Rev Biomed 1999; 10:33-55.

Symposium on obesity (First Part).

Symposium

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1) INTRODUCTION.

RA Bastarrachea-Sosa.

Obesity is a major health problem throughout the world. It is a complex, multifactorial disease with an increasing prevalence (1). Obesity is the most common nutritional disorder in the developed world and is associated with significant chronic diseases (hypertension, non insulin dependant diabetes mellitus, hypercholesterolemia) as well as stroke, sleep apnea, joint diseases and certain cancers (2). Obesity is a significant cause of morbidity and is having an increasing negative impact on the health care systems in both the developed and the developing world. Although treatment (e.g. diet, exercise, drugs) is available and most people can achieve significant medical weight loss (5 - 10% initial body weight), the long term maintenance of that weight loss is, unfortunately, very rare. Thus, obesity remains a poorly managed medical condition that is a major cause of morbidity and mortality (3, 9).

The problem of obesity is relatively easy formulated; simply stated, obesity is the result of a positive energy balance resulting from an increased ratio of caloric intake to energy expenditure. However, the origins of obesity are poorly understood. Prejudices that the condition is due to slothfulness, gluttony and lack of will power remain prevalent even among the medical profession (4). However recent work has identified important genetic (5) and environmental etiological factors (6). The relative importance of these newly elucidated genetic factors is currently an issue of intense speculation. Bearing in mind these recent findings, another important observation which continues to defy explanation is the very specific population distribution of the condition. In numerous studies it has been confirmed that the

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Received November 10, 1998; Accepted December 4, 1998.
This paper is also available at http://www.uady.mx/~biomedic/rb991016.html

highest levels of obesity are to be found in the lowest socio-economic groups, particularly among immigrants and aboriginal people living in Western societies (7).

Several recent sudies have demonstrated that obesity is, at its basis, a disease of biological dysregulation. That is, the steady-state body weight of an individual is taught to result from an integration of multiple biological factors, which are at least partially genetically determined. Perturbations in body weight in both directions (increased, dicreased) from this steady-state weight are resisted and corrected by robust physiological mechanisms in laboratory rodents and humans (8). The physiological mechanisms that resist changes in body fat content are responsible for the unfortunate and very frustrating weight regain that usually follows weight loss (9).

Obesity has rapidly become a major health issue in modern society due to the growing awareness of the magnified risks of developing diabetes, cardiovascular disease, dyslipidemia, and a number of other chronic diseases in overweight individuals. These risks have highlighted the need for increased medical intervention in the management of obesity. This simposyum has been prepared by interested physicians, specialists and obesity researchers internationally considered experts in every topic presented, and comprises of a number of sections addressing obesity and the medical or scientific issues associated with it. Each issue is designed to be an easily accesible yet fully referenced section dedicated to a key topic area.

Most of the material included in this symposium is drawn from the proceedings and presentations given at the international meeting called "Etiology, pathogenesis, prevention and treatment of human obesity", a continuing medical education (CME) course, presented in Merida, Yucatan, Mexico on November 7 - 8, 1997, jointly sponsored by the North American Association for the Study of Obesity (NAASO) and the American Society for Clinical Nutrition.

This symposium examines the genetic and environmental influences that affect the development of obesity, its association with other morbidities as well as options for its future management and the metabolic and regulatory events affecting energy balance.

2) THE GENETICS OF HUMAN OBESITY

C Bouchard.

Scientists involved in the study of the causes of human obesity have become optimistic about the possibility of identifying the genes associated with the predisposition to this disease. There are good reasons to believe that this new enthousiasm is justified. Our growing understanding of the human genome, the high degree of homology between humans and common laboratory mammal models for a large number of genes and chromosomal regions, and the availability of a whole variety of technologies and tools to study and manipulate DNA in the laboratory are among the most important reasons for the present level of hope in the obesity research community.

Heritability level. The level of heritability has been considered in a large number of twin, adoption and family studies. The level of heritability is simply the fraction of the population variation in a trait (e.g. BMI) that can be explained by genetic transmission. Results obtained by a good number of investigators indicate that the heritability level estimates depend on how the study was conducted and on the kinds of relatives upon which they were based. Recent studies incorporating individuals with a wide range of BMI together with information obtained on their parents, siblings and spouses suggest that the genetic contribution to obesity may be around 25 to 40 percent of the individual differences in body mass or body fat (2).

The single gene hypothesis. It is commonly observed that severely or morbidly obese persons are, on the average, about 10 to 12 BMI units heavier than their parents, brothers or sisters. Several studies have reported that a single major

gene for high body mass was segregating from the parents to their children. However, three studies did not find support for Mendelian transmission unless age and/or gender variations in the major gene were taken into account. From this small body of data, the trend seems to be for a major recessive gene accounting for about 20 to 25 percent of the variance, but with age-associated effects, with a gene frequency of about 0.2 to 0.3 (2). These results must be viewed with great caution as they are based only on the unmeasured genotype approach and the gene(s) has(have) not been identified yet.

The risk of becoming obese. A number of studies have reported that obese children had frequently obese parents. Thus, in about 30 percent of the cases, both parents of obese children are obese, with a range in frequency of about 5 to 45 percent. It has also been estimated that about 25 to 35 percent of the obese cases occur in families with normal weight parents despite the fact that the risk of becoming obese is higher if the person had obese parents. The level of risk (the so-called lR value) for a first-degree relative of an overweight, a moderately obese or a severely obese person in comparison to the population prevalence of the condition reaches about 2 to 3 (1).

Gene-energy balance interaction effects. It is generally recognized that there are some individuals who are prone to excessive accumulation of fat and for whom losing weight represents a continuous battle. On the other hand, there are others who seem relatively well protected against such a menace. In order to examine whether such differences could be accounted for by genetic factors, differences in the sensitivity of individuals to gain body weight or fat when chronically exposed to positive energy balance or to lose weight or fat under prolonged negative energy balance conditions, and the dependence or independence of such differences on the genotype were considered. (3).

Twelve pairs of male identical twins who consumed a caloric surplus of 1000 kcal per day, 6 days per week, for 100 days, were studied (total surplus of 84,000 kcal). Significant increases in body weight and fat mass were observed after the overfeeding. There were considerable interindividual differences in the adaptation to excess calories and the observed variation was not randomly distributed. For instance, there was at least 3 times more variance in response between pairs than within pairs for gains in body weight and fat mass. The within identical twin pair response to the standardized caloric surplus suggests that the amount of fat stored is likely influenced by the genotype.

In a second experiment, 7 pairs of young adult male identical twins completed a negative energy balance protocol during which they exercised on cycle ergometers twice a day, 9 out of 10 days, over a period of 93 days while being kept on a constant daily energy and nutrient intake. The mean total energy deficit reached 58,000 kcal. Mean body weight loss was 5.0 kg and it was entirely accounted for by the loss of fat mass. Intrapair resemblance was observed for the changes in body weight and body fat, with about 7 times more variance between pairs than within pairs in the response pattern. Even though there were large individual differences in response to the negative energy balance and exercise protocol, subjects with the same genotype were more alike in responses than subjects with different genotypes particularly for body fat and body energy losses. These results are remarkably similar to those of the overfeeding protocol.

Evidence for a role of single gene. Support for a role of specific genes in human obesity or variation in body fat content has been obtained from the following lines of evidence: Mendelian disorders (MIM) having obesity as one of the clinical features, single gene rodent models, quantitative trait loci from crossbreeding experiments, association studies and linkage studies.

