

EFFECTS OF LOW CARBOHYDRATE DIETS AND FASTING
ON BODY COMPOSITION

by

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INTRODUCTION

Fat storage in the body is desirable because fat deposits function as a concentrated energy reserve, a physical protector of vital organs, a thermal insulator, and a contributor to a smooth body contour. However, excess body fat contributes to a poor appearance and is associated with many diseases, such as cardiovascular and renal diseases, diabetes, degenerative arthritis, gout, and gallbladder disease. The basic method of preventing excess body fat accumulation is to ingest no more calories than needed for energy expenditure. The basic method of ridding oneself of excess body fat is to ingest fewer calories than needed for energy expenditure.

Mayer and Thomas (1) emphasized that excess body weight, as determined by life insurance height-weight tables, is not a satisfactory criterion for obesity; heavy, lean football players could then be considered obese. They defined obesity as a condition in which body fat is accumulated in abnormal excess.

The average obese person who desires to lose excess fat wants the process to be as rapid as possible. His major criteria for choosing a diet is that it promises quick body weight reduction. When he loses weight he assumes he is losing fat; he fails to realize that weight loss is composed of a combination of fat, lean tissue, and water.

A good weight reduction plan reduces body fat stores without reducing lean tissue; this is a gradual process. However, there are many popular

low carbohydrate reducing diets which promise rapid weight reduction. The purpose of this paper was to review: a) popular low carbohydrate reducing diets, b) physiological processes of body fat accumulation and reduction, c) methods of measuring body composition, and d) composition of weight loss produced by low carbohydrate diets and by fasting.

POPULAR LOW CARBOHYDRATE REDUCING DIETS

The low carbohydrate diet approach to weight reduction has been advocated for over a century. In 1864, an English surgeon, William Harvey, devised a diet for obesity that specifically ruled out sweet and starchy foods, while permitting meat ad libitum. One of his obese patients lost 50 pounds in one year (2). During the last 20 years, there has been a cyclic recurrence of similar diets with three major features: a low to very low carbohydrate content, no restriction of protein and fat, and unrestricted calories.

Several popular low carbohydrate diets and their allowable carbohydrate levels are: Air Force Diet (3), 60 g; Dr. Stillman's Diet (4), 0 g; Dr. Yudkin's Diet (5), 50 g; Drinking Man's Diet (6), 60 g; and Dr. Atkins's Diet (7), 0 g during first week and 5-8 g added each week thereafter until the individual's critical carbohydrate level is reached. The average American consumes 200-300 g carbohydrate daily (8).

Dr. Atkins (7) explained the logic of low carbohydrate diets as follows:

a) Obese people are victims of disturbed carbohydrate metabolism. Obesity is an allergy to carbohydrates. Ingested carbohydrates release

a flood of surplus insulin, which Dr. Atkins called the "fattening hormone." Insulin promotes the conversion of sugar into fat by initiating the manufacture of fatty acids. Somehow, insulin also prevents stored fat from breaking down.

b) Obese people can lose weight by decreasing the amount of carbohydrates they ingest. When very small amounts of carbohydrates are ingested, stored fat is mobilized as the body's energy source. As this fat is burned very rapidly for energy, the breakdown products, ketones, are discarded in the urine and breath. Dr. Atkins (7) believed that a fat mobilizing hormone (FMH) is responsible for this process. He described the process:

By cutting carbohydrates, this marvelous natural body substance--FMH--this magic bullet--is released by the pituitary and circulated in the bloodstream.

And the production of FMH is the whole purpose of this diet--and the reason it works when all other diets fail.

The presence of FMH circulating in your bloodstream guarantees that you are being continuously fed fuel which originates from your own unwanted stores of fat. This is so because the FMH makes your fat storage depots continually available to your body as fuel.

He also stated:

The ketones in your urine and on your breath represent incompletely burned calories. This means that when you excrete or breath out ketones you are sneaking calories out of the body. It's one of the reasons you can eat more calories than you burn up and--as long as no carbohydrates are present--still lose weight.

The ketones are the secret of this seemingly biochemical sleight-of-hand.

Hundreds of calories are sneaked out of your body every day in the form of ketones and a host of other incompletely broken down molecules of fat. You are disposing of these calories not by work or violent exercise--but just by breathing and allowing your kidneys to function. All this is achieved merely by cutting out your carbohydrates.

c) Fat and protein may be consumed ad libitum because carbohydrates, not calories, are the cause of overfat. In clinical experience with many patients over the years Dr. Atkins has shown that, with a low carbohydrate diet, weight is lost even when the calories taken in far exceed the calories expended. Fat in the diet helps to keep the patient from being hungry, helps to stabilize blood sugar levels, and helps to sneak off calories by causing excretion of ketone bodies.

PHYSIOLOGICAL PROCESSES OF BODY FAT ACCUMULATION AND REDUCTION

Vander et al. (9) categorized the physiological processes involved in body fat accumulation and reduction into two functional states: the absorptive state, during which ingested nutrients are entering the bloodstream from the gastrointestinal tract, and the postabsorptive (or fasting) state, during which the gastrointestinal tract is empty and energy must be supplied by the body's endogenous stores. Vander et al. (9) and Guyton (10) described these processes.

