Attached are randomized-controlled trials and other evidence supporting the case for carbohydrate-restriction as the primary intervention to reverse obesity, fix type 2 diabetes and minimise cardiovascular disease.


Comments, criticisms, questions, compliments, whatever are welcome
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Dietary carbohydrate restriction as the first approach in diabetes management: Critical review and evidence base


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DOI: 10.1016/j.nut.2014.06.011

Abstract: This is a critical review of the evidence supporting the use of low-carbohydrate diets in the management of diabetes. The benefits of carbohydrate restriction in diabetes are immediate and well documented. Concerns about the efficacy and safety are largely theoretical and anecdotal rather than data driven. Dietary carbohydrate restriction reliably reduces high blood glucose, does not require weight loss (although it is still best for weight loss), and leads to the reduction or elimination of medication. It has never shown side effects comparable to those seen in intensive pharmacologic treatment.

Highlights:
- We present major evidence for low-carbohydrate diets as a first approach for diabetes.
- Such diets reliably reduce high blood glucose, the most salient feature of diabetes.
- Benefits do not require weight loss although weight loss is better for weight reduction.
- Carbohydrate-restricted diets reduce or eliminate need for medication.
- There are no side effects comparable to those seen in intensive pharmacologic treatment.

Graphical abstract:

Abstract:

The inability of current recommendations to control the epidemic of diabetes, the specific failure of the prevailing low-fat diets to improve obesity, cardiovascular risk, or general health and the persistent reports of some serious side effects of commonly prescribed diabetic medications, in combination with the continued success of low-carbohydrate diets in the treatment of diabetes and metabolic syndrome without significant side effects, point to the need for a reappraisal of dietary guidelines. The benefits of carbohydrate restriction in diabetes are immediate and well documented. Concerns about the efficacy and safety are largely theoretical and anecdotal rather than data driven. Dietary carbohydrate restriction reliably reduces high blood glucose, does not require weight loss (although it is still best for weight loss), and leads to the reduction or elimination of medication. It has never shown side effects comparable to those seen in intensive pharmacologic treatment. Here we present 12 points of evidence supporting the use of low-carbohydrate diets as the first approach to treating type 2 diabetes and as the most effective adjacent to pharmacology in type 1. They represent the best-documented, least controversial results. The insistence on long-term randomized controlled trials as the only kind of data that will be accepted is without precedent in science. The side effects of diabetes require that we evaluate all of the evidence that is available. The 12 points are sufficiently compelling that we feel that the burden of proof rests with those who are opposed.
Comparison of the Atkins, Zone, Ornish, and LEARN Diets for Change in Weight and Related Risk Factors Among Overweight Premenopausal Women

The A TO Z Weight Loss Study: A Randomized Trial

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The Ongoing Obesity Epidemic, along with its health costs and consequences and the health benefits of weight loss, have been well established. National dietary weight loss guidelines (ie, energy-restricted, low in fat, high in carbohydrate) have been challenged, particularly by proponents of low-carbohydrate diets. However, limited evidence has been available to effectively evaluate other diets.

Several recent trials compared low-carbohydrate vs traditional low-fat, high-carbohydrate weight-loss diets. A meta-analysis that pooled the results of these early trials concluded that low-carbohydrate, non-energy-restricted diets were at least as effective as low-fat, high carbohydrate diets in inducing weight loss for up to 1 year. However, most of these trials were limited by combinations of small sample sizes, high rates of attrition, short durations, or limited diet assessment.

For the A TO Z (Atkins, Traditional, Ornish, Zone) Weight Loss Study, we selected 4 diets—3 popular and substantially different diets and 1 diet based on national guidelines—representing a spectrum of carbohydrate intake: Atkins (very low in carbohydrate), Zone

Context

Popular diets, particularly those low in carbohydrates, have challenged current recommendations advising a low-fat, high-carbohydrate diet for weight loss. Potential benefits and risks have not been tested adequately.

Objective

To compare 4 weight-loss diets representing a spectrum of low to high carbohydrate intake for effects on weight loss and related metabolic variables.

Design, Setting, and Participants

Twelve-month randomized trial conducted in the United States from February 2003 to October 2005 among 311 free-living, overweight/obese (body mass index, 27–40) nondiabetic, premenopausal women.

intervention

Participants were randomly assigned to follow the Atkins (n=77), Zone (n=79), LEARN (n=75), or Ornish (n=76) diets and received weekly instruction for 2 months, then an additional 10-month follow-up.