The evidence drawn from these lines of clinical and experimental research can be summarized as follows. First, at least 12 loci linked to Mendelian disorders exhibiting obesity as one clinical feature are known. Second, 5 loci causing obesity in rodent models of the disease have been recognized. Third, 23 quantitative trait loci, identified by crossbreeding

experiments with informative strains of rodents, have been defined. Fourth, several candidate genes exhibiting a statistical association with BMI or body fat have been reported in human subjects. Fifth, many loci found to be linked to a relevant phenotype are known and in 5 cases the evidence for linkage is rather strong. The latter markers are mapped to 2p21, 6p21, 7q31, 11q21 and 20q12-13. Finally, a good number of studies have concluded that there was no association or linkage with a given marker or gene. The compendium of markers and genes related to obesity or body fat is likely to grow significantly in the coming years (4).

The complexity of human obesity. Obesity is a complex multifactorial trait evolving under the interactive influences of dozens of affectors from the social, behavioral, physiological, metabolic, cellular and molecular domains. Segregation of the genes is not easily detected in familial or pedigree studies and whatever the influence of the genotype on the etiology, it is generally attenuated or exacerbated by nongenetic factors. From the research currently available, a good number of genes seem to have the capacity to cause obesity or increase the likelihood of becoming obese. This may be a reflection of how most human obesity cases come about. In other words, the susceptibility genotypes may result from allelic variations at a good number of genes. With the advent of a comprehensive human genetic linkage map, linkage studies with a large number of markers covering most of the chromosomal length of the human genome are likely to be helpful in the identification of putative obesity genes or chromosomal regions. Recent progress in animal genetics, transfection systems, transgenic animal models, recombinant DNA technologies applied to positional cloning, and methods to identify loci contributing to quantitative traits have given a new impetus to this field. The stage is now set for major advances to occur in the understanding of the genetic and molecular basis of complex diseases such as human obesity.

3) HUMAN OBESITY AND ITS RELATIONSHIP WITH PSYCHOLOGICAL ADJUSTMENTS

A Stunkard.

Recent research has transformed our understanding of human obesity. For many years obesity has been viewed as a disorder with strong behavioral determinants—psychopathology manifested as overeating. The obese were believed to overeat in response to negative feelings including frustration, sadness, or insecurity, and food was seen as providing comfort in the absence of other sources of solace. They were frequently portrayed as having problems with food because of their inability to establish satisfactory interpersonal relationships. As one author noted, obesity is a "particular way of handling one's difficulty in human relationships and, even more, one's poor relationship with oneself" (1). this view has changed 180 degrees in direction. When psychopathology is observed in obese individuals, it is now seen as a consequence rather than a cause—a consequence of the prejudice and discrimination to which the overweight are subjected.

Prejudice and Discrimination. Obese individuals in America and other industrialized nations suffer significant prejudice and discrimination (2, 3). Such prejudice has been observed in children as young as 6 years of age, who described silhouettes of an overweight child as "lazy, dirty, stupid, ugly, cheats and lies" (4). When shown black and white line drawings of an obese child and children with various handicaps, including missing hands and facial disfigurement, both children and adults rated the obese child as the one they least wished to play with (5). Regrettably, overweight individuals display this same prejudice (6).

Sadly, health-care professionals appear to share this prejudice. Physicians consider their obese patients to be "weak-willed, ugly and awkward" (7). Patients are fully aware of such attitudes. In a recent study, 80% of persons who underwent

surgery for their obesity reported that they had "been treated disrespectfully by the medical profession because of my weight" (8).

Psychopathology and Obesity. In view of the prejudice and discrimination to which they are subjected, overweight persons could be expected to show higher levels of psychological disturbance. Studies of this topic, however, have yielded some surprising findings.

Population studies have generally failed to find significant differences between obese and non-obese persons in psychological status (as measured by self-report inventories) (2,3). In contrast to population data, studies of overweight persons seeking weight reduction suggest that emotional disturbance is common in the obese. Thus, clinicians can anticipate that a significant minority of persons seeking treatment for their obesity will experience significant psychological distress that may require treatment by psychotherapy or other means.

Body Image Disparagement. Although weight dissatisfaction is so common among adolescent girls as to approach a "normative discontent" (9), it is more severe in obese girls. Many feel that their bodies are "ugly and despicable and that others view them with hostility and contempt" (10). The problem of body image disparagement occurs most commonly in young Caucasian women of upper-middle socioeconomic status, in whom prevalence of obesity is very low (i.e., 5%) and the sanctions against it very high (9).

Racial differences in preferred body type may also affect psychological responses. African Americans and Mexican Americans, for example, do not appear to value thinness to the same extent as Caucasians (11, 12), a finding that may explain their high prevalence of obesity and low prevalence of eating disorders (13).

Binge Eating Disorder. During the past 10 years there has been a revival of interest in binge eating by obese persons (14), culminating in the proposal of a new diagnostic category—"binge eating disorder."

An important consequence of the delineation of this disorder was the discovery that it is a surprisingly prevalent problem affecting as many as 15% of persons entering treatment for obesity. Binge eaters tend to be heavier than non-bingers and report significantly greater psychological distress (on standard measures of psychopathology) and have a higher lifetime prevalence of psychiatric illness (particularly affective disorders) (15). Thus, 50% of obese binge eaters report a lifetime diagnosis of depression compared to 5% of obese persons who do not binge. Despite these psychological problems, obese binge eaters do as well in weight loss programs as do non-bingers.

Weight Loss and Depression. Early reports described a large incidence of depression in obese persons in weight reduction programs and there is still some concern that dieting may produce depression. More recent studies that used better methods of assessment have provided reassurance in this matter. Depression occurs very rarely in the course of weight reduction diet and most dieters actually show a lessening in levels in depression. This good news is countered my bad news. Not only is weight regained all-too-often following treatment, but weight regain has a very negative effect on patients' satisfaction with their appearance, self-esteem, self-confidence, and happiness as well as on their physical health.
Patients routinely report that they feel disappointed if not disgusted with themselves and ashamed to meet with their health professionals and friends who helped them to lose weight. The more times the patient has lost and regained weight, the greater the burden of shame and failure the individual carries. Unfortunately, repeated gaining of lost weight makes the individual less likely to seek treatment, feeling that he or she does not deserve it or that "nothing will help anyway."

Personality and Psychopathology. In any group of ten patients receiving treatment, we usually observe several persons who are outgoing, socially skilled and productive group members. They participate constructively in sessions,

providing comfort and useful suggestions to other patients. Groups often include, however, individuals with a personality disorder (16). These persons are frequently popular during the early stages of treatment. They disclose intimate thoughts and feelings about themselves and develop strong relationships with other group members. Over time, however, they often flood the group with reports of their troubled personal relationships and difficulties coping with life's demands. In addition, they tend to be emotionally labile in meetings and develop intense positive and/or negative feelings toward staff and other group participants. The behavior of such persons, if not controlled, can have a very negative effect on group morale and functioning.

Persons with a passive-aggressive personality style have a different effect on group dynamics. They are likely to drain the group's energy by their constant complaints that they re not "getting anything out of treatment" and that none of the suggestions offered by the therapist is helpful. Such persons were first identified in traditional group psychotherapy, where they achieved recognition as "helping-rejecting complainers."

Thus, our obese patients display a wide range of personality styles. The great majority of individuals display normal psychological functioning, but there clearly are exceptions. It is important that they receive appropriate care, which may differ from that required for the management of their obesity. In many cases, adjunctive psychotherapy may be indicated.

The Experience of Dieting. Early Dieting: Most patients report that, after the first 2 to 3 weeks of dieting, they feel happier and more self-confident (17). These verbal reports are confirmed by patients' increased attention to their dress and appearance and often by more active social calendars. The rapidity with which these changes occur is surprising, because they do so before patients have lost enough weight to be noticeable to others. For many, the improved mood simply reflects relief that they have been able to diet successfully, a demonstration of self-control that brings hope of long-term weight management. Binge eaters, in particular, frequently report with pride that, as a result of being provided a structured meal plan, they have stopped binging for the first time in months.

