## Low-Carbohydrate Diets and Realities of Weight Loss

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Obesity is a worldwide epidemic and will be followed by a worldwide epidemic of diabetes.<sup>1</sup> While diet, lifestyle, and exercise are the cornerstones of current approaches to treat obesity, they have been ineffective in stemming the current epidemic. In this issue of THE JOURNAL, the article by Bravata et al<sup>2</sup> systematically reviews and synthesizes the literature on the use of low-carbohydrate diets for treatment of obesity. Their findings add to the review of popular diets published by Freedman et al.<sup>3</sup> Among the principal findings in the analysis by Bravata et al are that lower-carbohydrate ( 60 g/d of carbohydrate) diets were associated with reduced calorie intake and that weight loss was predicted by calorie intake, diet duration, and baseline body weight, but not by carbohydrate content. At the end of their analysis, Bravata et al note several gaps in the current literature on low-carbohydrate diets, including the need for better follow-up and for use of intent-to-treat analyses.

These findings are helpful in understanding whether low-carbodydrate diets work for obese individuals, but first several important lessons about obesity must be considered.<sup>4</sup> First, obesity is a chronic, relapsing, neurochemical disease that occurs in genetically susceptible people. Second, obesity can be conceptualized as an epidemiological disease with food as the agent that acts on the host to produce disease. Third, current treatments do not cure obesity and thus are only palliative. Fourth, 2 kinds of treatment are available for obesity: cognitive and noncognitive. Cognitive treatments, such as lifestyle change, diet, and exercise, produce weight loss when they are being used but when they are stopped, relapse occurs. Noncognitive treatments include drugs, surgery, and some environmental manipulations, and they may produce long-term weight loss. Fifth, as with most treatments for weight loss, a plateau is reached when the body's neurochemical counter-regulatory systems counterbalance the weight loss. Sixth, obesity is a stigmatized disorder, especially among women, which may explain why women predominate in seeking treatment. Seventh, treatments must be very safe because many individuals want to lose weight, even though they may be within the normal weight range. And eighth, even modest weight loss in high-risk individuals, such as those with cardiovascular disease or type 2 diabetes mellitus, is beneficial.

The analysis by Bravata et al of existing data on low-carbohydrate diets, a similar diet that was originally introduced by Banting in 1863, <sup>5</sup> is enlightening, particularly when the authors compare the homogeneous groups of studies. The study findings illustrate that calories do count and that low-carbohydrate diets produce weight loss by reducing calorie intake, thus reaffirming that the first law of thermodynamics articulated by von Helmholtz<sup>6</sup> still applies to humans.<sup>2</sup> These findings also reaffirm the point made years ago by Yudkin and Carey<sup>8</sup> that without carbohydrate-containing foods in the diet (eg, breads), less fat is ingested because few people eat much fat by itself. Thus, as noted by Bravata et al<sup>2</sup> and Freedman et al, <sup>3</sup> low-carbohydrate diets reduce calorie intake. The analysis by Bravata et al also reaffirms that individuals who are significantly overweight lost more weight, and

that low-carbohydrate diets do not have deleterious effects on either serum lipid or fasting serum glucose levels.

Given the lack of evidence supporting the use of low-carbohydrate diets, why have these diets been such a persistent theme for authors of diet books and such "cashcows" for publishers for the past 140 years? One reason is that some of these diets produce quick weight loss, something prized by dieters and diet promoters alike. Removing carbohydrates from the diet and thus lowering caloric intake requires the body to mobilize endogenous glycogen stores from liver and muscle to provide glucose while gluconeogenesis is being activated. Since glycogen stores can account for 5% of liver weight and 1% of muscle weight,<sup>9</sup> their weight loss produces solutefree intracellular fluid that needs to be excreted—thus, the rapid weight loss from low-carbohydrate diets is largely by diuresis. After 7 to 14 days, diuresis ends and the phase of rapid weight loss slows.<sup>10</sup> The ketosis produced by low-carbohydrate diets that contain less than 50 g/d of carbohydrate is an index of fatty acid utilization and can be used to monitor adherence to the diet. One potential concern about ketosis is its effect on bone mineralization. Excretion of the ketotic anions requires cations that must come from either food or body stores, of which the largest stores are found in the bones. Prolonged ketosis may run the risk of leaching cationic minerals from bone. However, a potential advantage of very lowcarbohydrate diets is that removing sweet-tasting solutions from the diet can reduce the gustatory stimulation sweets produce, which easily leads to overconsumption.<sup>11</sup>

Bravata et al<sup>2</sup> also are concerned about the role of low-carbohydrate diets in the maintenance of weight loss, although this concern may be somewhat misguided. Diets do not cure obesity. If they did, Banting's diet would have eliminated overweight and obesity and made the need for new diet revolutions unnecessary. When weight loss reaches a plateau, as it must when neurochemical compensatory mechanisms come into play, many patients become frustrated with the results of their treatment, particularly if they have not lost much weight. At this point, many patients conclude that their diet is not working because they have not reached their goal weight. This frustration may well lead to discontinuing treatment and relapse may follow. Thus, maintaining body weight at a lower plateau over time means that the treatment is working.

