What's Wrong With Carbohydrates?

BARBARA BIRCHWOOD, MD

SUMMARY

A review of carbohydrates in metabolism is presented. The recent rash of books on diet and weight reduction implicate carbohydrates as the root cause of obesity and abnormal metabolism, but they actually play a positive role in normal metabolism. They are involved in energy metabolism, water balance and a host of other functions in the body. Used with intelligence and not with indulgence, they will continue to be good sources of calories, contributing to total nutritional health and well-being.

Dr. Birchwood is on the teaching staff of the University of Toronto and practices internal medicine, specializing in nutrition and metabolism.

CARBOHYDRATES have recently become the target of severe and adverse criticism both by food faddists, many of whose major qualification is that they eat, and by health professionals who should know better but don't. Carbohydrate is a constituent of the normal diet and there is nothing inherently wrong with it. In many parts of the world it provides the major source of calories and together with an adequate amount of protein and fat it can maintain good health.

Role of Carbohydrates in Nutrition

Carbohydrates may be divided into simple (mono and disaccharides) and complex (polysaccharides). Simple carbohydrates are derived from such sources as fruit, honey, corn syrup, table sugar and milk, whereas complex carbohydrates are derived from grains and vegetables. Carbohydrate is an active body fuel which is stored only in very small quantities in the form of glycogen in the liver, smooth and cardiac muscles. Excess calories are deposited in adipocytes as reserve material.

Certain tissues, namely brain, constantly require carbohydrates under all physiological conditions.[1] In the event that this fuel source is curtailed, even for a short period, severe disability may result. Given time however, the body is able to convert protein and fat into glucose, and the brain is able to adapt its metabolism to utilize ketone bodies as a supplement to the small amount of glucose that the liver is able to supply.[2]

In a review of the fuel for muscular exercise, Gemmill[3] found a slight increase in efficiency on a carbohydrate diet and prolongation of muscular effort when carbohydrate was ingested.

In the liver, carbohydrate is not only stored as fuel in the form of glycogen, but it protects the body against toxic agents such as alcohol, carbon tetrachloride and bacterial toxins. It also provides acetyl groups for detoxification of such substances as sulfanilamide, and its presence in adequate amounts protects protein from being broken down to be used as fuel. In the same way its presence inhibits lipolysis, thus maintaining fuel reserve. It is therefore obvious that the fat cell is intimately involved in carbohydrate homeostasis. In the normal post-absorptive state and during fasting when the body receives no carbohydrate, lipolysis is activated.[4] Similarly, in diabetes where there is inadequate insulin supply or where insulin is ineffective, fat cells are broken down in an attempt to supply needed calories. The importance of insulin in the control of all aspects of energy metabolism cannot be overemphasized.[5] Carbohydrate, more than any other metabolic fuel, is able to influence insulin levels.[6] As such it determines the degree of preservation and utilization of other fuel material.

Insulin is secreted in response to a carbohydrate meal. It enhances the uptake of amino acids by peripheral tissues[5, 7, 8] and promotes the conversion of amino acids into protein while at the same time reducing protein catabolism.[9] This does not occur in isolation; because of the system's degree of integration its presence also promotes lipogenesis and inhibits lipolysis.[5, 10] Carbohydrates constitute about 50 percent of the total calories ingested daily in North America. During periods of deprivation when insulin levels are low, consumption of metabolic fuel shifts, to preserve lean body mass and promote utilization of fat.[6] Carbohydrate is the main fuel for endurance work. It can be the limiting factor in such work and can be stored in greater amounts if the preceding diet is high in carbohydrate.[11, 12]

Problems of Carbohydrate Metabolism

Obesity

Fifty percent or more of Canadians are obese. In view of the adverse effects obesity has on health – diabetes, coronary heart disease, arthritis, accidents etc. – this is a problem of some magnitude. Yudkin,[13] in an evolutionary

appraisal of food intake patterns, has found that since the turn of the century there has been a steady increase in the consumption of refined carbohydrate which now constitutes about 33 percent of total carbohydrate consumed. Easy availability of these highly refined carbohydrate foods makes them very attractive for snacks. These snack foods contain very concentrated calories; in a mechanized society where energy expenditure is minimal and where the ingestion of cakes, pies, potato chips does not preclude the eating of regular meals, the excess calories are stored as fat.

