

Protein is an Effective Food for Weight Loss and Maintaining Body Composition

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Abstract

Obesity is rapidly becoming a public health concern in modern society. In the U.S., national survey data suggests that more than 60% of adults are overweight with more than half of these obese individual. [1,2] In 1991 only 4 out of 45 states that participated in the study had an adult obesity prevalence rate of 15–19%. In 2004, 7 states had obesity prevalence rates of 15–19%, 33 states had rates between 20 and 24%, and 9 states had rates of more than 25%. [3] increase in obesity has occurred in both sexes, in all age groups, and across all ethnic groups [4] As of 2004, over 24% of Americans were obese. [5] Being obese or overweight can increase risks for secondary health diseases such as hypertension, dyslipidemia, and diabetes [6] Currently, the standard definition of an overweight adult is a BMI between 25–29.9 kg/m², whereas obesity is defined as a BMI of ≥30. When defining obesity as a measure of body fat, men and women are considered obese at 25% and 33%, of men and women we are considered obese respectively.

The economic has expanded past health into the pocketbooks of many Americans. Indirect and direct costs were estimated to be \$117 billion in 2000, with slightly more money contributing to the direct cost. Direct costs refer to services that involve the treatment, prevention, or diagnosis of obesity and overweight, whereas indirect costs are associated with wages lost due to the inability to work in the present and future. [7] There are various possibilities for the cause and prevention of obesity. Although obesity may have genetic links, the epidemic increase in obesity during the past 20 years appears to stem from consistent over consumption of calories [8,9] and chronic inactivity. [10]

Key words: carbohydrate-restricted diet; fat-restricted diet; high-protein diet; mediterranean diet; fasting

Introduction

Macronutrient Levels and Weight Loss

With the popularity of obesity and weight loss books, it's no surprise that 4,444 of the majority of American adults say "diet." [11] So, which meal plan is the best? Regarding weight loss, the agreement is that successful weight loss requires fewer calories than needed to maintain weight. However, debate continues regarding the impact of different micronutrients on weight loss on food plans. Several studies have shown that the macro nutrient composition of calorie-controlled meal plans play an important role in the treatment of obesity. [12-14] In the past, high-fat diet plans were seen as the root cause of obesity. [15] According to this theory, weight loss recommendations include reducing food intake to 60 grams per day or less. [16]

By default, Americans increased their carbohydrate intake by [17] and increased their total energy intake. 6 The result is decreased body fat oxidation, [18,19] increased blood triglycerides, [20] and decreased satiety

[21] causing many people not to know the direction when choosing what to eat.

The above study has led to increased evidence of obesity and the association of with excess calories from a bulk diet plan. [22] High carbohydrate consumption raises blood sugar and causes an increased release of insulin in the blood, which increases tissue glucose or decreases blood (circulating) glucose. Increased insulin output [23] and a potential postprandial hypoglycemic response, excessive exercise, and energy balance may be contributing factors. Other researchers have recommended a more protein-rich diet plan than other weight loss. [24,25] Eating more protein helps maintain muscle mass, but does not appear to increase blood sugar that carbohydrates do. [26] A meal plan high in protein and low in carbohydrates reduces postprandial glucose and insulin responses and consistently provides an amino acid substrate for glucose production in the liver, which helps

stabilize blood glucose. [27] This new study leads us to rethink the importance of protein in nutrition programs.

Discovering Protein

Proteins are vital to life; a fundamental component of the meal plan necessary for physical development and organ and cell functions. Proteins are labeled as micronutrients, like carbohydrates and fat. Until recently the role of specific proteins and amino acids as functional ingredients has been limited more to the weightlifting and bodybuilding communities focused on muscle development. Furthermore, protein intakes are more than the Recommended Dietary Allowance (RDA) are often stated as potentially detrimental to renal function and bone mineralization. These concepts have been challenged and are being replaced by a new understanding of the

Protein Source	Amount of Protein (g)	Type of Protein	Amino Acids Profile
Egg Albumin	12.6g per 100g	Complete Protein	Rich in all essential amino acids
Whey Protein	20-25g per serving	Fast Digesting	High in BCAAs (Leucine, Isoleucine, Valine)
Casein Protein	24g per 100g	Slow Digesting	High in Glutamine, Essential Amino Acids
Chicken Breast	31g per 100g	Complete Protein	High in Leucine, Glutamine
Soy Protein	36g per 100g	Plant-Based Protein	Rich in Glutamine and Arginine

Source: Adapted from Rooseboom, C., Sports Nutrition: A Guide for the Professional Working with Active People, 3rd ed., The American Dietetic Association, Chicago, IL, 2000.

Overview Of Protein

Protein is a general term used to refer to a diverse category of molecules that contain amino acids. Proteins can be as small as the hormone insulin, containing 51 amino acids, or as large as myosin, a structural component of muscle containing 6,100 amino acids. Whereas the body contains a large array of proteins in structures, enzymes, and hormones, each protein is constructed from just 22 individual amino acids. These 22 amino acids are assembled in different amounts and different sequences to give each protein a unique size, shape, and function.

Amino acids are categorized into two groups as shown in Table 20.1. Nine amino acids are termed essential or indispensable for humans because they must be present in the daily meal plan, whereas 13 amino acids are considered nonessential or dispensable because the body can make them in adequate quantities; they are not required in the daily meal plan. [29] Quantity and quality of proteins differ among food sources due to the amino acid amount and types present in each protein. In general, foods from animal sources contain more protein and provide a more complete amino acid mixture than foods from plant sources (Table 20.2). A complete protein such as egg albumin contains adequate amounts of each of the essential amino acids in proper ratios, whereas an incomplete protein, such as wheat gluten does not have all the essential amino acids in adequate amounts or correct proportions

Roles Of Amino Acids and Proteins

Amino acids and the resulting proteins have multiple bodily functions. The essential roles of the body

importance of dietary protein for adult health. Over the past decade or so, protein has emerged as a functional food ingredient for several health areas including weight loss and diabetes. The weight loss industry in the U.S. is quickly approaching 50 billion dollars in annual revenue, and it will continue to grow as the number of overweight and obese individuals continues to increase.[28] Through a maze of fad diets and supplements, protein has emerged as a critical nutrient for improving body composition and a core ingredient for weight loss products. This chapter provides an overview of protein with special attention to protein levels and sources, as well as amino acids with unique metabolic roles that are particularly intriguing about weight loss.

- Structure and function include serving as:
- Structures in cell membranes, muscles, and bones
- Enzymes to help regulate chemical reactions
- Antibodies for the immune system
- Hormones as regulators of metabolic processes
- Clotting factors in the blood
- Blood proteins for transporting nutrients and oxygen
- Receptors on cells
- Enzymes for digestion and absorption of food
- Unique metabolic regulators (such as leucine) in protein synthesis and arginine in

Nitrous oxide

An important energy source for muscle, liver, and the intestine among the many diverse roles of amino acids, some of the most noteworthy effects have been observed with the branched-chain amino acid (BCAA) leucine. [30,31,32] Leucine participates in numerous metabolic processes, its obvious role being as an indispensable amino acid for new protein synthesis. Leucine also functions as a critical regulator of translation initiation of protein synthesis, a modulator of the insulin-PI3 kinase signal cascade, and a nitrogen donor for muscle production of alanine and glutamine. The potential for leucine to impact protein synthesis, insulin signaling, and production of alanine and glutamine is dependent on dietary intake and increasing leucine concentration in skeletal muscle.