Absorptive State

During absorption of a normal meal, glucose provides the major energy source. Some amino acids and fat are used to resynthesize the continuously degraded body proteins and structural fat, respectively. Most of the amino acids and fat as well as the large quantity of carbohydrate not oxidized for energy are transformed into adipose tissue fat. Thus, body fat accumulation takes place during the absorptive state.

Fat. The primary function of fat in human nutrition is to supply the body's most concentrated source of energy. Fat is ingested primarily

as triglycerides. In the process of absorption, some triglycerides are broken down into mono- and diglycerides and unesterified fatty acids. All of these compounds, if not utilized by body cells as an immediate source of energy, are resynthesized into triglycerides by the liver or adipose tissue and stored in the adipose tissue.

Carbohydrates. The primary function of carbohydrates is to supply energy to body cells. Most of the ingested carbohydrate is converted to glucose for absorption into cells. Glucose is a more readily available form of energy than fat because it is absorbed into the bloodstream faster. A small amount of the glucose that is not utilized by the cells as an immediate energy source is converted to glycogen and stored as reserve energy in the skeletal muscles and liver. The rest is converted to either the glycerol or fatty acid moieties of the triglyceride molecule. The triglycerides formed are stored as fat in the adipose tissue.

Protein. The primary function of dietary protein is to promote growth and maintenance of tissue. Protein normally plays a minimal role as a source of energy. Amino acids which are not utilized as body building blocks are deaminated, and the non-nitrogen residues can be utilized as either carbohydrates or fat, depending upon the individual amino acid. Whether they are used as immediate forms of energy or are stored as fat depends upon the body's need for energy.

Postabsorptive State

Body fat is broken down during the postabsorptive state in which no source of energy is being absorbed into the bloodstream from the intestinal tract. Carbohydrate is synthesized in the body, but its utilization for

energy is greatly reduced; the oxidation of endogenous fat provides most of the body's energy supply; fat and protein synthesis are curtailed and net tissue breakdown occurs.

Glucose. During the postabsorptive period no glucose is being absorbed from the intestinal tract, yet the plasma glucose concentration must be maintained because the nervous system is unable to oxidize any other nutrient for energy. Lack of adequate glucose supply to the brain can cause damage, coma, and death within minutes. There are several sources of glucose which maintain blood glucose concentration during this fasting state. Glycogen stores, particularly in the liver, are mobilized quickly and can supply the body's needs for several hours, but they are inadequate for longer periods. The major source of blood glucose during prolonged fasting comes from protein. Large quantities of protein in muscle and some other tissues are not absolutely essential for cell function and can be catabolized without serious cellular malfunction. There are limits to this process and continued protein loss ultimately leads to functional disintegration, sickness, and death. Another source of glucose is adipose tissue triglyceride breakdown. The catabolism of triglycerides yields glycerol and fatty acids. Glycerol is liberated into the blood, circulates to the liver, and is converted into glucose. The small amount of glucose liberated from the above three sites cannot provide all of the body's energy needs. The nervous system utilizes this glucose while all other organs and tissues markedly reduce their oxidation of glucose and depend primarily on fat as their energy source, thus sparing the glucose produced by the liver to serve the obligatory needs of the nervous system.

Fatty Acids. The fatty acids liberated into the blood upon catabolism of adipose tissue triglycerides are picked up by all other tissues, are metabolized to acetyl CoA molecules, enter the Krebs cycle, and are oxidized to produce carbon dioxide, water, and energy. Over 50 per cent of the initial degradation of fatty acids to acetyl CoA occurs in the liver. The liver cannot use all of these fatty acids for its own metabolic processes; rather two acetyl CoA molecules combine to form one molecule of acetoacetic acid, which diffuses through the liver cell membrane and is transported by the blood to the peripheral tissues. Here it is broken down to two acetyl CoA molecules which are oxidized for energy via the tricarboxylic acid cycle. Normally, the total plasma concentration of acetoacetic acid is small because it is transported rapidly. Large quantities of acetoacetic acid occasionally accumulate in the blood and interstitial fluids--a condition called ketosis. This occurs during periods when no carbohydrates are metabolized--such as when carbohydrate intake is low. Fat must be used for energy, and large quantities of acetoacetic acid are found in the bloodstream because the cells cannot metabolize them as rapidly as they are formed. Acetoacetic acid is readily converted into two other compounds, beta-hydroxybutyric acid and acetone--these three compounds are called ketone bodies, and they are excreted via the urine and breath. A large accumulation of ketone bodies in body fluids causes severe acidosis because they decrease the blood pH, and because strong cations, such as sodium, are excreted with them.

METHODS OF MEASURING BODY COMPOSITION

Most methods of measuring body composition are based on the assumption that the body consists of two compartments of relatively constant but distinctly different composition. These compartments are body fat, which includes the entire content of chemical fat or lipids in the body, and fat free mass, which includes all the rest of the body apart from fat. A comparison of the density, potassium, and water content of these two compartments (11) is as follows:

<u>Factor</u>	<u>Fat Tissue</u>	<u>Fat Free Tissue</u>
Density	0.9	1.1
Potassium, mEq/kg	0	68.0
Water, g/kg	0	720.0

Researchers have utilized both direct and indirect methods of assessing body composition changes in persons on a low carbohydrate diet. The major direct methods are body density, potassium-40, and total body water. The major indirect methods are anthropometric measurements, weight changes and energy balance, nitrogen balance, serum and urine uric acid levels, serum and urine sodium and potassium levels and water balance, and abnormal metabolites in the blood and urine.

