Low Carbohydrate and Low-Fat Diets

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In the 1940s, the diet-heart hypothesis proposed that high dietary saturated fat and cholesterol intake promoted coronary heart disease in “at-risk“ individuals. This hypothesis prompted federal recommendations for a low-fat diet for “high risk” patients and as a preventive health measure for everyone except infants. The low carbohydrate diet, first used to treat type 1 diabetes, became a popular obesity therapy with the Atkins diet in the 1970s. Its predicted effectiveness was based largely on the hypothesis that insulin is the causa prima of weight gain and regain via hyperphagia and hypometabolism during and after weight reduction, and therefore reduced carbohydrate intake would promote and sustain weight loss. Based on literature reviews, there are insufficient randomized controlled inpatient studies examining the physiological significance of the mechanisms proposed to support one over the other. Outpatient studies can be confounded by poor diet compliance such that the quality and quantity of the energy intake cannot be ascertained. Many studies also fail to separate macronutrient quantity from quality. Overall, there is no conclusive evidence that the degree of weight loss or the duration of reduced weight maintenance are significantly affected by dietary macronutrient quantity beyond effects attributable to caloric intake.

obesity weight loss weight gain diet fat carbohydrate macronutrient

1. Introduction

According to the Center for Disease Control and Prevention, in 2015–2016, 39.8% of American adults were obese (BMI> 30 Kg/m^2) [1]. The negative multi-system (health, fat bias, economic) impact of obesity on individual and population health is well-documented [2]. The question remains, is there an optimal diet for weight management and metabolic health?

In 1977, the United States Senate Select Committee on Nutrition and Human Needs presented the Dietary Goals of the United States (the McGovern Report) to the 95th Congress [3]. This report emphasized the health benefits of lower fat higher carbohydrate diets. The USDA partnered with the Department of Health and Human Services to issue the Dietary Guidelines for Americans, which eventually became the UDSA Food Pyramid [4]. These guidelines seem ineffective since the prevalence of obesity and its co-morbidities have continued to increase [5]. Theoretical formulations implicating dysmetabolic consequences of high carbohydrate diets on insulin-adipocyte physiology have resulted in increasing interest in the actively debated hypothesis that obesity and its co-morbidities can be restrained by reducing dietary carbohydrates [6][7][8]. Currently, low carbohydrate and ketogenic diets, once reserved for those managing epilepsy [9], or as treatment for type 1 diabetes [10] prior to the discovery of insulin, are gaining popularity. However, the debate continues over whether a low carbohydrate or low-fat diet is best for

preventing weight gain, supporting weight loss, preserving weight maintenance and optimizing cardiovascular and metabolic health.

With the exception of several inpatient studies of the ingestion of diets of varying composition before, during or after weight loss [11], most examinations of the effects of diet composition on weight loss, gain and regain have been outpatient studies in which it is difficult to disassociate physiological effects of a diet from those related to the degree of dietary adherence [6][12][13][14].

