Journal of Scientific Exploration, Vol. 18, No. 1, pp. 83–134, 2004 0892-3310/04

REVIEW ESSAY

Low-Carbohydrate Diets

JOEL M. KAUFFMAN

University of the Sciences in Philadelphia
600 South 43rd Street, Philadelphia, PA 19104

Dr. Atkins' *New* Diet Revolution (3rd ed.) by Robert C. Atkins, MD.

Life Without Bread: How a Low-Carbohydrate Diet Can Save Your Life by Christian B. Allan, PhD and Wolfgang Lutz, MD.

Dr. Bernstein's Diabetes Solution (revised ed.) by Richard K. Bernstein, MD.

Dangerous Grains: Why Gluten Cereal Grains May be Hazardous to Your Health by James Braly, MD and Ron Hoggan, MA.

The Protein Power LifePlan by Michael R. Eades, MD and Mary Dan Eades, MD.

Eat Fat Get Thin! by Barry Groves.

Homo Optimus by Jan Kwasniewski, MD and Marek Chylinski with Engl. transl. by Bogdan Sikorski, PhD.

The Heart Revolution: The Extraordinary Discovery that Finally Laid the Cholesterol Myth to Rest by Kilmer S. McCully, PhD, MD and Martha McCully.

The No-Grain Diet by Joseph Mercola, DO with Alison Rose Levy.

The Modern Nutritional Diseases: heart disease, stroke, type-2 diabetes, obesity, cancer, and how to prevent them by Alice Ottoboni, PhD and Fred Ottoboni, MPH, PhD.

Enter the Zone by Barry Sears, PhD and Bill Lawren.

Going Against the Grain: How Reducing and Avoiding Grains Can Revitalize Your Health by Melissa Diane Smith.

"There is no nonsense so arrant that it cannot be made the creed of the vast majority by adequate governmental action."

Bertrand Russell, *An Outline of Intellectual Rubbish*, 1950
(Ottoboni & Ottoboni, 2003: 173).

Abstract—For about the past 50 years, most government agencies in many countries and many non-governmental associations have recommended high-carbohydrate diets, and restricted intake of fats, especially animal fats, because of their content of saturated fats and cholesterol, as well as plant oils high in saturates. This campaign was intensified in the past 20 years, coinciding with a massive increase in the proportion of people in the USA and UK who are overweight, obese, or diagnosed with adult-onset diabetes. The simple rationales and supposedly scientific evidence for such advice is shown to be flawed and, at no time in the past 50 years, have diet experts had a consensus. Reasoned objections to high-carbohydrate low-fat diets co-existed for the entire period; but these were overwhelmed by the financial power of the high-carbohydrate advocates.

Conversely, other sources from the medical and nutritional literature, based on studies and trials, show benefits of low-carbohydrate diets, both in weight loss and in improved values in conventional tests for hypertension, ''hyperlipidemia,'' and inflammation. This is often found to be the case in the actual data, even in papers on studies designed to provide evidence of the opposite. The conclusions in the abstracts of many such papers often do not match their actual data. Many studies also show that adherence to low-carbohydrate diets is better than for other types of diet.

The 20 authors of the 12 books reviewed herein have recommended low-carbohydrate diets, some with no restrictions on intakes of fat and/or protein. Not only do all of the books have a plausible biochemical rationale for their advice, but they present histories of extremely long adherence to low-carbohydrate diets by individuals, many with medical degrees, with good health being the result. Clinical observations by 9 of the authors who have extensive experience treating obese and diabetic patients support the value of low-carbohydrate diets. Many other advantages of low-carbohydrate diets are shown by improvements in a diverse area of afflictions, from Crohn's and celiac diseases, to cancer, multiple sclerosis, and arthritis. Even the performance of athletes who believed they were better served by high-carbohydrate diets was improved with low-carbohydrate diets. However, many other aspects of diet advice in these books are not in agreement, and a majority of them contain technical errors.

This review essay also shows that the energy content of specific foods, as determined by burning them, has little relation to their varied levels of digestibility and actual energy content as foods. The misguided diet stance of agencies and foundations is shown to have led to food labels which are misleading.

Low-carbohydrate diets, even if maintained for decades, have been demonstrated to be safe and effective. Nevertheless, they are still considered to be alternative, and thus targets of attack by certain associations and governmental agencies.

