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METABOLIIC EFFECT OF LOW CARBOHYDRATE DIETS

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Abstract: *Low-carbohydrate diets may promote greater weight loss than does the conventional low-fat, high-carbohydrate diet. Low-carbohydrate diets indeed provide "metabolic advantage," a greater weight loss/fat loss per calorie consumed compared to isocaloric (having similar caloric values) high-carb diets. Low-carbohydrate diets (LChD) have become very popular among the general population. These diets have been used to lose body weight and to ameliorate various abnormalities like diabetes, nonalcoholic fatty liver disease, polycystic ovary syndrome, narcolepsy, epilepsy, and others. Reports suggest that body weight reduction and glycemic control could be attained while following LChD.*
Keywords- *low-carbohydrate diets, ketosis, metabolic*

Introduction

A low-carbohydrate intake results in a lower circulating insulin/glucagon ratio, which promotes a high level of serum no esterified fatty acids (NEFAs) used for oxidation and production of ketone bodies. It is assumed that when the carbohydrate availability from liver glycogen and the exogenous carbohydrate supply is reduced during a short-term period to a significant amount, the body will be stimulated to maximize fat oxidation for energy needs. The ketone bodies, mainly produced from the oxidation of NEFAs and ketogenic amino acids such as leucine and lysine, comprise three compounds: acetoacetate (AcAc), 3-hydroxybutyrate (3HB), and acetone. Acetone is exhaled, while AcAc and 3HB are the most important ketone bodies synthesized and transported by the blood to extrahepatic tissues such as the brain, kidneys, and heart. In these tissues, AcAc and 3HB are oxidized in the citric acid cycle to meet most of the energy requirements. Ketosis results from serum elevation of ketone bodies; ketoacidosis and ketosis should not be used as synonyms since ketoacidosis is a threatening condition most common in untreated type 1 diabetes. In addition, reduced intake of carbohydrates during a VLChD promotes glycolysis inhibition and acceleration of gluconeogenesis. The latter happens in order

to provide glucose to tissues that require it as their sole or major fuel source, such as the renal medulla, central nervous system, gonads, and erythrocytes **(Aría-Eugenia *et al.*, 2011**

During very low carbohydrate intake, the regulated and controlled production of ketone bodies causes a harmless physiological state known as dietary ketosis. Ketone bodies flow from the liver to extra-hepatic tissues (e.g., brain) for use as a fuel; this spares glucose metabolism via a mechanism similar to the sparing of glucose by oxidation of fatty acids as an alternative fuel. In comparison with glucose, the ketone bodies are actually a very good respiratory fuel. Indeed, there is no clear requirement for dietary carbohydrates for human adults. Interestingly, the effects of ketone body metabolism suggest that mild ketosis may offer therapeutic potential in a variety of different common and rare disease states. Also, the recent landmark study showed that a very-low-carbohydrate diet resulted in a significant reduction in fat mass and a concomitant increase in lean body mass in normal-weight men. Contrary to popular belief, insulin is not needed for glucose uptake and utilization in man. Finally, both muscle fat and carbohydrate burn in an amino acid flame.

Much of the controversy in the study of LCDs stems from a lack of a clear definition. The rationale of carbohydrate restriction is that, in response lower glucose availability, changes in insulin and glucagon concentrations will direct the body away from fat storage and toward fat oxidation. There is a suggestion of a threshold effect, which has led to the clinical recommendation of very low concentrations of carbohydrate (20–50g/d) in the early stages of popular diets. This typically leads to the presence of measurable ketones in the urine and has been referred to as a very-low-carbohydrate ketogenic diet (VLCKD) or a low-carbohydrate ketogenic diet (LCKD). Potent metabolic effects are seen with such diets but, beyond the threshold response, there appears to be a continuous response to carbohydrate reduction. The nutritional intake of 200 g carbohydrate/d has been called an LCD, but most experts would not consider that to provide the metabolic changes associated with an LCKD. We suggest that LCD refers to a carbohydrate intake in the range of 50–150 g/d, which is above the level of generation of urinary ketones for most people **(Eric C Westman et al., 2007).**

Metabolic Effects of Low-Carb Diet

The hormonal changes associated with a low-carbohydrate diet include a reduction in the circulating levels of insulin along with increased levels of glucagon. These changes indeed favor gluconeogenesis. However, the body limits glucose utilization to reduce the need for gluconeogenesis. When the rate of mobilization of fatty acids from fat tissue is accelerated, as, for example, during low carbohydrate intake, the liver produces ketone bodies: acetoacetate and 3-hydroxybutyrate. However, the liver cannot utilize ketone bodies and thus, they flow from the

liver to extra-hepatic tissues (e.g., brain, muscle) for use as a fuel. This spares glucose metabolism via a mechanism similar to the sparing of glucose by burning of fatty acids as an alternative fuel. Indeed, the use of ketone bodies replaces most of the glucose required by the brain. In comparison with glucose, the ketone bodies are, in fact, very good fuel. Importantly, catabolism (breakdown) of lean body mass is reduced by ketones, which probably explains the preservation of lean tissue observed during very low carbohydrate diets **(Feinman *et al*., 2013).**