Treatment sessions reinforce patients' optimism and resolve during this early period (17). Many report that the group gives them greater strength and tenacity than they have ever experienced while dieting alone. The group is also a source of support and suggestions for the one or two individuals (our of a group of ten) who have difficulty "getting started" on the diet and are understandably discouraged. Brief individual meetings and telephone contacts between weekly meetings frequently help such individuals by providing greater structure and identifying impediments to adherence.

Later dieting: A significant minority of patients enjoy a very smooth course of dieting during 20 to 25 weeks of treatment. They report few adherence problems, lose weight regularly, and are often heard remarking that "I haven't felt this good in years."

A majority of patients, by contrast, have a generally favorable course of treatment but report that adherence to the diet and program of lifestyle change gets more difficult after the first 10 or so weeks. Complaints about keeping diet and exercise diaries increase over time, as do frustration and annoyance associated with failure to lose weight, despite reportedly good adherence. Most, however, maintain their efforts, continue to lose weight and, overall, are satisfied with their progress.

Most groups contain one or two individuals out of ten) who, as treatment progresses, become discouraged by their slow weight loss or inability to control binge eating (or related difficulties). Despite efforts by the group and therapist to support them, these persons frequently appear to "give up" at some point, convinced that their efforts are futile. This resignation frequently is associa-

ted with increased feelings of hopelessness and despair, which, in turn, lead to overeating and perpetuation of a vicious cycle (18). We encourage these individuals to remain in group treatment but also arrange individual care to meet their additional needs.

Sexuality: Most patients volunteer that after weight loss they feel more sexually attractive and are happier with their sex lives. A majority report increased libido well before they have achieved their full weight loss. There are, however, a handful of persons for whom weight loss was associated with marked distress about dating and sexuality. In some cases, patients stopped losing weight. Further investigation of the problems precipitated by weight loss is needed to determine their prevalence (which is quite low in our experience) and how to respond to them.

The growing recognition that obesity, in many persons, is a chronic condition requiring long-term care makes it imperative that practitioners concern themselves with their patients' self-esteem. Only by limiting their patients' experiences of shame and guilt concerning their weight can practitioners hope to create a trusting relationship to which patients can repeatedly return for the help they need. Years after treatment, many of our patients identify such a relationship as the most important and lasting aspect of their care.

Summary. The old view, that emotional disorders cause obesity has changed 180 degrees in direction. Obese persons have emotional problems but they are now seen as a consequence of being obese in a culture that disparages and discriminates against obesity. The major manifestations of emotional disorder are binge eating disorder and disparagement of the body image. The treatment of obesity requires a sensitive and supportive, professional approach. Weight loss is normally accompanied by an improvement in mood and emotional disorder.

4) OBESITY AND THE REGULATION OF ENERGY BALANCE, THE CONTROL OF FOOD INTAKE AND THE CONTROL OF ENERGY EXPENDITURE.

H Laviada-Molina.

Energy balance is the result of the control of ingestive behavior, energy expenditure and energy storage in adipose tissue (1). In most adults, both body weight and body fat content remain constant over many years or decades despite a very large flux of energy intake and expenditure (approximately 1 milion calories/year). Energy intake is a discrete process as individual meals are separated by intermeal intervals, while energy expenditure and storage in adipose tissue are continuous physiological processes (2). However, the complex molecular mechanisms by which discrete ingestive behavior, continuous energy expenditure and dynamic energy storage in adipose tissue are integrated and matched remain largely unknown. Two candidate signals are brain insulin and OB protein (1).

Feeding behavior is the result of complex central nervous system (CNS) integration of central and peripheral signals relating to brain and metabolic states. Meals are initiated, maintained and terminated by specific sets of these central and peripheral signals several times a day separated by intermeal intervals without food intake (3). These signals include patterns of: neural afferent traffic, metabolites (glucose), energy flux (fatty acid oxidation, ATP) and hormones (insulin) concentrations in plasma and brain, leptin concentrations in plasma and neuropeptide concentrations in brain. One hypothesis for this integrated control of food intake postulates the interaction of four classes of signals: 1) hypothalamis neuropeptides (neuropeptide Y or NPY, galanin, cholecystokinin or CCK, corticotropin releasing hormone or CRH, and enterostatin), 2) brain insulin, 3) leptin or OB protein, 4) ascending and descending neural inputs (4). These signals interact and provide the central/ peripheral integration necessary to regulate food

intake and body energy balance.

Total daily energy expenditure can be partitioned into resting metabolic rate, dietary and cold induced thermogenesis and the energy cost of physical activity (5). The energy cost of various kinds of voluntary physical activity (e.g. walking, jogging, swimming) and the thermogenesis due to digestion and absorption of food as well as thermogenesis induced by a cold environment have been calculated for both men and women as a function of lean body mass (or "fat free mass"). However, the regulation of resting metabolic rate is a complex function of energy intake, energy balance and hormonal and autonomic neural activity. When an individual reduces calorie intake and shifts into negative energy balance, the resting metabolic rate also decreases. This regulatory adaptation appropriately reduces obligatory energy expenditure when energy intake is reduced. However, this same adaptation causes a deceleration of weight loss following voluntary caloric restriction for the purpose of weight loss and contributes to the difficulty of maintaining weight loss, once achieved, over time (6).

5) BODY COMPOSITION AND OBESITY

SB Heymsfield.

Overview. Quantifying the amount and distribution of adipose tissue and its related components is integral to the study and treatment of many diseases. Body composition research is a field devoted specifically to the development and extension of methods for the in vivo quantification of adipose tissue, as well as other biochemical and anatomical components of the body. This field has progressed during the last 40 years from the whole body, "somatic" or organism level, to anatomic dissections, to biophysical approaches for the in vivo estimation of components, and most recently to underlying genetic and molecular mechanisms determining variability in composition. In this presentation I focus on methods of estimating body composition components in the context of evaluating disease states and their associated risks. This article first describes the organization of human body composition with an emphasis on muscle mass and fatness. A general overview is then provided on approaches to measuring body composition components, especially muscle and fat mass. The concepts developed in this section are then used to describe each of the available whole-body and regional methods for assessing body composition.

Body composition levels. The human body may be considered to consist of multiple components distributed across five basic levels of organization: atomic, molecular, cellular, anatomic or - more precisely - "tissue-system", and whole body (Figure 1). The thirty five to forty primary components at the five levels of organization are summarized in Table 1 (1, 2). Each component is considered discrete without overlap with other components at the same level. Components, however, may overlap across levels. The sum of the components at a level equals body weight or mass. These facts allow the formulation of explicit body composition equations for estimating unknown components from measured ones and body weight. Some of these are given in Table 2.

A complete assessment of the disordered body composition known clinically as "obesity" involves the quantification of components at all five levels of body composition. The following sections consequently review our model of the five body levels of composition organization as a prelude to a more detailed discussion of contemporary methods for assessing body composition in obesity. This includes the merits and limitations of various in vivo methods when applied to obese patients in both clinical and epidemiological settings and issues related to the use of the measured variables in evaluating effects of treatment, prognosis and risk of morbidity and mortality.

The Five Level Model of Body Composition. As shown if Figure 1, we conceive of human body composition as organized into discrete components at five basic levels of organization. An understan-

Table 1
Main Body composition Components.

