A New Approach to Obesity Management

Dennis W. Remington, M.D.

Why am I so fat when I eat so little? Why can't I lose weight? The harder I try to diet, the fatter I seem to get. Please help me, doctor." How often do you hear such heart-rending pleas from unhappy, unhealthy patients? Until very recently, we physicians have had almost nothing to offer the patient suffering from obesity. They have turned in great numbers to the commercial weight loss programs and to various forms of formula products in a desperate, futile, and often dangerous attempt to lose weight. New research and careful evaluation of the problem has led to some exciting new concepts and treatment principles for obesity.

Old Concepts of Obesity

For hundreds of years, obesity has been considered to be a product of excess food intake. Those people with the obese bodies who claimed to eat very little were thought to be either liars or to have very efficient bodies which were still being fed excessively. These ideas came from a direct application of one of the Laws of Thermodynamics, which states that "energy input into a system equals energy output." Several basic assumptions were made based on this law. Since fat is a form of stored energy, it was concluded that excess fat stores must be the result of excess energy (food) intake. It was also concluded that the only way to eliminate the excess fat is to create a negative energy balance by forcing the obese person to eat less food than is required to maintain homeostasis, thus making the body rely on the stored fat for its energy needs.

These basic assumptions seem very logical and on superficial examination seem to hold true. We have probably all gained a few pounds by eating excessively on a vacation or over the Christmas holidays. Most of us have been able to lose a few pounds merely by cutting back our food intake a little after these weight gains. These basic assumptions have seemed so obvious that they have been taught as gospel truths for many years without even attempting to verify them with proper scientific experiments.

In recent years, these basic assumptions have finally been tested with a number of different studies. Several studies have compared the eating habits of obese people to those of thin people and have shown that there is no difference between the two groups (Johnson 1956; Stefanik 1959; Coll 1979; Wooley 1979). In fact, some studies done in summer camp situations have shown that thin kids eat considerably more than the fat kids—a difference which cannot be explained by a difference in

activity levels (Stefanik 1959).

Force feeding studies have been done with animals as well as humans in which the subjects were given huge excesses of food (Sims 1973; Goldman 1976; Passmore 1971). Despite eating 10,000 calories per day, some of these subjects didn't gain weight. Those who did only gained about 14 percent above their previous weight, and then leveled off even with continuous excess food intake. When the force feeding was stopped, the weight quickly returned to the prestudy level with no attempt at all to diet.

Food deprivation studies have shown that when rats are given a reduced caloric diet, weight is lost, but as soon as they are allowed to eat "normally," the weight quickly returns. Even when these restricted rats are not allowed any more food than the amount eaten by nonrestricted controls, their weight will soon reach the same level as the controls (Brooks 1946; Levitsky 1976). Rats who are fasted for several days lose weight and then have to be kept on only 60 percent of their previous food intake to keep stable at the new "artificial" weight. People respond in the same way—weight lost through food restriction can be kept off only through continuing to severely limit calories.

Since it is well known that some obese people will not lose weight while eating less than 1,000 calories daily and some thin people will not gain weight while eating in excess of 10,000 calories daily, food intake is only part of the problem. In fact, the number of calories eaten may have a negative correlation with obesity—the less you eat, the fatter you may become (Bennett 1982; Wood 1983). This statement may seem ridiculous in view of traditional concepts about obesity but should seem entirely logical when viewed in the light of new information. The problem with obesity seems not to be a problem of excess food intake but rather a problem of the regulating mechanism that controls the weight.

Regulation of Body Weight

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Most parameters within the body are kept closely regulated. Body temperature, acid-base balance of the blood, electrolyte balance, and many other measurements are constantly kept within very narrow limits. It makes sense that the body would also try to keep body weight within a narrow range, and this in fact seems to happen. Few people would challenge the concept that at least some people maintain a remarkably constant body weight with no effort whatsoever to control their food intake. These people appear to have a weight regulating mechanism that effectively controls the weight at a constant level. Obese people also seem to have a regulatory mechanism that closely controls their weight as long as they eat according to the hunger drives from their feeding centers. Marked fluctuations are often observed as they frequently restrict their food intake by dieting. If observed closely, the majority of them soon return to their "natural" weight. The weight level chosen by the weight regulating mechanism has been referred to as the "setpoint," a term which has become very popular in recent magazine articles.