2. Overview of Low Fat and Low Carbohydrate Diets

2.1. Physiological Basis for Low Fat Diet (Figure 1)

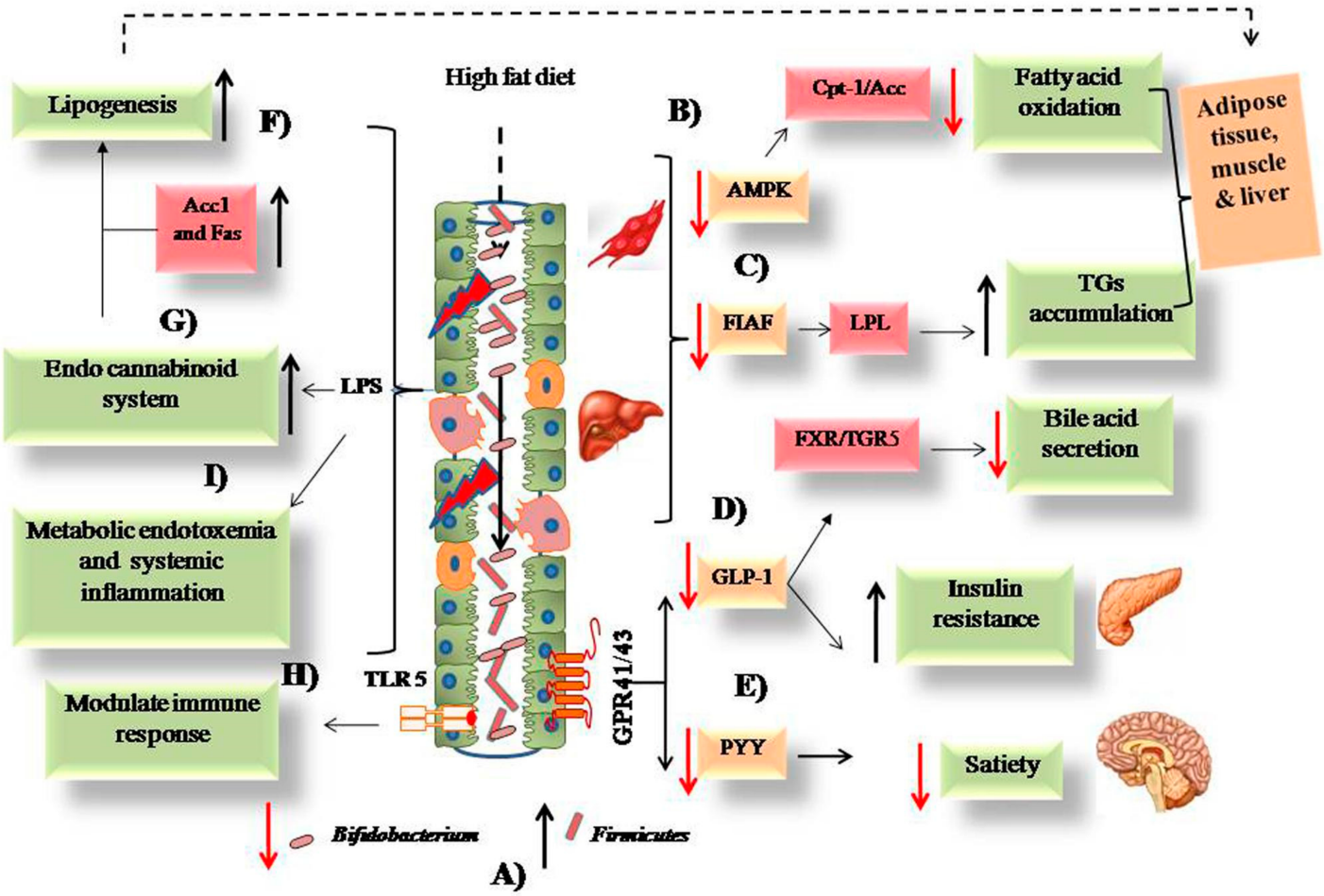

Figure 1. Possible mechanisms associated with the intake of high fat diet and obesity. (A) A high fat diet causes an alteration in intestinal microbiota from low to high Firmicutes and high to low Bifidobacterium. (B) The low expression of adenosine monophosphate kinase (AMPK) leads to decreased fatty acid oxidation. (C) Fasting induce adipose factor (FIAF) expression causes activation of lipoprotein lipase (LPL) that leads to triglyceride (TG) accumulation. (D) Low glucagon-like peptide 1 (GLP-1) leads to increased insulin resistance and decreased bile acid secretion from liver. (E) Decreased peptide YY (PYY) causes low satiety in obese host. (F) Increased lipogenesis via upregulated acetyl-CoA carboxylase (Acc1) and fatty acid synthase (Fas) enzymes. (G) The

activation of endo cannabinoid loop via release of lipopolysaccharide (LPS) due to damages intestinal epithelium. (H) The modulation of intestinal immune response via toll-like receptor 5 (TLR-5) downstream signaling. (I) The systemic inflammation caused by inflammatory cytokines and bacterial [15]. Cpt-1 - carnitine palmitoyltransferase, GPR – G-protein coupled receptors, FXR – Farnesoid X Receptor.