	LEVEL			
	Atomic	**Molecular**	**Cellular**	**Tissu-Syste**
Components	O, C, H, N, Ca, P, S, K, Na, Cl, Mg	fat, water, protein bone mineral, non-bone tissue mineral, glycogen, fat-free body mass, fat-free solids.	fat cells, cell mass, intracellular fluid, extracellular fluid, extracellular solids, body cell mass.	adipose tissue subcutaneous visceral AT, b skeletal muscle skeleton.
Number of Components	11	8	6	6

From reference 2, with permission.

ding of the theoretical and empirical bases of these levels, and of the inter-relationships among the components at different levels, is essential to correct application of contemporary body composition methodology.

Atomic. The human body is comprised of 11 elements that account for over 99.5 percent of body weight (1). Three of these elements, carbon, hydrogen, and oxygen are found in storage triglycerides (3). The elemental stoichiometry of some common triglycerides found in humans are shown in table 3. The average proportions of these as carbon, hydrogen, and oxygen are considered stable at approximately 76.7%, 12.0%, and 11.3%,

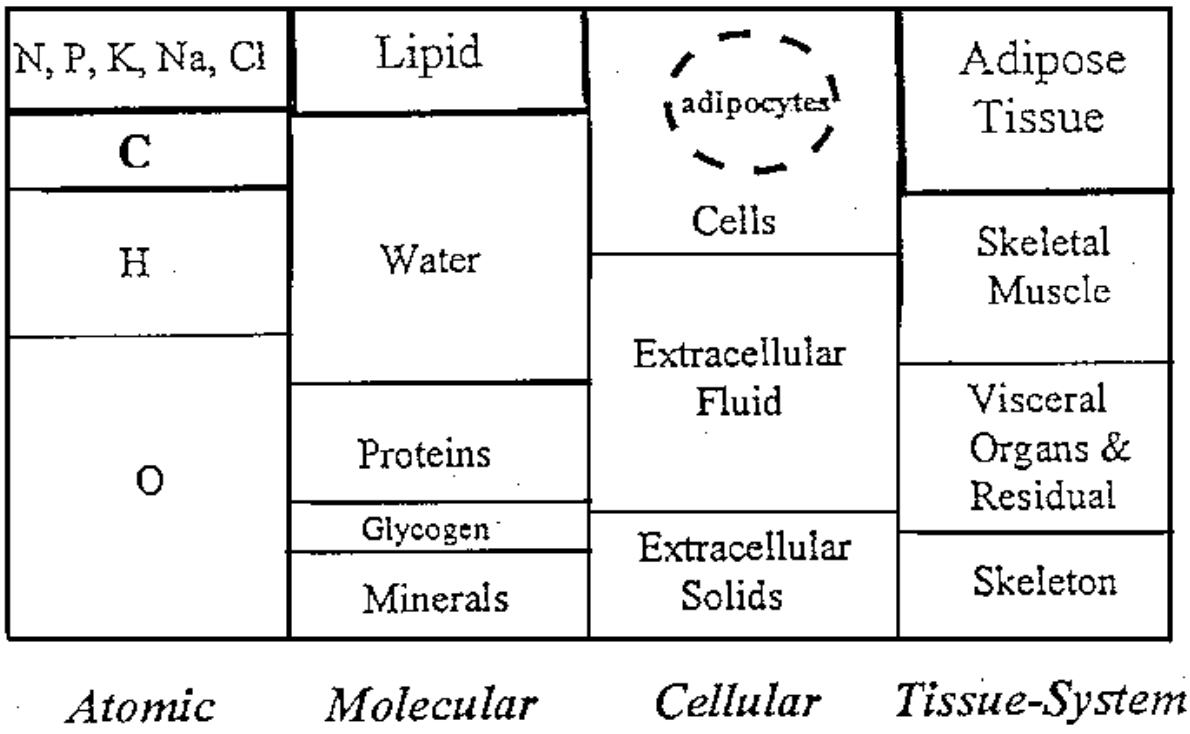


Figure 1. A model of human body composition.

respectively (3, 4). These stable elemental proportions of triglycerides allow the development of methods for deducing total body fat from total body carbon and other elements (5).

Molecular. The above elements, including trace elements that occur in low, but essential concentrations, combine to form various chemical compounds that may be grouped into the broad classes that define the molecular level of body composition. The main components of the molecular level are shown in Table 1, and include water, lipids, proteins, minerals, and carbohydrates. Each of the non-aqueous components represents many different but closely related chemical compounds. For example, the "protein" component consists of several hundred different compounds of protein.

The major molecular level components can be formulated into various models as summarized in Table 2. Generally, it is not feasible to measure all components at the molecular level. The direct quantification of body "fat" or lipid, in particular, has proven difficult historically. As a result a variety of methods have been developed for estimating this component indirectly using measurements of other components in various models. Two, three, and four component models are widely used in body composition research and these will be presented in later sections. Given the introduction of new methods of quantifying body fat directly, some of these models have been reoriented in recent years towards estimating other components that are difficult to measure, for example total body protein. Lipid is the main molecular level component of interest in the study of human obesity. The term lipid refers to all chemical compounds that are insoluble or weakly soluble in water, but are soluble in organic solvents such as chloroform and diethyl ether (6, 7). Lipids isolated from human tissues include triglycerides, sphyngomyelin, phospholipids, steroids, fatty acids, and terpenes. Triglycerides, commonly referred to as "fats", are the primary storage lipids in humans and comprise the largest fraction of the total lipid component (3, 4)(Table 3). At present there is limited information on the exact proportion of total lipids as triglycerides,

Table 2
Body Composition Equations at Differnt Body Body Composition Levels.

Level	Equation	
Atomic	BW = O + C + H + N + Ca + P + K + S + Na + Cl + Mg	11 components
Molecular	BW = F + A + Pro + Ms + Mo + G	6 components
	BW = F + A + Pro + M	4 components
	BW = F + A solids	3 components
	BW = F + Mo + residual	3 components
	BW = F + FFM	2 components
Cellular	BW = CM + ECF + ECS	
	BW = F + BCM + ECF + ECS	
Tissue-system	BW = AT + SM + bone + other tissues	

Abreviations: A, water; AT, adipose tissue; BCM, body cell mass; BW, body weight; CM, cell mass; ECF; extracellelar fluid; ECS, extracellular solids; F, fat; FFM, fat-free body mass; G, glycogen; M, mineral; MO, bone mineral; Ms, soft tissue mineral; Pro, Protein; SM, skeletal muscle. Frome reference 1, with permission.

or the amount of within and between-person variability. The "Reference Man", however, is considered to consist of 13.5 kg of total lipid of which 12.0 kg, or 89%, is "fat" (4).

A summary of molecular level component characteristics is presented in Table 4. These characteristics are used in developing body composition methods and their application will be presented in later sections.

Cellular. The cellular level includes three main components, cell mass, extracellular fluid, and extracellular solids. Cells can be divided into specific types such as connective, epithelial, nervous, and muscular. Adipocytes or fat cells serve as the primary storage site for triglycerides. It is often desirable to exclude inert, storage triglycerides in the estimation of "body cell mass". This component may be grouped separately as "fat mass" or combined with "extracellular solids" as a "metabolically-inert" component. The concept of body cell mass was originated by Moore and refers to the mass of materials composing cells that are actively involved in energy consumption and heat production (8). This concept has considerable clinical and physiological significance, but the exact definition and measurement of body cell mass is usually difficult in practice.

Table 3
Structure and Elemental Stoichiometry of Representative Tryglicerides.