Direct Methods of Measuring Body Composition

Body Density. Various methods have been used to determine body density. Among these are underwater weighing and body volume. Underwater weighing is based on the principle that lean tissue is more dense

than water and fat tissue is bouyant. The degree to which a person has positive or negative bouyancy, once it is corrected for the lift force of air in the lungs, is used to determine his body density (12). Body volume is measured by the amount of water displaced when a subject is submerged in a small, water filled tank or by gas dilution. Body density can be computed from known body volume (13).

Potassium-40. This measurement is based on the principle that the potassium content of lean body tissue is constant and fat tissue contains no potassium. Total body potassium can be calculated from the determination of the amount of naturally occurring radioactive potassium as assayed in the whole body scintillation counter. Radioactive potassium is naturally present in a constant ratio to ordinary potassium. Once potassium content of the body has been determined, one can, by use of appropriate constants, determine lean body mass and thus total body fat by subtraction from weight. Body potassium is lost whenever lean tissue is broken down (14-17).

Total Body Water. Total body water can be measured by injecting heavy hydrogen (deuterium or tritium) into the bloodstream. This substance diffuses into the cellular and extracellular fluid spaces. After two to three hours, when equilibrium of exchange or distribution of hydrogen occurs, a blood sample is drawn and the heavy water concentration is measured by specific gravity or by infrared spectrophotometry (10). Lean body mass and fat content can be derived from the value of total body weight (17).

Indirect Methods of Estimating Body Composition

Anthropometric Measurements. Body composition can be estimated by the measurement of skinfold thickness, bone length and diameter, and body circumference, along with equations based on these measurements (11, 18-20).

Weight Changes and Energy Balance. According to the basic laws of thermodynamics, a caloric intake that exceeds caloric expenditure indicates a storage of calories within the body. Likewise, a caloric intake that is much lower than caloric expenditure indicates that calories stored in the body are being metabolized for energy. Grande (21) criticized several investigators for concluding that a particular diet resulted in body fat loss because they did not show that caloric expenditure exceeded caloric intake.

Nitrogen Balance. More than 90% of urinary nitrogen results from deamination of amino acids in the body. When caloric intake is fully adequate and protein intake is just sufficient to cover the repletion of body tissues, urinary nitrogen is at its lowest level. When nitrogen intake equals nitrogen excretion, a state of nitrogen equilibrium exists. A negative nitrogen balance (greater nitrogen excretion than intake) results when caloric input is drastically decreased so that body proteins must be broken down for energy. Thus, a negative nitrogen balance indicates lean tissue breakdown (8).

Serum and Urine Uric Acid Levels. Uric acid is an end product of purine metabolism. It is formed from purine bases derived from nucleoproteins. As nucleoproteins are broken down in tissue degeneration, uric acid levels are elevated (22, 23).

Serum and Urine Sodium and Potassium Levels and Water Balance.

Sodium and potassium play key roles in the maintenance of osmotic pressure and fluid balance; sodium is the principle cation in extracellular fluid and potassium the principle cation within the cell. An abnormally low serum or urinary level of these electrolytes indicates loss of body fluids. Potassium also leaves the cells and is excreted during periods of protein catabolism or glycogenolysis. Fluid intake and excretion can also be measured to determine body water loss (8).

Abnormal Metabolites in the Blood and Urine. As previously discussed, an excessive amount of serum ketone bodies indicates excessive body fat metabolism (9, 10).

EFFECTS OF LOW CARBOHYDRATE REDUCING DIETS AND FASTING ON BODY COMPOSITION

Numerous studies have been conducted to determine the effects of both low carbohydrate diets and fasting on weight change and body composition. These two weight reducing regimens have similar physiological effects on the body. In both cases the body is forced to use fat and protein as energy sources. When fat and protein are provided in the diet, the body uses exogenous along with some endogenous fat and protein for energy. When a person fasts, his body uses only endogenous stores of fat and protein. It is to be expected, of course, that more body tissue, and thus weight, will be lost with a fasting diet than with a low calorie diet, because no outside calories are being provided to spare endogenous protein and fat tissue.

Low Carbohydrate Diets and Water Loss

Early studies on the effects of fasting and low carbohydrate diets on water balance were reviewed by Bloom (24). Hippocrates, from the seventh section of Aphorisms, stated that, "fasting should be prescribed for those persons who have humid flesh for fasting dries bodies." The effect of carbohydrates on water balance was noted in 1928 by William Stark, who gained eight pounds in five days after changing his diet from meat to flour. This phenomenon of rapid weight gain with addition of carbohydrate to the diet was confirmed by Bischoff and Voit in 1860 and Benedict and Milner in 1907.

Benedict in his classical studies on the faster, Levanzin, demonstrated that early in a fasting, weight loss exceeds that which can be explained on a caloric basis. This change was attributed to a change in water balance. In 1932, Gamble et al. confirmed this observation with three fasting children; there was a definite sodium loss related to the water loss of fasting.

During World War I, Gamble became interested in the problem of survival rations and for the first time the water and salt regulating role of carbohydrate in the diet was demonstrated. A striking difference in sodium excretion was observed when "life raft survivors" were supplied with water with or without 100 g of glucose. Those volunteers receiving glucose excreted far less sodium than their fasting counterparts despite the fact that there was only a 400 kcal difference in their energy balance. In 1952 Hervey and McCance carried out a series of experiments which yielded data comparable to Gamble's and indicated the important role of carbohydrate in conservation of water and salt in men without food or sufficient water.