Main Outcome Measures

Weight loss at 12 months was the primary outcome. Secondary outcomes included lipid profile (low-density lipoprotein, high-density lipoprotein, and non–high-density lipoprotein cholesterol, and triglyceride levels), percentage of body fat, waist-hip ratio, fasting insulin and glucose levels, and blood pressure. Outcomes were assessed at months 0, 2, 6, and 12. The Tukey studentized range test was used to adjust for multiple testing.

Results

Weight loss was greater for women in the Atkins diet group compared with the other diet groups at 12 months, and mean 12-month weight loss was significantly different between the Atkins and Zone diets (P<.05). Mean 12-month weight loss was as follows: Atkins, −4.7 kg (95% confidence interval [CI], −6.3 to −3.1 kg), Zone, −1.6 kg (95% CI, −2.8 to −0.4 kg), LEARN, −2.6 kg (−3.8 to −1.3 kg), and Ornish, −2.2 kg (−3.6 to −0.8 kg). Weight loss was not statistically different among the Zone, LEARN, and Ornish groups. At 12 months, secondary outcomes for the Atkins group were comparable with or more favorable than the other diet groups.

Conclusions

In this study, premenopausal overweight and obese women assigned to follow the Atkins diet, which had the lowest carbohydrate intake, lost more weight and experienced more favorable overall metabolic effects at 12 months than women assigned to follow the Zone, Ornish, or LEARN diets. While questions remain about long-term effects and mechanisms, a low-carbohydrate, high-protein, high-fat diet may be considered a feasible alternative recommendation for weight loss.

Trial Registration

clinicaltrials.gov Identifier: NCT0079573

JAMA. 2007;297:949-957

Author Affiliations: Stanford Prevention Research Center and the Department of Medicine, Stanford University School of Medicine, Stanford, Calif. Corresponding Author: Christopher D. Gardner, PhD, Hoover Pavilion, N225, 211 Quarry Rd, Stanford, CA 94305-7505 (gardner@stanford.edu).
Weight Loss with a Low-Carbohydrate, Mediterranean, or Low-Fat Diet


ABSTRACT

BACKGROUND
Trials comparing the effectiveness and safety of weight-loss diets are frequently limited by short follow-up times and high dropout rates.

METHODS
In this 2-year trial, we randomly assigned 322 moderately obese subjects (mean age, 52 years; mean body-mass index [the weight in kilograms divided by the square of the height in meters], 31; male sex, 86%) to one of three diets: low-fat, restricted-calorie; Mediterranean, restricted-calorie; or low-carbohydrate, non-restricted-calorie.

RESULTS
The rate of adherence to a study diet was 95.4% at 1 year and 84.6% at 2 years. The Mediterranean-diet group consumed the largest amounts of dietary fiber and had the highest ratio of monounsaturated to saturated fat (P<0.05 for all comparisons among treatment groups). The low-carbohydrate group consumed the smallest amount of carbohydrates and the largest amounts of fat, protein, and cholesterol and had the highest percentage of participants with detectable urinary ketones (P<0.05 for all comparisons among treatment groups). The mean weight loss was 2.9 kg for the low-fat group, 4.4 kg for the Mediterranean-diet group, and 4.7 kg for the low-carbohydrate group (P<0.001 for the interaction between diet group and time; among the 272 participants who completed the intervention, the mean weight losses were 3.3 kg, 4.6 kg, and 5.5 kg, respectively. The relative reduction in the ratio of total cholesterol to high-density lipoprotein cholesterol was 20% in the low-carbohydrate group and 12% in the low-fat group (P=0.01). Among the 36 subjects with diabetes, changes in fasting plasma glucose and insulin levels were more favorable among those assigned to the Mediterranean diet than among those assigned to the low-fat diet (P<0.001 for the interaction among diabetes and Mediterranean diet and time with respect to fasting glucose levels).

CONCLUSIONS
Mediterranean and low-carbohydrate diets may be effective alternatives to low-fat diets. The more favorable effects on lipids (with the low-carbohydrate diet) and on glycemic control (with the Mediterranean diet) suggest that personal preferences and metabolic considerations might inform individualized tailoring of dietary interventions. (ClinicalTrials.gov number, NCT00160108.)

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Nutrition & Metabolism

Research
The effect of a low-carbohydrate, ketogenic diet versus a low-glycemic index diet on glycemic control in type 2 diabetes mellitus
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Abstract
Objective: Dietary carbohydrate is the major determinant of postprandial glucose levels, and several clinical studies have shown that low-carbohydrate diets improve glycemic control. In this study, we tested the hypothesis that a diet lower in carbohydrate would lead to greater improvement in glycemic control over a 24-week period in patients with obesity and type 2 diabetes mellitus.