Structure	Carbon	Hydrogen	Oxygen
C57H104O6	77.4	11.8	10.9
C51H98O6	75.9	12.2	11.9
C55H102O6	76.9	11.9	11.2
C55H104O6	76.7	12.1	11.2
"Average" Trygliceride	76.7	12.0	11.3

Data from references 3 and 4

Tissue-System. The main components at this level are adipose tissue, skeletal muscle, bone, and visceral organs (e.g., liver, kidneys, heart, etc.). The adipose tissue component includes adipocytes with collagenous and elastic fibers, fibroblasts, capillaries, and extracellular fluid. Adipose tissue can be classified by distribution into four types, subcutaneous, visceral, interstitial, and yellow marrow (4). The introduction of computerized axial tomography (CT) over the past decade allowed the first accurate quantification of visceral adipose

Table 4
Characteristics of Molecular Level Components.

Component	Density (g/cm3)	Elemental Stoichiometry
Water	0.99371 at 36º C 0.994 at 37º C	0.111 H; 0.889 O
Protein	1.34 at 37ª C	0.532 C; 0.070 H; 0.161 N; 0.227 O; 0.01 S.
Glycogen	1.52 at 37º C	0.444 C; 0.062 H; 0.494 O
Minerals	3.042 (weighted avg of bone & non-bone)	0.398 Ca; 0.002 H; 0.185 P; 0.414 O
Bone*		
Non-bone	2.982 at 36 - 36.7º C 3.317 at apx 40º C	
Lipid tissue		0.767 C; 0.120 H; 0.113 O.
Adipose extracts	0.9007 at 36º C	

* Calculated from the largest component of bone mineral, calcium hydroxyapatite (Ca3(PO4)2)3Ca(OH)2. Other small elemental contributions to bone, such as Na, are recognized. Data from reference 8.

tissue (VAT)(9). More recently, magnetic resonance imaging (MRI) has been developed for this purpose as described below.

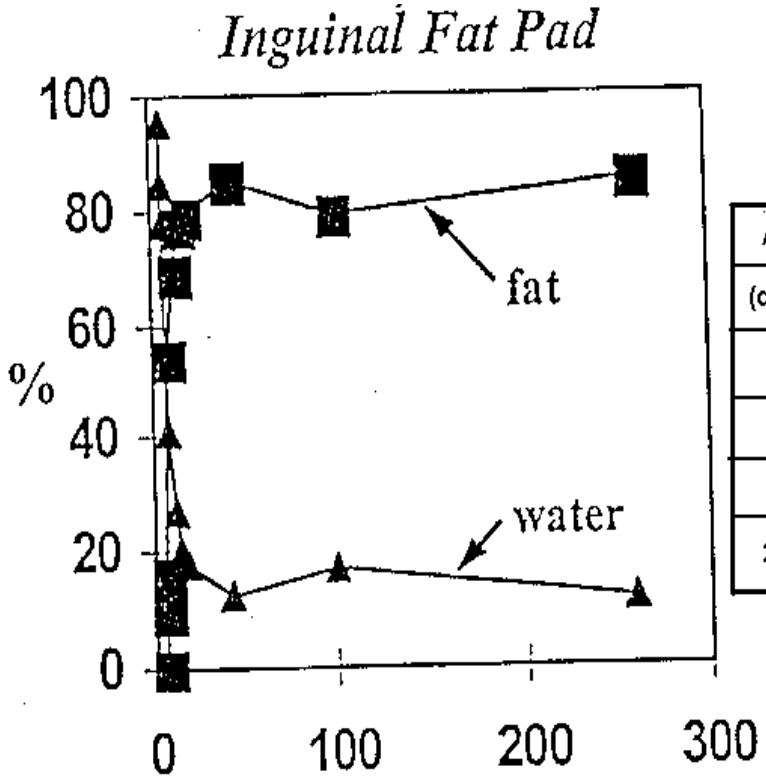
Human adipose tissue is often assumed to have an approximate average composition consisting of 80% lipid, 14% water, 5% protein, and <1% mineral, and a density of 0.92 g/cm3 at body temperature (10). Adipocytes, however, range in lipid content from a negligible amount in connective tissue precursors to a high lipid content in mature adipocytes observed in morbidly obese subjects. According to Martin and colleagues, for every 10 percent increase in relative adiposity there is a corresponding rise in adipose tissue lipid fraction of 0.124 (11). Variation in adipocyte fat content is shown for the rat in figure 2. As the rat matures there is a progressive increase in adipose tissue fat content and a corresponding relative reduction in water. The changes in fat content with age in the rat result is a lowering of adipose tissue density, a phenomenon which is important to consider when attempting to convert measured adipose tissue volume to mass.

A notable feature of adipose tissue is the relatively large extracellular fluid compartment relative to cell mass. Of the 14% of average adipose tissue samples as water, 11% is extracellular water (11).

Whole-Body. Skinfolds, circumferences, and linear dimensions are all measurements at the whole body level. These measurements are often used with prediction equations to estimate components at the other four body composition levels (1).

The human body can thus be divided into discrete components that are distributed into five increasingly complex levels. The study of human obesity involves investigation of many components at all five body composition levels. In the sections that follow our main emphasis will be on methods that are used to quantify "fatness" or "adiposity".

Body composition methods. Body composition methods can be organized as outlined in figure 3 (12). Methods can be broadly divided into in vivo and in vitro. This chapter will focus only on *in vivo*



Age (days)	BW (g)	Dens (g/cc)
0	5.6	1.007
3	7.4	0.952
14	18.5	0.924
280	260	0.918

Figure 2. Variation in adipocyte fat content in the rat in relation to age.

body composition methods.

In vivo body composition methods can be classified into six categories as outlined in figure 3. These categories are based on the basic body composition methodology formula (12), C = f (Q) [1] where C is unknown component, Q is a measurable quantity, and f is the mathematical function that links Q to C. All body composition methods share this basic formula in common. The formula indicates that body composition methods can be organized according to measurable quantity and mathematical function (12).

Measurable Quantities. Property-Based Methods. Property-based methods all apply the general formula C = f (P), where P is a measurable property. A relatively small number of physical, chemical, and biological properties are used in body composition assessment. These include anthropometric dimensions, electrical resistance, radioactive decay profiles, weight, volume, oxygen consumption, infra-red interactance, fat soluble gas uptake, and x-ray attenuation.

Examples of property-based methods include estimation of total body fat from measured skinfold thicknesses (13, 14), body volume (by underwater weighing) (15), and x-ray attenuation (by dual-energy x-ray absorptiometry) (16).

All property-based methods ultimately rely on the measurement of one or more properties. Property-based methods are the foundation of body composition methodology.

Component-Based Methods. Component-based methods all apply the general formula, Cu = f (Ck), where Cu is an unknown component and Ck is a known component. The known component must first be derived using a property-based method. An example of a component-based method is estimation of total body fat from total body carbon and other elements (5).

