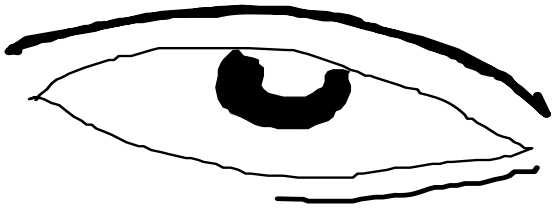

JOYFUL LIVING SERVICES

Overweight And Underweight

Obesity is a major malnutrition problem. It is one of the most important and least understood areas in the science of nutrition. Everyone knows roughly what it is. If you are too fat, you are overweight; if much too fat, you are obese. But why and how obesity occurs and what can be done about it are matters for much speculation, debate, and frustration. For the obese person who has earnestly tried every known means of losing weight only to fail, frustration can turn to despair.

Less well recognized is the problem of underweight, which can be equally mysterious. A "skinny" person finds it hard to gain a pound as a fat person does to lose one.

This session emphasizes the problems of overweight and obesity, partly because they have been more intensively studied and partly because they are a more widespread health problem in the developed countries. This does not imply that the underweight person faces a less difficult problem. The concluding section shows that what we know about the one extreme sometimes applies equally well to the other. Two subjects are not covered fully in this session but are reserved for Session 14: anorexia nervosa and bulimia.

Overweight and underweight both result from unbalanced energy budgets. The overweight person has consumed more food energy (kcalories) than he has expended and has banked the surplus in his fat cells. The underweight person has not consumed enough and so has depleted his fat stores. Energy itself doesn't weight anything and can't be seen, but when it exists in the form of chemical bonds in nutrients or body fat, the material that it holds together is both heavy and visible.

The amount of fat you might deposit or withdraw from "savings" on any given day depends on your energy balance for that day – the amount you consume (energy-in) versus the amount you expend (energy-out). As Session 6 shows, you can reduce your fat deposits by withdrawing more energy from them than you put in. A pound of body fat stores 3,500 kcalories. To lose a pound of body fat you must experience a deficit; you must take in 3,500 kcalories less than you expend. To lose that pound in a week, you need to achieve an average deficit of 500 kcalories a day.

Ideal Weight

Ideal weight is a misnomer; not the desirable but the average weight given in insurance tables for persons of a given sex and height in the United States – not necessarily ideal for a given individual.

Fatfold Test

A fatfold test is a clinical test of body fatness in which the thickness of a fold of skin on the back of the arm (triceps), below the shoulder blade (subscapular), or in other places is measured with an instrument called a caliper. The older, less preferred, term for this is skinfold test.

Frame Size

The frame size is the size of a person's bones and musculature. A person with a large frame should weight more than one with a small frame.

Ideal Weight and Body Fatness

How fat is too fat? And how thin is too thin? It isn't always possible to tell from the bathroom scales, because body weight says nothing about body composition. The relative amounts of lean and fat tissue vary widely from one person to the next. A dancer or an athlete, whose muscles are well developed and whose bones have become dense from constant stress, may weigh much more than a sedentary person with a similar figure. What is needed is a measure of body fatness – not of body weight. Ideally, by a very rough approximation, fat makes up about 18 percent of a man's body weight and about 22 percent of a woman's, with the remainder contributed by water (55 to 60 percent), muscle and other lean tissue (10

to 20 percent), and bone minerals (6 to 8 percent). But there is no easy way to look inside a person to see the bones and muscles.

Several laboratory techniques for estimating body fatness have been developed. One way is to determine the body's density (weight compared with volume). Lean tissue is denser than fat tissue, so the more dense a person's body is, the more lean tissue it must contain. Weight is easy to measure with a scale, but volume measurement involves submerging the whole body in water and measuring the amount of water displaced; this requires a large tank and takes up too much space to be practical for use in say, a doctor's office. Another way is to inject a water-soluble substance that is easy to detect and measure, and allow it to penetrate into the lean tissue (it will not mix into the fat tissues). A blood sample taken soon after will show the extent to which the substance has been diluted, providing an estimate of the amount of lean tissue.

A direct measure of the amount of body fat can be obtained by lifting a fold of skin from the back of the arm, from the back, or from other body surfaces and measuring its thickness with a caliper that applies a fixed amount of pressure. The fat under the skin in these regions is roughly proportional to total body fat. A fold over an inch thick indicates overfatness; under a half-inch reflect underweight. This technique – the fatfold test – is a practical diagnostic tool in the hands of trained people and is in increasingly wide use.

A still simpler test is the mirror test. Undress and stand before a mirror. If you look too fat, you may be too fat. (A notoriously poor judge of this, however, is the teenage girl, who often thinks any amount of fat, no matter how small, is a serious blemish. It may be that she needs to change her self-image – not to go on a diet.)

The scales are not necessarily an accurate indicator of body fatness, then, but you most probably use them anyway. After weighing yourself, you turn to the tables published by the insurance companies. You then discover that for a person your height and sex, three weight ranges are suggested: one for a small frame, one for medium, and one for large. Don't forget your shoes: you are assumed to be wearing one-inch heels. (Thus a person who stands 5 feet 10 inches tall in bare feet would look up the range for a person 5 feet 11 inches.) Finally, if you weigh yourself nude, you must adjust for clothing (the tables assume 5 pounds for clothes). All these steps involve a lot of guesswork. How do you decide on your frame size, for example?