Research design and methods: Eighty-four community volunteers with obesity and type 2 diabetes were randomized to either a low-carbohydrate, ketogenic diet (<20 g of carbohydrate daily, LCKD) or a low-glycemic, reduced-calorie diet (500 kcal/day deficit from weight maintenance diet, LGID). Both groups received group meetings, nutritional supplementation, and an exercise recommendation. The main outcome was glycemic control, measured by hemoglobin A1c.

Results: Forty-nine (58.3%) participants completed the study. Both interventions led to improvements in hemoglobin A1c, fasting glucose, fasting insulin, and weight loss. The LCKD group had greater improvements in hemoglobin A1c (-1.5% vs. -0.5%, p = 0.03), body weight (-11.1 kg vs. -6.9 kg, p = 0.008), and high density lipoprotein cholesterol (+5.6 mg/dL vs. 0 mg/dL, p < 0.001) compared to the LGID group. Diabetes medications were reduced or eliminated in 95.2% of LCKD vs. 62% of LGID participants (p < 0.01).

Conclusion: Dietary modification led to improvements in glycemic control and medication reduction/elimination in motivated volunteers with type 2 diabetes. The diet lower in carbohydrate led to greater improvements in glycemic control, and more frequent medication reduction/elimination than the low glycemic index diet. Lifestyle modification using low carbohydrate interventions is effective for improving and reversing type 2 diabetes.
Carbohydrate restriction has a more favorable impact on the metabolic syndrome than a low fat diet.

Volek JS¹, Phinney SD, Forsythe CE, Quann EE, Wood RJ, Puglisi MJ, Kraemer WJ, Bibus DM, Fernandez ML, Feinman RD.

Author information

Abstract
We recently proposed that the biological markers improved by carbohydrate restriction were precisely those that define the metabolic syndrome (MetS), and that the common thread was regulation of insulin as a control element. We specifically tested the idea with a 12-week study comparing two hypocaloric diets (approximately 1,500 kcal): a carbohydrate-restricted diet (CRD) (%carbohydrate:fat:protein = 12:59:28) and a low-fat diet (LFD) (56:24:20) in 40 subjects with atherogenic dyslipidemia. Both interventions led to improvements in several metabolic markers, but subjects following the CRD had consistently reduced glucose (-12%) and insulin (-50%) concentrations, insulin sensitivity (-55%), weight loss (-10%), decreased adiposity (-14%), and more favorable triacylglycerol (TAG) (-51%), HDL-C (13%) and total cholesterol/HDL-C ratio (-14%) responses. In addition to these markers for MetS, the CRD subjects showed more favorable responses to alternative indicators of cardiovascular risk: postprandial lipemia (-47%), the Apo B/Apo A-1 ratio (-16%), and LDL particle distribution. Despite a threefold higher intake of dietary saturated fat during the CRD, saturated fatty acids in TAG and cholesteryl ester were significantly decreased, as was palmitoleic acid (16:1n-7), an endogenous marker of lipogenesis, compared to subjects consuming the LFD. Serum retinol binding protein 4 has been linked to insulin-resistant states, and only the CRD decreased this marker (-20%). The findings provide support for unifying the disparate markers of MetS and for the proposed intimate connection with dietary carbohydrate. The results support the use of dietary carbohydrate restriction as an effective approach to improve features of MetS and cardiovascular risk.

PMID: 19082851 [PubMed - indexed for MEDLINE]
Effects of Low-Carbohydrate and Low-Fat Diets: A Randomized Trial

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Background: Low-carbohydrate diets are popular for weight loss, but their cardiovascular effects have not been well-studied, particularly in diverse populations.

Objective: To examine the effects of a low-carbohydrate diet compared with a low-fat diet on body weight and cardiovascular risk factors.

Design: A randomized, parallel-group trial.

Setting: A large academic medical center.

Participants: 148 men and women without clinical cardiovascular disease and diabetes.

Intervention: A low-carbohydrate (<40 g/d) or low-fat (<30% of daily energy intake from total fat [<7% saturated fat]) diet. Both groups received dietary counseling at regular intervals throughout the trial.

Measurements: Data on weight, cardiovascular risk factors, and dietary composition were collected at 0, 3, 6, and 12 months.