Combined Methods. Some methods are based on both a measurable property and known component. These combined methods all apply the general formula, Cu = f (P, Ck). Examples of combined methods include estimation of total body

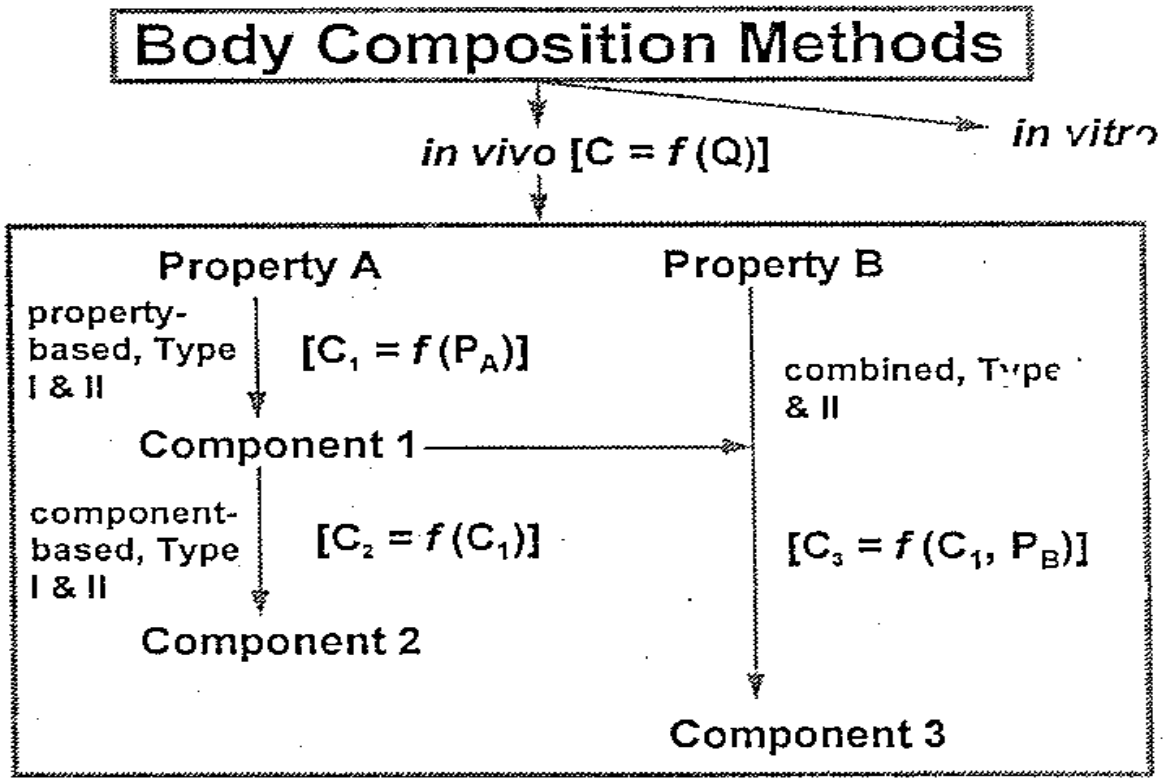


Figure 3. *In vivo* body composition methods.

fat from 1. body volume (a measurable property) and total body water (a known component), and 2. body weight (a measurable property) and total body potassium (a known component)(12).

Mathematical Functions. As noted earlier, mathematical functions applied in the fundamental body composition equation can be broadly classified into two types. The creation of these two function types is somewhat arbitrary, although in practice we found the distinction between type I and II functions useful in understanding method development and errors.

Type I Methods. Type I methods are all based on statistically derived regression equations (12). These methods share in common three characteristics: a reference method for measuring the component of interest, a well characterized subject group, and the application of statistical methods to derive the function for predicting the component from the measured properties or components. Because they are developed on discrete subject groups, type I prediction equation methods are often "population specific". This means that an equation does not necessarily provide accurate estimates when applied to people who differ in terms of sex, ethnicity, age, or health status from those included in the sample from which the equation was derived. As a result, estimates from prediction equations should be validated against those from an established laboratory-based method in a random subsample of participants or patients before the equation is applied to the entire study population. Some approaches that may be used in the development of prediction equations and the potential errors that might arise were reviewed recently by Roche and Guo (17).

An example of a type I prediction method is the estimation of total body water (TBW) using the bioelectric impedance approach, as found in Lukaski et al. (18). Bioelectric resistance, stature, and TBW by deuterium dilution were measured in a group of subjects. Simple linear regression analysis was used to derive an equation for predicting TBW (kg) from the two measurable properties height (H, cm) and bioelectric resistance (R, ohm): TBW = 0.63 H2/R + 2.03, r = 0.95, p< 0.0001. Although the general formula for relating body water volume to height and resistance is based on a theoretical model that relates conductive volume to length (L) and resistance (V = rL2/R), the coefficient r cannot be derived a priori and must be estimated statistically. This contrasts with the approach taken in type II methods.

Type II Methods. Type II methods are all formulated from what we generally refer to as models. A "model" in this context denotes the a priori specification of the function relating a component to a property or another component based on the assumption of one or more constant physical, biochemical, physiological, anatomical or other structural relationships between components (12). Most models specify functions in the form of proportions or ratios. Some examples include TBW/FFM (0.73 kg/kg), carbon/fat (0.767 kg/kg), total body potassium/FFM (0.00266 kg/kg), and nitrogen/protein (0.16 kg/kg)(1). In contrast to type I methods, the values for the ratios or proportions used in these models are formulated a priori based on data from chemical analyses of human tissues or animal experiments and do not require statistical methods in their development. Nevertheless, type II methods may be population-specific also and their application to a new group sometimes requires validation of the assumptions made in the underlying model.

An example of a classic type II method is estimation of FFM (kg) from TBW (kg): FFM = 1.37 x TBW. This method was first popularized by Pace and Rathbun (19) who observed a relatively constant hydration of FFM in animals (0.73) and others subsequently observed similar hydration in humans (20). The method gained widespread clinical use due to the ease and low cost of isotope dilution methods for TBW measurement.

Applications to the study of clinical conditions: critical analysis. Most body composition research is based on the molecular level (fat and FFM) and tissue-system level

(adipose tissue, adipose tissue-free body mass, and skeletal muscle). Measurements of interest include whole-body and regional components. The main available methods are outlined in Table 5. I have selected methods for review that are in current use and that are conside mred appropriate for clinical and research applications. The table is divided by type I versus type II methods and the range of whole body and regional estimates at molecular and tissue-system levels is shown for each method. An important feature of type I methods is that they are generally capable of providing predictions of a wide range of components using relatively simple, inexpensive measurements. As a result, they are preferred "field methods" for larger populations studies. Their accuracy, as noted above, depends in part on that of the more cumbersome and expensive type II, or "laboratory-based", methods they are usually calibrated against. The following is a brief overview of an important current topic in the field of obesity, assesment of adipose tissue distribution. References for this section are widely available from 21 to 37, or upon request to Dr. Heymsfield.

Numerous epidemiological and clinical studies have established that centralized obesity, in which fat is stored preferentially in adipocytes on and within the trunk rather than the extremities, represents the obesity phenotype that conveys the largest risk for morbidity and mortality from the major chronic diseases: heart disease, cancer, and diabetes. A variety of anthropometric approaches have been developed to grade or classify centralized adipose tissue distribution. Recent efforts have

Table 5
Whole Body (W) and Regional (R) Measurement Methods Used in Assesing Adiposity related Components.

	Adiposity-Related Component					
Method	**Fat**	**FFM**	**SAT**	**VAT**	**ATFM**	**SM**
Type I						
Body Weight	W	W				
Anthropometry	W,R	W,R	W,R	W	W,R	W,R
Bioimpedance and Conductivity	W,R	W,R				W,R
Ultrasound	W,R	W,R	W,R	W	W,R	W,R
Type II						
Isotope Dilution (total body water)	W	W				
Hydrodensitometry	W	W				
Dual Energy X- Ray Absorptiometry	W,R	W,R	R			R
Whole Body Counting /IVNA	W	W				W
Imaging (CT,MRI)			W,R	W,R	W,R	W,R

Abreviations: ATFM, adipose tissue-free body mass; CT, computed tomography; FFM, fat-free body mass; IVNA, in vivo neutron activation analysis; MRI, magnetic resonance imaging; SAT, subcutaneous adipose tissue; SM, skeletal muscle; VAT, visceral adipose tissue.

been focused on developing type I equations for predicting the amount of visceral adipose tissue (VAT), which is believed to be the main aspect of centralized obesity associated with risk. The following section reviews the merits and limitations of these anthropometric approaches.