Keywords: diet—calories—carbohydrate—fat—lipids—cholesterol—protein—obesity—diabetes—insulin—atherosclerosis—stroke—cancer—arthritis—gluten—celiac disease

Introduction

One of the fiercest battles in medicine is about nutritional advice on which diets will best preserve and prolong health, especially to curb obesity and related conditions. Now we are living in an advice war on how much

carbohydrate (carb) should be in our diets. Seemingly every government agency and private association recommends high-carb diets (more than 50% of metabolic energy from carbs, the rest from fat and protein), while every book examined in this review recommends the opposite! For the past half-century, high-carb diets have been recommended in order to limit intakes of fat and cholesterol in the simplistic belief that eating fat makes one fat, and that eating cholesterol leads to atherosclerosis (also referred to as cardiovascular disease, CVD). Also, these agencies and associations control the mass media and the major sources of research funding; some results of such control will be discussed below. True, there are many books, articles, pamphlets, and broadcasts that adhere to the mainstream view; this will be shown to be due to confirmation bias.

A previous book review in the *Journal of Scientific Exploration* addressed the lack of evidence for the concerns over eating saturated fat and cholesterol (Kauffman, 2001). This review essay will attempt to further resolve the controversy by examining the rationales and the results obtained in books authored by those recommending low-carb diets, as well as the results of controlled trials in the medical literature. This is followed by reviews of the individual books, which were chosen because all contain recommendations for low-carb diets and were written for educated readers.

In order to limit the number of citations to individual research papers, a citation without a date of publication will refer to one of the books under review. In most cases, the book will have cited the original peer-reviewed articles.

For definitions, composition of, and physical and chemical changes in food, an excellent textbook exists (de Man, 1999), which is without bias or judgment or recommendations. For example, it explains that the technical term for ''complex carbohydrates'' is polysaccharides (de Man, 1999: 183–206). Most are polymers of glucose (a monosaccharide, also called dextrose, blood sugar, grape sugar, and corn sugar [not to be confused with corn syrup]), which is a distinct entity different from fructose (fruit sugar) or sucrose (common table sugar or cane sugar).

The quantities of carbs in food, as shown on food labels, are not assayed directly, as are fat, protein, ash, or water, but are calculated by difference, so these quantities are not so accurate (Atkins, 2002: 173). All carbs are not alike, either in digestibility or in their effects on blood glucose and insulin levels.

Some indigestible polysaccharide amounts are listed on US and UK labels as fiber, or fibre, or dietary fiber, for which no calories are counted. This is what is also called ''crude dietary fiber'' and was determined by an older simple assay. This method missed soluble fiber, which is 2–16 times greater (de Man, 1999: 203–206), thus the caloric content for carbs on US food labels tends to be too high, because the quantity of soluble fiber is not listed, nor is it totally digested. The caloric values of the main food groups were originally determined by oxidizing foods in a bomb calorimeter under pressurized oxygen to form nitric

acid, water, and carbon dioxide, thus the honest older designation for the caloric values was "fuel values" (Atwater & Snell, 1903). These simple end-products are not all that is formed in our bodies, except from digestible carbs. The older caloric values were: carb, 4.0 kcal/g; fat, 9.3 kcal/g; and protein, 5.25 or 5.6 kcal/g (Wiley & Bigelow, 1898). Now it is understood that the net metabolizable energy (NME) from carbs in humans ranges from 0 to 2 kcal/g from fiber (Livesy, 2001) through 3.9 for sucrose to 4.2 for starches (Ottoboni & Ottoboni, 2003: 79). For example, the US Food and Drug Administration (FDA) food label on celery stalks gives 15 "calories" (should be kcal) per 65 g serving, while the Geigy Tables give 11 kcal (Lentner, 1981: 246), which is 38% lower. Natural fats have different digestibilities, so their NME ranges from 5.5 to 8.5 kcal/g (see Table 1). Proteins have been recognized to have an NME of 4.1 kcal/g (Kekwick & Pawan, 1969). So, for clarity, most of the discussion below is based on the conventional values of carb, 4; fat, 9; and protein 4, knowing that 2 of 3 are usually wrong.

The glycemic index (GI) is the effect of a food on serum glucose level compared with the effect of glucose as a standard with a value of 100. Food values range from 0 (fat) to 115 for tofu non-dairy frozen dessert. "Several prospective observational studies have shown that the chronic consumption of a diet with a high glycemic load (GI $\times$ dietary carbohydrate content = GL) is independently associated with an increased risk of developing type 2 diabetes, cardiovascular disease, and certain cancers" (http://wave.prohosting.com/rmendosa/gi.htm; Foster-Powell et al., 2002).