Role	Function	Examples
Structural Components	Form structures in cell membranes, muscles, and bones	Collagen, Myosin
Enzymes	Catalyze biochemical reactions in the body	Amylase, Protease
Antibodies	Help the immune system fight off infections	Immunoglobulins
Hormones	Regulate metabolic processes	Insulin, Growth Hormone
Clotting Factors	Facilitate blood clotting and prevent excessive bleeding	Fibrinogen, Prothrombin
Blood Proteins	Transport nutrients, oxygen, and other vital substances	Hemoglobin, Albumin
Receptors	Act as binding sites on cells to receive signals	Insulin Receptors, Neurotransmitter Receptors
Metabolic Regulators	Regulate processes like protein synthesis and blood flow	Leucine (protein synthesis), Arginine (nitric oxide)
Energy Source	Provide energy for muscle, liver, and intestines, especially during fasting	Glutamine, Alanine

Source: ESHA Research, Professional Nutrition Analysis Software and Databases v. 9.6.1 2002–2003 ESHA Research.

Figure modified from the Food and Nutrition Board, National Academies of Sciences, 1994

Protein Requirements

Beginning in 1943, the RDAs have been used as a standard for nutrition guidelines. These guidelines were developed as minimal standards for health policy. By definition, the RDAs are designed to be (simply) adequate for most healthy people;{33} they are intended as guidelines to prevent deficiencies. In the past decade, Americans have become increasingly dissatisfied with nutrition guidelines defined to be simply adequate. In response to these concerns, the Food and Nutrition Board (FNB) of the National Academies of Sciences developed a broader concept of nutrition intakes. In 2002, the FNB published the Dietary Reference Intakes (DRIs) for macronutrients. The U.S. and Canada DRIs were established as reference values, quantitative estimates of nutrient intake, and a dietary planning tool for healthy people ensuring a sufficient intake of essential nutrients. These references are associated with reduced risks of chronic diseases.

The DRIs define safe ranges for nutrient intakes, ranging from minimum intake for the prevention of deficiencies (RDA) up to an upper limit (UL), defined as a safe intake below any adverse effects of excess intake (Figure 20.1). The DRI protein range is 0.8 g/kg up to ~2.0 g/kg, a range expressed as 10% to 35% of energy intake. It is important to recognize that the protein intake range relates to body weight and not energy intake. A dietary intake of 90 g/d (~1.1 g/kg for an 80 kg person) represents 12% of energy intake at 3000 kcal/d but 24% of energy intake at 1500 kcal/d. It is important to recognize that protein intake relates to body weight. At low energy intakes, protein might represent a higher percentage of daily energy, whereas, at high energy intakes, protein may represent a lower percentage of daily intake. This is a fundamental concept that is not adequately characterized in current health guidelines and leads to misrepresentations of nutrition plan quality.

The DRIs provide a concept of a safe range. The RDA represents the minimum level to avoid a deficiency and the UL represents the maximum safe level to avoid toxicity. There is a need for DRIs based on metabolic outcomes, the optimum levels of amino acids for growth or metabolic outcomes, the optimum intake for individuals engaged in strength training or cardiovascular exercise such as fitness enthusiasts or competitive athletes, and the optimum protein intake to maintain muscle and bone health in the elderly.{34} Further, in a society exposed to excess energy and epidemic increases in obesity and diabetes, it is unclear if a meal plan designed to provide the minimum amount of protein to prevent a deficiency is consistent with lifelong health. Figure Metabolism of branched-chain amino acids.

1. Introduction to BCAAs

Branched-chain amino acids consist of:

- Leucine
- Isoleucine
- Valine

These amino acids are essential, meaning they must be obtained from the diet.

2. Initial Metabolic Pathway

Step 1: Transamination

- BCAAs undergo transamination to form branched-chain keto acids (BCKAs).
- Enzyme involved: Branched-Chain Aminotransferase (BCAT).

Step 2: Oxidative Decarboxylation

- The BCKAs are then converted into their respective CoA derivatives through oxidative decarboxylation.
- Enzyme involved: Branched-Chain Keto Acid Dehydrogenase Complex (BCKDC).

3. Final Products

The CoA derivatives formed include:

- Acetyl-CoA (from leucine)
- Propionyl-CoA (from isoleucine)
- Succinyl-CoA (from valine)

These products can then enter the tricarboxylic acid (TCA) cycle to produce energy.

4. Physiological Importance

BCAAs are crucial for:

- Muscle protein synthesis
- Energy production during exercise
- Regulation of metabolic pathways (e.g., activation of mTORC1).

5. Health Implications

Dysregulation in BCAA metabolism is associated with:

- Obesity
- Type 2 diabetes
- Muscle wasting disorders

Protein Sources

There are different types of dietary protein within food sources. For example, a primary protein bread contains gluten, eggs contain albumin, and milk contains casein and whey. Many of them protein types are families or chemically associated protein molecules. Eggs for example albumin includes ovalbumin, Ovo transferrin, ovomucoid, ovomucin, and lysozyme. Meantime, whey includes β -lactoglobulin, α -lactalbumin, immunoglobulins, bovine serum albumin, lactoferrin, and lactoperoxidase, as well as glycomacropeptide (GMP), a protein in derived in from in casein in cheese in whey In. On the other hand, the main casein fractions are α (s1) and α (s2)-caseins, β -casein and kappa-casein. Milk has evolved as a unique protein for mammals, in part because of its protein composition. Casein, the main protein in milk, makes up about 80% of the total milk protein and is considered a slow-acting protein.

Due to the complex chemical nature of the casein protein, digestion and absorption of its amino acids can take up to several hours, depending on the amount consumed.³⁰ Therefore, casein would provide a slow, steady rise in blood levels and uptake of amino acids into circulation. Whey, on the other hand, is more digestible, allowing for a quick increase in blood amino acid levels and an increase in protein synthesis.^[35] Whey combination of a casein protein (fast and slow acting proteins) is considered beneficial in muscle regeneration, especially the time immediately following strength training. When whey protein is mentioned, the first thought that usually comes to mind is its use by bodybuilders or athletes due to its popularity as a protein supplement. But also, whey is a major part of infant formula due to its nutritional value. Whey consists of calcium, phosphorus, lactose, water, magnesium, fat, and, of course, protein.^[36] What's more, whey protein is also considered more satisfying than casein due to circulating amino acid levels after eating a meal.^[37]

Whey is rich in essential amino acids, especially branched-chain amino acids (BCAAs), leucine, iso leucine, and valine. These amino acids are major contributors to skeletal muscle replenishment following exercise³¹ or short-term periods of food restriction, such as overnight fasting. If the diet is adequate in leucine, then the muscles can build or maintain muscle protein. However, if the diet is inadequate for protein/leucine, then muscle protein synthesis is blocked and muscle breakdown can be used to maintain metabolic functions occur. This is one of the reasons why whey is believed to increase muscle mass in the strength-training individual. Although whey

has many other agreed benefits, the ability to increase muscle mass is still controversial.[38]

Protein Digestion and Absorption

The process of converting dietary protein into amino acids for use in the body is a complex process involving the stomach, small intestine, and liver. Although the process is complex, it is highly efficient with nearly 100% of dietary protein digested and absorbed into the intestinal cells, known as enterocytes. Protein digestion begins in the stomach as gastric acids denature complex protein structures, and pepsin begins to cleave protein chains. The resulting polypeptides are released into the small intestine where proteases derived from the pancreas and enterocytes continue protein digestion. Ultimately, protein digestion produces a mixture of free amino acids and di- and tri peptides in ratios of approximately 1:1:1. These amino acid mixtures and small peptides are absorbed into the enterocytes by amino acid and peptide transporters. Once inside enterocytes, the remaining peptides are hydrolyzed to amino acids before being released into the portal circulation.