How to Determine Your Body Frame by Elbow Breadth

To make a simple approximation of your frame size: Extend your arm and bend the forearm upward at a 90-degree angle. Keep the fingers straight and turn the inside of your wrist away from the body. Place the thumb and index finger of your other hand on the two prominent bones on either size of your elbow. Measure the space between your fingers against a ruler or a tape measure. Compare the measurements with the following standards.

These standards represent the elbow measurements for medium-framed men and women of various heights. Measurements smaller than those listed indicate you have a small frame and larger measurements indicate a large frame.

	Men	
Height in 1-in Heels		**Elbow Breadth**
5 ft 2 in to 5 ft 3 in		2 ½ to 2 7/8 in
5 ft 4 in to 5 ft 7 in		2 5/8 to 2 7/8 in
5 ft 8 in to 5 ft 11 in		2 ¾ to 3 in
6 ft 0 in to 6 ft 3 in		2 ¾ to 3 1/8 in
6 ft 4 in and over		2 7/8 to 3 ¼ in
	Women	
Height in 1-in Heels		**Elbow Breath**
4 ft 10 in to 4 ft 11 in		2 ¼ to 2 ½ in
5 ft 0 in to 5 ft 3 in		2 ¼ to 2 ½ in
5 ft 4 in to 5 ft 7 in		2 3/8 to 2 5/8 in
5 ft 8 in to 5 ft 11 in		2 3/8 to 2 5/8 in
6 ft 0 in and over		2 ½ to 2 ¾ in

After finding the applicable weight, you have to apply the most important judgment factor of all. Ask yourself whether the weight range you have singled out is really ideal for you. At what weight are you most healthy? Does your family tend to be most healthy at the heavier, or lighter, end of the weight ranges? A recent reinterpretation of the insurance company statistics suggests that many people are healthiest at weight slightly above those thought to be ideal in the past.

Ideal weight probably changes with age. Many people typically become less active as they grow older. Their muscles get smaller, and their bones decrease in density. Thus a person who at 25 was lean and muscular might weight the same at 65 and yet have become considerably fatter. Such a person should either gradually lose weight as time goes on or, preferably, maintain a program of vigorous physical activity to preserve muscle mass and bone strength. Not much is known about ideal weights at older ages, but clearly, people do tend to gain about 20 to 30 pounds during adulthood. Perhaps for a person who maintains muscle mass, this weight gain may be consistent with good health if it does not precipitate high blood pressure.

With all their limitations, the weight tables are often used to draw arbitrary lines between too much and too little body weight. A person who is more than 10 percent above the weight on the table is considered overweight; if 20 percent or more, obese. (Some authorities say obesity is 15 percent above the table weight, some say 25 percent.) Similarly, a person who is more than 10 percent below the table weight is considered underweight.

Overweight
Overweight is when body weight is more than 10 percent above average (insurance company table) weight.

Obesity
Obesity is excessive body fatness; often loosely defined as a condition of being overweight by 15 or 20 percent or more.

Underweight
Underweight is when body weight is more than 10 percent below normal or average weight.

Juvenile-Onset Obesity
This is obesity arising in childhood; also called developmental obesity.

Adult-Onset Obesity
This is obesity arising after adolescence; sometimes called reactive obesity if it appears to arise in response to a specific traumatic life event.

The Problem of Obesity
However you define it, obesity does occur to an alarming extent and is increasing in the developed countries. For example, in the United States some 10 to 25 percent of all teenagers and some 25 to 50 percent of all adults are obese.

Some people become fat in childhood, and others later on. Few of either type lose the excess weight. There is no specific age that divides juvenile-onset obesity from adult-onset obesity, but as the terms imply, there is a distinction between the two types. A child who is obese will develop sturdy muscles and bones as she grows, to support her excess weight. Thus as an adult she will have more lean body mass and more body fat than the average person and will likely always be stocky, even after losing her excess fat. People who become obese, as children are also less likely to be able to reduce successfully than people whom become obese as adults.

Research on fat cells suggests a possible reason why early-onset obesity is especially resistant to treatment. Simply stated, early overfeeding is thought by some researchers to stimulate fat cells so that they increase abnormally in number. The number of fat cells is thought to become fixed by adulthood; if it is, then a gain in weight thereafter can take place only through and increase in the size of the fat cells. A person with an abnormally large number of fat cells is thought likely to be abnormally hungry and to overeat for that reason. On the other hand, a person who gains weight in adulthood supposedly has a normal number of fat cells and needs only to reduce the size of the cells.

This theory has been heavily criticized on several groups. Fat cells are hard to count, and researchers disagree as to whether new cells are being formed at certain periods or small, empty fat cells are being recruited as new storage cells. Even the critics agree, however, that there are certain periods in life when body fat increases more rapidly than lean tissue: early infancy (up to about two years), again during preadolescence (and throughout adolescence in girls), and possibly again during the third trimester of pregnancy. These are critical periods, in the sense that some developmental events that take place at these times are irreversible. Prevention of obesity would be most important during these times. There is also agreement that fat is hard to lose no matter when it is gained.

