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High-fat diets: healthy or unhealthy ?

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Abstract

In current dietary recommendations for the treatment and prevention of Type 2 diabetes and related complications there is flexibility in the proportion of energy derived from monounsaturated fat and carbohydrate as a replacement for saturated fat. Over the past few years, several population studies have shown that subjects eating a lot of refined grains and processed foods gain a much larger increase in waist circumference than those following a diet higher in monounsaturated fat, protein and carbohydrates rich in fibres and whole grains. In this issue of *Clinical Science*, Sinitskaya and co-workers have demonstrated that, in normal-weight rodents categorized into three groups of high-fat, medium-carbohydrate (53/30 ratio of fat/CHO % of energy, 4.7 kcal/g), high-fat, low-carbohydrate (67/9 ratio of fat/CHO % of energy, 5.2 kcal/g) and high-fat, carbohydrate-free (75/0 ratio of fat/CHO % of energy, 5.9 kcal/g) diets, the high-fat diets that also contained carbohydrates were both obesogenic and diabetogenic while the very high-fat, carbohydrate-free diet was not obesogenic but led to insulin resistance and higher risk of cardiovascular disease. This finding may indicate that high-fat diets could easily be precipitated with carbohydrates to an unhealthy diet, pointing at the significance of macronutrient composition rather than caloric content in high-fat diets.



Comment

Dietary advice is vital for patients with Type 2 diabetes. Initial dietary advice was aimed at controlling the glucose concentration in the blood and the urine. A high-fat, low-carbohydrate diet was advocated for the treatment of Type 2 diabetes by Dr W. Osler and by other distinguished physicians at that time [1]. The diet recommended for Type 2 diabetics in 1923 contained approximately 75% of total energy intake from fat, 20% from protein and 5% from carbohydrates [1]. The current recommendations of the American Diabetes Association, (ADA) and the European Association for the Study of Diabetes, (EASD), on dietary composition to the treatment and prevention of diabetes mellitus are < 35% of total energy intake from fat (< 10% from saturated and transunsaturated fat, < 10% from n-6 and n-3 polyunsaturated fat, 10-20% from cis monounsaturated fat), 10-20% from protein and 45-60% from carbohydrates [2, 3]. Thus, current low-fat, high-carbohydrate diet recommendations are dramatically different from the high-fat, low-carbohydrate dietary recommendations from the 1920's.

Excess weight is the principal risk factor for Type 2 diabetes. Visceral fat produces inflammatory activators, which, in turn, evoke insulin resistance. Obesity therefore promotes insulin resistance and glucose intolerance [4]. The cardiovascular and other metabolic complications resulting from abdominal visceral obesity and Type 2 diabetes are a significant cause of premature morbidity and mortality in industrialized countries [5]. Not surprisingly, the risk of contribution of high-fat food for weight gain and cardiovascular complications has been thoroughly investigated in population studies. Data from an 8-year follow-up in the Nurses Health Study (NHS) showed that overall, percent of calories from fat only had a weak positive association with weight gain [6]. Increased intake of monosaturated and polysaturated fat were not associated with weight gain: however, percentage of fat from animal, saturated, and trans fat showed associations with greater weight gain. Furthermore, diets lower in carbohydrate and higher in vegetable sources of fat and protein have been shown to reduce glycaemic burden and the risk of coronary heart disease in women [7].

In this issue of *Clinical Science*, Sinitskaya et al. [8] have demonstrated that, in rodents categorized into groups with high-fat, medium-carbohydrate (53/30 ratio of fat/CHO % of energy, 4.7 kcal/g), high-fat, low-carbohydrate (67/9 ratio of fat/CHO % of energy, 5.2



kcal/g) and high-fat carbohydrate-free (75/0 ratio of fat/CHO % of energy, 5.9 kcal/g) diets, the high-fat diets that contained carbohydrates produced weight gain and abdominal obesity. Interestingly, the high-fat, carbohydrate-free diet did not produce significantly higher body mass index or % adiposity than the control with a low-fat, high-carbohydrate, low-caloric (12/64 ratio of fat/CHO % of energy, 3.7 kcal/g) diet. Instead, the high-fat diets with low and medium-carbohydrate produced significantly higher BMI and % adiposity than the control diet. However, authors do not comment upon the fact that the high-fat, medium-carbohydrate diet (53/30 ratio of fat/CHO % of energy, 4.7 kcal/g) also contained 7-8% less metabolizable energy content in the form of protein compared to the other diets, possibly confounding the conclusions. In fact, population studies have shown that a lower content of protein in diet may not be favourable in terms of trying to maintain a thinner waist, instead a high energy intake from protein predicts an abdominal decrease [9]. Also, those eating a lot of refined grains and processed foods show a much larger increase in waist circumference than those eating carbohydrates rich in fibres and whole grains [9]. Furthermore, fat-rich diets can acutely induce insulin resistance, especially in subjects with already low adiponectin levels [10]. Thus, it is possible that the restriction of dietary fat and a diet high in carbohydrates might be particularly effective in subjects with low adiponectin levels such as individuals with obesity and Type 2 diabetes. The high-fat diets in this study may, in part, be precipitated or accelerated in combination with carbohydrates and low protein content to an unhealthy diet, pointing at the significance of macronutrient composition rather than merely the caloric content in high-fat foods.