There is a great deal of proof for the existence of a weight regulating mechanism. Experimental subjects who undergo either force feeding or starvation quickly return to their previous weight (Sims 1973; Goldman 1976; Brooks 1946; Levitsky 1976). Lesion places in the hypothalamic area in the brain of experimental animals cause these animals to either gain or lose a considerable amount of weight, depending on the location of the lesion (Powley 1970; Powley 1978), and then to defend that weight in the same manner as normal rats defend their setpoint weight (Keesey 1973).

The extremely poor success rate for weight control through dieting is another proof of controlled weight. Earlier statistics have suggested that less than 5 percent of those who begin a diet will successfully lose forty or more pounds, and up to 98 percent of those who lose weight will have regained it all in less than two years (Stunkard 1977). Statistics for more recent programs are more impressive but may be a product of better patient selection rather than any real improvement in treatment methods (Stunkard 1977).

The weight regulating mechanism seems to function by influencing food intake as well as by directing the body to either waste or conserve energy.

Control of Food Intake

There are a great number of factors that influence food intake, but all of them seem to influence a "feeding center" and a "satiety center" within the hypothalamus of the brain (Leibowitz 1970; Bray 1976; Jordan 1969; Bray 1973). When the body has a biological need for food, the feeding center is stimulated and the person has a desire to eat. Usually eating will soon begin and continue until adequate food intake has occurred for the body's needs. Then the satiety or satisfaction center will be activated, a feeling of fullness follows, and then eating ceases. These centers in

Dennis W. Remington, M.D., is a physician in bariatrics with a private practice in Provo. He serves as a counselor in the BYU 134th Ward bishopric.

the brain act like computers, receiving information from many parts of the body and then using this information to "decide" whether to give us a message of hunger or satiety.

When food is eaten, the smell, taste, mouth feel, chewing, and swallowing are measured and that information is sent to the brain. The amount of stomach distension created by the food is determined, and that information is sent to the brain. An interesting study was done in which a tube passed directly into the stomach of volunteers was hooked up to a food pump. The subjects were instructed to keep pumping food until they felt completely satisfied. When the smell, taste, and chewing were bypassed, it took considerably more food to produce satiety than when food was eaten in a conventional manner (Jordan 1969).

From the stomach, food soon enters the intestines where a vast number of hormones, enzymes, and regulatory substances are released which further influence the eating. The amount of food energy as well as the composition of the food seems to be measured and that information is sent to the brain. From the intestines, nutrients are absorbed into the blood stream, and many receptors throughout the body measure the sugar, fat, and protein levels. Stores of fat, sugar, and possibly protein are measured, and that information also influences the brain centers.

This food regulating system seems to be a fail-safe system that has many checks to assure adequate intake of nutrients occurs. This system is very difficult to fool although many dietary efforts have attempted to do so. As an example, some reducing diet programs suggest eating huge volumes of low calorie vegetables to fill the stomach, thus inducing satiety. Anyone who has tried this knows it just doesn't work, and hunger gets progressively worse as more and more measuring systems report their needs are not being satisfied. As the weight starts to fall, the hunger drive gets more and more intense until the person becomes preoccupied with food and finds it almost impossible to resist the powerful self-preservation drives. Very restrictive diets can artificially suppress the hunger drive for a time, but almost invariably extreme hunger will again return to defeat the dieter.

Control of Energy Balance

While the hunger drive is getting stronger and more unmanageable, many other changes are occurring within the body in response to food restrictions. The body has an amazing number of biological systems that can be activated and developed to conserve energy and protect the fat stores in response to perceived starvation (Jessen 1980; Nicholls 1979; Rothwell 1980; Rothwell, Stock 1980; Bray 1979). The metabolic rate goes down dramatically as the various bodily processes slow down to conserve energy. There is a decrease in brown fat activity. Brown fat allows thin people to burn large amounts of energy to produce heat which is then lost from the system as an alternative to storing that energy as fat. In obese people, the low brown fat activity may conserve energy but results in an impaired thermogenesis in response to cold (Jessen 1980; Nicholls 1979; Rothwell 1980).