Amino acids within the enterocyte can be used for intestinal enzyme synthesis, e.g., proteases, used for energy, or transported to portal blood for use by the rest of the body. The use of amino acids by the intestine varies greatly among amino acids. Dietary glutamine and glutamate are completely removed by the enterocyte as fuels; neither one of these amino acids, from a meal (or supplements), reaches the blood. In total, the enterocytes remove approximately 25% of dietary amino acids before they reach the blood and become available to other tissues.

Amino acids leave the intestine via the portal blood to the liver. The liver is the most active amino acid metabolism tissue in the body. Amino acids that reach the liver can be used for protein synthesis, as an energy source, or released into the blood. Similar to the intestine, the liver removes nearly 25% of dietary amino acids for energy. Surprisingly, the primary energy sources for both the intestine and the liver are amino acids. This means that less than one-half of dietary amino acids ever reach the blood or a majority of tissues. Although the liver and intestines use amino acids for energy, amino acids are not removed uniformly. The enterocyte is active in removing glutamine, glutamate, asparagine, and aspartate, whereas the liver is capable of metabolizing most of the remaining amino acids. The major exemptions to the intestine and liver are the BCAAs. These three amino acids are unique in that the liver lacks the necessary enzymes to metabolize them; the net result, BCAAs appear in the blood in nearly the exact amounts present in a food

Protein Turnover

Amino acids enter the blood, move throughout the body, are transported into cells, and become available for the synthesis of new proteins. Proteins within the body are constantly being made and destroyed (see Figure 20.3). Some proteins such as enzymes have a lifespan of only a few hours, whereas other structural proteins such as connective tissues are retained for as long as 6 months. Hence, the body has a daily need to replace most enzymes, whereas a sprained ankle may take 4 to 6 months to be completely repaired. The process of synthesis and degradation of proteins is called protein turnover. Each day, the body makes and degrades over 250 g of protein. The magnitude of this turnover is surprising as few people consume more than 100 g of protein per day. The lack of a direct relationship between the amount of dietary protein and the level of daily protein turnover emphasizes the difficulty in defining protein requirements. Body protein quantity is largely determined by the balance of protein synthesis and degradation. Although the daily turnover is greater than 250 g/d, the actual potential to accumulate new proteins is very limited. During maximum growth, protein turnover is positive, i.e., synthesis is greater than degradation, but the net balance is less than 10 g/d. Protein turnover balance appears to be largely regulated by protein synthesis change.

Protein synthesis is a complex process that assembles the 22 amino acids into hundreds of individual proteins. This complex process is regulated by the gene expression of mRNA (the blueprints for each new protein), the availability of amino acids and energy, and regulatory proteins called

initiation factors. These controls allow the body to make new proteins in the correct cells at the correct time. A review of protein synthesis is beyond the scope of this chapter; however, new research has shown an important link between insulin and leucine in the regulation of protein synthesis that appears to be a key to understanding the management of body weight and composition.

Bcaas — Specifically, Leucine and Weight Loss

BCAAs are mainly used for protein synthesis [39] and are participants in signal transduction pathways, which may help provide the anabolic effect protein has on muscle tissue.[40] Leucine, in particular, has shown the same effects as amino acid mixtures[40] and therefore will be the focus of the BCAA section.

The BCAA leucine has multiple roles in metabolism, including being a substrate for protein synthesis,[41] a fuel for skeletal muscle [42] and a nitrogen donor for the production of alanine and glutamine in skeletal muscles.[43] These roles are dependent on the dietary intake of leucine.[41] Due to leucine metabolism, the levels consumed are relative to the levels that reach the skeletal muscle. Leucine's contribution to the stimulation of protein synthesis is supported by human studies.[44] Leucine or even a mixture of the BCAAs can stimulate protein synthesis during energy restriction.[45] Low-calorie controlled high-protein meal plans (one providing 10 g of leucine per day equivalent to around 125 g of dietary protein per day) compared to USDA Food Guide Pyramid recommendations, showed a greater loss of weight and improved body composition (increase in body fat loss and decrease in muscle mass loss) with the high-protein meal plan. See Table 20.2 for a breakdown of BCAAs in food sources.

Protein And Glycemic Control

Dietary protein plays a role in blood glucose regulation via its effects on insulin [46] and increased availability of substrates (amino acids) for gluconeogenesis.[47] Janey and colleagues [48] demonstrated that 50–80 g of glucose could be generated from 100 g of ingested protein, while Jungas and colleagues stated that the primary liver fuel source in the fasted state is amino acids.[27] Amino acids are produced by protein breakdown in the muscle. They transfer to the liver, deaminate, and become carbon skeletons for gluconeogenesis.[47] Common substrates for gluconeogenesis include alanine and glutamine, which are deaminated into pyruvate and glutamate, respectively. Gluconeogenic substrate availability is thought to be proportional to dietary amino acid consumption.[49]

Therefore, an increase in dietary protein would lead to increased availability of gluconeogenic substrates, also relative to BCAA amount. The fasted state is accompanied by a decrease in insulin and an increase in glucagon, which causes an increase in hepatic glucose production and degradation of glycogen, via a series of dephosphorylations, to produce fuel for the body.[50] Glucagon is also known to be a stimulator of gluconeogenesis.[46] With increased substrate availability and stimulation of hepatic glucose production, a moderate protein meal plan increases the role of the liver in blood glucose control.[51,52] This blood glucose control method has been used for regulation in type 2 diabetes for years. [53]

Energy Metabolism

Meal consumption stimulates a series of physiological and metabolic processes. When food is consumed, the metabolic state changes from a catabolic to an anabolic state, due to an increase in protein synthesis and a decrease in protein breakdown.[54] Generally, during the absorptive period, there is a rise in blood glucose levels. Insulin will aid in the uptake and utilization of glucose into muscle and adipose, decrease hepatic glucose output, increase glycogen synthesis, decrease lipolysis, and decrease protein degradation. Therefore, within a few hours after a meal, dietary glucose is either stored or oxidized, [55] whereas fat is either used or stored for future use, and protein is either used for the previously mentioned functions or made into glucose and then stored as glycogen.[27]

Once the food has been absorbed, the body relies on endogenous energy sources for fuel. Maintained blood glucose levels sustain the brain and glycolytic requirement of glucose. As the exogenous supply of glucose decreases, insulin secretion follows suit, whereas glucagon secretion increases. Glucagon stimulates liver glycogenolysis to release glucose into circulation. Liver glycogen as a glucose resource will also be prolonged by tissues such as skeletal muscle, increasing the use of alternative fuel sources. The insulin level fall allows for an increase in adipose tissue lipolysis, resulting in the release of free fatty acids (FFA) into circulation. This decrease in glucose and insulin, and an increase in FFA availability for fuel, is known as the glucose–fatty acid cycle or Randle cycle.[56] When inadequate blood glucose-producing carbohydrate is consumed, blood glucose is maintained through glycogen breakdown and gluconeogenesis. Liver glycogen is the primary glycogen tissue, whereby the derived glucose can be released into the blood. However, liver glycogen is limited for adults and easily exhaustible. For instance, liver glycogen levels can be reduced to nadir levels within the first day of starvation. During semi starvation, glycogen levels are dramatically decreased, and the role of hepatic glycogenolysis in maintaining blood glucose levels is lessened relatively.