Results: Sixty participants (82%) in the low-fat group and 59 (79%) in the low-carbohydrate group completed the intervention. At 12 months, participants on the low-carbohydrate diet had greater decreases in weight (mean difference in change, −3.5 kg [95% CI, −5.6 to −1.4 kg]; P = 0.002), fat mass (mean difference in change, −1.5% [CI, −2.6% to −0.4%]; P = 0.011), ratio of total-high-density lipoprotein (HDL) cholesterol (mean difference in change, −0.44 [CI, −0.71 to −0.16]; P = 0.002), and triglyceride level (mean difference in change, −0.16 mmol/L [−1.41 mg/dL]; CI, −0.31 to −0.01 mmol/L [−27.4 to −0.8 mg/dL]; P = 0.038) and greater increases in HDL cholesterol level (mean difference in change, 0.16 mmol/L [7.0 mg/dL] [CI, 0.08 to 0.28 mmol/L [3.0 to 11.0 mg/dL]]; P < 0.001) than those on the low-fat diet.

Limitation: Lack of clinical cardiovascular disease and points.

Conclusion: The low-carbohydrate diet was more effective for weight loss and cardiovascular risk factor reduction than the low-fat diet. Restricting carbohydrate may be an option for persons seeking to lose weight and reduce cardiovascular risk factors.

Primary Funding Source: National Institutes of Health.

Topics
carbohydrate; diet; baseline; dietary fat; carbohydrate restricted diet; changes in diet; obesity; fat-restricted diet; weight reduction
A Randomized Trial
of a Low-Carbohydrate Diet for Obesity

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and Samuel Klein, M.D.

BACKGROUND
Despite the popularity of the low-carbohydrate, high-protein, high-fat (Atkins) diet, no randomized, controlled trials have evaluated its efficacy.

METHODS
We conducted a one-year, multicenter, controlled trial involving 63 obese men and women who were randomly assigned to either a low-carbohydrate, high-protein, high-fat diet or a low-calorie, high-carbohydrate, low-fat (conventional) diet. Professional contact was minimal to replicate the approach used by most dieters.

RESULTS
Subjects on the low-carbohydrate diet had lost more weight than subjects on the conventional diet at 3 months (mean [SD], -6.8±5.0 vs. -2.7±3.7 percent of body weight; P<0.001) and 6 months (-7.0±6.5 vs. -3.2±5.6 percent of body weight, P<0.02), but the difference at 12 months was not significant (-4.4±6.7 vs. -2.5±6.3 percent of body weight, P=0.26). After three months, no significant differences were found between the groups in total or low-density lipoprotein cholesterol concentrations. The increase in high-density lipoprotein cholesterol concentrations and the decrease in triglyceride concentrations were greater among subjects on the low-carbohydrate diet than among those on the conventional diet throughout most of the study. Both diets significantly decreased diastolic blood pressure and the insulin response to an oral glucose load.

CONCLUSIONS
The low-carbohydrate diet produced a greater weight loss (absolute difference, approximately 4 percent) than did the conventional diet for the first six months, but the differences were not significant at one year. The low-carbohydrate diet was associated with a greater improvement in some risk factors for coronary heart disease. Adherence was poor and attrition was high in both groups. Larger and longer studies are required to determine the long-term safety and efficacy of low-carbohydrate, high-protein, high-fat diets.
A Call for a Low-Carb Diet That Embraces Fat

By ANAHAD O'CONNOR   SEPT. 1, 2014

People who avoid carbohydrates and eat more fat, even saturated fat, lose more body fat and have fewer cardiovascular risks than people who follow the low-fat diet that health authorities have favored for decades, a major new study shows.

The findings are unlikely to be the final salvo in what has been a long and often contentious debate about what foods are best to eat for weight loss and overall health. The notion that dietary fat is harmful, particularly saturated fat, arose decades ago from comparisons of disease rates among large national populations.

But more recent clinical studies in which individuals and their diets were assessed over time have produced a more complex picture. Some have provided strong evidence that people can sharply reduce their heart disease risk by eating fewer carbohydrates and more dietary fat, with the exception of trans fats. The new findings suggest that this strategy more effectively reduces body fat and also lowers overall weight.

The new study was financed by the National Institutes of Health and published in the Annals of Internal Medicine. It included a racially diverse group of 150 men and women — a rarity in clinical nutrition studies — who were assigned to follow diets for one year that limited either the amount of carbs or fat that they could eat, but not overall calories.

“To my knowledge, this is one of the first long-term trials that’s given these diets without calorie restrictions,” said Dariush Mozaffarian, the dean of the Friedman School of Nutrition Science and Policy at Tufts University, who was not involved in the new study. “It shows that in a
Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease1–5

Patty W Siri-Turino, Qi Sun, Frank B Hu, and Ronald M Krauss

ABSTRACT

Background: A reduction in dietary saturated fat has generally been thought to improve cardiovascular health. The objective of this meta-analysis was to summarize the evidence related to the association of dietary saturated fat with risk of coronary heart disease (CHD), stroke, and cardiovascular disease (CVD; CHD inclusive of stroke) in prospective epidemiologic studies.