Obese people also have reduced levels of sodium-potassium ATPase, an enzyme which powers the sodium-potassium pump mechanism (Rothwell 1980). This pump is responsible for pumping potassium into the cells and sodium out of the cells to keep the proper electrolyte balance. Thin people can waste energy by merely pumping sodium and potassium across the cell membranes in a futile cycle in which nothing useful is accomplished except the wasting of energy. The decreased pump activity may save energy, but may also contribute to some of the other problems observed with obesity. For example, low levels of intracellular potassium may be a major factor in the insulin resistance seen almost universally in the obese (Anderson 1969).

With restricted calorie intake, especially with low carbohydrate intake, some interesting changes occur with thyroid hormones. T3 levels drop rather dramatically, but reverse T3 levels increase so that conventional lab tests would not detect any changes (Sussman 1982). There is resistance to the action of T3 at the nuclear and mitochondrial membranes, resulting in a slowdown of activity within these cellular organelles (Danforth 1980). The dieter becomes clinically hypothyroid, but the lab tests don't detect it, and adding thyroid doesn't help much if any. Exogenous thyroid stimulates gluconeogenesis and valuable muscle tissue is lost to provide the glucose required by the body to replace that missing from the diet (Bray 1973).

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Obese people develop resistance to insulin which results in higher insulin levels—four to six times higher on a fasting basis than the levels of thin people (Driesberg 1976). High levels of insulin promote obesity by converting glucose from the blood stream into fat and then storing the fat inside the fat cells (Bray 1976). Insulin also inhibits the metabolism of fat for fuel and thus causes the obese person to rely more on sugars for their primary fuel source. Insulin by itself will make experimental animals fat even when given only enough food that would keep their litter mates lean

(Renold 1972). Persistent dieting or consistently eating mineral-depleted foods contributes to the problem, as deficiencies in potassium, zinc, chromium, calcium, magnesium and probably other minerals all cause insulin resistance. The insulin resistance may be controlled by the weight regulating mechanism, as this is an ideal way to conserve the limited food intake and protect the fat stores.

An enzyme called adipose tissue lipoprotein lipase also seems to play a major role in protecting the fat stores. This enzyme is found in higher levels in the obese than in the thin (Greenwood 1980; Schwartz 1980). Its role is to remove fat from its protein carrier in the blood and then to place it in the fat cells for storage. An interesting study (Renold 1972) with moderately obese men showed that when weight was lost, this enzyme became elevated ninefold and stayed markedly elevated as long as the weight remained down. A year after this study was completed, these men were again evaluated, and all of them except one had regained the lost weight. In fact, the average weight was 105 percent of their previous weight, a gain of nearly ten pounds for each person. The lipase levels had fallen, and were now only slightly higher than they were before. This enzyme may be a major factor in causing fat to be regained twenty times faster after food restriction than it would otherwise be gained (Boyle 1978).

These biological systems that conserve energy and protect the fat stores are beautifully orchestrated defense mechanisms to protect our bodies from starvation. Any restriction in food intake whether actual starvation, dieting, or prolonged periods without eating will all trigger the development of these defense mechanisms and ultimately program the body to conserve energy and store fat more effectively. For example, rats fed only once daily will get twice as fat as those who are allowed to eat all day long, even though the food intake is the same (Fábry 1967). The same thing probably happens in people, as the vast majority of the grossly overweight people that we have seen are essentially one-meal-a-day eaters. Diets seem to function the same way, as chronic dieters train themselves to live on very little food and seem to gain a little extra weight with each new dietary effort (Sclafani 1979).

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Factors That Influence the Setpoint

The human body seems to be designed with two great advances over some lower forms of life; the ability to be very mobile, and the ability to store energy. These two functions are not at all compatible; the more energy that is stored, the less mobile a person becomes. The weight regulating mechanism seems to be programmed to choose a weight level suitable for our needs based on the kind of information it gets from the various sensors throughout the body. If it receives frequent messages that caloric needs or needs for vitamins and minerals are not being met, then it chooses to raise the setpoint and store more fat as soon as any extra energy can be diverted into fat stores. Thus dieting or missing meals to save calories unwittingly programs the body to become fatter in the long term.