Non-insulin-dependent diabetes mellitus (NIDDM) is a result of excessive carb consumption, which leads to excessive insulin production in people genetically so disposed; it is also called adult-onset or Type-II diabetes. Milder cases are called insulin resistance or Syndrome X. IDDM is a result of destruction of most of the insulin-producing beta cells of the pancreas, and injected insulin is an essential component of successful treatment. "Prior to the availability of insulin ... [in 1925] ... people with Type I diabetes usually died within a few months of diagnosis. Their lives could be prolonged somewhat with a diet that was very low in carbohydrate and usually high in fat. Sufferers from ... Type II diabetes frequently survived on this type of diet ... When I became diabetic [IDDM in 1947] ... many people were still following very low carbohydrate, high-fat diets" (Bernstein, 2003: 310). About this time it was noted that the induction of "atherosclerosis" in vegetarian animals dosed with animal fats led to the assumption that the complications of diabetes were caused by high fat diets. "Therefore, I and many other diabetics ... [were] treated with a high-carbohydrate, low-fat diet ... as adopted by the AD[b]A, AHA and other groups around the world. On the new diet many of us had even higher serum cholesterol levels, and still developed the grave complications of diabetes. Seemingly unaware of the importance of blood sugar control, the AD[b]A raised the recommended carbohydrate content to 40% of calories, and then more recently to 60%" (Bernstein, 2003: 310).

TABLE 1
Calculated[a] Fuel Value (9 kcal/g) vs. Actual Caloric Availability of Fats in Rodents or Other Animals

Type of fat or oil	Caloric availability (kcal/g) (NME)
Cocoa butter	5.7[b]
	5.5[c]
	6.5[d]
	6.8[f]
Coconut oil	7.8[e]
Corn oil	8.5[e] (humans)
Lard	8.3[f]
	8.4[g] (pigs)
	8.5[h] (turkeys)
	7.9[i] (chickens)
	8.4[j] (chickens)
Tallow	7.3[f]
	5.9[e] (pigs)
	6.5[g] (turkeys)
	7.1[h] (chickens)
	7.4[k] (chickens)
	7.7[e]

[a] *CRC Handbook,* 35th ed., 1953–1954, p. 1794.
[b] Chen et al., 1989.
[c] Apgar et al., 1987.
[d] Hoagland & Snider, 1942.
[e] Finley et al., 1994a.
[f] Finley et al., 1994b.
[g] Carlson & Bayley, 1968.
[h] Whitehead & Fisher, 1975.
[i] Peterson & Vik-Mo, 1968.
[j] Crick et al., 1988.
[k] De Schrrijver et al., 1991.

Recommendations and Evidence *Against* Low-Carb Diets

Official Recommendations

"With nearly 70,000 members, the American Dietetic Association [ADA, here ADtA] is the nation's largest organization of food and nutrition professionals. The ADtA serves the public by promoting optimal nutrition, health and well-being." The ADtA endorses the Food Guide Pyramid of the United States Department of Agriculture (USDA) unequivocally, thus recommending high-carb diets with 75% carb, 10% fat, and 15% protein. None of the books under review here appear on the ADtA reading list (http://www.eatright.org/Public/NutritionInformation/92_fgp.cfm).

In USDA Bulletin No. 42 it is written that "... as the chief function of the fats and carbohydrates is to serve as fuel, it is of more importance that they should be in sufficient amount than that they should be in definite relative proportions to each other" (Williams, 1907). How times change! The latest Food Guide Pyramid now has carbs in 5 different places, and seemingly recommends

20 servings of them to just 1 serving of fats (http://www.nal.usda.gov.8001/py/pymap.htm).

