	No

body. The beams are attenuated to differing degrees by tissues with different density, so that a differentiation can be made between lean body mass, fat and mineral (Gotfredsen *et al.* 1986; Goodsit, 1992; Mazess *et al.* 1992). This method, using standards derived from the same population as the individual being measured, is very accurate. However, the accuracy decreases in individuals weighing > 100 kg, and as individuals get fatter they cannot be accurately scanned, because they do not fit on the examining tables presently being manufactured.

Other methods are available, but not really readily accessible to clinicians. These methods include total body ⁴⁰K measurement in a whole-body counter (Pierson *et al.* 1982), total body water by isotope dilution (Schoeller, 1996), neutron activation (Dilmanian *et al.* 1990), and air-displacement plethysmography (Nuñez *et al.* 1999; see Table 4).

Body fat distribution

Not only is the total amount of fat that an individual carries important, but also where the fat is distributed in the body. Numerous studies have shown that fat in a central or upper body (android) distribution is most related to health risk (Larsson *et al.* 1984; Ohlson *et al.* 1985; Seidell *et al.* 1987). The health risks that are particularly evident with central obesity are insulin resistance with glucose intolerance, hyperinsulinaemia, diabetes mellitus, high blood pressure, dyslipidaemia and cardiovascular disease (Pi-Sunyer, 1993). There is risk from central obesity even when BMI is not markedly increased (Ohlson *et al.* 1985).

While the best and most accurate way to measure central obesity is by magnetic resonance imaging (Albu *et al.* 1997) or computer-assisted tomography scanning (Abate *et al.* 1996), this approach is too expensive for routine use. As a result, simple anthropometric measurements can be used. The waist circumference:hip circumference has been widely used, particularly in epidemiological studies. The rationale behind this ratio is that the hips are relatively constant, while the waist increases more with accretion of central fat. In fact, however, the hips are a problematical measurement. For instance, in African-Americans, the hips are smaller than those in Caucasian-Americans, making for a higher waist:hip ratio for a given amount of central fat (Marcus *et al.* 1998). As a result of differences like these in various racial and ethnic groups, the waist:hip ratio has fallen into some disfavour.

The waist circumference alone is often used, and is now considered superior as a reflection of central fatness than the waist:hip ratio (Lemieux *et al.* 1996). Abnormally high values are >1020 mm for men and >880 mm for women (World Health Organization, 1997; National Institutes of Health, 1998). These values should be considered a risk factor for insulin resistance, diabetes mellitus and cardiovascular disease. In fact, in some population groups abdominal obesity is a better marker of metabolic and cardiovascular risk factors (Després *et al.* 1989; Haffner *et al.* 1991; Fujimoto *et al.* 1995). Some caution is necessary with regard to following a patient by measures of fat distribution. The propensity to central fatness is greater in men than women. This factor is partly related to the influence of testosterone,

but other unknown factors may come into play. Stress and hypothalamic-pituitary imbalance has been suggested (Björntorp, 1993). Central fatness also increases with age (Marcus *et al.* 1998). In addition, after the menopause women tend to put on fat more centrally. Oestrogen levels clearly protect them from this accretion in the menstrual years, since women who take hormone-replacement therapy after menopause have less central fat accumulation (Haarbo *et al.* 1985).

Aetiology

The aetiology of most human obesity is not clearly known. There are a number of neuro-endocrine disorders that have been described as causing obesity, and these disorders must be ruled out in patients presenting for treatment. This screening process can be usually done by taking a careful medical history and doing a good physical examination. Injury to the hypothalamus by trauma, tumour, surgical damage or inflammatory disease can cause uncontrolled hyperphagia and severe obesity (Bray & Gallagher, 1975). The most common endocrinological cause of obesity, with a predominance of central fat distribution, is Cushing's syndrome. While hypothyroidism is often invoked as a cause of obesity, the fat gain with this condition is usually minor. Polycystic ovarian syndrome is often associated with obesity, but has not been shown to be an aetiological factor. Drug-induced weight gain is common. Drugs associated with weight gain include antipsychotics (Allison *et al.* 1999a) and antidepressants (Lamberti *et al.* 1992; Stanton, 1995), steroids and anti-diabetic drugs, including insulin (UK Prospective Diabetes Study Group, 1998). A very common cause of weight gain is cessation of smoking (Flegal *et al.* 1995).

A clear genetic predisposition for obesity has been found. Recent studies, done with the collaboration of obese patients, their parents, siblings and spouses, suggest that the genetic contribution to obesity is between 25 and 40 % of the individual differences in BMI (Bouchard, 1994). While there are a number of single gene mutations in mice and rats, there have been very few patients described to date with obesity due to the mutation of a single gene. These single mutations include a mutation of the leptin gene (Montague *et al.* 1997), or of the leptin receptor (Clément *et al.* 1998). Also a mutation of peroxisome proliferator-activated receptor γ has been described leading to obesity (Ristow *et al.* 1998). In addition, there has been some suggestive evidence that in the β 3-adrenergic receptor, a mutation at W64R changing tryptophan into arginine, may be related to a propensity to obesity (Walston *et al.* 1995) and that a mutation of uncoupling protein 3 also leads to obesity (Argyropoulos *et al.* 1998). It is clear that for most patients the genetic predisposition will not be defined by one gene but by multiple genes and their interaction with the environment. There are many candidate genes at the present time, both for body fatness and also for fat distribution (Bouchard, 1994). Eventually, classification of obesity may be done by genetic means, but this approach will require much more knowledge of the human genome and its significant mutations than is now available. Also, the response of the obese phenotype to environmental change

depends on the genotype of an individual. This factor greatly complicates the predictability of fatness and fat distribution in relation to genotypic make-up.

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