In addition to the amount of food having an effect on the setpoint level, the type of food also plays an important role. High fat levels in the diet are well known to cause increased body fat in animals as well as humans (Sclafani 1979). This fat gain may occur as a direct result of dietary fats raising the setpoint (Keesey 1980). High fat foods have an extremely high caloric density, most of them being ten to eighty times more concentrated than some of the vegetables. By the time enough chewing, swallowing, and gastric distension occurs to satisfy our needs, excessive amounts of high fat foods may be ingested—more than can be wasted—leaving no alternative but to store the excess as fat. High fat intake is also associated with a dramatic decrease in voluntary activity. We may have all experienced this after a large holiday dinner high in fat content following which we feel like just sitting and doing nothing for a number of hours.

Refined carbohydrates (especially sugar) may also directly raise the setpoint (Sclafani 1979; Keesey 1980). In experimental animals, sugars are well known to cause excess fat gain. Refined sugars are absorbed very quickly and stimulate high insulin levels which quickly drive these sugars into fat stores. Sugar that should have provided enough energy for many hours may be absorbed and placed into fat storage within one hour causing strong hunger drives and the need to eat a short time later. Continued eating in this fashion then causes the ingestion of more calories than can be wasted, again leading to excess fat storage. Obtaining many of the needed calories by using these refined foods which have been stripped of their minerals may also play a major role in causing obesity by causing insulin resistance and interfering with metabolism.

Activity levels seem to play a major role in determining the level of the setpoint (Keesey 1980). A body with high mobility and very little fat storage provides definite survival value in some situations. A tribe of Indians in Mexico and a North African tribe are hunters who have a unique way of obtaining food. They chase the animals on foot

until they drop from exhaustion. Something about that type of activity seems to convince the weight regulating mechanism of the need for a streamlined body with very little fat stores. The same situation seems to be present in long distance runners and other endurance athletes—very little body fat with no need to restrict or in any way control the calories. Inactivity results in just the opposite effect; the storage of extra fat. Many people have reported gains of twenty to thirty pounds or so after an injury forces them to be inactive or after giving up some type of active work. It is as if the body is naturally programmed for a moderate amount of fat storage and is only influenced to store less fat when the person is very active. Thus an inverse relationship exists between activity and fat stores—the less active a person is, the more fat storage occurs. The chronic dieter whose body gives messages to move slowly and conserve energy just doesn't feel like exercising and the ensuing inactivity together with frequent food restriction combine to make the problem worse.

Stress also seems to play a major role in moving the setpoint to a higher level (Keesey 1980). Very frequently stressful situations result in rapid weight gains even when the patient claims to be eating no more food than usual. The body seems to respond to certain situations by putting on excess fat as if that would provide some form of protection. Genetics also influence the setpoint level (Keesey 1980). Certain drugs like cortisone and anti-depressants also seem to raise the setpoint level. In addition to the effect of the setpoint, there are other unrelated factors that influence body weight.

Other Factors That Influence Obesity

Although the weight regulating mechanism has a powerful influence over eating behavior, humans have a remarkable capacity to disregard hunger and satiety drives and eat for other reasons. By itself this is not a problem, because thin people seem to overeat, eat because of stress, use food for entertainment or rewards, and disregard satiety drives just as often as do obese people. In obese people this nondirected eating may cause more energy intake than the body can waste while thin people seem to have the capacity to merely waste the excess. This type of eating probably only causes relatively small weight gains, but most obese people seem to feel guilty about this type of behavior and alternate it with periods of dieting, missed meals, or other forms of deprivation. These periods of dieting are probably far more responsible for weight gain in the long term than is the overeating.

Eating food or drinking calories in response to thirst may also be a major problem in inducing obesity.

Eating food or drinking calories in response to thirst may also be a major problem in inducing obesity. Large numbers of excess calories can be ingested in this manner. Even worse than the number of calories is the fact that most drinks provide very little satisfaction and are already processed or refined so that the nutrients are absorbed quickly, leading to high insulin output with all its obesity inducing properties. Caffeine contained in many drinks also is well known to stimulate insulin production (Briggs 1979) and may play a role in fat production. Even artificial sweeteners affect the weight through directly raising the setpoint, causing insulin production, or in some other way causing weight gain. Experimental rats given artificial sweeteners in their water ate more and became fatter than control rats (Sussman 1982).