The recommendations for dietary fat restriction arose from the observation that diets high in saturated fat and cholesterol were associated with coronary heart disease [16][17]. Ancel Keys suggested a low-fat diet would help prevent cardiovascular disease [18]. The American Heart Association's (AHA) subsequent low-fat diet recommendations were also initially intended for those at risk for cardiovascular disease based on family history or their own morbidities [19]. Coupled with the knowledge that fats are more calorically dense than carbohydrates or protein, the AHA recommendations to replace animal fats with non-tropical vegetable oils, also acknowledged the importance of obesity as a risk factor for cardiovascular disease [20]. In 1977, the U.S. Senate's Select Committee on Nutrition and Human Needs, led by Senator George McGovern, gave a clear government sponsored endorsement for the diet-heart hypothesis [3]. When results from the Framingham Study confirmed an association of obesity and cardiovascular disease risk [21], the diet-heart hypothesis was deemed applicable to adults on the assumption that the lower caloric density and higher thermic effect of nutrients [22] of the low-fat diet would also prevent obesity [23]. Recently, some studies have suggested that high fat diets promote alterations in the gut microbiome (decreased Bifidobacteria and increased Firmicutes) that promote inflammation and decrease satiation [15] (**Figure 1**).

2.2. Physiological Basis for Low CHO Diet: Insulin-Carbohydrate Model (Figure 2)

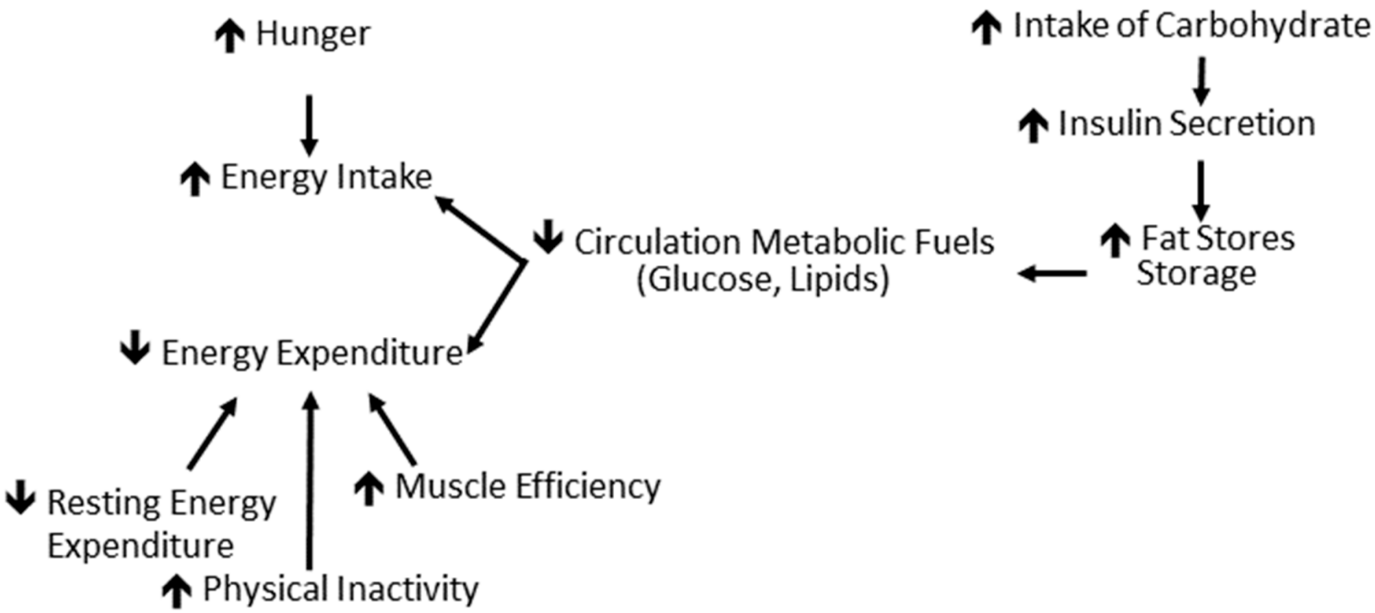

Figure 2. Schematic of the insulin-carbohydrate model. Increased carbohydrate intake promotes increased insulin secretion resulting in depletion of circulating concentrations of metabolic fuels that are used in lipogenesis. The decreased circulating glucose and lipids results in adaptive thermogenesis and hyperphagia which promote weight gain or regain (Based on Ludwig [6]).