In 1932 Lynn and Dunlop found that weight loss of obese people was inversely proportional to the carbohydrate content of 1000 kcal diets. In 1944 Anderson found that there was a rapid decrease in weight on a high fat, low carbohydrate, 650 kcal diet; when the composition of the diet was changed to include carbohydrate, there was no loss of weight over a period of nine days. Anderson suspected salt and water balance differences but his methods were not definitive enough to establish this conclusion.

The major conclusion of these studies reviewed by Bloom was that the weight loss observed on low carbohydrate diets of short duration (usually ten days or less) was due to water loss. Methods utilized to determine this were weight change, energy balance, and sodium excretion.

Defect in Carbohydrate Metabolism

In the 1950's, Pennington (25) suggested that obese people gained weight because of a metabolic defect. He said that they were unable to metabolize carbohydrate completely, so that it was oxidized only as far as pyruvic acid. High amounts of pyruvic acid supposedly inhibit fat oxidation and stimulate fat synthesis. Severe restriction of dietary carbohydrate, then, would lead to mobilization of body fat, even in the presence of large amounts of dietary fat.

Kekwick and Pawan (26, 27) supported the metabolic defect idea. For periods of 8-10 days they gave obese patients 1000 kcal diets, in which 90% of the calories came either from protein, fat, or carbohydrate. The patients lost the most weight with the fat diet and lost little or no weight, or even gained weight with the carbohydrate diet. Thirty to 50%

of the weight loss was water loss and the remainder (50-70%) was fat loss. Nitrogen equilibrium was maintained throughout the study. Kekwick and Pawan stated (25), "It would appear that some aspects of metabolism in the obese differ from those in the non-obese, and that alteration in the composition, as distinct from the total caloric value, of the diet, may induce changes in the energy output of the obese individual."

Pennington (25) believed that a diet allowing unlimited fat consumption and restricted carbohydrate consumption, and placing no restriction on total calories, could produce weight loss. On this basis he refuted the orthodox contention that weight loss can only be achieved by calorie restriction. Kekwick and Pawan (26) supported Pennington's theory of luxury consumption. They said that if the carbohydrate in the diet is less than 60 g, the obese patient will lose weight even if his caloric intake considerably exceeds that on which he had previously remained overweight. They believed that energy expenditure is increased by protein and especially by fat, whereas it is decreased by carbohydrate. The crucial point of their work was the observation that four out of five obese patients lost 1-2.6 kg when, for a week, a normal composite diet of 2000 kcal was replaced by a diet of 2600 kcal given mainly as fat and protein. Some of their patients maintained their weights on high carbohydrate diets of 1000 kcal (which is decidedly lower than the basal metabolism of a euthyroid adult), while others lost weight on high fat diets of 2600 kcal. In determining total body water, they found that the ratio of body weight to body water remained remarkably constant during weight loss on the various diets.

Generally accepted physiological facts do not support the assumptions of Pennington (25) and Kekwick and Pawan (26, 27) about the effect of foods

on energy expenditure. Classical reports on specific dynamic action of foodstuffs do not point to any increase of metabolism due to fat; on the contrary, the action of fat seems to be smaller than that of carbohydrate. It is known, also, that an intact digestive canal absorbs large amounts of fat as effectively as it does normal amounts. The theory of a metabolic defect in the obese is unlikely, based on studies which clearly show that fluid loss can account for the majority of early weight loss occurring in subjects fed low carbohydrate diets (24). According to Astrand and Rodahl (28), carbohydrate binds water when it is stored as glycogen (each gram of glycogen binds two to four grams of water); thus as glycogen stores are depleted, water is lost.

With the publication of Kekwick and Pawan's studies in 1956 (26) the low carbohydrate diet began to attract the attention of the popular press. Their claim that an obese person could lose body fat quickly by restriction of dietary carbohydrate to 60 g and no restriction of fat, protein, or total calories, was used by many people as a basis for weight reduction diets.

Low Carbohydrate Versus High Carbohydrate Diets

The research of Pennington (25) and Kekwick and Pawan (26, 27) spurred many other workers to set up similar studies over longer time periods. Pilkington et al. (29) alternated high fat and high carbohydrate 800-1000 kcal diets for obese patients during a four month period. They found significant upward swings of weight loss during the first few days with the high fat diet as compared to the high carbohydrate diet. These swings lasted up to ten days and weight loss amounted to up to 2.5 kg. After the tenth day the rate of weight loss was the same on both diets. The patients

were in negative nitrogen balance throughout the study--more so when fed a high fat diet, which indicated there was more lean tissue loss with the high fat diet. There was also a moderate degree of ketosis on this diet, which indicated fat tissue loss. The swings in weight loss were attributed to variations in fluid balance, observed by measuring fluid intake and output. The changes in fluid balance were too small to be detected accurately by measurements of total body water.

Olesen and Quade (30) treated eight obese women with a high fat, high calorie diet and a high carbohydrate, low calorie diet, successively, for a total diet period of 14-29 days. They observed a weight loss for the first few days when the high fat, high calorie diet was fed, but the weight loss ceased after a few days. They found that weight remained unchanged when the high carbohydrate, low calorie diet was fed. The patients' basal metabolic rates showed that the high carbohydrate 1250 kcal intake was less than their minimum energy requirement. Therefore, they must have been drawing on their energy stores. They attributed failure of weight loss to water retention.