Hazards of Obesity

Insurance companies report that fat people die younger from a host of causes including heart attacks, strokes, and complications of diabetes. In fact, gaining weight often appears to precipitate diabetes. Fat people more often suffer high levels of blood fat, hypertension, coronary heart disease, post-surgical complications, gynecological irregularities, and the toxemia of pregnancy. The burden of extra fat strains the skeletal system, causing arthritis – especially in the knees, hips, and lower spine. The muscles that support the belly may give way, resulting in abdominal hernias. When the leg muscles are abnormally fatty, they fail to contract efficiently to help blood return from the leg veins to the heart; blood collects in the leg veins, which swell, harden, and become varicose. Extra fat in and around the chest interferes with breathing, sometimes causing severe respiratory problems. Gout is more common and even the accident rate is greater for the severely obese.

Beyond all these hazards is the risk incurred by millions of obese people throughout much of their lives – the risk of ill-advised, misguided dieting. Some fad diets are more hazardous to health than obesity itself. One survey of 29,000 claims, treatments, and theories for losing weight found fewer than 6 percent of them effective – and 13 percent dangerous.

Once a person becomes obese, the situation tends to perpetuate itself. When fat cells enlarge, they become sluggish in responding to insulin, the hormone that promotes the making and storage of fat. The excess glucose remains in the bloodstream longer than normal and stimulates the insulin-producing cells of the pancreas to multiply and secrete more insulin. When the fat cells finally respond, they store more fat than normal in response to the raised insulin level. As if this were not enough, the enlarged fat cells are also less sensitive to other hormones that promote fat breakdown. Weight loss restores insulin levels to normal, but it first has to be achieved against these odds.

Not only physical but also social and economic disadvantages plague the fat person, who is less often sought after for marriage, pays higher insurance premiums, meets discrimination when applying for a job, can't find attractive clothes so easily, and is limited in choice of sports. Fat girls have only a third the chance of being accepted into college than lean girls have. The fat child often suffers ridicule from his classmates and the unbearable humiliation of having the captain choose him last for the team.

The many disadvantages justify our calling obesity a severe physical handicap. However, it is unlike other handicaps in two important ways. First, mortality risk is not linearly related to excess weight. Instead, there is a threshold at which risk dramatically increases. Being only a few pounds above this threshold weight may cause blood pressure, blood glucose, and blood lipids to zoom upwards. Second, obesity is reversible, and if it is corrected in time, some of its risks are, too. Mortality rates (from insurance data) are no higher for the formerly obese than for the never obese.

Ideally a person would never have to struggle with the problem of obesity, because he would never have become obese to begin with. Preventive efforts are needed, especially in vulnerable groups: infants, preadolescents, adolescents, and women before they are pregnant. (This is in no way meant to imply that a woman who is pregnant should attempt to lose weight. Weight loss during pregnancy requires skilled medical supervision if it is done at all.) Where prevention has failed, treatment is urgently needed. But how to treat? Before turning to the matters of diet, drugs, exercise, and other means of attacking the problem, it is necessary to try to figure out what causes obesity.

"Pull" versus "Push" Theories of Obesity

The "pull" theory of obesity proposes that a subtle disorder inside the person increases food intake either by affecting signals transmitted to a "satiety center" or by altering the sensitivity of the satiety center to such signals. The "push" theory proposes that the obese person "force-feeds" himself, over-eating for non-physiological reasons.

Longitudinal Study

A longitudinal study is one in which the subjects are studied over time – for example, in 1960 and again in 1970 and in 1980.

Causes of Obesity

Kcalories are not stored in fat until the body's energy needs have been met. Excess body fat can accumulate only when kcalories are eaten beyond those needed for the day's metabolic, muscular, and digestive activities. To put it bluntly, obesity results from overeating.

In fact, however, this statement neither explains the cause of obesity nor suggests a cure. Why do people overeat? Is it a hunger problem? An appetite problem? A satiety problem? Is it genetic? Metabolic? Environmental? Is it a matter of habits learned in early childhood? Is it psychological? Might all these facts play a role? To tell the truth, we do not know the cause.

In general, two schools of thought address this problem. One attributes it to inside-the-body causes the other to environmental factors. One currently popular inside-the-body theory is the so-called set-point theory. Noting that many people who lose weight on reducing diets subsequently return to their original weight, some researchers have suggested that the body "wants" to maintain a certain amount of fat and regulates eating behaviors and hormonal actions to defend its "set point." While this theory is compelling in its simplicity, there is at present little scientific evidence supporting its reality in humans.

The other point of view is that obesity is environmentally determined. Proponents of this view hold that we overeat because we are pushed to do so by factors in our surroundings – foremost among them, the availability of a multitude of delectable foods. The two views are not mutually exclusive, and research with animals suggests that both are possible. Some obesity may arise from one, some from the other, cause. The two possibilities were humorously illustrated years ago by two obesity researchers (the "pull" versus "push" theories of obesity definition above); there is no reason why they should not both be operating, even in the same person.