Gluconeogenesis creates glucose from non-carbohydrate substrates, namely pyruvate, lactate, glycerol, alanine, and glutamine. Lactate and alanine carbons are recycled between the brain and liver or skeletal muscle and liver, respectively, are, respectively, called the Cori and glucose–alanine cycles. The major gluconeogenic precursors are amino acids alanine and glutamine, which are derived mainly from proteolysis in skeletal muscle. This combination of actions helps maintain blood glucose levels during a fasting period.⁵⁵ Thus, dietary protein is an important fuel consideration during caloric imbalances.

Weight Loss and Energy Intake

Weight loss is often stated as a matter of simple energy economy. When calories out exceed calories in, there must be a net energy expenditure resulting in a body mass reduction. A calorie is the cumulative amount of the calories consumed, which, by and large, are carbohydrates, proteins, fat, and alcohol. On the opposite hand, daily caloric expenditure is a reflection of resting metabolism, lifestyle (daily activities), and exercise. When considering strategies for weight loss, both sides of the energy balance equation must be considered. Increased food availability (e.g., portion sizes, buffets, convenience stores, etc.) partly explains the caloric consumption increase. In addition, public perception of caloric consumption vs. Certain foods can be a contributing factor. For instance, the American Institute for Cancer Research surveyed to gauge public perception as to which was more important for weight management, the amount or type of food eaten.[57] Of those surveyed, 78% of the respondents said that eating certain foods was more crucial to weight management success than the actual amount of food consumed.⁵⁷

Weight loss can be accomplished via a calorie restriction and/or an increase in caloric expenditure (exercise). Researchers have reported positive study results with the prevention or treatment of adult obesity that focuses on modifying calorie intake [58,59] Energy restriction also reduces secondary health risks associated with obesity.[60] In support, energy restriction positively influences fasting blood glucose, hepatic glucose production, and blood insulin values with effects seen within 7–10 d of initial energy restriction. [61–63] All of these can enhance weight loss success. Blood glucose control, in fed and fasted states, is important when maintaining and/or losing weight. As discussed above, the fed state produces a blood glucose increase which causes an increase in insulin secretion via the pancreas. Insulin causes translocation of the intracellular glucose transporters to the plasma membrane for tissue glucose uptake,[64] suppression of hepatic glucose production in the liver,⁶⁵ and synthesis of glycogen.[66] Gluconeogenic precursors (alanine, pyruvate, glutamine) are shifted toward glycogen formation,[67] and insulin manages glucose uptake. Humans adapt to restricted energy intake using numerous mechanisms.[68] One adaptation is the conservation of energy via metabolic responses.[69] An adult will adapt to energy restriction with reduced hepatic glucose

production, [70,71] a decrease in basal metabolic rate, and a reduction in weight and activity. Control of blood glucose is important as many obese people exhibit chronic hyperinsulinemia, insulin resistance, and dysfunction of oxidative and non-oxidative glucose disposal pathways. This may be a result of how the body handles consistent high carbohydrate eating patterns.²² It is logical to ask whether the meal plan composition can influence these conditions.

Protein And Weight Loss

As previously stated, protein is used for many metabolic and physiological reasons. Generally, during a state of weight loss, there is an emphasis on a caloric intake decrease and a caloric expenditure (activity) increase. During an energy imbalance favoring weight loss, the dependence on body protein to sustain its energy needs increases. Being the largest and most accessible protein resource, muscle mass is targeted. The weight loss plan does not provide enough protein to service the anatomical and physiological needs as well as provide energy through the weight loss period.

Dietary protein would need to compensate for the additional protein needed in general, but also provide adequate amounts of the essential amino acids. As discussed above, one of the adaptations to an energy-restricted meal plan is a decrease in hepatic glucose production. If this continues, it can produce a drop in blood glucose levels if gluconeogenesis does not increase the production of glucose in the blood. Meal plans high in protein have shown an increase in PEPCK mRNA, a key enzyme in gluconeogenesis, [72,73] which causes an increase in glucose production. This suggests that maintenance of glucose homeostasis during energy restriction may depend on meal plan composition, and provides a link between energy restriction and glucose homeostasis, which is important in obesity prevention/treatment.²¹ Modifications in energy intake, exercise, and specific macronutrients composition can decrease body weight, fasting plasma glucose, and insulin concentrations closer to homeostatic values.⁶⁰ An increase in dietary protein and a decrease in dietary carbohydrates has been shown to produce glucose homeostasis, increase lean body mass, increase fat loss, and improve blood lipid profiles.¹⁴ These studies suggest that meal plans with a reduced amount of carbohydrates and an increase in protein, can increase weight loss and loss of body fat, and reduce loss of lean tissue.²⁰ For instance, one study involved overweight women assigned to one of two groups: a high carbohydrate or a moderate

protein meal plan for 10 weeks.¹⁴ The higher carbohydrate group received a plan similar to the Food guide Pyramid which had a carbohydrate-to-protein ratio of 3.5 (~68 g protein/day). The moderate protein group received a carbohydrate-to-protein ratio of ~1.4 (~125 g protein/day). Both groups lost weight, but the moderate protein group had a significantly higher loss of fat/lean tissue ratio. This meant that more fat was lost and more lean muscle mass was preserved (protein-sparing) than in the high carbohydrate meal plan. The high carbohydrate group also had an increased meal insulin response and postprandial hypoglycemia when compared to the moderate protein group. An animal model with similar meal plans, but no energy restriction, resulted in comparable glucose and insulin outcomes. Basal hepatic glucose production was greater in the moderate protein group as compared to the high carbohydrate group; increased hepatic glucose production was influenced by the increased amount of dietary protein consumed.⁶⁷ Increased hepatic glucose production has been reported to be important in blood glucose maintenance in the fasted state.[74],⁵¹ Other studies show similar beneficial results in blood lipids and overall body composition with dietary substitution of protein for carbohydrates.^{13,19,20,40,[75]}

Protein And Appetite

Appetite can be influenced by biological, environmental, and behavioral factors. The biological factor that drives an individual to consume food is hunger. Food that inhibits further consumption produces satiety and a delay in the onset of the next meal. Food that is considered to have a high level of satiety produces a long period between feelings of hunger. Macronutrients at equivalent calorie levels have been shown to have different satiety effects.

[76,77] Higher protein intake is often thought to reduce appetite, which can lead to reduced caloric consumption. A review of energy density (calories/gram) noted a hierarchical effect on satiety in the order of protein carbohydrates fat.[78-80] Participants in one study were fed protein, carbohydrate, and fat contributing either 29%, 61%, and 10% of energy, respectively (higher in protein and carbohydrate), or 9%, 30%, and 61% of energy (higher in fat), respectively, both in energy balance.[81] Diet-induced thermogenesis (DIT) and satiety were higher in the high protein/carbohydrate diet than in the high-fat group. Researchers in another study fed protein, carbohydrates, and fat contributing 10%, 60%, and 30% of energy, respectively, or 30%, 40%, and 30% to healthy women in energy balance.[82] The researchers reported an increase in sleeping metabolic rate, DIT, and satiety, and a lower 24-h hunger (calorie consumption) and respiratory quotient (RQ) in the high protein group. They also found incidental relationships between satiety and ghrelin, and glucagon-like peptide 1, but only with the higher protein intake.

