Exercise, Obesity, and Weight Control

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HIGHLIGHT

"Physical inactivity is certainly a major, if not the primary, cause of obesity in the United States today. A certain minimal level of activity might be necessary for us to accurately balance our caloric intake to our caloric expenditure. With too little activity, we appear to lose the fine control we normally have to maintain this incredible balance. This fine balance amounts to less than 10 kcal per day, or the equivalent of one potato chip!"

INTRODUCTION

It is ironic that while millions of people are dying of starvation each year in most parts of the world, many Americans are dying as an indirect result of an overabundance of food. Further, billions of dollars are spent each year overfeeding the American public, which then leads to the spending of billions of dollars more each year on various weight loss methods. This review will investigate various aspects of overweight and obesity, and show how they are affected by physical activity. But first, we must define and differentiate between the terms overweight and obesity.

OVERWEIGHT, OBESITY AND THEIR ASSESSMENT

The terms *overweight* and *obesity* are often used interchangeably, but this is technically incorrect as they have different meanings. Overweight is defined as a body weight that exceeds the normal or standard weight for a particular person, based on his or her height and frame size. These standards are established solely on the basis of population averages. It is quite possible to be overweight according to these standard tables and yet have a body fat content that is average or even below average. For example, almost all college and professional football players are overweight by these tables, but few are overfat. There are also people who are within the normal range of body weights for their height and frame size by the standard tables, but who have, in fact, excessive body fat.

Obesity is the condition in which the individual has an excessive amount of body fat. This means that the actual amount of body fat, or its percentage of a person's total weight, must be assessed or estimated. A number of laboratory and field assessment techniques can provide reasonably accurate estimates of a person's body composition. Exact standards for allowable fat percentages, however, have not been established. But there is general agreement among clinicians and scientists that men over 25% body fat and women over 35% should be considered obese, and that relative fat values of 20% to 25% in men and 30% to 35% in women should be considered borderline obese.

PREVALENCE OF OBESITY AND OVERWEIGHT

The prevalence of obesity and overweight in the United States has increased dramatically over the past 30 years. On the basis of data from a large study conducted between 1976 and 1980 by the National Center for Health Statistics (National Center for Health Statistics, 1986), 28.4% of American adults aged 25 to 74 years are overweight. Between 13% and 26% of the U.S. adolescent population, 12 to 17 years of age, are obese, depending on gender and race, and an additional 4% to 12% are superobese. This represents a 39% increase in the prevalence of obesity when compared with data collected between 1966 and 1970. Equally alarming, there has been a 54% increase in the prevalence of obesity among children 6 to 11 years of age (Gortmaker et al., 1987).

It has also been demonstrated that the average individual in this country will gain approximately one pound of additional weight each year after the age of 25 years. Such a seemingly small gain, however, results in 30 pounds of excess weight by the age of 55 years. Since the bone and muscle mass decreases by approximately one half pound per year due to reduced physical activity, fat is actually increasing by 1.5 pounds each year. This means a 45 pound gain in fat over this 30-year period! It is no wonder that weight loss is a national obsession.

THE CONTROL OF BOD Y WEIGHT

It is important to have a basic understanding of how body weight is controlled or regulated in order to better understand how one becomes obese. The issue of how body weight is regulated has puzzled scientists for years. It is rather remarkable that the body takes in an average of about 2,500 kcal per day, or nearly one million kcal per year. The average gain of 1.5 pounds of fat each year, which we just discussed, represents an imbalance between energy intake and expenditure of only 5,250 kcal per year (using 3,500 kcal to represent the energy equivalent of a pound of adipose tissue), or less than 15 kcal per day. Even with a weight gain of 1.5 pounds of fat, the body is able to balance the food intake to within one potato chip per day of what is expended! That is truly remarkable.

The ability of the body to balance its intake and expenditure has led scientists to propose that body weight is regulated within a narrow range similar to the way in which body temperature is regulated. There is excellent evidence for this in the animal research literature (Keesey, 1986). When animals are force-fed or starved for various periods of time, their weight will increase or decrease markedly, but they will always return to their original weight, or to the weight of the control animals (for those animals that naturally continue to increase weight throughout their life span), when allowed to go back to their normal eating patterns.