The observation of increasing obesity in society has created a rash of books on the subject of diet and weight loss. Two recent ones written by physicians, Stillman,[14] and Atkin[15] implicate carbohydrates as the sole source of obesity. In Atkin's book, carbohydrate is referred to as a "poison" which promotes hypoglycemia, diabetes, atherosclerosis and obesity. Both these diets were critically studied and reviewed.[16, 17] There is absolutely no evidence that carbohydrate is a "poison". Indeed, if this were so there would be no third world, since more than half of the world's population subsists on high carbohydrate foods. In the same context, in countries where carbohydrate intake constitutes most of the ingested calories, obesity and heart disease are uncommon.

Yudkin and Carey,[18] in a carefully controlled study of six obese adults, provide the explanation for weight loss on a ketogenic diet. When carbohydrate in the diet was voluntarily reduced to about 50 g/day, the deficit was not made up by increased intake of protein and fat but there was a reduction of total caloric intake ranging from 13 percent to 55 percent, with ensuing weight loss. A repeat study with 11 subjects[19] confirmed the previous finding that reduction in carbohydrate intake was not accompanied by increase in protein and fat but resulted in a decrease in total caloric intake.

Diabetes

This is a condition characterized by the production of inadequate amounts of insulin for need. The effect of high carbohydrate vs. low carbohydrate diet in the management of diabetes continues to engender a great deal of controversy. Ford[20] et al demonstrated that diets high in carbohydrate improved oral glucose tolerance in normal persons. In the mild to moderate diabetic Brunzell et al[21, 22] and Stone et al[23] showed improved glucose tolerance when a high carbohydrate diet (85 percent of calories) was fed for shorter or longer periods of time. Swan et al[24] have shown that ingested glucose has a more deleterious effect on carbohydrate tolerance than ingested starch.

In a crossover study of 18 non-insulin-dependent, normal weight diabetic patients, using diets containing 60 percent carbohydrate, 25 percent fat, and 40 percent carbohydrate with 45 percent fat for a duration of 40 weeks, Weinsier et al [25] found no compromise of diabetic control on high carbohydrate intake, and no alteration in fasting plasma insulin. They did however find a significant increase in glycosuria in two patients with severe diabetes who had persistent hyperglycemia of more than 200 mg/100 ml.

It therefore appears that both in the insulin and non-insulin dependent diabetic, restriction of total carbohydrate intake is less important than the type of carbohydrate present in the diet. Simple sugars adversely affect carbohydrate tolerance and should be limited in the diabetic diet. In the severe diabetic, restriction of total carbohydrate in the diet is still the treatment of choice.

Reactive Hypoglycemia

In the last decade, reactive hypoglycemia has been awarded a diagnostic prominence it does not deserve. It has been implicated as the cause of a series of disorders from schizophrenia to impotence. It has received such publicity that any individual who feels tired or somewhat depressed feels he/she has hypoglycemia and demands a five hour glucose tolerance test. In many instances a diagnosis of hypoglycemia is made without convincing clinical evidence or laboratory support. There is no documented scientific evidence that the majority of tired people have hypoglycemia. The diagnosis is based on low blood sugars, either random or during a glucose tolerance test, of 40-45 mg/100 ml, with symptoms of neuroglycopenia at that time. It is important that symptoms be confirmed at the time of low blood sugar values, since many people are asymptomatic with blood sugar levels below defined normal limits.

Reactive hypoglycemia may be the forerunner of true diabetes mellitus and in many cases a family history of diabetes may be elicited.[26] Symptoms usually appear two to four hours after a high carbohydrate meal. Treatment is directed at reducing the proportion of carbohydrate in the meal, particularly simple sugars which are absorbed quickly, and substituting protein and fat in several small feedings daily.

Another condition which improves with the restriction of simple carbohydrate and multiple small feedings is tachyalimentation in post-gastrectomy patients. Symptoms of hypoglycemia occur one to two hours after a carbohydrate meal due to rapid passage of glucose into the small intestines, increased rate of entry of sugar into the blood stream and excessive release of insulin.

Carbohydrate and Water Balance

In 1952, Hervey and McCance[27] demonstrated the role of carbohydrate in conserving water and salt in men without food or sufficient water. Bloom[28] noted that fasting obese patients promptly ceased to excrete sodium in their urine even though they remained in significant negative calorie balance. Individual tests with salt, protein, fat or carbohydrate demonstrated that only carbohydrate produced sodium retention with accompanying water retention. These results have been confirmed by other investigators and there is no evidence for the erroneous belief that the salt loss of carbohydrate deficiency is the result of ketosis, since the maximum salt loss occurs during the first four days of fasting when ketone excretion is at its lowest. The initial sodium loss during carbohydrate restriction in a diet is only temporary; within a few days a new homeostatic fluid and electrolyte level is achieved and sodium and water excretion diminishes. This new adjustment results from a decrease in extra cellular volume and heart size.[29] Often this results in postural hypotension.