Design: Twenty-one studies identified by searching MEDLINE and Embase databases and secondary referencing qualified for inclusion in this study. A random-effects model was used to derive composite relative risk estimates for CHD, stroke, and CVD.

Results: During 5–23 y of follow-up of 347,747 subjects, 11,006 developed CHD or stroke. Intake of saturated fat was not associated with an increased risk of CHD, stroke, or CVD. The pooled relative risk estimates that compared extreme quintiles of saturated fat intake were 1.07 (95% CI: 0.96, 1.19; P = 0.22) for CHD, 0.81 (95% CI: 0.62, 1.05; P = 0.11) for stroke, and 1.00 (95% CI: 0.89, 1.11; P = 0.95) for CVD. Consideration of age, sex, and study quality did not change the results.

Conclusions: A meta-analysis of prospective epidemiologic studies showed that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD. More data are needed to elucidate whether CVD risks are likely to be influenced by the specific nutrients used to replace saturated fat. Am J Clin Nutr 2010;91:535–46.

INTRODUCTION

Early animal studies showed that high dietary saturated fat and cholesterol intakes led to increased plasma cholesterol concentrations as well as atherosclerotic lesions (1). These findings were supported by associations in humans in which dietary saturated fat correlated with coronary heart disease (CHD) risk (2, 3). More recent epidemiologic studies have shown positive (4–10), inverse (11, 12), or no (4, 13–18) associations of dietary saturated fat with CVD morbidity and/or mortality.

A limited number of randomized clinical interventions have been conducted that have evaluated the effects of saturated fat on risk of CVD. Whereas some studies have shown beneficial effects of reduced dietary saturated fat (19–21), others have shown no effects of such diets on CVD risk (22, 23). The studies that showed beneficial effects of diets reduced in saturated fat replaced saturated fat with polyunsaturated fat, with the implication that the CVD benefit observed could have been due to an increase in polyunsaturated fat or in the ratio of polyunsaturated to saturated fat (P:S), a hypothesis supported by a recent pooling analysis conducted by Jakobsen et al (24).

The goal of this study was to conduct a meta-analysis of well-designed prospective epidemiologic studies to estimate the risk of CHD and stroke and a composite risk score for both CHD and stroke, or total cardiovascular disease (CVD), that was associated with increased dietary intakes of saturated fat. Large prospective cohort studies can provide statistical power to adjust for covariates, thereby enabling the evaluation of the effects of a specific nutrient on disease risk. However, such studies have censuses, including a reliance on nutritional assessment methods whose validity and reliability may vary (25), the assumption that diets remain similar over the long term (26) and variable adjustment for covariates by different investigators. Nonetheless, a summary evaluation of the epidemiologic evidence to date provides important information as to the basis for relating dietary saturated fat to CVD risk.

SUBJECTS AND METHODS

Study selection

Two investigators (QS and PS-T) independently conducted a systematic literature search of the MEDLINE (http://www.ncbi.nlm.nih.gov/pubmed) and Embase (http://www.embase.com) databases through 17 September 2009 by using the following search terms: ("saturated fat" or "dietary fat") and ("coronary" or "cardiovascular" or "stroke") and ("cohort" or "follow up").

1 From the Children’s Hospital Oakland Research Institute, Oakland, CA (PWS-T and RMK), and the Departments of Nutrition (QS and FBH) and Epidemiology (PS-T), Harvard School of Public Health, Boston, MA.

2 PWS-T and QS contributed equally to this work.

3 The contents of this article are solely the responsibility of the authors and do not necessarily represent the official view of the National Center for Research Resources (http://www.ncrr.nih.gov) or the National Institutes of Health.

4 Supported by the National Dairy Council (PWS-T and RMK) and made possible by grant U11 RR02413-01 from the National Center for Research Resources, a component of the National Institutes of Health (NIH), and NIH Roadmap for Medical Research (PWS-T and RMK). QS was supported by a Postdoctoral Fellowship from Unilever Corporate Research. FBH was supported by NIH grant HL07012.

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The Questionable Link Between Saturated Fat and Heart Disease

Are butter, cheese and steak really bad for you? The dubious science behind the anti-fat crusade

By NINA TEICHLER
Updated May 8, 2014 10:25 a.m. ET

“Saturated fat does not cause heart disease”—or so concluded a big study published in March in the journal Annals of Internal Medicine. How could this be? The very cornerstone of dietary advice for generations has been that the saturated fats in butter, cheese and red meat should be avoided because they clog our arteries. For many diet-conscious Americans, it is simply second nature to opt for chicken over sirloin, canola oil over butter.