The insulin-carbohydrate model is based on the known action of insulin to increase the cellular uptake of glucose and fatty acids, stimulate lipogenesis and inhibit lipolysis [6]. According to this hypothesis, a high carbohydrate diet stimulates insulin release and the resultant decrease in circulating glucose and free fatty acids is then sensed by the central nervous system and other cellular systems regulating energy homeostasis as a state of undernutrition. This invokes subsequent hypometabolism and hyperphagia as well as the preferential storage of ingested calories, such as fat. Clinically, the result is weight/fat gain and increased difficulty in weight management.

2.3. Overview of Energy Balance

In adults, there is remarkable consistency of body weight and composition over time due to a complex interplay of genetic, physiological and behavioral factors [24]. Body weight fluctuates around a set point in a given environment which is influenced by non-homeostatic mechanisms including the hedonic regulation of food intake, and homeostatic mechanisms that regulates the short and long-term energy balance driven by hunger, satiation and changes in adiposity [25]. As an example of the coordination of homeostatic systems regulating energy intake and output, it has been estimated that Americans consume 570 Kcal more per day than 35 years ago, but only 10–20 Kcal per day are stored as additional body weight [25][26][27]. The control of energy stores is achieved through the coordinated regulation of energy intake and expenditure mediated by signals emanating from adipose, gastrointestinal and other endocrine tissues. These signals are then integrated by the liver and by regulatory (hypothalamus, brainstem), hedonic–emotional (amygdala, ventral striatum, orbitofrontal cortex), and executive–restraint (cingulate, middle frontal, supramarginal, precentral, and fusiform gyri) elements of the central nervous system (CNS). Changes in these signals are involuntary and largely due to the reduction in circulating leptin as a result of the loss of fat mass and of the negative energy balance [27]. The consequence is that during and following weight loss, most individuals experience hypometabolism, hyperphagia, neuroendocrine changes (decreased circulation concentrations of bioactive thyroid hormones and leptin) and autonomic changes (decreased sympathetic and increased parasympathetic nervous system tone) that work in concert to favor the return to usual body weight [25][28].

These considerations identify the means by which the dietary macronutrient content could meaningfully affect energy balance. Specifically, macronutrient composition would have to either disproportionately decrease appetite and/or increase energy expenditure to promote weight loss and reverse some of the metabolic, behavioral, endocrine and/or autonomic changes that occurred because of weight loss if it is to prevent weight regain. Investigations in this area are further complicated by the possible confounding effects of whether the hypothesis being tested regarding dietary macronutrient balance is examining initial weight gain, weight loss, reduced weight maintenance, or weight regain and whether diets differ in macronutrient quality or quantity. The effects of energy stores versus energy balance on therapeutic results is exemplified by the adipocyte derived hormone leptin. The administration of leptin has little effect on individuals with and without obesity at their usual weight. Leptin repletion has a small effect on appetite but does not affect neuroendocrine function or energy expenditure during weight loss, but at least partially reverses most of the metabolic, behavior, autonomic and neuroendocrine changes that otherwise favor weight regain during reduced weight maintenance [29].

3. Current Insights on Low Carbohydrate and Low-Fat Diets

The major conclusion is that there is insufficient evidence to conclude that a low carbohydrate or low-fat diet is superior as a means to prevent obesity or to achieve or maintain weight loss. The lack of controlled prolonged inpatient studies raises uncertainties as to whether the mechanistic consequences predicted from these diets (e.g., increased energy expenditure and decreased appetite on a low carbohydrate diet) are of physiological significance. The difficulty in dietary supervision raises uncertainties regarding any study comparing diet efficacy on an outpatient basis unless compliance can be accurately assessed and included as a covariate in the analysis.