Bortz[30] isocalorically substituted fat for carbohydrate in the diet of ten obese patients for 24 day periods with a period of equilibration between. Apart from early rapid weight loss of the first hypocaloric period, there was no significant difference in the mean daily weight loss of both periods. These studies support the known facts of the equivalency of foodstuffs and energy balance. They show very convincingly that carbohydrate restriction has no metabolic superiority over total caloric restriction in accomplishing weight loss.

The mechanism of the shift in sodium and water balance

has been hypothesized but not confirmed. It may be that introduction of carbohydrate into the diet after a period of deprivation stimulates increase in aldosterone secretion with sodium and water retention, or it may be that carbohydrate per se reduces the urinary solute load. It should be noted however that even on diets containing carbohydrate, the predominant metabolite is endogenous fat when the total calories are restricted.

Carbohydrate and Hyperlipidemia

Carbohydrate and lipid metabolism are closely interwoven. Where fat is excluded from the diet and replaced by carbohydrate, endogenous synthesis of fatty acid increases in both adipose tissue and liver. On a diet containing 40 percent of calories as fat, only a very small amount of dietary carbohydrate is converted to fatty acid. When a fast is broken by a high carbohydrate diet, the rate of fatty acid synthesis from two carbon units and hydrogen derived from glucose increases sharply.

During fasting, exercise, stress or uncontrolled diabetes, triglyceride stored in adipose tissue is broken down with release of glycerol and fatty acid. The glycerol is mainly metabolized in the liver where it is phosphorylated and either reutilized for triglyceride synthesis or used for gluconeogenesis.

The importance of carbohydrate in lipid metabolism derives its importance from the association of hyperlipoproteinemia and coronary heart disease.

Atherosclerosis is a metabolic disease characterized by alterations in plasma and tissue lipids. Epidemiological evidence suggests that elevated lipid levels associated with atherosclerosis are related to prolonged consumption of excessive calories, saturated fats, cholesterol and refined carbohydrates.[31]

Studies on the role of complex and simple carbohydrates in the genesis of hyperlipidemia have confirmed previous suspicions that excessive simple sugars are hypertriglyceridemic and particularly so in susceptible individuals. This is significant, since Albrink et al[32] suggest that triglycerides may be more important than cholesterol in the development of atherosclerosis and heart disease, and this has been reiterated by others.

MacDonald,[33] having determined that sucrose rather than starch increases triglyceride levels in normal man, hypothesized that it was the fructose moiety in sugar, rather than glucose, which was hypertriglyceridemic. He later confirmed this in monkeys using labelled glucose and fructose.[34] Work undertaken by Winitz et all[35] corroborated MacDonald's finding that fructose is the hyperlipidemic component. In Winitz's studies, cholesterol decreased when the carbohydrate in the diet was glucose and increased when sucrose was substituted for part of the carbohydrate.

In a series of pilot studies, Hodges and Krehl[31] provide unequivocal evidence that a high intake of simple sugars is accompanied by an increase in the concentration of lipids, whereas an equivalent intake of complex carbohydrate is accompanied by a decrease in the concentration of lipids.

Birchwood et al[36] in a study of the interrelationship between carbohydrate and fat in hyperlipoproteinemic patients found no difference in the effect of starch and sucrose on serum lipids when the diet was high in polyunsaturated fat.

The effect of complex carbohydrates on plasma lipids may be influenced by other components within the complex molecule, namely pectins, sitosterol and other plant sterols which increase fecal excretion of bile acids. In addition the type and amount of dietary fat included in the diet will influence the lipid level. It appears that substitution of polyunsaturated fat in the diet attenuates the hyperlipidemic effect of simple sugar. The different effect of complex and simple sugar on lipids may be explained in part by the differences in absorption rates; simple sugars are rapidly absorbed, stimulate insulin production and triglyceride synthesis, whereas the absorption rate of complex carbohydrate allows for a more orderly progression of lipid response.

Considering the rather high incidence of coronary heart disease in North America and the association of elevated lipids with atherosclerosis, these differences in effect of sucrose and starch on plasma lipids become relevant. The findings of Yudkin et al[37] that serum levels and platelet adhesiveness correlated with sucrose intake in patients with peripheral vascular disease, and of Antar[38] who found acceleration of blood coagulability in vitro when sucrose was isocalorically substituted for starch in four males and four females, leave little doubt that the type and amount of carbohydrate in the diet influences the vascular pathology.