In current dietary recommendations for people with Type 2 diabetes, there is flexibility in the proportion of energy derived from monounsaturated fats and carbohydrate [2, 3]. Provided that energy intake is controlled, the use of monounsaturated fat instead of carbohydrate as a replacement for saturated fat may cause an increase, as opposed to a decrease, in HDL-cholesterol levels, which is of particular benefit in Type 2 diabetes and abdominal obesity. Thus, monounsaturated fats are promoted as the main source of dietary fat because of their lower susceptibility to lipid peroxidation and consequent lower atherogenic potential [11]. The rationale for modifying fat distribution and reducing weight is based on an expected improvement in insulin sensitivity, β -cell function and insulin-sensitizing factors [12]. Adiponectin, which is exclusively produced and secreted from the adipocyte, has putative anti-atherogenic properties and appears to ameliorate insulin resistance in both muscle and



liver [10, 12]. This protein has now been extensively explored, and it is possible that a low plasma adiponectin concentration may be a convenient marker for identifying subjects with the metabolic syndrome who may progress to prediabetes on an unhealthy diet. Ways of preventing severe obesity, especially in childhood, by providing healthy dietary recommendations for individuals at risk, and also the role of calorically sweetened soft drinks and the increased total intake of caloric sweeteners contribution to the current obesity epidemic need to be subjected to intense scrutiny.

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References

1. Osler, W., McCrae, T. (1923). *The Principles and Practice of Medicine*. New York, Appleton and Co.
2. American Diabetes Association Task Force for Writing Nutrition Principles and Recommendations for the Management of Diabetes and Related Complications: American Diabetes Association position statement: evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. (2002). *J. Am. Diet. Assoc.* **102**, 109-118.
3. Mann, J., I., De Leeuw, I., Hermansen, K. et al. (2004). The Diabetes and Nutrition Study Group (DNSG) of the European Association: Evidence-based nutritional approaches to the treatment and prevention of diabetes mellitus. *Nutr. Metab. Cardiovasc. Dis.* **14**, 373-394.
4. Weiss, R., Dufour, S., Taksali, S., E. et al. (2003). Prediabetes in obese youth: a syndrome of impaired glucose tolerance, severe insulin resistance, and altered myocellular and abdominal fat partitioning. *Lancet* **362**, 951-957.
5. Simone, G., Devereux, R., B., Chinali, M. et al. (2007). Prognostic impact of metabolic syndrome by different definitions in a population with high prevalence of obesity and diabetes. The Strong Heart Study. *Diabetes Care* **30**, 1851-1856.
6. Field, A., E., Willett, W., C., Lissner, L., and Colditz, G., A. (2007). Dietary fat and weight gain among women in the Nurses Health Study. *Obesity (Silver Spring)* **15**, 967-976.
7. Halton, T. L., Willett, W., C., Liu, S., Manson, J., E., Albert, C. M., Rexrode, K., and Hu, F. B. (2007). Low-carbohydrate-diet score and the risk of coronary heart disease in women. *N. Eng. J. Med.* **355**, 1991-2002.
8. Sinitskaya, N., Gourmelin, S., Schuster-Klein, C., Guardiola-Lemaitre, B., Pevet, P., and Challet, E. (2007). Increasing fat-to-carbohydrate ratio in a high-fat diet prevents the development of obesity but not prediabetic state in rats. *Clin. Sci.* **?**, ?-?.
9. Halkjær, J., Tjønneland, A., Thomsen, L. et al. (2006). Intake of macronutrients as predictors of 5-y changes in waist circumference. *Am. J. Clin. Nutr.* **84**, 789-797.
10. Thamer, C., Haap, M., Bachmann, O. et al. (2004). Serum adiponectin levels predict the effect of short-term dietary interventions on insulin sensitivity in humans. *Diabetologia* **47**, 1303-1305.



11. Garg, A. (1998). High-monosaturated fat diets for patients with diabetes mellitus: a meta-analysis. *Am. J. Clin. Nutr.* **67**, 577S-582S.
12. Guldstrand, M., Adamson, U., and Ahrén, B. (2003). Improved β -cell function after standardized weight reduction in severely obese subjects. *Am. J. Physiol. Endocrinol. Metab.* **284**, E557-E565.

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