Weight Loss and Exercise

Positive study results of adult obesity prevention/treatment focus on increased exercise. [83,84] The NIH guidelines for weight management emphasize the need for both proper nutrition and increased physical activity (minimum of 30 min per day of moderate intensity for exercise 7 d a week) for weight control. In 2003, 59% of adults did not engage in vigorous leisure-time physical activity, whereas only 26% of adults engaged in vigorous leisure-time physical activity 3 or more times per week.[85] Exercise has been projected to be important in the production of weight/fat loss, [86] prevention/treatment of obesity, [87] maintenance of blood glucose,[88] decreases of plasma insulin concentrations, [89] maintenance of muscle mass. Exercise is known to induce a fall in circulating insulin levels with an increase in glucose utilization and increased insulin sensitivity.[90] Paffen Barger and colleagues [91].

Reported epidemiological studies showing the association between decreased obesity risks when physical activity was performed. Satabin and colleagues [92] used male Wistar CF rats with a high Protein diet compared to a control, high in carbohydrates. Rats were exercised on a treadmill for 60 Min/d for 3 weeks. Blood glucose was measured and found to remain in homeostasis, via an increase in liver gluconeogenesis, with rats on the high protein diet. Previous in vitro studies have shown an increase in insulin's action on glucose uptake when muscular contractions are present.[93] Ji and colleagues [94] showed that after 10 weeks of training, there was a significant increase in gluconeogenic enzyme activity. This produced an increase in glucose Production, through gluconeogenesis, supporting the stabilization of blood glucose. Holm and colleagues [95] conducted a study involving obese women who exercised for 1 h at 70% of their Maximal working capacity. Subjects fasted for 16–18 h, then tested for blood values. The Results showed a decrease in plasma insulin and triglycerides a few days following exercise. Rodnick and colleagues also demonstrated this in a study with rats that were exercise-trained in wheel cages for 6 weeks [96] Results showed a significant difference in fasting serum insulin between the exercise-trained group and the sedentary group, with the exercise-trained group having a lower insulin value than the sedentary group. Thus, exercise appears to improve insulin sensitivity [97] leading to glucose uptake, which is further enhanced in trained skeletal muscle.[96]

Protein, Exercise, And Weight Loss

The debates continue whether eating the recommended RDA for protein is adequate for a person who exercises regularly. Some 38 feel it is adequate but others [98] think it can lead to an increase in protein breakdown and a decrease in protein synthesis, possibly leading to an increase in protein needs. Over time, not eating enough protein can lead to a decrease in muscle mass[99] and physical performance.[99] During exercise, the BCAA leucine is mainly used by the muscles,[100] with an increase in leucine oxidation [101] After exercise, leucine stimulates muscle recovery.[102] Layman and colleagues [103] conducted a 4-month, 2 × 2 weight loss study with adult obese (determined by BMI) women. Meal plans were either a high protein

or a high carbohydrate meal plan with or without exercise. The dietary composition of the carbohydrate group consisted of 0.8 grams of protein per kilogram body weight per day (~15% of energy intake) and ~30% of energy intake from dietary fat. The dietary composition of the protein group consisted of 1.6 g of protein per kilogram body weight per day (~30% of energy intake) and ~30% of energy intake from dietary fat. The exercise treatments consisted of walking 5 d per week for 30 min per day with an additional 2-d-a-week 30-min resistance training session. The non-exercise groups followed the NIH guidelines and exercised (walked) for 30 min, 5 d a week. The high protein meal plan with and without exercise produced greater weight loss after 16 weeks than the carbohydrate meal plan. The higher protein meal plan with exercise eliminated the most body fat. All groups lost weight on these calorie-controlled meal plans, but subjects in the protein groups lost more total weight and body fat and maintained more muscle mass than the carbohydrate groups. The protein group with exercise appeared to experience an additive effect on body composition and weight loss.[103]

Research Method

The research form division outlines the systematic approach used to conduct the review. It starts accompanying a detailed writing of the search action working to identify appropriate studies. Various photoelectric databases, such as PubMed, MEDLINE, and Google Scholar, concede possibility have happened utilized, accompanying distinguishing search agreements related to protein consumption, burden loss, and crowd arrangement. Inclusion tests were established to select studies gathering fixed criteria, containing randomized regulated trials (RCTs), potential comrade studies, and meta-reasonings focusing on the belongings of protein devouring on weight administration effects. Studies including diverse member public, including men of various age groups, genders, and health statuses, were deliberate to capture a general of evidence.

The traits of included studies, in the way that sample breadth, study design, participant head count, mediation pacts (e.g., protein portion of drug or other consumable, event), control groups, and outcome measures (for instance, changes in crowd weight, corpse fat allotment, lean bulk mass, metabolic stones), were carefully reviewed and combined. Potential biases and disadvantages owned by the selected studies, to a degree disclosure bias, participant devotion to abstinence from food interventions, and confusing variables, were accepted and focused on to enhance the lawfulness and generalizability of the review verdicts.

Results

The results section presents a inclusive combination of the verdicts from the reviewed studies concerning the belongings of protein intake on burden misfortune and body arrangement. Quantitative dossier, containing mean changes in body burden, bulk fat percentage, lean frame bulk, and metabolic limits, were extracted and resolved. Across the contained studies, a consistent current towards approving outcomes guide raised protein use was observed. Specifically, bigger protein diets were guide greater reductions in frame burden, specifically fat mass, distinguished to lower protein diets. Additionally, maintenance of lean body bulk and betterings in metabolic markers, to a degree insulin feeling and lipid descriptions, were commonly stated accompanying higher protein consumption.

Statistical studies, containing meta-analyses place appropriate, were performed to decide the importance and significance of the noticed belongings. Subgroup studies may have happened attended to explore potential modifiers of protein's belongings, to a degree age, grammatical rules applying to nouns that connote sex or animateness, baseline metabolic rank, and digestive adherence.

Discussion

In the dispute portion, the implications of the review verdicts are precariously resolved in the context of existent history and theoretical foundations. The methods latent protein's beneficial belongings on pressure

management, containing raised thermogenesis, reinforced feeding, and maintenance of lean body bulk, are elucidated established physiological and metabolic law. Limitations of the inspected studies, to a degree methodological distinctness's, variety in participant traits, and challenges in abstinence from food assessment and agreement listening, are approved and discussed. Suggestions for future research endeavors, containing well-devised RCTs with more protracted effect periods and patterned outcome measures, are projected to address existent gaps and improve the strength of evidence.

Practical implications of the review judgments for healthcare pros, nutritionists, and things seeking pressure administration strategies are described. Recommendations for combining enough protein into daily abstinence from food consumption, such as stressing lean protein beginnings (e.g., fowl, bait, legumes) and classifying protein intake proportionately during the whole of the day, are supported to amend pressure loss and bulk arrangement outcomes.

Conclusions

Ongoing research will continue to support the need for customized food-plans. Weight-loss methods need to be considered on an individual basis, including personal choices in lifestyle and how the food will affect individual metabolic outcomes. Nutrition plans with increased levels of protein and leucine, present in high levels in animal proteins can be used to substitute for high glycemic carbohydrates and have been shown to enhance insulin sensitivity,⁴⁵ stimulate muscle protein synthesis,^{102,104} reduce the role of insulin in glycemic control,⁴⁵ and stimulate the role of the liver in the stabilization of blood glucose.⁴⁵ In these studies, the net effects of these changes are lower body fat, increased lean muscle mass, increased insulin sensitivity, increased hepatic gluconeogenesis, Stabilization of fasting blood glucose, and reduced serum triglycerides.¹⁴ As previously stated, if these types of meal plans are sustainable throughout a person's lifespan, fit into a person's lifestyle, and taste good, then that person may benefit from a high-protein food Plan during weight loss. The choice is up to individual bodies.