Carbohydrate and Dental Caries

During recent years the etiology of dental caries has been more clearly established. Of all the components in the diet, carbohydrate provides the necessary substrate for bacterial activity in the oral cavity. The form of carbohydrate ingested determines the extent of caries. Taken as a liquid even at high levels, there seems to be little effect on teeth. However when carbohydrate is ingested in the form of candy, chocolates, etc., marked increase in dental caries occurs. This may be reduced if the same simple sugar is taken together with a meal rather than taken between meals. Complex carbohydrate such as rice and cooked cereal have also been shown to be cariogenic. The effect of carbohydrate, both simple and complex, can be attenuated by good dental hygiene.

Congenital Abnormalities of Carbohydrate Metabolism

Hereditary fructose intolerance, lactose intolerance and glycogen storage disease are all disorders due to abnormalities in carbohydrate metabolism, characterized by symptomatic hypoglycemia, weakness, and fatigue. They have in common a deficiency of the appropriate enzyme for metabolizing carbohydrate. These disorders are rare; in fructose and lactose intolerance, identification of the offending carbohydrate and its withdrawal from the diet causes immediate and remarkable improvement in health. If the diagnosis is not made early, severe disability may result. In galactosemia, irreversible mental retardation occurs early in life. ◀

References

1. SOSKIN, S.: The Blood Sugar: its origin, regulation and utilization. Physiol. Revs. 21:140, 1941.

2. OWEN, O. E., MORGAN, A. P., KEMP, H. G., SULLIVAN, J. M., HERRERA, M. G., and CAHILL, G. F. Jr.: Brain Metabolism During Fasting. J. Clin. Invest. 46:1589, 1967.