A review of the literature regarding low fat and low carbohydrate diets is complicated by the fact that currently there are no standard definitions of these diets. The American Academy of Family Physicians has defined a low carbohydrate diet as having <20% of calories from carbohydrate [30]. The Atkins diet that was popularized in the 1970s restricts carbohydrate intake to 15–20g per day during the induction phase [31]. Whereas ketogenic diets encourage 90% of calories to come from fat, 1g/Kg of protein and minimal (<15g per day) carbohydrates [32]. The traditional ketogenic diet has a ratio of fats to protein and carbohydrate of 4:1, but there have been numerous adjustments to this with the development of diet offshoots, such as modified Atkins and low glycemic index diet [32]. Similarly, there is not a standard low-fat diet definition, but generally <30% Kcal from fat can be considered a low-fat diet and <20% a very low-fat diet [33]. Without standard definitions, the idea that these diets could become universally prescriptive under any circumstances is unlikely.

Highly controlled and precise diet studies are inherently difficult to execute. Outpatient studies lack the required oversite to ensure that dietary interventions are appropriately followed and intakes are accurately reported. Dietary self-reports have an important role in epidemiological and nutrition research, however, they should not be used as a measure of true energy intake or for determining diet-health associations [34]. The errors in exercise and activity self-reports also have the potential for introducing error in energy balance studies. At this time, large-scale inpatient trials of low carbohydrate and low-fat diets are lacking in the literature. However, even when executed to perfection (and often at great expense), inpatient interventions do not always have feasible real-world applications.

References

1. Adult Obesity Facts. 13 August 2018. Available online: https://www.cdc.gov/obesity/data/adult.html (accessed on 1 August 2019).
2. Djalalinia, S.; Qorbani, M.; Peykari, N.; Kelishadi, R. Health impacts of obesity. Pak. J. Med. Sci. 2015, 1, 239–242.
3. United States Senate Select Committee on Nutrition and Human Needs. Dietary Goals for the United States; U.S. Government Printing Office: Washington, DC, USA, 1977.

4. Watts, M.; Hager, M.; Toner, C. The art of translating nutritional science into dietary guidance: History and evolution of the Dietary Guidelines for Americans. Nutr. Rev. 2011, 69, 404–412.
5. Gow, M.L.; Ho, M.; Burrows, T.L.; Baur, L.A.; Hutchesson, M.J.; Cowell, C.T.; Collins, C.E.; Garnett, S.P. Impact of dietary macronutrient distribution on BMI and cardiometabolic outcomes in overweight and obese children and adolescents: A systematic review. Nutr. Rev. 2014, 72, 453–470.
6. Ludwig, D.S. The glycemic index: Physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. JAMA 2002, 287, 2414–2423.
7. Ludwig, D.S.; Ebbeling, C.B. The carbohydrate-insulin model of obesity: Beyond “calories in, calories out”. JAMA Intern. Med. 2018, 178, 1098–1103.
8. Hall, K.D.; Guyenet, S.J.; Leibel, R.L. The carbohydrate-insulin model of obesity is difficult to reconcile with current evidence. JAMA Intern. Med. 2018, 178, 1103–1105.
9. Cooder, H.R. Epilepsy in children-with particular reference to the ketogenic diet. Calif. J. West Med. 1933, 39, 169–173.
10. Allen, F.M.; Fitz, R.; Stillman, E. Total Dietary Regulation in the Treatment of Diabetes; Rockefeller Institute for Medical Research: New York, NY, USA, 1919.
11. Leibel, R.L.; Hirsch, J.; Appel, B.E.; Checani, G.C. Energy intake required to maintain body weight is not affected by wide variation in diet composition. Am. J. Clin. Nutr. 1992, 55, 350–355.
12. Hall, K.D. A review of the carbohydrate-insulin model of obesity. Eur. J. Clin. Nutr. 2017, 71, 323–326.
13. Ebbeling, C.B.; Swain, J.F.; Feldman, H.A.; Wong, W.W.; Hachey, D.L.; Garcia-Lago, E.; Ludwig, D.S. Effects of dietary composition on energy expenditure during weight loss maintenance. JAMA 2012, 307, 2627–2634.
14. Makris, A.; Foster, G.D. Dietary approaches to the treatment of obesity. Psychiatr. Clin. N. Am. 2011, 34, 813–827.
15. Dahiya, D.K.; Puniya, M.; Shandilya, U.K.; Dhewa, T.; Kumar, N.; Kumar, S.; Puniya, A.K.; Shukla, P. Gut microbiota modulation and its relationship with obesity using prebiotic fibers and probiotics: A review. Front. Microbiol. 2017, 8, 563.
16. Oppenheimer, G.M. Becoming the Framingham Study 1947-1950. Am. J. Public Health 2005, 95, 602–610.
17. Keys, A. Seven Countries: A Multivariate Analysis of Death and Coronary Heart Disease; Harvard University Press: Cambridge, MA, USA, 1980.