The inside-the-body idea is supported by the fact that animal strains do exist that are genetically fat, and they tend to be fat in any environment – that is, no matter what kind or variety of food is offered. The environmental obesity model is supported by experiments with "cafeteria rats." Ordinary rats, fed regular rat chow, are of normal weight (for rats), but if those very same rats are offered free access to a wide variety of tempting, rich, highly palatable foods, they greatly overeat and become obese.

It seems likely that both environmental and hereditary factors influence obesity in humans. The average adult in our society gains about 30 pounds between the ages of 20 and 50, but people in non-Western societies do not. This suggests that all people may have inherited the capacity or tendency to gain weight, but that our surroundings have allowed it to be realized, while conditions in other countries prevent it.

One way to test whether human obesity is inherited is to study identical twins raised in different families, one family fat and the other thin. If genes determine fatness, then both twins will become equally fat or thin. But if the environment is responsible, the twins will resemble their respective families. Another approach is to study adopted children, to see whether they resemble their natural or adoptive parents. Studies of both kinds suggest that the tendency to obesity is inherited, but that the environment is influential in the sense that it can prevent or permit the development of obesity when the potential is there.

Inheritance of the tendency to obesity is probably very complex and governed by many different genes. To complicate the situation further, these genes probably occur with different frequencies in different populations.

A related question is, "Do fat babies become fat adults?" Ten years ago, most nutrition experts might have answered "Yes (probably)." Today, however, the results of several longitudinal studies are available that suggest the answer "Not necessarily; some do, some don't." Clearly, not all fat babies are fated to become fat adults; many grow up elegantly thin. Nor is a thin baby immune to becoming fat later on.

Still, if obesity is not programmed in by inheritance or by early, critical developmental events, it nonetheless seems to persist from childhood in many instances. Many researchers have the impression that early food habits exert a powerful influence on lifelong tendencies to overeat. Food centered families encourage such behaviors as overeating at mealtimes, rapid eating, excessive snacking, and eating to

meet needs other than hunger. Children readily imitate overeating parents, and their behavior at the table tends to persist outside the home. Obese children have been observed to take more bites of food per interval of time and to chew them less thoroughly than their nonobese schoolmates.

People who eat small but frequent meals may tend to store less fat than those who eat large meals at irregular intervals. Thus families that allow their children to skip meals may be promoting obesity.

Hunger
Hunger is the physiological need to eat; a negative, unpleasant sensation.

Appetite
Appetite is the desire to eat, which normally accompanies hunger; by itself a pleasant sensation.

Satiety (sat-EYE-uh-tee)
Satiety is the feeling of fullness or satisfaction at the end of a meal, which prompts a person to stop eating.

Glucostatic Theory
This is a theory of hunger regulation: the theory that blood levels of glucose determine when people eat.
Gluco = glucose
Stasis = staying the same

Lipostatic Theory
This is a theory that the body's total fat stores are fixed and that when they are depleted, eating behavior is turned on.
Lipo = fat

Purinergic Theory
This is a theory that circulating purines regulate eating behavior.
Erg = driving force

Hunger and Appetite Regulation
Whatever sets the stage for excess fat accumulation, the fat is gained because we put food into our mouths. A vast amount of research has been devoted to finding out what stimulates and governs eating behavior. Why do we start to eat? Why do we eat as much as we do? Why do we stop?

An important distinction was made early between hunger, appetite, and satiety. Hunger is said to be physiological – an inborn instinct – whereas appetite is psychological – a learned response to food. The two are not the same. We have all experienced appetite without hunger: "I'm not hungry, but I'd love to have a piece." The too-thin person may often experience the reverse, hunger without appetite: "I know I'm hungry, but I don't feel like eating." Hunger is a negative experience (and we may eat in order to avoid it); appetite is positive. As for satiety, which signals that it is time to stop eating, it vies with hunger and appetite for the distinction of being recognized as the primary regulator of eating behavior. One view holds that eating behavior is turned "on" all the time, except when the satiety signal turns it off. But the exact nature of the satiety signal is not known.

The stomach participates in signaling satiety. Nerves responsive to stretch of the stomach wall fire when the stomach is full, transmitting a message to the brain. Even animals without stomachs get hungry, though, so it is clear that an empty stomach is not the only cue to hunger.

Whether hunger, appetite, or satiety regulates eating behavior (and there are other possibilities discussed below), two questions arise. First, what molecular or other messengers make us feel these sensations? Second, where in the body are they received? Many theories have been put forth to answer the first question. The glucostatic theory of hunger regulation proposes that the blood glucose level determines whether we are hungry or sated; the lipostatic theory states that the size of our fat stores dictates how much we eat; and the purinergic theory proposes that the circulating levels of purines, molecules found in DNA and RNA, govern hunger. Careful measurement of blood levels of glucose show that it does not account for the starting and stopping of eating, however, and glucose researchers are now pursuing the possibility that exhaustion of liver glycogen may somehow convey the signal. "Eat." If fat stores regulate hunger in some way, the messenger they send to the brain to do so has yet to be identified. As for the purinergic theory, its proponents confess that they proposed it "somewhat tongue in cheek"; it is new, and the other three are old, familiar, and frustratingly unsatisfactory to account for what is observed.