Obesity Scenario

The progression of typical obesity may occur something like this: A high school girl decides that she is too fat compared to current fashion standards and believing that the problem has resulted from excess food intake begins to cut back either with a formal diet or by starting to skip meals. Some weight is quickly lost, but the hunger becomes difficult to overcome. After a few days, the weight loss slows down and she begins to stray from her diet as the hunger gets harder to deny. The weight then gradually returns, usually to a level a little higher than before.

With each new dietary effort it becomes more difficult to lose weight and a more restrictive diet needs to be followed. The weight seems to return more quickly each time, and she then feels the need to seek help through a commercial weight loss group or with some extreme form of diet such as a semistarvation formula drink. She becomes progressively more tired, weak, and inactive and begins to get very discouraged. She gets hungrier and becomes more and more obsessed with food. Frequent binges begin to occur, and she relies more and more on the high fat, high sugar foods that seem to be so much more satisfying to her. She begins to feel very guilty and is concerned that she must have a character disorder because of her inability to control her weight and her eating. She becomes depressed and doesn't feel good physically; her headaches become worse, and she may develop problems like hypertension, hyperlipidemias, diabetes, cholelithiasis, and a myriad of other problems.

Her friendly neighborhood doctor diagnoses her problems and tells her how weight loss would really help. He tells her that she really needs to stop

overeating and may even suggest a diet program to follow. She looks at the 1200 calorie exchange diet sheet that he has given her and patiently explains that she is already eating less than that. She can tell by his tactful response that he simply doesn't believe her. She is told that she needs to be more honest with herself, count her calories more carefully, be even more restrictive, or go someplace else for help with her diet. This makes her feel even more guilty and inadequate, so she stops on the way home at the donut shop to feed the constant aching hunger within her, to drown her sorrows, and to provide a little boost in her energy to enable her to drag through the rest of the day.

The depression so often seen with obesity may influence or even produce alterations in the neurohormones in response to malnourishment from dieting.

The etiology and pathogenesis of obesity and its associated problems are now much better understood. Obesity itself is a product of a high setpoint. Dieting stimulates many adaptive responses which effectively conserve energy, make the body more efficient, and protect the fat stores. It becomes harder to lose weight and easier to gain it as the body becomes programmed for more fat storage. At the beginning of a diet, weight is lost easily and quickly as the body gives us a considerable amount of muscle protein with its attendant water to provide needed fuel. This may be in part an adaptive response to make the body more streamlined by getting rid of nonessential muscle, which requires a lot of energy to maintain. This muscle wasting is enhanced by the inactivity usually associated with dieting.

Further muscle is lost as the body takes steps to inhibit the metabolism of fat for the body's energy needs, leaving muscle as a major fuel source. Fatigue, lethargy, weakness, and decreased stamina result from muscle loss, from the metabolic changes that conserve energy by not allowing us to generate enough energy for our nonessential activities, and from "messages" from the body saying, "slow down, move more slowly, conserve energy, don't move any more than you have to, etc." Chronic dieting may deplete the body of essential minerals that may also conserve energy by enhancing insulin resistance. Low zinc levels are thought to interfere with metabolism of carbohydrate, allowing the partly metabolized glucose molecules to be diverted into fat stores which further contributes to the fatigue and difficulty in losing weight (Collipp 1981).

Even the diseases associated with obesity may be more a product of dieting than of the obese state itself. Cholelithiasis, for example, may be precipitated by the dehydration and bile concentration usually associated with dieting. This is probably made worse by the recent emphasis on proteins as diet food. A recent review article on hypertension in May 1983 (American College of Physicians 1983) has described a number of dietary influences that contribute to hypertension. Low levels of dietary calcium, potassium, magnesium, and fiber all have been shown to be responsible for increased blood pressure. High intake of protein saturated fats, sodium, or alcohol raise blood pressure. Inactivity also raises blood pressure. All of these factors would be made worse by traditional dieting with its accompanying inactivity.

Another contributor to hypertension has been thought to be high levels of epinephrine and norepinephrine. These hormones are anti-insulin, and seem to be required at times in the obese to counteract the effects of the high insulin levels. These high insulin levels are due to insulin resistance which in turn is a defense mechanism in response to food restriction. Aldosterone levels are markedly influenced by dietary carbohydrate (Kolanowski 1980), and they may be adversely affected by the large variations in carbohydrate intake so often seen in the obese as they alternate carbohydrate restriction with carbohydrate binges. The depression so often seen with obesity may influence or even produce alterations in the neuro-hormones in response to malnourishment from dieting. If the substrate for producing these hormones or the enzymes or cofactors responsible for manufacturing them are deficient, it seems logical that altered neurohormone levels could result. Some of these neurohormones are involved in directing nutrient choices, and if their levels need to become higher or lower to counteract poor food choices, then this disruption may contribute in some way to depression and other neural disturbances.