3. GEMMILL, C. L.: The Fuel For Muscular Exercise. Physiol. Revs. 22:32, 1942.

4. LEVINE, R., and HAFT, D. E.: Carbohydrate Homeostasis. New. Engl. J. Med. 283:175, 1970.

5. CAHILL, G. F. Jr.: Physiology of Insulin in Man. Diabetes, 20:785, 1971.

6. FLATT, J. P. and BLACKBURN, G. L.: The Metabolic Fuel Regulatory System: implications for protein-sparing therapies during caloric deprivation and disease. Amer. J. Clin. Nutr. 27:175, 1974.
7. LOTSPEICH, W. D.: The Role of Insulin in the Metabolism of Amino Acids. J. Biol. Chem. 179:175, 1949.
8. MANCHESTER, K. L.: The Control by Insulin of Amino Acid Accumulation in Muscle. Biochem. J. 117:457, 1970.
9. WOOL, I. G., STIREWALT, W. S., KURIHARA, K., LOW, R. B., BAILEY, P., and DYER, D.: Mode of Action of Insulin in the Regulation of Protein Biosynthesis in Muscle. Recent Progr. Hormone Res. 24:139, 1968.
10. JUNGAS, R. L., and BALL, E. G.: Studies on the Metabolism of Adipose Tissue XII. The effect of insulin and epinephrine on free fatty acid and glycerol production in the presence and absence of glucose. Biochem. 2:383, 1963.
11. HERMANSEN, L., HULTMAN, E., and SALTIN, B.: Muscle Glycogen During Prolonged Severe Exercise. Acta Physiol. Scand. 71:129, 1967.
12. LEWIS, S., and GUTIN, B.: Nutrition and Endurance. Amer. J. Clin. Nutr. 26:1011, 1973.
13. YUDKIN, J.: Patterns and Trends in Carbohydrate Consumption and Their Relation to Disease. Proc. Nutr. Soc. 23:149, 1964.
14. STILLMAN, I., and BAKER, S.: Doctor's Quick Weight Loss Diet. Prentice – Hall Inc., Englewood Cliffs, N.J. 1967.
15. ATKINS, R. C.: Dr. Atkin's Diet Revolution: the high calorie way to stay thin forever. David McKay Co., Inc. New York, N.Y. 1972.
16. RICKMAN, F., MITCHELL, N., DINGMAN, J., and DALEN, J. E.: Changes in Serum Cholesterol During the Stillman Diet. J. Amer. med. Assn. 228:54, 1974.
17. A Critique of Low Carbohydrate Ketogenic Weight Reduction Regimens. A review of Dr. Atkin's diet revolution. J. Amer. med. Assn. 224:1415, 1973.
18. YUDKIN, J., and CAREY, M.: The Treatment of Obesity by the "High Fat" Diet. The inevitability of calories. Lancet. 2:939, 1970.
19. STOCK, A. L., and YUDKIN, J.: Nutrient Intake of Subjects On Low Carbohydrate Diet Used in Treatment of Obesity. Amer. J. Clin. Nutr. 23:948, 1970.
20. FORD, S. Jr., BOZIAN, R. C., and KNOWLES, H. C. Jr.: Interactions of Obesity and Glucose and Insulin Levels in Hypertriglyceridemia. Amer. J. Clin. Nutr. 21:904, 1968.
21. BRUNZELL, J. D., LERNER, R. L., HAZZARD, W. R., PORTE, D. Jr., and BIERMAN, E. L.: Improved Glucose Tolerance with High Carbohydrate Feeding in Mild Diabetes. New. Engl. J. Med. 284: 521, 1971.
22. BRUNZELL, J. D., LERNER, R. L., PORTE, D. Jr., and BIERMAN, E. L.: Fallacy of Dietary Carbohydrate Restriction in Diabetes Mellitus. Diabetes. 19:379, 1970.
23. STONE, D. B., and CONNOR, W. E.: The Prolonged Effects of a Low Cholesterol, High Carbohydrate Diet Upon the Serum Lipids in Diabetic Patients. Diabetes. 12:127, 1963.
24. SWAN, D. C., DAVIDSON, P., and ALBRINK, M. J.: Effect of Simple and Complex Carbohydrates on Plasma Nonesterified Fatty Acids, Plasma Sugar and Plasma-Insulin During Oral Carbohydrate Tolerance Tests. Lancet. 1:60, 1966.
25. WEINSIER, R. L., SEEMAN, A., HERRERRA, M. G., ASSAL, J. P., SOELDNER, J. S., and GLEASON, R. E.: High and Low Carbohydrate Diets in Diabetes Mellitus. Study of effects on diabetic control, insulin secretion, and blood lipids. Ann. Intern. Med. 80:332, 1974.
26. SELTZER, H. S., FAJANS, S. S., and CONN, J. W.: Spontaneous Hypoglycemia as an Early Manifestation of Diabetes Mellitus. Diabetes. 5:437, 1956.
27. HERVEY, G. R., and McCANCE, R. A.: The Effects of Carbohydrate and Sea Water on the Metabolism of Men Without Food or Sufficient Water. Roy. Soc. London Proc. Series B, 139:527, 1952.
28. BLOOM, W. L.: Inhibition of Salt Excretion by Carbohydrate. Arch. Intern. Med. 109:26, 1962.
29. BLOOM, W. J., AZAR, G. and SMITH, E. G., Jr.: Changes in Heart Size and Plasma Volume During Fasting. Metab. 15:409, 1966.
30. BORTZ, W. M., WROLDSON, A., MORRIS, P., and ISSEKUTZ, B. Jr.: Fat, Carbohydrate, Salt and Weight Loss. Amer. J. Clin. Nutr. 20:1104, 1967.
31. HODGES, R. E., and KREHL, W. A.: The Role of Carbohydrates in Lipid Metabolism. Amer. J. Clin. Nutr. 17:334, 1965.
32. ALBRINK, M. J., MEIGS, J. W., and MANN, E. B.: Serum Lipids, Hypertension and Coronary Artery Disease. Amer. J. Med. 31:4, 1961.
33. MacDONALD, I., and BRAITHWAITE, D. M.: The Influence of Dietary Carbohydrates on the Lipid Pattern in Serum and in Adipose Tissue. Clin. Sci. 27:23, 1964.
34. MacDONALD, I., and ROBERTS, J. B.: The Serum Lipid Response of Baboons to Various Carbohydrate Meals. Metab. 16:572, 1967.
35. WINTIZ, M., GRAFF, J., and SEEDMAN, D. A.: Effect of Dietary Carbohydrate on Serum Cholesterol Levels. Arch. Biochem. 108:576, 1964.
36. BIRCHWOOD, B. L., LITTLE, J. A., ANTAR, M. A., LUCAS, C., BUCKLEY, G. C., CSIMA, A., and KALLOS, A.: Interrelationship Between the Kinds of Dietary Carbohydrate and Fat in Hyperlipoproteinemic Patients. Atherosclerosis. 11:183, 1970.
37. YUDKIN, J., SZANTO, S., and KAKAAR, V. V.: Sugar Intake, Serum Insulin and Platelet Adhesiveness in Men With and Without Peripheral Vascular Disease. Postgrad. Med. J. 45:608, 1969.
38. ANTAR, M. A.: Studies of Thrombus Formation and Fatty Acid Composition of Serum Lecithins and Cephalins in Man During High Sugar and Starch Diets. J. Atheroscler. Res. 8:569, 1968.

College of Family Physicians of Canada

Development Program

Percentages achieved to date of quotas for special gifts from provincial chapters