The new study’s conclusion shouldn’t surprise anyone familiar with modern nutritional science, however. The fact is, there has never been solid evidence for the idea that these fats cause disease. We only believe this to be the case because nutrition policy has been derailed over the past half-century by a mixture of personal ambition, bad science, politics and bias.

Related Video

Our distrust of saturated fat can be traced back to the 1950s, to a man named Ancel Benjamin Keys, a scientist at the University of Minnesota. Dr. Keys was formidable persuasive and, through sheer force of will, rose to the top of the nutrition world—even gracing the cover of Time magazine—for relentlessly championing the idea that saturated fats raise cholesterol and, as a result, cause heart attacks.

This idea fell on receptive ears because, at the time, Americans faced a fast-growing epidemic. Heart disease, a rarity only three decades earlier, had quickly become the nation’s No. 1 killer. Even

http://online.wsj.com/news/articles/SB10001424052702303678404579533760760461486
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Is dietary carbohydrate essential for human nutrition?
Eric C Westman

Dear Sir:

I read with interest the article by Dewailly et al (1) regarding diet and cardiovascular disease in the Inuit of Nunavik, but I was disappointed that no information regarding macronutrient intake was presented or considered in the estimation of cardiovascular risk. The traditional Inuit diet consists primarily of protein and fat, somewhat similar to the low-carbohydrate diets promoted in popular weight-reducing diets (2). These diets have caused concern among nutritionists because of the metabolic changes and health risks associated with limited carbohydrate consumption (3). However, in exploring the risks and benefits of carbohydrate restriction, I was surprised to find little evidence that exogenous carbohydrate is needed for human function.

The currently established human essential nutrients are water, energy, amino acids (histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine), essential fatty acids (linoleic and α-linolenic acids), vitamins (ascorbic acid, vitamin A, vitamin D, vitamin E, vitamin K, thiamine, riboflavin, niacin, vitamin B-6, pantothenic acid, folic acid, biotin, and vitamin B-12), minerals (calcium, phosphorus, magnesium, and iron), trace minerals (zinc, copper, manganese, iodine, selenium, molybdenum, and chromium), electrolytes (sodium, potassium, and chloride), and ultratrace minerals (4). (Note the absence of specific carbohydrates from this list.)

Although one current recommended dietary carbohydrate intake for adults is 150 g/d, it is interesting to examine how this recommendation was determined at a recent international conference (5):

"The theoretical minimal level of carbohydrate (CHO) intake is zero, but CHO is a universal fuel for all cells, the cheapest source of dietary energy, and also the source of plant fiber. In addition, the complete absence of dietary CHO entails the breakdown of fat to supply energy [glycerol as a gluconeogenic substrate, and ketone bodies as an alternative fuel for the central nervous system (CNS)], resulting in symptomatic ketosis. Data in childhood are unavailable, but ketosis in adults can be prevented by a daily CHO intake of about 50 g. This value appears to approximate the quantity of glucose required to satisfy minimal glucose needs of the CNS and during starvation. The Group therefore concluded that the theoretical minimum intake of zero should not be recommended as a practical minimum.... about 100 g of glucose/d are Irreversibly oxidized by the brain from the age of 3-4 y onward. However, this excludes recycled carbon, gluconeogenic carbon, for example from glycerol, and it does not account for glucose used by other non-CNS tissues. For example, in the adult, muscle and other non-CNS account for an additional 20-30 g of glucose daily. For this reason a safety margin of 50 g/d is arbitrarily added to the value of 100 g/d and the practical minimal CHO intake set at 150 g/d beyond the ages of 3-4 y."

Thus, although carbohydrate could theoretically be eliminated from the diet, the recommended intake of 150 g/d ensures an adequate supply of glucose for the CNS. However, it appears that during starvation (a condition in which the intakes of carbohydrate, protein, and fat are eliminated), an adequate amount of substrate for the CNS is provided through gluconeogenesis and ketogenesis (6). The elimination of dietary carbohydrate did not diminish the energy supply to the CNS under the conditions of these experiments. Second, carbohydrate is recommended to avert symptomatic ketosis. In the largest published series on carbohydrate-restricted diets, ketosis was not typically symptomatic (7).