The theories just described (at least the first two) have been discussed and researched for several decades. Newer ideas as to what molecules might be the regulators of eating behavior include the endogenous opiates and a variety of hormones. It has long been known that the GI tract produces several hormones that serve to notify the pancreas, the gallbladder, and the intestine that food is present and must be dealt with. A flurry of findings during the early 1980'' brought forth many reports that these same hormones, now numbering some 20 or 30, are also produced in the brain after meals. Perhaps, in the brain, they signal satiety.

That brings us to the second question: where in the brain are these messages received (whatever they are)? One brain area stands out as a regulator for food behavior – the hypothalamus – but it is not the only one involved. At one time, it was thought that the front-central hypothalamus was the "satiety center," and that the sides were the "hunger centers." Now, however, that idea has been exposed as an oversimplification. The hypothalamus integrates many kinds of signals received from the rest of the body, including information about the blood's temperature, sodium content, and glucose content. It is certainly important in regulating eating, because damage to the hypothalamus produces derangements in eating behavior and body weight – in some cases causing severe weight loss, in others vast overeating. In the person with a normal hypothalamus, however, eating behavior seems to be a response not to a single signal arriving at some one location in the hypothalamus but to a whole host of signals. Somehow these many inputs become integrated into a "final common path" – the act of eating.

Thermogenic Theory

Related to body weight regulation, there is also a thermogenic theory – but this deals with how energy is spent, not how hunger is regulated. The thermogenic theory suggests that the amount of heat generated in response to food determines how much fat is stored.
Thermo = heat
Genic = arising from

Cholecystokinin (COAL-ee-sis-to-KINE-in)

Among the hormones produced in the brain as well as the GI tract after meals are cholecystokinin, the messenger that communicates the arrival of fat to the gallbladder and pancreas, and calcitonin, a hormone that responds to blood calcium levels.

Hypothalamus (high-po-THALL-uh-mus)

The hypothalamus is a brain center that integrates signals about the blood's temperature, glucose content, and other conditions.

The Behavior of Eating

The word behavior has been used many times in this discussion as if it were a simple thing that everybody understands, but the study of behavior offers unique insight into the problem of overeating by viewing it as a conditioned response to a variety of stimuli.

To begin with, certain behavior patterns, whether innate or learned, occur appropriately in response to certain stimuli. For example, dogs salivate in response to the smell of food – but, as everyone knows, they can "learn" (be conditioned) to respond to the same way to the sound of a bell. As another example, an appropriate self-care behavior of animals is grooming, which can involve quite a complex pattern of motions – licking, scratching, nibbling the fur – but grooming behavior can suddenly appear unexpectedly at an inappropriate time. In the midst of a hostile confrontation, for example, one contestant will suddenly stop posturing and begin to groom himself intensively as if the wrong switch had been accidentally pushed. It is possible that people also displace one behavior with another when they are threatened. Rather than grooming, the behavior selected may be eating – and if this response occurs often enough, the consequence is obesity.

Displacement may explain some cases of obesity, but even if it doesn't the view given here is useful. It presents a picture of eating as a sort of package of behavior that can be triggered by any of many different stimuli. Some researchers focus on "external cues" as the triggers; others on "stressors," with connection to the production of endogenous opiates.

Displacement Activity

Biologists give the name displacement activity to the substitution of one behavior for another under stress.

External Cue Theory

This is the theory that some people eat in response to such external factors as the presence of food or the time of day rather than to such internal facts as hunger.

External Cues

Some obese people are unconscious eaters. Rather than responding only to internal, visceral hunger cues, they seem to respond helplessly to such external factors as the time of day ("It's time to eat") or the availability, sign, and taste of food. This is the basis of the external cue theory.

Of interest in this connection is the report of an experiment in which lean and fat people were housed in a metabolic ward and were offered their meals in monotonous liquid form from a feeding machine. The lean people at enough to maintain their weight, but the fat people drastically reduced their food intake and lost weight. When kcalories were added to the formula, the lean people adjusted their intake to continue maintaining weight as if they had an internal kcalorie counter. The obese people were unaware of the change, continued drinking the same amount of formula as before, and stopped losing weight. External cues were the only signals the obese people had to go by, and they responded the same way to the same environmental situation, regardless of how many kcalories they were getting.

For the person who responds to external cues, today's environment provides abundant stimuli to promote eating behavior. Restaurants, TV commercials, the display of food in our markets, vending machines in every office building and gas station – all prompt us to eat and drink high kcalorie foods. There are no "vegetable houses" on our main streets, only steak houses. Kitchen appliances such as the hamburger cooker and the doughnut maker make high-kcalorie foods easy to prepare and thus quickly available.

Arousal

The term arousal has been used several times. The general meaning is self-evident, but in the sense, in which it is used here, it refers to heightened activity of certain brain centers associated with excitement and anxiety.

Stressors and Arousal

Anything that excites or disturbs the equilibrium of an organism can be termed a stressor. The terms stressor and stress response have specific meanings, but they are being used differently by some researchers today to apply to many of the subjects being discussed here. "Stressors" include pain, anxiety, arousal, excitement, and even the presence of food.