This new understanding of obesity opens up some exciting avenues for treatment possibilities. Now that we understand the nature of the problem, we have been able to design a treatment program that can attack the problem directly rather than relying on older methods which although can produce reasonable short term results, merely aggravate the problem in the long term.

Selecting An Ideal Weight

There is considerable variability with body build as with most other human conditions. Society's standards dictate that all women must be extremely thin to be ideal. Many women who are ideal according to medical standards have perceived themselves as being too fat and have damaged their health, made themselves fatter, and become very depressed while their self-esteem has been lowered by getting into the vicious diet trap. Muscular, large-boned men have done much the same thing

when convinced by the inadequate, outdated Metropolitan Life Insurance height-weight charts that they are considerably overweight when in fact they were ideal for their particular build. Current medical research suggests that the healthiest weights are from ideal to 20 percent or so above the older Metropolitan Life Insurance tables. More recent height-weight tables are available which reflect this change in attitude. Even these upgraded tables are not nearly as accurate as determining an ideal weight based on a percent fat determination. These determinations can be done with skin calipers, hydrostatic weighing, or special fat composition tables which use girth measurements (Remington 1983).

Regardless of what ideal weight is calculated, it is not necessary for everyone to fit within the guidelines. The physician can do much good by making his patients realize that he accepts them and cares for them regardless of how much they weigh. A new understanding and sympathy by the physician for the obese patient can do much to improve doctor-patient relationships. Too often obese patients have been treated as if their condition was the end result of laziness, gluttony, or not trying hard enough to lose weight. This only adds to the feelings of guilt, inadequacy, and frustration suffered by these unfortunate victims.

Reprogramming Patients to Be Thin

Garth Fisher, Ph.D., exercise physiologist; Edward Parent, Ph.D., psychologist, and I have evolved the above ideas after many years of experience working with obese people and after intensive research of the recent scientific literature. We recognized that obese people were programmed to be that way and that conventional dieting merely programmed them to be fatter and more effectively regain any lost fat and also produced health problems and unhappiness. Any permanent solution to the problem had to come through helping the patient change the biochemical and regulating problems that kept them fat—reprogram them, in effect, to be thinner. Understanding the basic pathophysiology of the obese process was the first essential step. Another important step was to analyze those who had effectively controlled their weight and identify what they had done to be successful. Using this information, we have formulated treatment principles which seem to lower the setpoint and make desirable biochemical changes.

The Role of Exercise

It became apparent that exercise was the most important component of the reprogramming process. Exercise accomplishes the following things:

1. Exercise lowers the setpoint directly (Fábry 1967).

2. Exercise increases the muscle mass and prevents the muscle loss which occurs with dieting.

3. Exercise increases the betaoxidation enzymes within the muscles which enable the muscles to burn fat for fuel. Muscle activity using these enzymes is the only effective way the body has of burning fat.

4. Exercise increases the metabolic rate both during exercise and for many hours afterwards. This helps the body be a high energy burner.

5. Exercise lowers insulin resistance, makes the tissues more sensitive to insulin and thus lowers the insulin levels necessary for the body to function more effectively. This then inhibits the process whereby the obese body diverts much of the food energy into fat production. It also encourages the body to use the fat for a fuel source.

6. Exercise lowers the level of various hormones, probably through metabolizing them (Bjorntrop 1980). Since many of these hormones have an adverse effect on insulin activity, this helps the obese process. It also allows the obese person to be more relaxed, less irritable, have a more even temperament, be more clearheaded, sleep better, and probably is responsible for many of the other positive benefits attributed to exercise.

7. Exercise burns up extra calories. Although this is the traditional reason given for why exercise is important to the weight loss process, it comes last on our list.

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The type of exercise that effectively makes the preceding changes is aerobic exercise that uses large muscle groups in a repetitive, rhythmic fashion. After thirty minutes or longer, these changes begin; thus only prolonged activity (preferably for one hour or more) is effective. Walking, running, swimming, bicycle riding, aerobic dancing, using a minitrampoline, and other similar activities are all acceptable.