Similar results have been found in humans, although the number of studies has been limited. Subjects placed on semi-starvation diets have lost up to 25% of their body weight, but regained that weight within months of returning to a normal diet (Keys et al., 1950). In overfeeding studies of Vermont prisoners, overfeeding resulted in weight gains of 15 to 25%, yet weight returned to its original level shortly after the completion of the experiment (Sims, 1976).

How is the body able to do this? The total amount of energy expended each day can be expressed in three categories: resting metabolic rate (RMR), the thermic effect of feeding (TEF), and the thermic effect of activity (TEA). RMR is your body's metabolic rate early in the morning following an overnight fast and eight hours of sleep. The term *basal metabolic rate* (BMR) is also used, but generally implies that the person sleeps over in the clinical facility where the metabolic rate measurement will be made. Most research today uses resting metabolic rate. It accounts for 60 to 75% of the total energy expended each day.

The TEF, which represents the increase in metabolic rate that is associated with the digestion, absorption, transport, metabolism, and storage of the ingested food, accounts for approximately 10% of the total energy expended each day. There is probably also a wastage component included in the thermic effect of a meal, where the body is able to increase its metabolic rate above that necessary for the processing and storage of the ingested food. This component may be defective in obese individuals. The TEA, which is simply the energy expended above resting metabolic rate levels necessary to accomplish a given task or activity, whether it be washing your face or taking a brisk 3-mile walk, accounts for the remainder.

The body makes very important adaptations in each of these three components of total energy expenditure when there are major increases or decreases in the energy intake. With very low calorie diets, there are decreases in the RMR, TEF, and TEA. The body appears to be attempting to conserve its energy stores. This is dramatically illustrated by the decreases reported in resting meta- bolic rate of 20 to 30% or more within several weeks after patients begin a very low calorie diet. Conversely, with overeating RMR, TEF, and TEA all increase to prevent the unnecessary storage of a large number of calories. It is quite possible that all of these adaptations are under the control of the sympathetic nervous system and play a major, if not the primary, role in controlling weight around a given set-point.

ETIOLOGY OF OBESITY

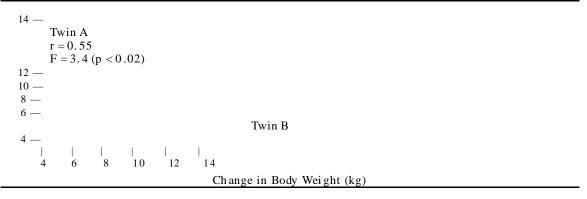
The results of recent medical and physiological research show that obesity can be the result of any one, or a combination of many, factors. Its etiology is not as simple or straightforward as was once believed. A number of experimental studies on animals have linked obesity to hereditary or genetic factors. Studies by Dr. Albert Stunkard at the University of Pennsylvania have shown a direct genetic influence on height, weight, and BMI (Stunkard et al., 1986a, 1986b, 1990).

A study from Laval University in Quebec, Canada, has provided possibly the strongest evidence of a significant genetic component in the establishment of obesity (Bouchard et al., 1990). With periods of overfeeding of identical monozygotic twins (1,000 kcal above maintenance levels, six out of every seven days), there was a threefold variation in the weight gained over 100 days between twin pairs, while there were relatively small differences within twin pairs. This is illustrated in Figure 15.1. Similar results were found for gains in fat mass, percentage body fat, and subcutaneous fat.

Obesity has also been experimentally and clinically linked with both physiological and psychological trauma. Hormonal imbalances, emotional trauma, and alterations in basic homeostatic mechanisms have all been shown to be either directly or indirectly related to the onset of obesity. Environmental factors, such as cultural habits, inadequate physical activity, and improper diets, also make major contributions to excessive fat gain. Thus, obesity is of complex origin, and the specific causes undoubtedly differ from one person to the next. Recognizing this fact is important both in the treatment of existing obesity and in the application of measures to prevent its onset.

FIGURE 15.1

Similarity within twin pairs of weight gain in response to a 1,000 kcal increase in dietary intake for 84 days of a 100 day period of study. Each point represents one pair of twins (A and B). The closer the points are to the diagonal line, the more similar the twins are to each other.