Ketosis

Insulin activates key enzymes in pathways, which store energy derived from carbohydrates, and when there is an absence or scarcity of dietary carbohydrates the resulting reduced insulin level leads to a reduction in lipogenesis and fat accumulation. After a few days of fasting, or of drastically reduced carbohydrate consumption (below 50g/day), glucose reserves become insufficient both for normal fat oxidation via the supply of oxaloacetate in the Krebs cycle (which gave origin to the phrase 'fat burns in the flame of carbohydrate') and for the supply of glucose to the central nervous system (CNS) **(A Paoli *et al*., 2013).**

Low-Carbohydrate Diets and Metabolic Advantage

Low-carb/high-protein diets indeed provide "metabolic advantage," a greater weight loss/fat loss per calorie consumed compared to isocaloric (having similar caloric values) high-carb diets. The idea that metabolic advantage might violate laws of thermodynamics ("Manninen dude, don't confuse me with the facts, a calorie is always a calorie") has some immediate appeal, but is not theoretically correct. The first law of thermodynamics can be written as follows: Change in energy stores = energy intake – energy expenditure although this principle always applies, the application to living organisms is certainly not simple. Indeed, the abovementioned equation only applies to closed systems. However, if matter can be exchanged between system and surroundings, the system is open. Thus, all living organisms that have ever existed are open systems. The system takes in food from the environment and uses it to maintain body temperature and power all the biochemical pathways of its body.

The second law of thermodynamics tells us that whenever energy is exchanged, the efficiency will be imperfect and some energy will escape— usually in the form of heat. Importantly, the metabolic pathways that macronutrients (i.e., carbs, fats, proteins) follow may be very different due to the differences in hormonal state and enzymatic activity. As noted above, the hormonal changes associated with a low carbohydrate diet include a reduction in the circulating levels of insulin along with increased levels of glucagon. These changes favor gluconeogenesis, which is, of course, an energy-consuming process. In addition, a low-carb diet increases turnover of body proteins; protein turnover spends significantly more energy than previously appreciated. Thus,

although supported by the prominent obesity authorities, the old nutritional mantra "a calorie is a calorie" is misleading. Different diets (e.g., low carb/high-protein vs. high-carb/low-protein) lead to different biochemical pathways (due to the hormonal and enzymatic changes) that are not equivalent when correctly compared through the laws of thermodynamics. The recent review by Drs. Richard Feiman and Eugene Fein published in the Nutrition Journal stated, "Metabolic advantage with low-carbohydrate diets is well established in the literature… The extent to which metabolic advantage will have significant impact in treating obesity is unknown and it is widely said in studies of low-carbohydrate diets that "more work needs to be done." However, if the misconception is perpetuated that there is a violation of physical laws, that work will not be done, and if done, will go unpublished due to editorial resistance. Attacking the obesity epidemic will involve giving up many old ideas that have not been productive. "A calorie is a calorie" might be a good place to start."

The basis of low-carbohydrate diets

The diet plans discussed here have one thing in common; they limit carbohydrate intake partly to control the release of insulin by the pancreas. Insulin is released in proportion to the rate at which glucose enters the bloodstream. Therefore, carbohydrates, especially the simple carbohydrates, raise insulin levels to a greater extent than either proteins or fats do. Insulin promotes glucose uptake by cells, a process necessary for survival. However, it also initiates signal transduction cascades that result in inhibition of lipolysis (fat breakdown), inhibition of fatty acid oxidation, and inhibition of glycogen breakdown by the muscle and liver.

Insulin also promotes synthesis of cholesterol, and, therefore, elevated insulin levels are also associated with heart disease. While insulin is secreted in response to elevated blood sugar, another hormone, glucagon, is secreted in response to low blood sugar. Glucagon acts on some of the same signal cascades as insulin, but with the opposite effect. It results in activation of enzymes that break down fuel while inhibiting the enzymes involved in energy storage **(Nicholas Arpaia, and Salvatore Priore 2005).**

Conclusion

Carbohydrate diets have become very popular and are currently used to achieve weight loss, to improve metabolic parameters, and to treat and prevent various pathologies. These regimens, which are not individualized plans, are found on the Internet and in books and common media and are often used without medical advice or follow-up. Of course, dietitians could choose these types of diets for certain patients in order to ameliorate a disease or to investigate its effects.

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