The brain seems to respond to many of these stimuli by producing endogenous opiates. They soothe pain and lessen arousal, and they have two effects on energy balance. They enhance appetite for palatable foods, and they reduce activity. Combine these effects with a tendency to be supersensitive to particular stressors anyway, and you are all the more likely to gain weight in response to stress.

The psychiatrist Dr. Hilde Bruch, who has devoted as much attention to the human hunger drive as Freud did to the sex drive, sees other links between eating behavior and human experience. She states that hunger and appetite are understandably mixed up together because both are intimately connected to deep emotional needs. Two factors that she finds most important in this connection are the fear of starvation and "the universal experience in the early life of every individual that food intake requires the cooperation of another person." Feeding behavior is a response not only to hunger or appetite but also to complex human sensations such as "yearning, craving, addiction, or compulsion."

Others agree that food is widely used for nonnutritive purposes, especially in a culture like ours where food is abundant. An emotionally insecure person might eat as a substitute for seeking love or friendship. Eating is less threatening than calling a friend and risking rejection. Often, especially in adolescent girls, eating is used to relieve boredom or to ward off depression. Some obese people respond to anxiety, or in fact to any kind of arousal, by eating. Significantly, however, if they are able to give a name to their aroused condition, thereby gaining a feeling that they have some control over it, they are not as likely to overeat.

Stress may act in another way to promote obesity. The hormones secreted in response to physical stress favor the rapid metabolism of energy stores (glycogen and fat) to fragments such as glucose and fatty acids that can be used to fuel the muscular activity of fight or flight. Under emotional stress the same hormones are secreted and blood concentrations of these same fuels rise. If a person fails to use the fuel in violent physical exertion, the body has no alternative but to turn many of these fragments to fat. If

glucose has been used this way, and transferred into fat, then the lowered glucose level or exhausted glycogen will signal hunger, and the person will eat again soon after.

Stress eating may appear in different patterns; some people eat excessively at night, while others characteristically go on an eating binge during an emotional crisis. The overly thin often react oppositely. Stress causes them to reject food and thus become thinner. It is not yet known why these behaviors occur, but clearly investigations of the chemical, hormonal, and neural mechanisms involved in the body's responses to different stimuli hold much promise for a future understanding of eating behavior.

Inactivity

The many possible causes of obesity mentioned so far all relate to the input side of the energy equation. What about output? A person may be obese because he eats too much, but another possibility is that he spends too little energy. It is probably that the most important single contributor to the obesity problem in our country is underactivity. The control of hunger/appetite actually works quite well in active people and only fails when activity falls below a certain minimum level. Obese people under close observation are often seen to eat less than lean people, but they are sometimes so extraordinarily inactive that they still manage to have a kcalorie surplus. One authority has noted that normal people actually swim 35 minutes during "an hour of swimming," whereas obese people swim only 7 minutes during that hour. Most of their time is spent sitting, standing, or lying in the sun.

Individuality

No two people are alike either physically or psychologically, and the causes of obesity may be as varied as the people who are obese. Many causes may contribute to the problem in a single person. Given this complexity, it is obvious that there is no panacea. The top priority should be prevention, but where prevention has failed the treatment of obesity must involve a simultaneous attack on many fronts.

Treatments of Obesity: Poor Choices

The only means of reducing body fat is to shift the energy budget so that energy-in is less than energy-out. This is most effectively done by eating less and exercising more. A later section in this session addresses appropriate strategies for losing weight, but because rumors of other means fly about, they will first be dispensed with briefly.

Caution:
This cruel fact is one many of u would like to circumvent. Isn't there an easier way? No, the hard truth is that the only way to lose body fat is to eat fewer kcalories than you spend. Magical alternatives that have been offered time and again over the centuries – ways to "shrink the stomach," to eat "negative kcalories," to "eat all you want and lose weight' – they prove to be born of wishful thinking. They are effective only when they directly affect the kcalorie balance. The success of these plans is not in their achievements but in their popularity. They sell easily to susceptible people who want something for nothing, who become enthusiastic practitioners (but only briefly), and who pass on the word to the next person. This type of reaction reflects a human characteristic that for all our scientific rationality we have failed to outgrow. We love magic. Many writers of fad diet books and sellers of fraudulent diet pills and formulas use this characteristic to their advantage. Watch out for such frauds. A sign of their presence is the appeal they make to magical thinking and the promise of something for nothing.

Diuretic (dye-you-RET-ic)

A diuretic is a drug that promotes water excretion.
Dia = through
Ure = urine

Water Pills

For the obese person, the idea that excess weight is due to water accumulation may be an attractive one. Indeed, temporary water retention, seen in many women around the time of the menstrual period, may make a difference of several pounds on the scale. Oral contraceptives may have the same effect. (They may also promote actual fat gain in some women. A woman who has this problem should consult her physician about switching brands.) In cases of severe swelling of the belly, as much as 20 pounds of excess body water may accumulate.