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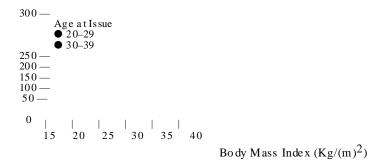
HEALTH IMPLICATIONS OF OVERWEIGHT AND OBESITY

There is an increased risk for general excess mortality associated with overweight and obesity. This relationship is curvilinear as is illustrated in Figure 15.2. A large jump in risk occurs when the body mass index (BMI) exceeds a value of 30 kg/m². The BMI is a simple ratio of body weight divided by height squared, and provides an estimate of obesity. The causes of the excess mortality associated with obesity and overweight include heart disease, hypertension, and diabetes.

It has been recognized since the 1940s that there are major gender differences in the way in which fat is stored or patterned in the body. Males tend to pattern fat in the upper regions of the body, particularly in the abdominal area, while females tend to pattern fat in the lower regions of the body, particularly in the hips, buttocks, and thighs. When obese, the male pattern is referred to as upper body, "apple-shaped," or android obesity, and the female pattern is referred to as lower body, "pear-shaped," or gynoid obesity. Research beginning in the late 1970s and early 1980s clearly established upper body obesity as a risk factor for heart disease, hypertension, stroke, elevated blood lipids, and diabetes (Björtorp et al., 1988). Further, upper body obesity appears to be more important as a risk factor for these diseases than total body fatness. With upper body obesity, the increased risk may be the result of the location of these depots in close proximity to the portal circulatory system.

FIGURE 15.2

Relation of body mass in dex to excess mortality.



From: Bray, G.A. (1985). Obesity: definition, diagnosis and disadvantages. *Medical Journal of Australia*, 142, S2–S8.

GENERAL TREATMENT OF OBESITY

In theory, weight control seems to be a very simple matter. The energy consumed by the body in the form of food must equal the total energy expended, which is the sum of the RMR, TEF, and TEA. The body normally maintains a balance between caloric intake and caloric expenditure. However, when this balance is upset, a loss or gain in weight will result. It would appear that both weight losses and weight gains are largely dependent on just two factors—dietary intake and habitual physical activity This now appears to be an oversimplification considering the results of the overfeeding study of monozygotic twins discussed earlier in this paper, where there was considerable variation in the weight gained for the same amount of overfeeding (Bouchard et al., 1990). Thus, not everyone will respond the same to the same intervention. This must be considered when designing treatment programs for individuals attempting to lose weight. Also, it is important for the individual trying to lose weight to understand this fact so that he or she will not get discouraged.

Many special diets have achieved popularity over the years, including the Drinking Man's Diet, the Beverly Hills Diet, the Cambridge Diet, the California Diet, Dr. Stillman's Diet, and Dr. Adkin's Diet. Each claims to be the ultimate in terms of effectiveness and comfort in weight loss. Some of the more recent diets have been developed for use either in the hospital or at home under the supervision of a physician. These are referred to as very low calorie diets, as they allow only 350 to 500 kcal of food per day. Most of these have been formulated with a certain amount of protein and carbohydrate in order to minimize the loss of fat-free tissue. Research has shown that many of these diets are effective, but no one single diet has been shown to be any more effective than any other. Again, the important factor is the development of a caloric deficit, while maintaining a balanced diet that is complete in all respects with regard to vitamin and mineral requirements. The diet that meets these criteria and is best suited to the comfort and personality of each individual is the best diet.

Generally, improper eating habits are at least partially responsible for most weight problems, so any given diet should not be looked on as a "quick fix." The individual should be instructed to make *permanent* changes in his or her dietary habits, particularly reducing the intake of fat and simple sugars. Just eating a low fat diet will gradually bring weight down to desirable levels for most individuals without restricting the quantity of food eaten. For most individuals, reducing the total caloric intake by not more than 250 to 500 kcal per day would be sufficient to accomplish the desired weight loss goals.

Behavior modification has been proposed as one of the most effective techniques for dealing with weight problems. By changing basic behavior patterns, many associated with eating, major weight losses have been achieved. Further, these weight losses appear to be much more permanent, in that the weight is less likely to be regained. This approach appeals to most people since the techniques seem to make sense. For example, individuals might not have to reduce the amount of food they eat but simply agree that all eating will be done in one location. Or, the individual will be allowed to eat as much as he or she wants, but it must be eaten with the first helping—no second helpings! There are a number of very simple things that can be done to regulate the individual's eating behavior which can result in substantial weight loss.