Fletcher et al. (31) tested obese patients for six weeks by feeding 500 kcal diets high in either protein, fat, or carbohydrate, given in sequence, each for two weeks. They found no significant difference in the amount or rate of weight loss in patients fed the different diets.

Kinsell et al. (32) studied five obese patients over a period of two months and found that in diets of varying fat, carbohydrate, and protein composition, rate of weight loss was essentially constant throughout the entire study. They said that there is good reason to believe that the satiety value of high fat diets is superior to that of high carbohydrate

diets, and hence, these diets may be associated with better dietary adherence. Unfortunately, many obese individuals with a pattern of compulsive eating tend to go on carbohydrate binges, regardless of the theoretically greater satiety value of a high fat regimen.

Brozek et al. (33) conducted a series of experiments feeding a group of men diets low in carbohydrate, calories, and water in order to determine possible length of survival and body changes in soldiers. In one experiment they fed the men 580 kcal as carbohydrate per day for 12 days, and in the following experiment they fed them 1010 kcal as carbohydrate per day for 24 days. Limb and trunk circumferences of the men showed significant decrements during severe caloric restriction. In the recovery period, which lasted 16 days in the first experiment and 20 days in the second, the abdominal circumference showed the largest gain (90%, vs. 41% for the upper arm). During caloric restriction, skinfold measurements and soft tissue roentgenograms indicated a reduction of 30-40% in the thickness of the subcutaneous fat layer. During recovery, there were substantial differences in increase of anthropometric measurements. Body weight decreased by a total of 5.9 kg in the first experiment and 7.6 in the second experiment. Estimates of the composition of the weight loss, derived from energy balance and nitrogen excretion, indicated that water composed a large part of the early weight loss during the first half of the restricted period, and that fat composed the largest portion of the weight loss during the last half of the restricted period.

Bell et al. (34) measured weight loss, uric acid levels, nitrogen balance, and ketosis in five obese women fed 400 kcal diets composed solely of either protein, carbohydrate, or fat, in alternating sequences, or 800

kcal combinations of these. They found that at 400 kcal/day, ketosis was least with carbohydrate and greatest with fat as the source of calories. Four hundred kcal/day of protein reduced the degree of ketosis. Four hundred kcal of carbohydrate plus 400 kcal of protein effectively prevented development of ketosis but the addition of carbohydrate to fat in the diet was not beneficial. They observed a serum uric acid rise and urine uric acid fall in subjects fed high fat diets, and the opposite when high carbohydrate diets were fed. Loss of weight, sodium, and potassium was much greater with diets based on fat than on carbohydrate. Nitrogen losses on 400 kcal/day of fat intake were greater than nitrogen losses on 400 kcal/day carbohydrate intake. Also, negative nitrogen balance in most of these obese individuals increased during five days of the fat diet, while it tended to decrease during five days of carbohydrate intake. When caloric content of the diet was raised to 800 kcal carbohydrate, two of the subjects achieved positive nitrogen balance and two others achieved a smaller deficit than when no supplemental source of calories was given. However, the addition of fat calories did not improve nitrogen balance. In fact, the combination of fat and protein at 800 kcal was associated with the same destruction of tissue protein as occurred at 400 kcal fat without protein in the diet. Exogenous fat provided no improvement over allowing the subject to use his own endogenous fat to support his energy needs. Bell et al. (34) concluded that carbohydrate is necessary to prevent clearance of uric acid, when endogenous fat is the chief source of energy. Exogenous protein can fulfill part of this function, but it is less effective than carbohydrate at equivalent low intakes.

Worthington and Taylor (35) compared subjects fed 5% and 35% carbohydrate, low calorie diets and found a definite increase in the amount of urinary ketones with the low carbohydrate diet. They also found that the first week weight loss was greater with the low carbohydrate diet, but the second week weight loss was similar with both levels of carbohydrate intake.

Yudkin and Carey (36) tested Pennington's hypothesis that a subject eating a "high fat" diet (limited in carbohydrate and unlimited in fat) with unlimited calories would actually lose weight. Their purpose was to find out what happened to the intake of calories, protein, and fat in diets restricted in carbohydrate. Subjects were instructed to reduce the carbohydrate in their diets to about 50 g daily and eat as much fat and protein as they liked for two weeks. Their daily caloric intake was found to be 200-1900 kcal lower than their normal intake, a reduction of between 13 and 55%. There was no significant change in protein intake, and either no change or a reduction in fat intake. Yudkin and Carey stated that the obese patients lost weight on this diet, not because of some peculiar metabolism of fat, but because of a reduction in calorie intake.

There were several major findings in these studies of low versus high carbohydrate diets. Low carbohydrate diets procued a greater initial weight loss than diets containing higher amounts of carbohydrate, but after one and a half to two weeks, total weight loss was similar with both diets. Ketosis occurred with low carbohydrate diets, indicating body fat loss. Uric acid levels rose and nitrogen balance was more negative with a low carbohydrate diet than with a higher carbohydrate

diet, indicating body protein loss. Electrolyte and fluid loss occurred with low carbohydrate diets. When given instructions to reduce carbohydrate intake and eat as many calories of fat and protein as desired, subjects also reduced their protein and fat consumption and thus their total caloric intake, and lost weight in the process.