Another approach to treatment includes the use of interventions that do not require conscious activity, but rather adjust body fat automatically, and do not demand cognitive approaches. These noncognitive strategies, those that act on an environment or on the host, such as drug therapy, are 2 approaches to this disease. For instance, the increase in consumption of high-fructose corn sweeteners in soft drinks is an environmental event that may enhance caloric intake leading to obesity.<sup>12-13</sup> High-fructose corn sweeteners were introduced into the food industry at exactly the same time as the prevalence of obesity began to rapidly rise,  $\frac{11}{11}$  about 1970. The increasing soft drink consumption parallels the decreasing milk consumption and the decreasing calcium intake, which has had an inverse relationship to body mass index.<sup>14</sup> Many low-carbohydrate diets have the advantage of reducing the ingestion of high-fructose corn sweeteners, but they do not deal with the preference many human beings have for sweet tastes. The aspect of carbohydrates as a preference and dieting is one that still needs to be addressed. Moreover, drugs affecting neurochemicals in the brain and intestinal lipase are the major therapeutic agents for treating obesity, although new agents are on the way.

The broader issue of whether a unique diet exists that will produce long-term weight loss has yet to be evaluated. Although the truth of "a calorie is a calorie" has been reaffirmed by the systematic review by Bravata et al, the question of whether patients can adhere more easily to one type of diet or another remains to be answered. One study<sup>15</sup> suggesting that diet composition can make a difference came from a 9-month randomized study in which 25% of dietary fat was replaced with the fat substitute olestra to provide diets with 25% available fat. A standard diet with 25% fat initially produced weight loss, but this loss was not maintained. By replacing dietary fat with olestra, weight loss continued over the entire 9 months of the trial. Thus, the possibility remains that some dietary components may provide long-term effects.

The conclusion from the analysis of the effects of low-carbohydrate diets on obesity by Bravata et al<sup>2</sup> is similar to the conclusion from another recent meta-analysis of the effects of low-fat diets on the treatment of obesity. "Further high quality research is needed to identify which type of weight loss diet is most efficacious in the long-term. Future trials would incorporate appropriate methods of randomisation and blinding of outcome assessors. Whilst drop-outs and withdrawals can not always be controlled, every effort should be made to ascertain the reasons for withdrawals so that factors affecting dietary adherence can be further elucidated."<sup>16</sup> Given the increasing prevalence of obesity, studies such as these deserve highest priority.

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## REFERENCES

<u>1.</u> Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US Adults, 1999-2000. *JAMA.* 2002;288:1723-1727. <u>ABSTRACT/FULL TEXT</u>

<u>2.</u> Bravata DM, Sanders L, Huang J, et al. Efficacy and safety low-carbohdyrate diets: a systemataic review. *JAMA.* 2003;289:1837-1846. <u>ABSTRACT/FULL TEXT</u>

<u>3.</u> Freedman MR, King J, Kennedy E. Popular diets: a scientific review. *Obes Res.* 2001;9(suppl 1):1S-40S. <u>ISI | MEDLINE</u>

<u>4.</u> Bray GA. Barriers to the treatment of obesity. *Ann Intern Med.* 1993;(7 pt 2):707-713.

<u>5.</u> Banting W. *Letter on Corpulence, Addressed to the Public*. 3rd ed. London, England: Harrison; 1864.

<u>6.</u> von Helmholtz H. *Uber die Erhaltung der Kraft*. Berlin, Germany: G Reimer; 1847.

7. Atwater WO, Benedict FG. *Experiments on the Metabolism of Matter and Energy in the Human Body, 1899-1900.* Washington DC: Government Printing Office; 1902. US Dept of Agriculture, Office of Experiment Stations Bulletin No. 109.

<u>8.</u> Yudkin J, Carey M. The treatment of obesity by the "high-fat" diet: the inevitability of calories. *Lancet.* 1960;7157:939-941.

<u>9.</u> Astrand P-O, Rodahl K. *Textbook of Work Philosophy*. New York, NY: McGraw Hill; 1970:467.

<u>10.</u> Bray GA. *The Obese Patient: Major Problems in Internal Medicine*. Vol 9. Philadelphia, Pa: WB Saunders Co; 1976:1-450.

<u>11.</u> Bray GA, Bray CA. *An Atlas of Obesity and Weight Control*. Boca Raton, Fla: Parthenon Publishing; 2003.

<u>12.</u> Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet.* 2001;357:505-508. <u>CrossRef</u> | <u>ISI</u> | <u>MEDLINE</u>

<u>13.</u> Elliott SS, Keim NL, Stern JS, Teff K, Havel PJ. Fructose, weight gain, and the insulin resistance syndrome. *Am J Clin Nutr.* 2002;76:911-922. <u>ABSTRACT/FULL TEXT</u>

<u>14.</u> Davies KM, Heaney RP, Recker RR, et al. Calcium intake and body weight. *J Clin Endocrinol Metab.* 2000;85:4635-4638. <u>ABSTRACT/FULL TEXT</u>

<u>15.</u> Bray GA, Lovejoy JC, Most-Windhauser M, et al. A 9-month randomized clinical trial comparing fat-substituted and fat-reduced diets in healthy obese men: the Ole Study. *Am J Clin Nutr.* 2002;76:928-934. <u>ABSTRACT/FULL TEXT</u>

<u>16.</u> Pirozzo S, Summerbell C, Cameron D, Glasziou P. Advice on low-fat diets for obesity (Cochrane Review). In: The Cochrane Library issue 1. Oxford, England: Update Software; 2003.