18. Keys, A.; Keys, M. How to Eat Well and Stay Well the Mediterranean Way; Doubleday: New York, NY, USA, 1975.
19. La Berge, A.F. How the ideology of low fat conquered America. J. Hist. Med. Allied Sci. 2008, 63, 139–177.
20. Report by the Central Committee for Medical and Community Program of the American Heart Association. Dietary fat and its relation to heart attacks and strokes. JAMA 1961, 175, 389–391.
21. Levy, D.; Brink, S. A Change of Heart; Knopf: New York, NY, USA, 2005.
22. Jéquier, E. Nutrient effects: Post-absorptive interactions. Proc. Nutr. Soc. 1995, 54, 253–265.
23. Bray, G.A.; Popkin, B.M. Dietary fat does affect obesity! Am. J. Clin. Nutr. 1998, 68, 1157–1173.
24. Jéquier, E.; Tappy, L. Regulation of body weight in humans. Physiol. Rev. 1999, 79, 451–480.
25. Yu, Y.H. Making sense of metabolic obesity and hedonic obesity. J. Diabetes 2017, 9, 656–666.
26. Duffey, K.J.; Popkin, B.M. Energy density, portion size, and eating occasions: Contributions to increased energy intake in the United States. PLoS Med. 2011, 8, e1001050.
27. Rosenbaum, M.; Leibel, R.L. Models of energy homeostasis in response to maintenance of reduced body weight. Obesity 2016, 24, 1620–1629.
28. Straznicky, M.; Lambert, E.; Nestel, P.; McGrane, M.; Dawood, T.; Schlaich, M.; Masuo, K.; Eikelis, N.; de Courten, B.; Mariani, J.; et al. Sympathetic neural adaptation to hypocaloric diet with or without exercise training in obese metabolic syndrome subjects. Diabetes 2010, 59, 71–79.
29. Rosenbaum, M.; Leibel, R.L. 20 years of leptin: Role of leptin in energy homeostasis in humans. J. Endocrinol. 2014, 223, T83–T96.
30. Last, A.R.; Wilson, S.A. Low-carbohydrate diets. Am. Fam. Physician 2006, 73, 1942–1984.
31. Atkins, R.C. Dr. Atkins' Diet Revolution; the High Calorie Way to Stay Thin Forever; D. McKay Co: New York, NY, USA, 1972.
32. Freeman, J.M.; Kossoff, E.H. Ketosis and the ketogenic diet, 2010: Advances in treating epilepsy and other disorders. Adv. Pediatr. 2010, 57, 315–329.
33. Austin, G.L.; Ogden, L.G.; Hill, J.O. Trends in carbohydrate, fat, and protein intakes and association with energy intake in normal-weight, overweight, and obese individuals: 1971-2006. Am. J. Clin. Nutr. 2011, 83, 836–843.
34. Subar, A.F.; Freedman, L.S.; Tooze, J.A.; Kirkpatrick, S.I.; Boushey, C.; Neuhouser, M.L.; Thompson, F.E.; Poitschman, N.; Guenther, P.M.; Tarasuk, V.; et al. Addressing current criticism regarding the value of self-report dietary data. J. Nutr. 2015, 145, 2639–2645.