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References

1. U.S. Department of Health and Human Services, National Center for Health Statistics, Centers for Disease Control and Prevention, Prevalence of Overweight and Obesity among Adults, Hyattsville, MD, 1999
2. U.S. Department of Health and Human Services, National Center for Health Statistics, Centers for Disease Control and Prevention. Prevalence of obesity among adults aged 20 years and over: U.S., 1997-2001. Hyattsville, MD; 2002
3. U.S. Department of Health and Human Services, National Center for Health Statistics, Centers for Disease Control and Prevention. Overweight and obesity: obesity trends: U.S. obesity trends 1985-2004. Hyattsville, MD
4. Variyam JN. (2002). Economic Research Service, USDA. *Food Rev.*;25(3):16-20
5. U.S. Department of Health and Human Services, National Center for Health Statistics, Centers for Disease Control and Prevention. Early release of selected estimates based on data from the January-June 2004 National Health Interview Survey (12/2004)
6. National Heart, Lung, and Blood Institute, National Institute of Diabetes and Digestive and Kidney Diseases. Obesity Education Initiative. In: Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence reports. Bethesda, MD: National Heart, Lung, and Blood Institute, in cooperation with the National Institute of Diabetes and Digestive and Kidney Diseases; 1998. p. 12-19. NIH Publication No. 98-4083
7. Wolf AM, Colditz GA. (1998). Current estimates of the economic cost of obesity in the U.S. *Obes Res.*; 6:97-106
8. McCance RA, Widdowson EM. (1962). Nutrition and growth. *Royal Society of London Proceedings.*; 158:326-337.
9. Hill JO, Peters JC. (1998). Environment contributions to the obesity epidemic. *Science.*; 280:1371-1374.
10. U.S. Department of Health and Human Services, Physical Activity and Health: A Report of the Surgeon General, Atlanta, GA, 1996.
11. Serdula, M.K., Mokdad, A.H., Williamson, D.F. et al., (1999). Prevalence of attempting weight loss and Strategies for controlling weight, *JAMA*, 282: 1353-1358.
12. Alford, B.B., Blankenship, A.C., Hagen, R.D., (1990). The effects of variations in carbohydrate, protein, fat content of the diet upon weight loss, blood values, and nutrient intake of adult obese women, *J Am Diet Assoc*, 90: 534-540.
13. Skov, A.R., Toubro, S., Ronn, B., Holm, L., Strup, A., (1999). Randomized trial on protein vs. carbohydrate in ad libitum fat reduced diet for the treatment of diabetes, *Int J Obes*, 23: 528-536.
14. Layman, D.K., Boileau, R.A., Erickson, D.J., Painter, J.E., Shiue, H., et al., (2003). A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles During weight loss in adult women, *J Nutr*, 133: 411-417.
15. Tremblay, A., (1995). Differences in fat balance underlying obesity, *Int J Obes*, 19(Suppl. 7): 10S-14S.
16. Nutrition and Your Health, Dietary Guidelines for Americans. U.S. Department of Agriculture, Washington, D.C.: U.S. Govt. Print. Off., 1980.
17. Jahoor, R., Peters, E.J., and Wolfe, R.R., (1988). Gluconeogenic precursors supply and glucose production, *FASEB J*, 2:1215.
18. McGarry, J.D., Kuwajima, M., Newgard, C.B., Foster, D.W., (1987). From Dietary Glucose to Liver glycogen, *Annu Rev Nutr*, 7: 51-73.
19. Wolfe, R.R., (1998). Metabolic interactions between glucose and fatty acids in humans, *Am J Clin Nutr*, 67(3 Suppl.): 519S-526S.
20. Parker, B., Nokes, M., Luscombe, N., Clifton, P., (2002). Effects of a high-protein, monounsaturated fat weight Loss diet on glycemic control and lipid levels in type 2 diabetes, *Diabetes Care*, 25: 425-430.
21. Ludwig, D.S., (2000). Dietary glycemic index and obesity, *J Nutr*, 130: 280S-283S.
22. Heller, R.F., Heller, R.F., (1994). Hyper-insulinemic obesity and carbohydrate addiction: the missing link is the carbohydrate frequency factor, *Med Hypothesis*, 42: 307-312.
23. Ullrich, I.H., Albrink, M.J., (1985). The effect of dietary fiber and other factors on insulin response: role in Obesity, *J Environs Pathol Toxicol Oncol*, 5(6): 137-155.