If water retention is a problem, it can be diagnosed by a physician, who will prescribe a diuretic (water pill) and possibly a mild degree of salt restriction. But the obese – that is, overfat – subject has a smaller percentage of body water than the person of normal weight does. If she takes a self-prescribed diuretic,

she has done nothing to solver her fat problem, although she may lose a few pounds on the scale for half a day and suffer from dehydration.

Diet Pills, Starch Blockers, and Glucomannan

Some doctors prescribe amphetamines ("speed") to help with weight loss. (The best known are dexedrine and benzedrine.) These reduce appetite – but only temporarily. Typically the appetite returns to normal after a week or two, the lost weight (and often more) is regained, and the user then has the problem of trying to get off the drug without gaining more weight. It is generally agreed that these drugs cause a dangerous dependency and are of little or no usefulness in treating obesity.

A multitude of other drugs are presently under investigation: hormones and hormone-like compounds, inhibitors of nutrient absorption; inhibitors of fat synthesis, promoters of fat breakdown, other modifiers of metabolism – in short, every kind of agent that researchers can imagine might be effective in any way against obesity. Tests in humans of any of these would be premature at present, and results in animals are not encouraging. Side effects, in many cases, are severe. In short, at present, no known drug is both safe and effective, and many are hazardous. Even diet pills, long thought safe and widely used, have been shown not to be safe for all users. Two cases of serious illness have been ascribed to taking of diet pills containing phenylpropanolamine. The only effective appetite-reducing agent to which tolerance does not develop in time is cigarette smoking – and that, of course, entails hazards of its own too numerous to mention.

Among popular drugs recently on the market, starch blockers not long ago attracted a lot of attention. They sounded like a dieter's dream come true – eat your favorite carbohydrate foods and derive no kcalories from them. Unfortunately, although the principle seems sound enough, it doesn't work out in practice. It has been known since 1943 that uncooked wheat and kidney beans contained inhibitors of the starch-digesting enzyme amylase. The inhibitor from kidney beans has been purified and fed to rats; with the result that they excreted some starch and gained less weight than controls. However, tests on humans have shown no inhibition of starch digestion whatsoever.

Nevertheless, 100 different starch blocker preparations were on the market as of the end of 1982, at the peak of their success people were swallowing over a million pills a day. Some people were even stockpiling the pills, expecting that the FDA soon would ban them. As expected, FDA has prohibited their being marketed; they have been found to cause nausea, vomiting, diarrhea and stomach pains, and not to block starch digestion.

People who don't want to use starch blockers are trying glucomannan, a preparation derived from vegetable (konjac tuber) used in Japanese cuisine. The Japanese are said to have used konjac for weight control for 1,500 years – but in a controlled experiment reported in 1982, glucomannan was ineffective.

Some day a pill may be developed that is effective against overeating and obesity. None of those described here is a likely candidate. One that may be promising is the opiate antagonist naloxone, which blocks stress-induced eating in animals, and possibly also in humans. Extensive testing will be required to determine whether naloxone can be safely used for this purpose.

Perhaps the most promising anti-obesity agent presently being tested is the artificial fat (sucrose polyester). It remains to be seen, however, whether long-term use will facilitate permanent weight loss or whether, like artificial sweeteners, sucrose polyester will become a mere addition to the diet.

Cellulite (SELL-you-leet)

Cellulite is supposedly a lumpy form of fat; actually, a fraud. The skin sometimes appears lumpy in fatty areas of the body because strands of connective tissue attach the skin to underlying structures. These points of attachment may pull tight where the fat is thick, making lumps appear between them. The fat itself is not different from fat anywhere else in the body. So, if you lose the fat there, you lose the lumpy appearance.

Health Spas

One of the biggest moneymaking schemes that profits from people's desires to lose weight the easy way is the health spa. The spa can be used to advantage. People who really exercise there reap the expected benefits. But health spas can be extremely costly, and most of their gimmicks offer no real health advantage other than the psychological boost the consumer herself supplies. Hot baths do not speed up the basal metabolic rate so that pounds can be lost in hours. Steam and sauna baths do not

melt the fat off the body, although they may dehydrate a person so that his weight on the scales changes dramatically. Machines intended to jiggle parts of the body while the person leans passively on them provide pleasant stimulation but no exercise and so no expenditure of kcalories.

Some people believe there are two kinds of body fat: regular fat and "cellulite." Cellulite is supposed to be hard and lumpy fat that yields to being "burned up" only if it is first broken up by methods like the massage or the machine typical of the health spa. The notion that there is such a thing as cellulite received wide publicity with the publication of a book by a certain Madame R of Paris, which sold widely during the 1970s. The Journal of the American Medical Association has published the statement that cellulite is a hoax.

Hormones

Because hormones are powerful body chemicals and many affect fat metabolism, it has long been hoped that a hormone might be found that would promote weight loss. Several have been tried. With testing, all have proven ineffective and often hazardous as well. Thyroid hormone, in particular, causes loss of lean body mass and heart problems except when medically prescribed for the correction of a thyroid deficiency – and thyroid deficiency is very seldom the cause of obesity.