The most direct way to determine whether carbohydrate is an essential nutrient is to eliminate it from the diet in controlled laboratory studies. In studies involving rats and chicks, the elimination of dietary carbohydrate caused no obvious problems (6-12). It was only when carbohydrate restriction was combined with glycerol restriction (by substituting fatty acids for triacylglycerol) that chicks did not develop normally (13). Thus, it appears that some minimum amount of a gluconeogenic precursor is essential—for example, glycerol obtained from fat (triacylglycerol) consumption. More subtle abnormalities from carbohydrate elimination might not have been observed in these studies. In addition, the
Professor uses 1000 mice to expose food folly

AAP NOVEMBER 21, 2013 12:00AM

BELIEF that single nutrients such as omega-3s, sugar or salt can cure or cause all ills is folly, says a leading health scientist.

The key, Stephen Simpson says, is for people to think about food as food and to seek a healthy balance between protein, carbohydrates and fat.

Too much of one for too long can make you fat and unhealthy, or even thin and unhealthy, says Professor Simpson, academic director of the new $500 million Charles Perkins centre set up at the University of Sydney to fight obesity, diabetes and cardiovascular disease.

“The balance really matters,” he told colleagues at an Australian Society for Medical Research conference in Victoria.

His team conducted a study in which 1000 mice were fed 30 different diets with different ratios of protein, carbohydrates and fat.

“If you want to lose weight as a mouse, you go onto a high-protein diet. But if you stay on that too long you will have poor circulating insulin and glucose tolerance.

“If you go too low on protein, you will drive over-consumption and be prone to obesity.”

A good balance for a mouse is about 20 per cent protein, about 60 per cent carbohydrates and about 20 per cent fat.

“And mice are not that different from humans,” he said.

An interesting finding was that a low-protein diet coupled with high carbohydrates led to obesity. But these mice lived longest and had a healthy balance in their gut.

Professor Simpson said he was concerned about the emphasis on micronutrients such as vitamins, sugar and salt.

“It is unhelpful when people argue everything is the fault of sugar or fat or salt or whatever when what we are dealing with is a balancing problem.”

The best type of carbohydrates and fat is limited amounts of sugar and complex, low GI, hard-to-digest foods.

Professor Simpson said healthy fats such as omega-3 were also important.

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Abstract

Objective: To describe the nutritional quality of community-level diets in remote northern Australian communities.

Design, setting and participants: A multisite 12-month assessment (July 2010 to June 2011) of community-level diet in three remote Aboriginal communities in the Northern Territory, linking data from food outlets and food services to the Australian Food and Nutrient Database.

Main outcome measures: Contribution of food groups to total food expenditure; macronutrient contribution to energy and nutrient density relative to requirements; and food sources of key nutrients.

Results: One-quarter (24.8%; SD, 1.4%) of total food expenditure was on non-alcoholic beverages; 15.6% (SD, 1.2%) was on sugar-sweetened drinks. 2.2% (SD, 0.2%) was spent on fruit and 5.4% (SD, 0.4%) on vegetables. Sugars contributed 25.7%–34.3% of dietary energy, 71% of which was table sugar and sugar-sweetened beverages. Dietary protein contributed 12.5%–14.1% of energy, lower than the recommended 15%–25% optimum. Furthermore, white bread was a major source of energy and most nutrients in all three communities. Mean: 61% carbs, including ~24% refined sugar.

Conclusion: Very poor dietary quality continues to be a characteristic of remote Aboriginal community nutrition profiles since the earliest studies almost three decades ago. Significant proportions of key nutrients are provided from poor-quality nutrient-fortified processed foods. Further evidence regarding the impact of the cost of food on food purchasing in this context is urgently needed and should include cost–benefit analysis of improved dietary intake on health outcomes.

Dietary Improvement for Indigenous Australians is a priority strategy for reducing the health gap between Indigenous and non-Indigenous Australians. Poor-quality diet among the Indigenous population is a significant risk factor for three of the major causes of premature death — cardiovascular disease, cancer and type 2 diabetes. The 26% of Indigenous Australians living in remote areas experience 40% of the health gap of Indigenous Australians overall. Much of this burden of disease is due to extremely poor nutrition throughout life.

Comprehensive dietary data for Indigenous Australians are not available from national nutrition surveys or any other source. Previous reports on purchased food in remote Aboriginal communities are either dated, limited to the primary store and/or short-term or cross-sectional in design. These studies have consistently reported low intake of fruit and vegetables, high intake of refined cereals and sugars, excessive sodium intake, and limited availability of several key micronutrients.

The aim of this study was to examine characteristics of the community-level diet in remote communities in the Northern Territory over a 12-month period.