Skinfold thicknesses have been used to describe primarily the distribution of subcutaneous adipose tissues. This aspect of the adipose tissue distribution has been called "fat patterning", to distinguish it from the more general form that includes the amounts and distribution of internal adipose tissues. Historically, three main approaches have been used to describe fat patterning: pattern-profile, ratio, and principal components methods. The pattern-profile method was first applied by Garn and compares two or more groups graphically for mean values of skinfold thicknesses across several anatomical sites. It provides a useful, visual comparison of differences or similarities between groups for anatomical variation in subcutaneous fat thickness. Cluster analysis provides a more sophisticated, statistical approach to defining pattern-profiles.

A variety of skinfold thickness ratios have been used to index fat patterning. The ratio of the subscapular to triceps skinfolds is one of simplest and most widely used. Some consider it to be important to include a skinfold on the leg, such as the lateral calf or medial thigh skinfold. The advantages of the ratio approach are that it requires few variables, simple computation, and provides a single continuous variable for grading subjects. Three problems can be identified, however, with ratio indices of fat patterning: 1) they tend to be correlated with total fat mass or percent body fat; 2) they may have poor sensitivity and validity with regard to the latent variable, subcutaneous adipose tissue distribution; and, 3) it may be difficult to determine whether a correlation with another variable (e.g. serum HDL cholesterol) is due to variation in the numerator or denominator of the ratio. The use of a greater number of skinfolds, and the sum of all skinfolds in the denominator, may partly alleviate these problems. Principal components analysis is a more sophisticated statistical approach to constructing fat pattern indices. This approach summarizes the information in several skinfold variables in a smaller number of new, statistically independent indices that are not correlated with total body fat. When this method is applied to data for several skinfold variables, it provides the best "criterion" measures for judging the validity and sensitivity of simpler ratio indices. The main criticism of skinfold thickness methods in the study of obesity is that they do not capture variation in the amounts of internal adipose tissues, especially those surrounding the viscera. "Visceral adipose tissue" has been recognized as the main aspect of body fat distribution that is associated with increased risk for chronic disease. As a result, many prefer indices based on circumferences that are believed to include variation in visceral adipose tissues. The most popular circumference index is the "waist/hip ratio" (WHR), followed by the "waist/thigh ratio" (WTR). The waist/hip ratio was the first used to assess the associations between adipose tissue distribution and chronic disease morbidity and mortality. Numerous studies have now shown that WHR is an independent predictor of metabolic disturbances including insulin resistance, hyperlipidemia, hypertension, and atherosclerosis. Similar associations have been also been reported for WTR, as well as for skinfold thickness indices and some other circumference indices such as the "conicity index". In general, the associations of risk factors, as well as morbidity and mortality, with circumference indices tends to be somewhat stronger than with skinfold thickness indices of adipose tissue distribution. This is generally thought to be due to either the increased measurement error in skinfold thicknesses, or the influence of VAT volume on waist circumference. As for BMI or skinfolds, cut-off values for the WHR have been recommended for defining "upper body obesity". The recommended values generally used are > 0.95 in men and > 0.80 in women. It is important to recognize that these values were selected based on the increase in risks with

increasing WHR and not on the association with adipose tissue distribution.

There are several problems in the use of circumference ratios as indices of adipose tissue distribution. First, standard definitions of the circumferences are not always followed, making it difficult to compare results across studies. For example, the "waist" circumference has been defined variously as at the level of: the smallest circumference on the torso below the sternum; the umbilicus; the lower margin of the ribs; and, the iliac crests. The "hip" circumference has been defined as at the level of: the iliac crests; the anterior iliac spines; the greater trochanters; or, the maximum posterior protrusion of the buttocks. There may be considerable differences among circumferences measured at these locations. Some may vary between subjects in relation to bone landmarks (e.g., smallest circumference on torso below the sternum), or may be difficult to identify in obese subjects. There is scarcely any difference between a "waist" circumference measured at the level of the umbilicus and a "hip" circumference measured at the level of the iliac crests in most subjects. Obviously, "waist" circumference in one study may be the same as "hip" circumference in another when both are defined as at the level of the iliac crests.

In obese subjects, the identification of the "waist" may be extremely subjective, if not impossible, and the measurement is more correctly defined as an abdominal circumference. The location of the abdominal circumference in relation to a soft-tissue landmark, such as the umbilicus, is not recommended because many obese subjects will have an extremely pendulous abdominal adipose panniculus. The umbilicus may be directed downward and located well below the horizontal, transverse plane of the midabdomen. This may lead to considerable variation among subjects in the definition of this measurement. In addition, a pendulous panniculus may result in overlapping of abdominal and hip circumferences or interfere with the standard measurement of hip circumference.

A second problem is that circumferences are influenced by variation in muscle and bone as well as adipose tissues, as noted previously. These influences may be particularly difficult to sort out when ratio indices are used. It has been assumed conventionally that increased WHRs mainly reflect increased VAT, based on studies that report significant correlations between WHR and VAT area, as measured using imaging methods (see below). Some studies, however, have reported significant correlations with measures of cross-sectional muscle area also, in particular those for the pelvis or hips. Thus, variation in WHR may reflect the effects increased VAT on waist circumference (numerator) as well as decreased gluteo-femoral muscle on hip circumference (denominator). This influence of muscle has been recognized increasingly and may be important in understanding the relationship of WHR to chronic disease risk. Larsson et al. reported that risk of heart disease was greatest in those with lower BMIs and high WHRs. Filipovsky et al. reported that all cause and cancer mortality over 20 years of follow-up in the Paris Prospective Study was greatest in those with low BMIs but high WTRs. The low BMIs in these study might reflect low muscle mass, rather than fat; subsequently, the high WHRs or WTRs might reflect a combination of increased VAT and muscle loss. It should be remembered that the phenotype originally described by Vague, who first drew attention to the association of body composition and metabolic disease, consisted of a expanded abdominal fat mass in conjunction with thin legs. The latter might reflect muscle atrophy associated with disease as much as lack of subcutaneous adipose tissue on the extremities. This potentially important association between visceral adiposity and skeletal muscle was recently re-emphasized by Bjorntorp.

A third problem is the strong correlations of circumferences and circumference ratios with total adiposity. This makes it difficult statistically to separate the effects of centralized adipose tissue distribution from obesity. This confounding is

further exacerbated by the moderate positive correlations of body fatness and centralized adipose tissue distribution with age. Many early studies that reported significant correlations between WHR and VAT did not control for the confounding influences of age and BMI. Seidell et al. reported that WHR did not correlate significantly with ratio of visceral to subcutaneous adipose tissue, as measured using CT, after adjustment for age and BMI. Similarly, Ross et al. reported that, after controlling for both age and adiposity, WHR explained only 12% of the variation in absolute levels of VAT in men. Furthermore, the observed relationship between WHR and relative VAT was non existent. Thus, while WHR is an independent predictor of numerous metabolic aberrations, its association with risk may not be attributed simply to its association with the amounts of either absolute or relative VAT.

Several investigators have argued that simple waist circumference is a better index of variation in VAT than WHR. It is important to note, however, that waist circumference is very highly correlated with total adiposity (r values > 0.90) in most populations. Also, the error of prediction of VAT from waist circumference, alone or in combination with other variables, is large. Ross et al. recently reported that the sensitivity and specificity of waist circumference for predicting absolute values of VAT was poor. These observations are explained in large measure by the intraindividual variation in the visceral to subcutaneous adipose tissue ratio.