Among the hormones advertised as promoting weight loss is HCG (human chorionic ganadotropin), a hormone extracted from the urine of pregnant women. HCG has legitimate uses; for example, it can stimulate ovulation in a woman who has had difficulty becoming pregnant. But it has no effect on weight loss and does not reduce hunger. A rash of "clinics" run by "doctors" that sprang up on the West Coast during the 1970s advertised tremendous success using HCG in the treatment of obesity. These outfits seem to have had one element in common. They prescribed an extremely rigid low-kcalorie diet, which accounted for their apparent effectiveness. The American Medical Association and the California Medical Association have concluded that the claims made for HCG are groundless and that the side effects are unknown and probably dangerous.

Surgery

Sheer desperation prompts some obese people to request surgery. One operation, bypass surgery, involves removing or disconnecting a portion of the small intestine to reduce absorption. Another involves stapling the stomach to make it smaller.

After a bypass operation, the person can continue overeating but will absorb considerably fewer kcalories. Side effects from this procedure are many and highly undesirable, including liver failure, massive and frequent diarrhea, urinary stones, intestinal infection, and malnutrition. Reports of mortality range from 2 to 10 percent. Still, in the United States, surgery has been reported to be effective more than half the time for treating the massively obese where all other methods have failed. It should probably be attempted only in otherwise healthy and cooperative people under 30 who weigh more than 300 pounds and who have tried everything else.

Gastric stapling is in increasing use in preference to bypass surgery, because it forces the person to eat less rather than causing malabsorption. Still, although the theory is pleasingly simple, stapling involves hazards in practice; stomach tissue is damaged, scars are formed, staples pull loose. The person contemplating surgery should think long and hard before submitting to it.

Weight-Loss Diets Compared

With a balanced perspective on foods and a sense of what's important in diet planning and what's not, you can evaluate the many different diets people consume. Here's a summary of the questions you might ask. Start with 100 points and subtract if any of these criteria are not met:

1. Does the diet provide a reasonable number of kcalories (enough to maintain weight; not too many; and if a reduction diet, not fewer than 1,200 kcalories for the average-sized person)? If not, give it a minus 10.
2. Does it provide enough, but not too much, protein (at least the recommended intake or RDA but not more than twice that much)? If not, minus 10.
3. Does it provide enough fat for satiety but not so much fat as to go against current recommendations (say, between 20 and 35 percent of the kcalories from fat)? If not, minus 10.
4. Does it provide enough carbohydrate to space protein and prevent ketosis (100 grams of carbohydrate for the average-sized person)? Is it mostly complex carbohydrate (not more than 20 percent of the kcalories as concentrated sugar)? If no to either, minus 5, if no to both, minus 10.

5. Does it offer a balanced assortment of vitamins and minerals from whole food sources in all four-food groups? If a food group is omitted (for example, meats), is a suitable substitute provided? The four food groups are milk/milk products; meat/fish/poultry/eggs/legumes; fruits/vegetables; grains. For each food group omitted and not adequately substituted for, minus 10 points
6. Does it offer variety, in the sense that different foods can be selected each day? If you'd class it as "monotonous," give it a minus 10.
7. Does it consist of ordinary foods that are available locally (for example, in the main grocery stores) at the prices people normally pay? Or does the dieter have to buy special, expensive, or unusual foods to adhere to the diet? If you'd class it as "bizarre" or "requiring unusual foods," minus 10.

Diet Name	KCalories	Protein	Fat	Carbohydrate	Food Groups	Variety	Ordinary Foods	Total Score
Atkin's	Yes	Minus 10	Minus 10	Minus 10	Minus 30	Minus 10	Yes	30 Points
Banana-Milk	Minus 10	Minus 10	Minus 10	Yes	Minus 20	Minus 10	Yes	40 Points
Beverly Hills	Minus 10	Minus 10	Minus 10	Minus 10	Minus 30	Minus 10	Minus 10	10 Points
Scarsdale	Yes	Minus 10	Minus 10	Minus 5	Minus 10	Yes	Yes	65 Points
Cambridge	Minus 10	Minus 10	Minus 10	Minus 10	Minus 40	Minus 10	Minus 10	0 Points
Fasting	Minus 10	Minus 10	Minus 10	Minus 10	Minus 40	Minus 10	Minus 10	0 Points
High Roughage	Yes	Yes	Yes	Yes	Minus 5	Yes	Minus 5	90 Points
I Love NY	Minus 5	Yes	Yes	Yes	Yes	Yes	Yes	95 Points
Kempner's Rice	Yes	Minus 10	Minus 10	Yes	Minus 20	Minus 10	Yes	30 Points
Magic Mayo (Grapefruit)	Minus 10	Minus 10	Minus 10	Minus 10	Minus 20	Minus 10	Yes	30 Points
New Canadian	Yes	Yes	Yes	Yes	Yes	Yes	Yes	100 Points
Simeons' HCG	Minus 10	Minus 10	Minus 10	Minus 10	Minus 25	Yes	Minus 10	25 Points
Dr. Stillman	?	Minus 10	Minus 10	Yes	Minus 20	Minus 10	Yes	50 Points
UCLA Diet	Yes	Yes	Yes	Yes	Yes	Yes	Yes	100 Points
Weight Watchers	Yes	Yes	Yes	Yes	Yes	Yes	Yes	100 Points