Hormones and drugs have also been used to assist patients in weight loss, mainly through increasing their RMR. Surgical techniques are also used in the treatment of extreme obesity, but only as a last resort when other treatment procedures have failed and the obesity constitutes a life-threatening situation.

THE ROLE OF PHYSICAL ACTIVITY IN WEIGHT REDUCTION AND CONTROL

Inactivity is a major cause of obesity in the United States. In fact, inactivity might be a far more significant factor in the development of obesity than overeating. Thus, exercise must be recognized as an essential component in any program of weight reduction or control.

The Excess Post-Exercise Oxygen Consumption (EPOC)

It has often been stated that physical activity has only a limited influence on changing body composition, and that even exercise of a vigorous nature results in the expenditure of too few calories to lead to substantial reductions in body fat. Yet, research has conclusively demonstrated the effectiveness of exercise training in promoting major alterations in body composition. How do we account for this apparent conflict?

When estimating the energy cost of an activity, it is typical to multiply the average or steady-state rate of energy expenditure for a specific activity times the minutes engaged in that activity. However, the metabolism remains elevated following exercise. This was, at one time, referred to as the oxygen debt, but is now referred to as the excess post-exercise oxygen consumption (EPOC). The recovery of the metabolic rate back to pre-exercise levels can require several minutes for light exercise, several hours for very heavy exercise, and up to 12 to 24 hours or even longer for prolonged, exhaustive exercise.

The EPOC can add up to a substantial energy expenditure when totaled over the entire period of recovery. If the oxygen consumption following exercise remains elevated by an average of only 50 ml/min or 0.05 liter/min, this will amount to approximately 0.25 kcal/min or 15 kcal/hr. If the metabolism remains elevated for five hours, this would amount to an additional expenditure of 75 kcal that would not normally be included in the calculated total energy expenditure for that particular activity. This major source of energy expenditure, which occurs during recovery, but is directly the result of the exercise bout, is frequently ignored in most calculations of the energy cost of various activities. If the individual in this example exercised five days per week, he or she would have expended 375 kcal, or lost the equivalent of approximately 0.1 pounds of fat in one week, or 1.0 pounds in 10 weeks, just from the additional caloric expenditure during the recovery period alone.

Changes in Weight and Body Composition with Exercise Training

A number of studies have shown major changes in both weight and body composition with exercise training. In one study, the researchers investigated the changes in body composition with diet, exercise, and a combination of diet and exercise (Zuti & Golding, 1976). A caloric deficit of 500 kcal per day was maintained by each of three groups of adult women during a 16-week period of weight loss. The diet-only group reduced their normal daily intake by 500 kcal per day, but did not alter their activity levels. The exercise-only group increased their activity by 500 kcal per day, but did not alter their diet. The combination group reduced their caloric intake by 250 kcal and increased their activity by 250 kcal. While there were similar decreases in body weight, the two groups that exercised lost substantially more body fat. A major difference between the two exercise groups and the diet-only group was the gain in fat-free body mass with exercise and its loss with diet-only

In a second study, 72 mildly obese male subjects were assigned to one of several treatment programs, which included either exercise or nonexercise in combination with different dietary treatments. While the exercise and nonexercise groups lost similar amounts of weight, the exercise group lost significantly more fat weight and did not lose fat-free mass. The nonexercising group lost a significant amount of fat-free mass (Pavlou et al., 1985).

Not all studies have been able to demonstrate such dramatic changes in weight and body composition with exercise training. However, most studies have found similar trends, in that total weight decreases, fat weight and relative body fat decrease, and fat-free mass is either maintained or increases (Ballor & Keesey, 1991; Stefanick, 1993; Wilmore, 1983). While most of these studies have used aerobic training, several studies have shown impressive decreases in body fat and increases in fat-free mass with resistance training. The evidence is clear that exercise is an important part of any weight loss program. However, it is also clear that to maximize decreases in body weight and body fat, it is necessary to combine exercise with decreases in caloric intake.