It is important to establish whether reductions in VAT are related to concurrent reductions WHR or waist circumference. Some have reported that WHR changes with weight loss, while others do not. Ross et al. recently reported that diet and exercise induced reductions in VAT (kg) that were significantly associated with reduced waist circumferences in obese male (r=0.69) and female (r=0.47) subjects. For the two groups combined, a 1 cm reduction in waist circumference was associated with a 4% reduction in VAT ($p<0.01$). The standard deviation associated with the 4% reduction in VAT per centimeter reduction in waist circumference, however, was also 4%. This suggests that the ability to quantify small changes in VAT volume from waist circumference is limited by inter-individual variations in the reduction of abdominal subcutaneous and lean tissue. Thus, whereas changes in visceral obesity are clearly associated with changes in waist circumference, it is not possible to accurately predict small changes in VAT.

Efforts to develop equations for predicting VAT have not generally been successful. The errors associated with these equations tend to be large: 25 to 40%. This level of accuracy is clearly insufficient for estimating changes in individuals and may be inadequate for comparing groups. Few of these equations have been cross-validated in independent samples. Future efforts to develop this approach have value, however, given the inadequacies described above for skinfold and circumference indices. At present, it would appear that new or different anthropometric measurements will be needed to increase the accuracy of prediction equations to acceptable levels. One measure that has been suggested is the sagittal thickness of the trunk or abdomen.

Sagittal Thickness. Several studies have suggested that sagittal trunk thickness correlates more highly than other anthropometric variables with the volume of visceral adipose tissue quantified by imaging methods. As a result, it may be useful both as a simple index, like waist circumference, or as an independent variable in equations for predicting VAT. There is presently no standardized technique for measuring sagittal trunk thickness and, to date, its use has been limited mostly to clinical studies.

Sagittal trunk thickness may be defined as the maximum diameter of the abdomen in the sagittal plane. As for circumferences, this measurement may be obtained technically in all subjects regardless of obesity level. Bony landmarks for the standard location of this measurement have not been identified: alternative possibilities include the

xiphoid process of the sternum, the fourth lumbar vertebrae, or the iliac crests. It is important to note that the choice between these landmarks will result in measurements at very different levels on the trunk or abdomen. Sliding calipers with long, parallel blades are necessary for this measurement.

Although sagittal trunk thickness may be taken with the participant standing, measurement in the supine recumbent position may be preferred to maximize the association with the latent variable, intra-abdominal adipose tissue volume. Theoretically, when a person with an enlarged intra-abdominal adipose tissue mass lies supine the mass shifts cranially causing anterior projection of the abdomen, which is measured as increased sagittal thickness. When a person is standing, gravity pulls the intra-abdominal adipose tissue mass downward and the maximum sagittal thickness may be located somewhat lower. It is important to keep in mind that the level of the maximum measurable diameter, either supine or standing, will likely vary among some subjects. The extent to which these measurements are influenced by the amount of subcutaneous abdominal adipose tissue, and shifts in its distribution between supine and standing measurements, is not well established.

Results for the use of sagittal thickness to grade or predict visceral adipose tissue volume in several Swedish studies have been summarized by Sjostrom. Visceral adipose tissue volume was estimated using seven cross-sectional CT scans of the abdomen. Sagittal thickness was measured on the CT image at the level of L4-5. Visceral adipose tissue (VAT) volume was regressed on sagittal thickness (ST) in 17 men producing an equation, VAT (L) = 0.731 x ST (cm) - 11.5, (R2 = 0.81). This equation was latter cross-validated in two independent samples of 7 and 13 men, respectively, with virtually indistinguishable results. A similar regression equation was developed using data for 10 women and cross-validated in 9 independently selected women: VAT (L) = 0.370 x ST(cm) - 4.85, (R2 = 80). Sjostrom and associates also showed that changes in VAT volume were accurately tracked by changes in sagittal thickness in 6 patients with Cushing's disease during treatment.

Pouliot et al. analyzed associations of sagittal thickness with visceral adipose tissue area on CT images at L4-L5 in 81 men and 70 women, 30 to 42 years of age. Sagittal thickness correlated better with VAT area than waist/hip ratio in both sexes; however, it was also correlated strongly with subcutaneous abdominal adipose tissue area. Taken together, the results of the studies by Sjostrom et al. and Pouliot et al. indicate that sagittal thickness is somewhat more sensitive than conventional indices such as waist/hip ratio for grading or predicting the amounts of visceral adipose tissue. The measurements of sagittal thickness in these studies were taken from the CT scans, rather than anthropometrically. Sjostrom and associates, however, reported that the squared difference between sagittal thicknesses measured anthropometrically and on the CT images in their studies was only 1.7%. In an independent study, Van der Kooy et al. reported a high correlation (r=0.94) between sagittal thickness measured with the subject standing and from MRI images at the same level. It may be important to consider that the errors of estimation for VAT averaged about 20% in these studies, which could allow for considerable misclassification when subjects are grouped by sagittal thickness. Lastly, the strong correlation with abdominal subcutaneous adipose tissue areas reported by Pouliot et al. is bothersome since it suggests that sagittal thickness may not accurately discriminate visceral from subcutaneous abdominal adipose tissue.

We have examined the association of sagittal diameter with VAT volume in elderly participants in the New Mexico Aging Process Study (unpublished data). Visceral adipose tissue volume was quantified using an MRI protocol developed by Ross et al.. Sagittal diameter was measured using sliding calipers while the participant was supine at a level of L4-5. The R2 for the regression of VAT volume on sagittal diameter was 0.75 in 9 elderly men, or similar to that reported by Sjostrom.

The corresponding R2 for 9 women, however, was only 0.09. In contrast, the R2 for the regression of sagittal thickness on subcutaneous adipose tissue volume for the trunk in the women was 0.33. This suggests that while sagittal diameter, measured anthropometrically, is a moderately sensitive index of VAT in elderly men, it may not be useful in elderly women. The mean sagittal diameter in our women was 20.8 (+ -) 2.9 cm, which is closely similar to the mean for women reported by Sjostrom. The corresponding mean VAT volume, however, was 1.51 - 0.59 L, which is about one-half that reported by Sjostrom. The difference between studies for mean VAT volume is partly attributable to a different definition of VAT in our study. This difference could explain the lack of correlation between sagittal diameter and VAT in the women in our study, and highlights issues related to definition and measurement of VAT that are addressed in greater detail in a subsequent section on measuring VAT from CT and MRI methods.

Whereas establishment of the association of sagittal diameter with the latent variable of interest, VAT, is important, it is also important to examine the sensitivity of this measure in relation to risk factors associated with visceral obesity. Richelsen and Pedersen recently analyzed associations of sagittal thickness and other indices of VAT with serum total cholesterol, triglyceride, LDL and HDL, fasting insulin and glucose concentrations in 58, middle-aged men. They concluded that sagittal thickness was slightly better correlated with an adverse lipid, insulin and glucose risk profile than waist/hip ratio. Similar findings were also reported in the study by Pouliot et al.. Taken together, these studies suggest that sagittal diameter may be preferred to other indices of VAT as a risk factor in epidemiologic studies of obesity-associated chronic diseases. In this regard, Seidell et al. reported that abdominal sagittal thickness was a strong predictor of mortality in younger adult men enrolled in the Baltimore Longitudinal Study. Further studies are needed to establish the usefulness of sagittal thickness for grading or predicting visceral adipose tissue and as a risk factor in different age, sex and ethnic groups.

Conclusion.

There are many methods of estimating body composition components, particularly those components related to obesity and malnutrition. Practically all major body composition components can be quantified in vivo. This lecture will focus on widely available methods or those of special conceptual interest.

The selection a component assessment method depends largely on the specific purpose for body composition assessment. The question posed will dictate the requirement for method accuracy and precision. Additional relevant factors include method cost, safety, practicality, and availability. These are all important considerations as most research centers now have multiple available methods ranging from CT and MRI to DXA, BIA, and anthropometry.

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