24. Cohen, D., Dodds, R., Viberti, G.C., (1987). Effect of protein restriction in insulin-dependent diabetics at risk of nephropathy, *Br Med J*, 294: 795–798.
25. Seney, F.D. Jr., Wright, F.S., (1985). Dietary protein suppresses feedback control of glomerular filtration in Rats, *J Clin Invest*, 75: 558–568.
26. Gannon, M.C., Nuttal, F.Q., Lane, J.T., Burmeister, L.A., (1992). Metabolic response to cottage cheese or egg white protein, with or without glucose in type 2 diabetic subjects, *Metabolism*, 41: 1137–1145.
27. Jungas, R.L., Halperin, M.L., Brosnan, F.T., (1992). Quantitative analysis of amino acid oxidation and related Gluconeogenesis in humans, *Physiol Rev* 72(2): 419–448.
28. Nutrition Business Journal, NBJs Sports Nutrition, and Weight Loss Report 2005.
29. Berdanier, C., (2000). Proteins, in Advanced Nutrition: Macronutrients, 2nd ed., Boca Raton, FL: CRC Press, 130–196.
30. Dangin, M. et al., (2001). The digestion rate of protein is an independent regulating factor of postprandial Protein retention, *Am J Physiol Endocrinol Metab.*, 280: E340–E348.
31. Blomstrand, E., Hassmen, P., Ekblom, B., Newsholme, E.A., (1991). Administration of branched-chain amino acids during sustained exercise — effects on performance and plasma concentration of some amino Acids, *Eur J Appl Physiol*, 63: 83–88.
32. Gibson, N.R., Fereday, A., Cox, M., Halliday, D., Pacy, P.J., Millward, D.J., (1996). Influences of dietary energy and protein on leucine kinetics during feeding in healthy adults *Am J Physiol*, 270: E282–E291.
33. Recommended Dietary Allowance, 10th ed., National Academy Press, Washington, DC, 1989.
34. Evans, W.J., (1998). Exercise and nutritional needs of elderly people: effects on muscle and bone, *Gerodontology*, 15: 15.
35. Dairy Council Digest, Health-Enhancing Properties of Dairy Ingredients, 72(2): 7–12, 2001.
36. Boire, Y. et al., (1997). Slow and fast dietary proteins differently modulate postprandial protein accretion, *Proc Natl Acad Sci USA*, 94: 14930–14935.
37. Hall, W.L., Millward, D.J., Long, S.J., Morgan, L.M., (2003). Casein and whey exert different effects on plasma amino acid profiles, gastrointestinal hormone secretion, and appetite, *Br J Nutr*, 89: 239–248.
38. Rosenbloom, C., (2000). Sports Nutrition: A Guide for the Professional Working with Active People, 3rd ed., *The American Dietetic Association*, Chicago, IL.
39. Holecek, M., Sprongl, L., Tilser, I., (2001). Metabolism of branched-chain amino acids in starved rats: the Role of hepatic tissue, *Physiol Res*, 50: 25–33.
40. Hutson, S.M., Harris, R.A., (2001). Leucine as a nutritional signal, *J Nutr*, 131: 839S–840S.
41. Layman, D.K., (2003). The role of leucine in weight loss diets and glucose homeostasis. Symposium: dairy Product components and weight reduction, *J Nutr*, 133: 261S–267S.
42. Wagenmaker, A.J.M., (1998). Muscle amino acid metabolism at rest and during exercise: role in human Physiology and metabolism, *Exerc Sport Sci Rev*, 26: 287–314.
43. Ruderman, N.B., (1975). Muscle amino acid metabolism and gluconeogenesis, *Annu Rev Med*, 26: 245–258.
44. Platell, C., Kong, S.E., McCauley, R., Hall, J.C., (2000). Branched-chain amino acids, *J Gastroenterol Hepatol*, 15: 706–717.
45. Layman, D.K., Shiue, H., Sather, C., Erickson, D., Baum, J., (2003). Increased dietary protein modifies glucose and insulin homeostasis in adult women during weight loss, *J. Nutr*, 133: 405410.
46. Muller, W.A., Foloona, G.R., Unger, R.H., (1976). The influence of the antecedent diet upon glucagon and Insulin secretion, *N Engl J Med*, 285: 1450–1455.
47. Jahoor, R., Peters, E.J., Wolfe, R.R., (1988). Gluconeogenic precursors supply and glucose production, *FASEBJ*, 2: 1215.
48. Janey, N.W., (1915). The metabolic relationship of the proteins to glucose, *J Biol Chem*, 20: 321–347.
49. Harper, A.E., Miller, R.H., Block K.P., (1984). Branch-chain amino acid metabolism, *Annu Rev Nutr*, 4: 409–454.
50. Hers, H.G., Van-Schaftingen, E., (1982). Fructose 2,6 bisphosphate 2 years after its discovery, *Biochem J*, 206: 1–12.
51. Katz, J., Tayek, J.A., (1998). Gluconeogenesis and the Cori cycle in 12-, 20-, and 40-h-fasted humans, *Am J Physiol*, 38: E537–E542.
52. Balasubramanyam, A., McKay, S., Nadkarni, P., Rajan, A.S., Farza, A., et al., (1999). Ethnicity affects the postprandial regulation of glycogenolysis, *Am J Physiol Endocrinol Metab*, 40: E905–E914.
53. Mayer, J., (1972). Dietary controls of diabetes. In Human Nutrition: Its Physiological, Medical and Social Aspects, Charles C Thomas, Springfield, IL, pp. 525–535.
54. Millward, D.J., Fereday, A., Gibson, N.R., Pacy, P.J., (1996). Post-preprandial protein metabolism, *Baillere's Clin Endocrinol Metab*, 10: 533–549.
55. Stipanuk, M.H., Regulation of fuel utilization, in Biochemical and Physiological Aspects of Human Nutrition, Stipanuk, M.H. (Ed.), W.B. Saunders, Philadelphia.
56. Randle, P.J., Garland, P.B., Hales, C.N., and Newsholme, E.A., (1963). The glucose fatty acid cycle: its role in insulin sensitivity and the metabolic disturbances of diabetes mellitus, *Lancet*, I: 785–794.
57. American Institute for Cancer Research, New Survey Shows Americans Ignore Importance of Portion Size in Managing Weight, March 24, 2000, American Institute for Cancer Research (accessed 26 July 2006).
58. Simon, E., Portillo, M., Fernandez-Quintela, A., Zulet, M., Martinez, J.A., et al., (2002). Responses to dietary macronutrients distribution of overweight rats under restricted feeding, *Ann Nutr Metab*, 46: 24–31.
59. Lean, Me.J., Han, T.S., Pravn, T., Richmond, P.R., Avenell, A., (1997). Weight loss with high and low carbohydrates 1,200 kcal diets in free living women, *Eur J Clin Nutr*, 51: 243–248.
60. Tuomilehto, J., Linstrom, J., Eriksson, J.G., Valle, T.T., Hamalainen, H., Ilane-Parikka, P., Keinanen Kiukaanniemi, S., Laakso, M., Louheranta, A., Rastas, M., Salminen, V., Uusitupa, M., (2001). For the Finnish Diabetes Prevention Study Group: Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance, *N Engl J Med*, 344: 1343–1350.
61. Wing, R.R., Blair, E.H., Bononi, P., Marcus, M.D., Watanabe, R., et al., (1994). Caloric restriction per se is a significant factor in improvements in glycemic control and insulin sensitivity during weight loss in obese NIDDM patients, *Diabetes Care*, 17: 30–36.
62. Henry, R.R., Scheaffer, L., Olefsky, J.M., (1985). Glycemic effects of intensive caloric restriction and isocaloric refeeding in non-insulin-dependent diabetes mellitus, *J Clin Endocrinol Metab*, 61: 917–925.
63. Dhahbi, J.M., Mote, P.L., Wingo, J., Rowley, B.C., Cao, S.X., et al., (2001). Caloric restriction alters the feeding response of key metabolic enzyme genes, *Mech Aging Dev*, 122: 1033–1048.
64. Karnieli, E., Zarnowski, M.J., Hissin, P.J. et al., (1981). Insulin-stimulated translocation of glucose transport systems in the isolated rat adipose cell, *J Biol Chem*, 256(10): 4772–4777.
65. Sheppard, P.R., Kahn, B.B., (1999). Glucose transporters and insulin action, *N Engl J Med*, 341: 248–257.