Mechanisms for Change in Body Weight and Composition

In looking for ways to explain the above changes in body weight and composition with exercise, it is important to consider both sides of the energy balance equation. When evaluating energy expenditure, it is useful to consider each of the three components of energy expenditure: RMR, TEF, and TEA. When evaluating energy intake, it is also important to consider the energy that is lost in the feces (energy excreted), which is generally less than 5% of the total calories ingested.

The Energy Balance Equation

Energy Intake 2 Energy Excreted 5 RMR 1 TEF 1 TEA

It has been contended that exercise will stimulate the appetite to such an extent that food intake will be unconsciously increased to at least equal that expended during exercise. Jean Mayer, world-famous nutritionist, reported a number of years ago that animals exercising for periods of from 20 minutes up to one hour per day had a lower food intake than nonexercising control animals (Mayer et al., 1954). He concluded from this and other studies that when activity is reduced to below a certain minimum level, a corresponding decrease in food intake does not occur and the animal or human begins to accumulate body fat. This has led to the theory that a certain minimum level of physical activity is necessary before the body can precisely regulate or fine-tune food intake to balance energy expenditure. A sedentary lifestyle might reduce the ability of the fine-tuning device to control food intake precisely, resulting in a positive energy balance and a weight gain.

Exercise does, in fact, appear to be a mild appetite suppressant, at least for the first few hours following intense exercise training. Further, studies have shown that the total number of calories consumed per day does not change when one begins a training program, even with a greatly increased caloric expenditure. While some have interpreted this as evidence that exercise does not affect appetite, it might be more accurate to conclude that appetite was affected in that caloric intake did not increase in proportion to the additional caloric expenditure resulting from the exercise program. In studies conducted on rats, the food intake of male rats appears to be reduced with exercise training, while female rats tend to eat the same or even more than nonexercising control rats (Oscai, 1973). There is no obvious explanation for this gender difference, and so far similar results have not been reported in humans.

It is possible that the decrease in appetite occurs only with intense levels of exercise in which the increased catecholamine levels might suppress the appetite. It is also possible that the increased body temperature that accompanies high intensity activity, or almost any activity performed under hot and humid conditions, leads to a decreased appetite. When the weather is hot, or when there is an elevated body temperature as a result of illness, there is a loss in the desire for food. This might also explain why there is little or no desire to eat after a hard running workout, but a relatively strong craving for food following a hard swimming workout. In the pool, providing the water temperature is well below core temperature, the heat generated by exercise is lost very effectively, making it possible to better regulate core temperature.

The effect of exercise on the components of energy expenditure became a major topic of interest among researchers in the late 1980s and early 1990s. Of obvious interest is how exercise training might affect the RMR, since it represents 60% to 75% of the total number of calories expended each day. If a 25-year-old male's total caloric intake was 2,700 kcal per day, and his RMR accounted for just 60% of that total (0.60 x 2700 = 1620 kcal RMR), just a 1% increase in his RMR would be an extra 16 kcal expended each day, or 5,913 kcal per year. This small increase in RMR alone would account for the equivalent of a 1.7 pound fat loss per year!

The role of physical training in increasing RMR has not been totally resolved. Several cross-sectional studies have found that highly trained runners have higher RMRs than individuals of similar age and size who are untrained. However, other studies have not been able to confirm this (Poehlman, 1989). Few longitudinal studies have been conducted where untrained individuals are trained for a period of time and their changes in RMR are determined. Those longitudinal studies that have been conducted suggest that there might be an increase in RMR following training, but the data are not conclusive (Broeder et al., 1992). Since RMR is closely related to the fat-free mass of the body, there is now interest in the possible use of resistance training to increase the fat-free mass in an attempt to increase RMR.

A number of studies have been conducted on the role of individual bouts of exercise and exercise training in increasing the TEF. It is reasonably clear that a single bout of exercise, either before or after a meal, increases the thermic effect of that meal. Less clear is the role of exercise training on the TEF. Studies have shown increases, others have shown decreases, and still others have shown no effect of exercise training on the TEF.