Low Calorie Diets Versus Fasting

Jones et al. (37) studied obese subjects during sequential periods of fasting and refeeding of 600 kcal diets. The calories in the three diets were furnished by fat or carbohydrate or an equal mixture of fat, carbohydrate, and protein. The study period was from 25-30 days. Each patient was fed a single nutrient or the mixed diet for five days at a time and then changed to another diet. Ingestion of the fat diet resulted in less negative nitrogen balances than those observed during fasting, but the carbohydrate diet and the mixed diet fed thereafter effected further, more striking, positive changes in the balances. The mixed diet was associated with more positive nitrogen balances than the carbohydrate diet. Plasma ketones increased during fasting periods and remained at high levels during ingestion of the fat diet; they fell quite rapidly toward normal when the carbohydrate diet was fed. The rate of decline and degree of reduction of plasma ketones was directly correlated with the carbohydrate content of the diet. Less weight loss occurred when the carbohydrate diet was ingested than during consumption of the fat diet; the mixed diet caused a slight weight gain. Sodium retention was greatest during the carbohydrate diet. Diets composed largely of fat resulted in sodium retention but at a slower rate and to a lesser degree than diets containing carbohydrate.

In a ten day fasting study of normal adult males, Consolazio et al. (38) found highly negative water balances that resulted in body hypohydration, negative nitrogen balances that indicated excessive body catabolism, marked ketosis, and large mineral and urea losses. They compared the results of this study to those of a second study (39), in which they fed normal adult males 420 kcal as carbohydrate per day for ten days, in an effort to diminish fasting symptoms. In the carbohydrate fed group, body water losses were still large, but less than in the starvation group.

Drenick et al. (40) studied obese patients who fasted 12-117 days. After one month of fasting, there was a constant protein loss of 25 g/day. Sodium and potassium losses were high initially, but decreased as the fast continued; this indicated the ability of the kidneys to preserve electrolytes when intake is low. Hyperuricemia developed in all patient within two weeks of fasting. Normal uric acid levels returned as soon as a positive protein balance was re-established. A substantial potassium deficit, as measured by the radiation counter, occurred in the majority of patients.

Gilder et al. (41) determined the components of weight loss in patients subjected to low calorie intake and then prolonged starvation. Lean tissue loss was determined by nitrogen balance and body fat loss by indirect calorimetry carried out intermittently throughout the study using a portable expired air analyzer. During the first week, reduced caloric intake resulted in a rapid rate of weight loss owing to excessive loss of sodium and water and a relatively large loss of lean tissue. The lean tissue loss gradually diminished with continued fast to a constant rate between 0.9 and 1.3 g/kg/day. Fat loss was constant throughout the fast. In seven patients the overall mean ranged from 1.5-2.1 g/kg/day.

Bloom et al. (42, 43) conducted a series of experiments relating carbohydrate to weight loss and sodium and water balance. They demonstrated that weight changes following renutrition after fasting are a function of sodium excretion. Patients were fed the following diets; sodium free, 600 kcal glucose, 600 kcal fat and protein, fasting plus 180 mEq sodium, or total fasting (42). Urinary sodium of fasting patients dropped to low levels immediately after carbohydrate ingestion, but urinary sodium increased after fat and protein or sodium ingestion. After four days of carbohydrate ingestion, weight loss during fasting was decreased or completely abolished in association with this sodium retention following carbohydrate ingestion, even though the patient remained in negative caloric balance. Exogenous NaCl or 600 kcal fat and protein did not alter the weight loss of fasting. A 600 kcal diet composed of glucose and 200 mEq sodium abolished weight loss and produced the same sodium retention found with carbohydrate alone. The authors concluded that the sodium and weight retaining effect of the diet must have been due to the carbohydrate content of the diet.

In another experiment (43) Bloom and Azar studied the metabolic effects of an exogenous mixture (fat and protein) of caloric constituents similar to the caloric mixture of fasting. They fed one group of volunteers a 2000 kcal, moderate sodium, high fat diet. They found that urinary nitrogen excretion, plasma ketones, and non-esterified fatty acids increased in all subjects despite adequate caloric intake. Comparable weight loss was observed in all subjects. The weight loss would logically have been attributed to salt and water excretion since the subjects consumed adequate calories. However, with the evidence of continued fat mobilization (NEFA

and ketone elevations) in these subjects, it was concluded that actual flesh weight also was lost. Further evidence to support flesh weight loss was the negative nitrogen balance. Thus, the weight loss must have been related to the absence of carbohydrate from the diet with the subsequent salt and water excretion as well as to continued fat mobilization from depots. This experiment suggested that a diet adequate in calories, protein, and fat, but lacking in carbohydrate, results in loss of body fat, salt, and water, similar to that resulting from fasting. The results of this and the above studies indicated that a fasting regimen produces the same type of results as does a low carbohydrate diet.