66. Cohen, P., (1983). In Control of Enzyme Activity, 2nd ed., Chapman and Hall, New York, pp. 42–71.
67. Rossetti, L., Rothman, D.L., DeFronzo, R.A., Shulman, G.I., (1989). Effects of dietary protein on in vivo insulin action and liver glycogen repletion, *Am J Physiol*, 257: E212–E219.
68. Mohan, P.F., Rao, B.S.N., (1983). Adaptation to underfeeding in growing rats: effect of energy restriction at two dietary protein levels on growth, feed efficiency, basal metabolism and overall body composition, *J Nutr*, 113: 79–85.
69. Lee, M., Lucia, S.P., (1961). Some relationships between caloric restriction and body weight in the rat. 1. Body composition, liver lipids, and organ weights, *J Nutr*, 74: 243–248.
70. Hellerstein, M.K., Letscher, A., Schwarz, J.M., Cesar, D., Shackleton, C.H., et al., (1997). Measurement of hepatic Ra UDP-glucose in vivo in rats; relation to glycogen deposition and labeling patterns, *Am J Physiol*, 272: E155–E162.
71. Christiansen, M.P., Linfoot, P.A., Nesse, R.A., Hellerstein, M.K., (2000). Effect of dietary energy restriction on glucose production and substrate utilization in type 2 diabetes, *Diabetes*, 49: 1691–1699.
72. Boisjoyeux, B., Chanez, M., Azzout, B., Peret, J., (1986). Comparison between starvation and consumption of a high protein diet: plasma insulin and glucagon and hepatic activities of gluconeogenic enzymes during the first 24 h, *Diabetes Metab*, 12(1): 21–27.
73. Peret, J., Chanez, M., Cota, J., Macaire, I., (1975). Effects of quantity and quality of dietary protein and variation in certain enzyme activities on glucose metabolism in the rat, *J Nutr*, 105(12): 1525–1534.
74. Pascual, M., Jahoor, F., Reeds, P.J., (1997). Dietary glucose is extensively recycled in the splanchnic bed of fed adult mice, *J Nutr*, 127: 1480–1488.
75. Mikkelsen, P.B., Toubro, S., and Astrup, A., (2000). Effect of fat-reduced diets on 24-h energy expenditure: comparisons between animal protein, vegetable protein, and carbohydrate, *Am J Clin Nutr*, 72: 1135–1141.
76. Blundell, J.E., Lawton, J.R., Cotton, J.R., Macdiarmid, J.L., (1996). Control of human appetite: implications for the intake of dietary fat, *Annu Rev Nutr*, 16: 285–319.
77. Holt, S.H., Miller, J.C., Petocz, P., Farmakalidis, E., (1995). A satiety index of common foods, *Eur J Clin Nutr*, 49: 675–690.
78. Stubbs, J., Ferres, S., Horgan, G., (2000). Energy density of foods: effects on energy intake, *Crit Rev Food Sci Nutr*, 40: 481–515.
79. Johnstone, A.M., Stubbs, R.J., Harbron, C.G., (1996). Effect of overfeeding macronutrients on a day-to-day food intake in man, *Eur J Clin Nutr*, 50: 418–430.
80. Blundell, J.E., Macdiarmid, J.L., (1997). Fat as a risk factor for overconsumption: satiety, satiety, and patterns of eating, *J Am Diet Assoc*, 97(Suppl.): S63–S69.
81. Westerterp-Plantenga, M.S., Rolland, V., Wilson, S.A., Westerterp, K.R., (1999). Satiety related to 24 h diet-induced thermogenesis during high protein/carbohydrate vs high-fat diets measured in a respiration chamber, *Eur J Clin Nutr*, 53(6): 495–502.
82. Lajeune, M.P., Westerterp, K.R., Adam, T.C., Luscombe-Marsh, N.D., Westerterp-Plantenga, M.S., (2006). Ghrelin and glucagon-like peptide 1 concentration, 24-h satiety, and energy and substrate metabolism during a high-protein diet and measured in a respiration chamber, *Am J Clin Nutr*, 83(1): 89–94.
83. Jeffery, R.W., Bjornson-Benson, W.M., Rosenthal, B.S., Linquist, R., Kurth, C.L., et al., (1984). Correlates of weight loss and its maintenance over two years of follow-up among middle-aged men, *Prev Med*, 13: 155–168.
84. Sherwood, N.E., Jeffery, R.W., French, S.A., Hannan, P.J., Murry, D.M., (2000). Predictors of weight gain in A pound of Prevention Study, *Int J Obes Relat Metab Disord*, 24: 395–403.
85. Summary Health Statistics for U.S. Adults: National Health Interview Survey, 2003, Table 29, U.S. Department of Health and Human Services, National Center for Health Statistics, Centers for Disease Control and Prevention, Available at
86. Segal, K.R. and Pi-Sunyer, F.X., (1989). Exercise and obesity, *Med Clin N Am*, 73(1).
87. Parizkova, J., (1963). Impact of age, diet, and exercise on man's composition, *Ann N Y Acad. Sci*, 110: 661–674.
88. Koivisto, V., Hendler, R., Nadel, E., Felig, P., (1982). Influence of physical training on fuel-hormone response to prolonged low-intensity exercise, *Metabolism* 31: 192–197.
89. Bjorntorp, P., Fahlen, M., Grimby, G., Gustafson, A., Holm, J., et al., (1972). Carbohydrate and lipid metabolism in middle-aged physically well-trained men, *Metabolism*, 21: 1037–1044.
90. Wasserman, D.H., Geer, R.J., Rice, D.E., Bracy, D., Flakoll, P.J., et al., (1991). Interaction of exercise and insulin action in humans, *Am J Physiol*, 260 (Endocrinol. Metab 23): E37–E45.
91. Paffenbarger, R.S., Hyde, R.T., Wing, A.L. et al., (1984). A natural history of athleticism and cardiovascular health, *JAMA*, 252: 491–495.
92. Satabin, P., Bois-Joyeux, B., Chanez, M., Guezennec, C.Y., Peret, J., (1989). Effects of long-term feeding of high-protein or high-fat diets on the response to exercise in the rat, *Eur J Appl Physiol*, 58: 583–590.
93. James, D.E., Kraegen, E.W., Chisholm, D.J., (1985). Muscle glucose metabolism in exercising rats: comparison with insulin stimulation, *Am J Physiol*, 248 (Endocrinol Metab 11): E575–E580.
94. Ji, L.L., Lennon, D.L.F., Kochan, R.G., Nagle, F.J., Hardy, H.A., (1986). Enzymatic model to physical schooling underneath B-blockade inside the rat: evidence for a B2-adrenergic mechanism in skeletal muscle, *J Clin makes investments*, 78: 771–778.
95. Holm, G., Bjorntorp, P., Jagenburg, R., (1978). Metabolism of carbohydrates, lipids and amino acids after bodily exercising in a guy, *J Appl Physiol: Respir Environ Exerc Physiol*, forty-five (1):128–131.
96. Rodnick, J., Reaven, G.M., Azhar, S., Goodman, M.N., Mondon, C.E., (1990). outcomes of insulin on carbohydrate and protein metabolism in loose-ranging rats, *Am J Physiol*, 259 (Endocrinol Metab 22): E706–E714.
97. Bofardus, C., Ravussin, E., Robbins, D.C., Wolfe, R.R., Horton, E.S., et al., (1984). results of bodily education and dietary therapy of carbohydrate metabolism in sufferers with glucose intolerance and non-insulin-based diabetes mellitus, *Diabetes*, 33: 311–318.
98. Wolf, R.R., Good enough, R.D., Wolfe, M.H., Royle, G.T., Nadel, E.R., (1982). Isotope analysis of leucine and urea metabolism in exercise human beings, *J Appl Physiol*, fifty-two: 458–466.
99. Lemon, P.W., (1996). Is extended dietary protein essential or beneficial for individuals with physically lively existence? *Nutr Rev*, 54: S169–S175.
100. younger, V.R., Bier, D.M., Pellet, P.L., (1989). A theoretical foundation for increasing present-day estimates of amino corporations' acid requirements in grownup men with an experimental guide, *Am J Clin Nutr*, 50: 80–92.
101. Bowtell, J.L., Lesse, G.P., Smith, okay., Watt, P.W., Nevill, A., et al., (1998). Modulation of whole-body protein metabolism, throughout and after a workout, by variant of dietary protein, *J Appl Physiol*, 85: 1744–1752.
102. Anthony, J.C., Anthony, T.G., Layman, D. k., (1999). Leucine supplementation will increase skeletal muscle recovery in rats after a workout, *J Nutr*, 129: 1102–1106.

103. Layman, D.k., Evans, E., Baum, J.I., Seyler, J.E., Erickson, D.J., Boileau, R.A., (2005). dietary protein and workout has additive effects on body composition for the duration of weight loss in adult girls, *J Nutr*, 135: 1903–1910.
104. Gautsch, T.A., Anthony, J.R., Kimball, S.R., Paul, G.L., Layman, D, et al., Eukaryotic availability of initiation element 4E regulates skeletal muscle protein synthesis during recuperation from exercising, *Am J Physiol*, 274:



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