With respect to the specific loss of body fat with exercise, several research studies have pointed to the possible role of human growth hormone as being responsible for the increased fatty acid mobilization during exercise. Growth hormone levels do increase sharply with exercise and remain elevated for up to several hours in the recovery period. Other research has suggested that with exercise the adipose tissue is more sensitive to the sympathetic nervous system, or to the levels of circulating catecholamines, which would result in increased lipid mobilization. More recent research suggests that a specific fat-mobilizing substance, which is highly responsive to elevated levels of activity, is responsible. Thus, it is impossible to state with certainty which factors are of greatest importance in mediating this response.

Spot Reduction, Other Myths, and Exercise Devices

Many individuals, including athletes, believe that by exercising a specific area of the body, the fat in that localized area will be utilized, thus reducing the locally stored fat. Several early research studies reported results that tended to support the concept of spot reduction. However, later research suggested that spot reduction is a myth and that exercise, even when localized, draws from all of the fat stores of the body, not just from the local depots.

One study utilized outstanding tennis players to investigate the phenomenon of spot reduction, theorizing that they would be ideal subjects for studying spot reduction since they could act as their own controls, in that the dominant arm exercises vigorously every day for several hours, while the nondominant arm is relatively sedentary (Gwinup et al., 1971). They postulated that if spot reduction was a reality, the nondominant (inactive) arm should have substantially more fat than the dominant (active) arm. In fact, while the arm girths were substantially greater in the dominant arm due to exercise-induced muscle hypertrophy, there were absolutely no differences between the arms in subcutaneous skinfold fat thicknesses. Another study reported no difference in the rate of change in fat cell diameters at the abdomen, subscapular and gluteal fat biopsy sites following a 27-day intense sit-up training program, indicating a lack of specific adaptation at the site of exercise training (Katch et al., 1984). Researchers now theorize that fat is mobilized from either those areas of highest concentration or equally from all areas, thus negating the spot reduction theory. Changes in girth, such as the abdominal girth, can occur with exercise training, but these changes are the result of increased muscle tone, not fat loss.

During the latter part of the 1980s and early 1990s, various professional exercise groups promoted low intensity aerobic exercise to increase the loss of body fat. It has been clearly established that the higher the exercise intensity, the greater the body's reliance on carbohydrate as an energy source. With high intensity aerobic exercise, carbohydrate might supply 65% or more of the body's energy needs. These groups theorized that low intensity aerobic training would allow the body to use more fat as the energy source, thus more effectively reducing the body's fat stores. While it is true that the body uses a higher percentage of fat for energy at lower intensities of exercise, the total number of calories expended from the use of fat is not different. Furthermore, there are substantially more calories expended during the higher intensity workout for the same period of time.

With the popularity of exercise increasing, there are many gimmicks and gadgets on the market. While some of these are legitimate and effective, many are of no practical value for either exercise conditioning or weight loss. Three such devices were evaluated to determine the legitimacy of their claims: the Mark II bust developer; the Astro-Trimmer exercise belt; and the Slim-Skins vacuum pants. The last two devices claimed to take inches off the abdomen, hips, buttocks, and thighs in a matter of minutes, while the first device claimed to add two to three inches to the bust within three to seven days. All three failed to produce any changes whatsoever when evaluated in tightly controlled scientific studies (Wilmore et al., 1985a, 1985b). To gain the benefits from exercise it is necessary actually to do the work!

REFERENCES

- Ballor, D.L., & Keesey, R.E. (1991). A meta-analysis of the factors affecting exercise-induced changes in body mass, fat mass and fat-free mass in males and females. *International Journal of Obesity*, 15, 717–726.
- Bjorntorp, P., Smith, U., & Lönnroth, P. (1988). Health implications of regional obesity. *Acta Medica Scandinavica Symposium Series* No. 4, Stockholm: Almqvist & Wiksell International.
- Bouchard, C., Tremblay, A., Després, J.-P., Nadeau, A., Lupien, P.J., Thériault, G., Dussault, J., Moorjani, S., Pinault, S., & Fournier, G. (1990). The response to long-term overfeeding in identical twins. New England Journal of Medicine, 322, 1477–1482.
- Bray, G.A. (1985). Obesity: definition, diagnosis and disadvantages. Medical Journal of Australia, 142, S2-S8.
- Broeder, C.E., Burrhus, K.A., Svanevik, L.S., & Wilmore, J.H. (1992). The effects of either high intensity resistance or endurance training on resting metabolic rate. *American Journal of Clinical Nutrition*, 55, 802–810
- Gortmaker, S.L., Dietz, W.H., Jr., Sobol, A. M., & Wehler, C.A. (1987). Increasing pediatric obesity in the United States. *American Journal of Diseases of Children*, 141, 535-540.