Hood et al. (44) determined the optimum amount of calories and carbohydrate which would produce a good rate of weight loss and fewest metabolic disturbances. They fed patients, in various sequences and for eight days in each instance, 1000 kcal diets in which 3, 6, 12, 25 or 50% of the calories were supplied by sucrose. They found no significant difference in weight loss with the varying proportions of carbohydrate, and more ketosis with the lower carbohydrate diets. The 25 and 50% carbohydrate diets were associated with much less negative nitrogen balances, despite the lower protein content of the diets. Based on the results of their study, they recommended a 1000 kcal/day diet with 1/3 of the calories from carbohydrate (333 kcal or 83 g carbohydrate) for a good rate of weight loss and least metabolic disturbance.

CONCLUSIONS

Low carbohydrate reducing diets and fasting regimens are not desirable methods of reducing body fat stores. Decreased carbohydrate

Intake results in increased metabolism of fat for energy, acidosis, and losses of body water and protein. A balanced diet containing carbohydrate, protein, and fat, and limiting in total calories would be advisable for fat loss without the undesirable body changes. A balanced diet develops good food habits which should be maintained throughout life.

SUMMARY

Low carbohydrate reducing diets are popular with overweight individuals because they produce rapid initial weight loss. The purpose of this paper was to review popular low carbohydrate diets, physiological processes of body fat accumulation and reduction, methods of measuring body composition, and research on the composition of weight loss produced by low carbohydrate diets and by fasting.

The theory of advocates of low carbohydrate reducing diets is that carbohydrates, not calories, are responsible for weight gain in the obese; thus, diets containing 0-60 g carbohydrate and unrestricted amounts of fat and protein are advised for weight loss. A review of physiological processes of body fat accumulation and reduction stressed that body weight cannot be lost unless fewer total kilocalories are consumed than are expended.

The following direct and indirect methods of measuring changes in body composition were discussed: body density, potassium-40, total body water, anthropometric measurements, weight changes and energy balance, nitrogen balance, serum and urine sodium and potassium levels and water balance, and abnormal metabolites of the blood and urine.

Studies of body composition of individuals consuming a low carbohydrate diet indicated that weight loss was rapid during the first few days, due mainly to fluid and electrolyte losses. Thereafter, the weight loss continued at a slower rate, matching the rate of weight loss produced by low calorie diets containing higher amounts of carbohydrate. Long term low carbohydrate diets generally resulted in negative nitrogen balance, rise in uric acid levels, and ketosis which indicated that body protein, as well as body fat, was lost.

Studies of the effect of fasting on body composition also were reviewed. The effects of fasting were similar to those of a low carbohydrate diet in that both forced the body to utilize fat and protein as energy sources. Losses of body protein and fat were greater with fasting than with the low carbohydrate intake.

LITERATURE CITED

1. Mayer, J. & Thomas, D. W. (1967) Regulation of food intake and obesity. *Science* 156, 328-337.
2. American Medical Association Council on Foods and Nutrition. (1974) A critique of low carbohydrate ketogenic weight reduction regimens. A Review of Dr. Atkins Diet Revolution. *Nutrition Reviews*: Supplement No. 1, pp. 15-21.
3. Air Force Diet. (1960) Air Force Diet Publishers, Toronto, Canada.
4. Stillman, I. M. & Baker, S. S. (1968) The Doctor's Quick Weight Loss Diet. Dell Publishing Co., New York.
5. Yudkin, J. (1964) The Complete Slimmer. Macgibbon & Kee, London.
6. Jameson, G. & Williams, E. (1964) The Drinking Man's Diet. Cameron & Co., San Francisco.
7. Atkins, R. C. (1972) Dr. Atkins Diet Revolution. David McKay Co., New York.
8. Robinson, C. Y. (1972) Normal and Therapeutic Nutrition. MacMillan Co., New York.
9. Vander, A. J., Sherman, J. H., & Luciano, D. S. (1970) Human Physiology: The Mechanisms of Body Function. McGraw-Hill, Inc., New York.
10. Guyton, A. C. (1966) Textbook of Medical Physiology. W. B. Saunders Co., Philadelphia.
11. Durnin, J. V. G. A. & Womersley, J. (1974) Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16-72 years. *Brit. J. Nutr.* 32, 77-97.
12. Gnaedinger, R. H., Reineke, E. P., Pearson, A. M., Van Huss, W. D., Wessel, J. A. & Montoye, H. J. (1963) Determination of body density by air displacement, helium dilution, and underwater weighing. *Ann. N.Y. Acad. Sci.* 110, 96-107.
13. Siri, W. E. (1961) Body volume measurement by gas dilution. In: *Techniques of Measuring Body Composition*. (Brozek, J., ed.) pp. 108-117, National Academy of Sciences of the National Research Council, Washington, D.C.
14. Forbes, G. B., Gallup, G. & Hursch, J. P. (1961) Estimation of total body fat from potassium-40 content. *Science* 133, 101-102.
15. Cureton, K. J., Boileau, R. A. & Lohman, T. G. (1975) A comparison of densitometric, potassium-40 and skinfold estimates of body composition in prepubescent boys. *Human Biol.* 47, 321-334.