Eating Right to Lose Weight Permanently

Our first efforts to change the setpoint emphasized using exercise and then using a strict diet along with it to further speed up the weight loss process and make patients thinner and more able to exercise effectively. Although we had good short-term results, after a modest weight loss, people would stop losing in spite of continuing a markedly reduced caloric regime. We also found it almost impossible to keep people faithfully exercising while on a re-

stricted food intake. It soon became apparent that the diet was negating all the positive effects of the exercise. We became increasingly aware of patient histories in which weight loss occurred right down to ideal through good exercise with no effort to diet, but heard of few people who lost to ideal while exercising at the same time as dieting. We then began to emphasize the importance of eating plenty of good nutritious food which satisfied the body's needs for calories as well as vitamins and minerals. We suggested greatly reducing those foods that had a known effect in raising the setpoint (high-fat foods and refined carbohydrates). We emphasized a dramatic increase in foods high in complex carbohydrate, and encouraged only small amounts of protein-rich foods. We began training people to eat in harmony with their hunger drives so that the defense mechanisms against starvation would not be triggered.

Preliminary results with this new technique have been extremely promising. Patients found it hard to believe that they were effectively losing weight while eating more than they had been able to eat for many years. Most people reported a rather dramatic increase in energy level as well as feeling much better in ways hard for them to define. Most began to lose their craving and their constant thinking about food, and began to develop a natural relationship with food where they would eat when hungry, have no trouble stopping when full, and then be able to go for a number of hours without even thinking about food. When presented with the new concepts, most patients stopped blaming themselves for their weight problems and began to develop a pride in their newfound self-control along with an increased self-esteem. When asked if they could live like this for a lifetime, most responded with a resounding "Yes!"

It was exciting to be able to work with people who were happy, full of enthusiasm, and feeling great while losing weight instead of encouraging people to stay with an unnatural starvation regime and try to keep them up when they felt tired and miserable. In particular it was refreshing to see people actually enjoy exercise and look forward to it.

It was no problem convincing people of the value of the new ideas, but it was a problem to get them to change their old thinking about dieting. It was hard to convince people bread and potatoes were okay to eat—in fact ideal—and cheese and meat were really not good diet food. It was difficult to convince people that they should eat until full and to snack if hungry and make sure not to miss any meals. There was often very little weight loss at the beginning, and it was hard to convince people that they were actually starting to make changes in their bodies and making good progress and the weight loss would come later. We still need to learn how to effectively teach the new ideas and help people to effectively make the necessary life-style changes to keep the setpoint at the lower level.

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Future of Weight Management

Other researchers are developing similar ideas about the setpoint and the futility of dieting. In a few years, there should be many sound programs developed that will take advantage of these new ideas to directly attack the obesity problem. Soon, informed physicians will not encourage counterproductive harmful diets but will be able to suggest effective, practical approaches for their patients to follow. In fact, with a new understanding of the obese process, we as physicians should be much more effective in preventing obesity by discouraging the type of behavior that makes people fat.

Reprogramming and Latter-day Saint Philosophy

For a number of reasons, the reprogramming plan fits in much more closely with Word of Wisdom principles than have other weight-management principles. The caffeinated drinks so often encouraged with diet systems are now known to be strong insulin stimulators which merely make the obese process worse and promote more hunger a short time later. In recent years there has been a great deal of reliance on proteins as a diet food. This type of approach causes relative dehydration, probably leeching out important mineral stores, reduces the intake of cancer preventing fiber and vitamins containing complex carbohydrate, and leads to the ingestion of harmful dietary fits. Word of Wisdom dietary principles are now accepted as being very healthy for over-all disease prevention as well as for good weight management.

Life is hard enough for most people without the added burden of having to live with the constant hunger, deprivation, fatigue and depression associated with regular dieting. Along with this goes the guilt associated with the inevitable eating binges. The new reprogramming plan allows the seasoned dieter an escape from the diet trap—the ability to finally eat in a natural way to avoid the chronic hunger and other associated problems. Now people can devote their energy and attention to more important facets of their lives. Future research should demonstrate the effectiveness of these gospel-compatible principles in promoting effective weight management as well as better physical and emotional health.

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