Methods

We examined purchased food in three remote communities in relation to:

- food expenditure;
- estimated per capita intake;
Aboriginal and Torres Strait Islander adults experience diabetes 20 years earlier than non-Indigenous adults

Aboriginal and Torres Strait Islander adults are more than three times as likely as non-Indigenous adults to have diabetes, and they experience it at much younger ages, according to new figures released by the Australian Bureau of Statistics today.

"Results from the largest ever biomedical collection for Aboriginal and Torres Strait Islander adults, which collected information on a wide range of chronic diseases and nutrition, reveal that diabetes is a major concern," said Dr Paul Jelfs from the ABS.

"The voluntary blood test results showed that in 2012–13, one in ten Aboriginal and Torres Strait Islander adults had diabetes. This means that, when age differences are taken into account, Aboriginal and Torres Strait Islander adults were more than three times as likely as non-Indigenous adults to have diabetes."

"What was even more striking was how much earlier in life Aboriginal and Torres Strait Islander adults experience diabetes. In fact, the equivalent rates of diabetes in the Aboriginal and Torres Strait Islander population were often not reached until 20 years later in the non-Indigenous population." said Dr Jelfs.

The survey revealed that diabetes was twice as common among Aboriginal and Torres Strait Islander adults living in remote areas. Around one in five in remote areas had diabetes compared with around one in ten in non-remote areas.

Also of interest was the fact that many Aboriginal and Torres Strait Islander adults with diabetes also had signs of other chronic conditions.

"More than half of all Aboriginal and Torres Strait Islander adults with diabetes also had signs of kidney disease. This compared with a third of non-Indigenous adults with diabetes", said Dr Jelfs.

"Given these findings, it is not surprising that the death rate for diabetes among Aboriginal and Torres Strait Islander people is seven times higher than for non-Indigenous people."

Other results released today suggest that many Aboriginal and Torres Strait Islander adults may not be aware they have high cholesterol, with one in four having high cholesterol levels, yet only one in ten being aware they had it.

Further information is available in Australian Aboriginal and Torres Strait Islander Health
SUGAR, HEART DISEASE AND STROKE

FACTS

- Heart disease and stroke are leading causes of death in Canada, responsible for 27.3% of all deaths. Over 1.3 million Canadians are living with heart disease and 315,000 Canadians are living with the effects of stroke.

- More than 60% of Canadian adults and 31% of children and youth aged 5 to 17 years are overweight or obese. Children who are obese are at increased risk of remaining overweight or obese as adults.

- Up to 80% of early heart disease and stroke can be prevented through adopting healthy behaviours including eating a healthy diet.

- Sugar is a carbohydrate that provides energy to the body. Other than providing energy, sugar has no other nutritional benefits.

- Sugar can occur naturally in milk, fruit, vegetables, starches, grains and most plant-based foods. Sugars can also be added to foods and drinks for flavour, as a sweetener, as a preservative or to enhance the texture of products.

- Free sugars include all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices.

- It is estimated that Canadians consume as much as 13% of their total calorie intake from added sugars. This added sugar estimate does not take into account the broader range of sugars captured by free sugars (which also include 100% fruit juice, honey, etc.). Consumption of free sugars among Canadians would be higher than 13%.

- Ten percent of total energy (calories) from free sugars in a 2,000-calorie-a-day diet is equivalent to about 48 grams (roughly 12 teaspoons) of sugar. Five percent of total energy is equivalent to about 24 grams (roughly 6 teaspoons) of sugar.

- Excess sugar consumption is associated with adverse health effects including heart disease, stroke, obesity, diabetes, high blood cholesterol and cancer and dental caries.

- Individuals who consume greater than or equal to 10% but less than 25% of total energy (calories) from added sugar have a 30% higher risk of death from heart disease or stroke when compared to those who consume less than 10%. For those who consume 25% or more of calories from added sugar, the risk is nearly tripled.

- While there are a variety of causes of obesity, researchers speculate that excess caloric intake may be the single largest driver. Larger portion sizes contribute to over consumption of calories and excess body weight.

- Sugar-sweetened beverages (SSBs) are the single largest contributor of sugar in the diet. A single 355 mL can of sugar-sweetened soda contains up to 40 grams (about 10 teaspoons) of sugar and no health benefits.

- The total volume of SSBs available to Canadians is 3.5 billion litres, the equivalent of 110 L per person per year or over 300 mL per day. A standard sized soft drink can is 355 mL.

- As children get older, they consume more sugar from soft drinks. Boys' average daily consumption of regular soft drinks is 68 grams at ages 4 to 8 years and increases to 76 grams at ages 14 to 18 years. Among girls the increase is from 47 to 179 g.

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