16. Barter, J. & Forbes, G. B. (1963) Correlation of potassium-40 data with antropometric measurements. *Ann. N.Y. Acad. Sci.* 110, 264-270.
17. Delwaide, P. A. & Crenier, E. J. (1973) Body potassium as related to lean body mass measured by total water determination and by anthropometric method. *Human Biol.* 45, 509-526.
18. Behnke, A. R. (1963) Anthropometric evaluation of body composition throughout life. *Ann. N.Y. Acad. Sci.* 110, 450-463.
19. Wilmore, H. J. & Behnke, A. R. (1968) Predictability of lean body weight through anthropometric assessment in college men. *J. Appl. Physiol.* 25, 349-355.
20. Seltzer, C. C. & Mayer, J. (1967) Greater reliability of the triceps skinfold over the subcapular skin fold as an index of obesity. *Am. J. Clin. Nutr.* 20, 950-953.
21. Grande, F. (1968) Energy balance and body composition changes: A critical study of three recent publications. *Ann. Int. Med.* 68, 467-480.
22. Taber's Cyclopedic Medical Dictionary (1973) (Thomas, C. L., ed.) F. A. Davis Co., Philadelphia.
23. Williams, S. R. (1973) Nutrition and Diet Therapy. C. V. Mosby Co., St. Louis.
24. Bloom, W. L. (1967) Carbohydrate and water balance. *Amer. J. Clin. Nutr.* 20, 157-161.
25. Pennington, A. W. (1953) A reorientation on obesity. *New Engl. J. Med.* 248, 959-964.
26. Kekwick, A. & Pawan, G. L. S. (1956) Calorie intake in relation to body weight changes in the obese. *Lancet* 271, 155-161.
27. Kekwick, A. & Pawan, G. L. S. (1957) Metabolic study in human obesity with isocaloric diets high in fat, protein, or carbohydrate. *Metab. Clin. Exptl.* 6, 447-459.
28. Astrand, P. E. & Rodahl, K. (1970) Textbook of Work Physiology. McGraw-Hill Book Co., New York.
29. Pilkington, T. R. E., Gainsborough, H., Rosenoer, V. M. & Carey, M. (1960) Diet and weight reduction in the obese. *Lancet* 1(2), 856-858.
30. Olesen, E. S. & Quade, F. (1960) Fatty foods and obesity. *Lancet* 1(3), 1048-1051.
31. Fletcher, R. F., McCririck, M. Y. & Crooke, A. C. (1961) Reducing diets; Weight loss of obese patients on diets of different composition. *Brit. J. Nutr.* 15, 53-58.

32. Kinsell, L. W., Gunning, B., Michaels, G. D., Richardson, J., Cox, S. E., & Lemon, C. (1964) Calories do count. *Metabolism* 13, 195-203.
33. Brozek, J., Grande, F., Taylor, H. L., Anderson, J. T., Buskirk, E. R., & Keys, A. (1957) Changes in body weight and body dimensions in men performing work on a low calorie carbohydrate diet. *J. Appl. Physiol.* 10, 412-420.
34. Bell, J. D., Margen, S. & Calloway, D. H. (1969) Ketosis, weight loss, uric acid, nitrogen balance in obese women fed single nutrients at low caloric levels. *Metab. Clin. Exptl.* 18, 193-208.
35. Worthington, B. S. & Taylor, L. E. (1974) Balanced low calorie vs. high protein/low carbohydrate reducing diets. *J. Amer. Diet. Ass.* 64, 47-55.
36. Yudkin, J. & Carey, M. (1960) The treatment of obesity by the "high-fat" diet: The inevitability of calories. *Lancet* 2(2), 939-941.
37. Jones, J. E., Albrink, M. J., Davidson, P. C. & Flink, E. B. (1966) Fasting and refeeding of various suboptimal isocaloric diets: Effects on mineral and nitrogen balance in obese patients. *Amer. J. Clin. Nutr.* 19, 320-327.
38. Consolazio, C. F., Matoush, L. O., Johnson, H. L. & Krzywicki, H. J. (1967) Metabolic aspects of acute starvation in normal humans (10 days). *Amer. J. Clin. Nutr.* 20, 672-682.
39. Consolazio, C. F., Matoush, L. O., Johnson, H. L., Krzywicki, H. J., Isaac, G. J. & Witt, N. F. (1968) Metabolic aspects of caloric restriction: Hypohydration effects on body weight and blood parameters. *Amer. J. Clin. Nutr.* 21, 793-801.
40. Drenick, E. J., Swendseid, M. E., Bland, W. H. & Tuttle, S. G. (1964) Prolonged starvation as treatment for severe obesity. *J. Amer. Med. Ass.* 187, 100-105.
41. Gilder, H., Cornell, G. N., Grafe, W. R., Macfarlane, J. R., Asaph, J. W., Stubenbord, W. T., Watkins, C. M., Reess, J. R. & Thorbjarnarson, B. (1967) Components of weight loss in obese patients subjected to prolonged starvation. *J. Appl. Physiol.* 23, 304-310.
42. Bloom, W. L. & Mitchell, W. (1962) Inhibition of salt excretion by carbohydrate. *Arch. Int. Med.* 109, 26.
43. Bloom, W. L., Azar, G. J. (1963) Similarities of carbohydrate deficiency and fasting. Weight loss, electrolyte excretion, and fatigue. *Arch. Int. Med.* 112, 333-337.
44. Hood, C. E., Goodhart, J. M., Fletcher, R. F., Gloster, J., Bertranc, P. V. & Crooke, A. C. (1970) Observations on obese patients eating isocaloric reducing diets with varying proportions of carbohydrate. *Brit. J. Nutr.* 24, 39-44.

EFFECTS OF LOW CARBOHYDRATE DIETS AND FASTING
ON BODY COMPOSITION

by

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