- Gwinup, G., Chelvam, R., & Steinberg, T. (1971). Thickness of subcutaneous fat and activity of underlying muscles. *Annals of Internal Medicine*, 74, 408–411.
- Katch, F.I., Clarkson, P.M., Kroll, W., McBride, T., & Wilcox, A. (1984). Effects of sit up exercise training on adipose cell size and adiposity. Research Quarterly for Exercise and Sport, 55, 242–247.
- Keesey, R.E. (1986). A set-point theory of obesity. In K.D. Brownell & J.P. Foreyt (Eds.), *Handbook of Eating Disorders: Physiology, Psychology, and Treatment of Obesity, Anorexia, and Bulimia* (pp. 63–87). New York: Basic Books.
- Keys, A., Brozek, J., Henschel, A., Mickelsen, O., & Taylor, H.L. (1950). *The Biology of Human Starvation*. Minneapolis: University of Minnesota Press.
- Mayer, J., Marshall, N.B., Vitale, J.J., Christensen, J.H., Mashayekhi, M.B., & Stare, F.J. (1954). Exercise, food intake, and body weight in normal rats and genetically obese adult mice. *American Journal of Physiology*, 177, 544–548.
- National Center for Health Statistics. (1986). *Health, United States, 1986.* DHHS Publ. No. (PHS) 87-1232. Public Health Service, Washington DC: U.S. Government Printing Office, December 1986.
- National Institutes of Health. (1985). Health implications of obesity: National Institutes of Health Consensus Development Conference Statement. *Annals of Internal Medicine*, 103, 1073–1077.
- Oscai, L.B. (1973). The role of exercise in weight control. Exercise and Sport Sciences Reviews, 1, 103-123.
- Pavlou, K.N., Steffee, W.P., Lerman, R.H., & Burrows, V. (1985). Effects of dieting and exercise on lean body mass, oxygen uptake, and strength. *Medicine and Science in Sports and Exercise*, 17, 466–471.
- Poehlman, E.T. (1989). A review: exercise and its influence on resting energy metabolism in man. *Medicine and Science in Sports and Exercise*, 21, 515–525.
- Sims, E.A.H. (1976). Experimental obesity, dietary-induced thermogenesis and their clinical implications. *Clinics in Endocrinology and Metabolism*, 5, 377–395.
- Stefanick, M.L. (1993). Exercise and weight control. Exercise and Sport Sciences Reviews, 21, 363-396.
- Stunkard, A.J., Foch, T.T., & Hrubec, Z. (1986a). A twin study of human obesity. *Journal of the American Medical Association*, 256, 51-54.
- Stunkard, A.J., Harris, J. R., Pedersen, N.L., & McClearn, G.E. (1990). The body-mass index of twins who have been reared apart. *New England Journal of Medicine*, 322, 1483–1487.
- Stunkard, A.J., Sorensen, T.I.A., Hanis, C., Teasdale, T.W., Chakraborty, R., Schull, W.J., & Schulsinger, F. (1986b). An adoption study of human obesity. *New England Journal of Medicine*, *314*, 193–198.
- Wilmore, J.H. (1983). Body composition in sport and exercise: Directions for future research. *Medicine and Science in Sports and Exercise*, 15, 21–31.
- Wilmore, J.H., Atwater, A.E., Maxwell, B.D., Wilmore, D.L., Constable, S.H., & Buono, M.J. (1985a). Alterations in body size and composition consequent to Astro-Trimmer and Slim-Skins training programs. *Research Quarterly for Exercise and Sport*, 56, 90–92.
- Wilmore, J.H., Atwater, A.E., Maxwell, B.D., Wilmore, D.L., Constable, S.H., & Buono, M.J. (1985b). Alterations in breast morphology consequent to a 21-day bust developer program. *Medicine and Science in Sports and Exercise*, 17, 106–112.
- Zuti, W.B., & Gelding, L.A. (1976). Comparing diet and exercise as weight reduction tools. *Physician and Sportsmedicine*, 4, 49–53.