The American Diabetes Association (ADbA) recommends 60% high-GI carbs in the diet without reservation: "The message today: Eat more starches! It is healthiest for everyone to eat more whole grains, beans, and starchy vegetables such as peas, corn, potatoes and winter squash. Starches are good for you because they have very little fat, saturated fat, or cholesterol. They are packed with vitamins, minerals, and fiber. Yes, foods with carbohydrate—starches, vegetables, fruits, and dairy products—will raise your blood glucose more quickly than meats and fats, but they are the healthiest foods for you. Your doctor may need to adjust your medications when you eat more carbohydrates. You may need to increase your activity level or try spacing carbohydrates throughout the day. On average, Americans eat around 40–45% of our calories as carbohydrate. This is a moderate amount of carbohydrate, not high. Currently some controversy about carbohydrates is raging due to a number of new diet books. These books encourage a low carbohydrate, high protein and moderate fat intake. These diets are not in synch with the American Diabetes Association nutrition recommendations, which are based on years of research and clinical experience. In addition, these trendy diets are hard to follow year after year" (http://www.diabetes.org/main/health/nutrition/foodpyramid/starches.jsp).

The FDA recommends high-carb diets with caloric content of 55% carb, 30% fat (⅓ each saturated, monounsaturated, polyunsaturated), and 15% protein. The daily value percentage of each on US food labels reflects this and utilizes the conventional caloric values of foods (http://www.cfsan.fda.gov/~dms/foodlab.html). Crude fiber is counted at 4 kcal/g instead of 0, and soluble fiber is not listed separately, or counted at 0–2 kcal/g, as it should be (Livesy, 2001).

The National Cholesterol Education Program (NCEP) sponsored by the National Heart, Lung and Blood Institute (NHLBI) of the National Institutes of Health (NIH), recommends high-carb diets with caloric content the same as those of the FDA. Many high-GI foods are recommended as well as margarine, mayonnaise, and vegetable oils, including for diabetics (www.nhlbisupport.com).

The American Heart Association (AHA) recommends the use of a food pyramid with about the same caloric content from each of the food groups as in the USDA pyramid. Differences are that the AHA recommends no egg yolks at all, and otherwise to avoid saturated fat and cholesterol intake at all costs; also, the positions of some foods are changed. The AHA favors small amounts of soft margarine, and large amounts of milk and low-fat milk and other dairy products, with no exceptions for diabetics that are apparent on the website. On their new web pages for diabetics: "Type 2 diabetes is a progressive disease that develops *when the body does not produce enough insulin* and does not efficiently use the insulin it does produce (a phenomenon known as insulin resistance) . . ." (Italics added). The AHA notes that the World Health Organization (WHO) Study Group recommends that 15% of total calories be derived from fat, and is concerned that certain key nutrient levels will not be met in certain population

groups at this level. The AHA still quotes the work of Ancel Keys, stating that the consumption of saturated fat in the diet is the best predictor of heart disease (www.americanheart.org). The overall diet recommendations of the AHA, the ADtA, and other groups were published as the result of a conference (Deckelbaum et al., 1999).

The American Medical Association (AMA) clearly recommends a low-fat diet (http://www.ama-assn.org/ama/pub/article/4197-6132.html).

Health Canada recommends very high-carb diets with caloric content of 78% carb, 9% fat, and 13% protein. The Food Rainbow (instead of a pyramid) favors grains, then fruits and vegetables without regard to GI or exceptions for diabetics, followed distantly by "Other Foods" such as milk products, meats, and meat substitutes such as beans and peanut butter! (http://www.hc-sc.gc.ca/hpfb-dgpsa/onpp-bppn/food_guide_rainbow_e.html)

In 1983–1984, 2 UK committees, the National Advisory Committee for Nutrition Education and the Committee on the Medical Aspects of Food Policy, recommended that the intake of fats be reduced "to prevent heart disease" (Groves, 1999: 25). Based on the food label from Walkers Ready Salted Crisps, the anonymous nutrition professionals of the UK recommend for average adults a daily fat-calorie consumption of 3.5% for women and 3.8% for men.

Evidence Presented

As early as the 1860s, in reaction to the success story of a low-carb diet (see below), Dr. Felix Niemeyer of Stuttgart, on fallacious thermodynamic grounds, recommended a high-protein diet with both carb and fat restricted, which is still the basis of some slimming diets to this day (Groves, 2003: 19).

Sir William Osler, an immensely influential professor of medicine about 100 years ago in both the USA and UK, "recognized" that atherosclerosis and angina pectoris were "afflictions of the better classes, not of the working classes," and ascribed the afflictions to consumption of eggs and milk rather than cereals, vegetables, fruit, fish, and meat. Atherosclerosis was not found in wild animals, but in lab animals fed cholesterol around 1911–1914. The writer of this bit of history, William Dock, MD, was positive that a diet with eggs, butter, and cream killed ⅓ of all physicians (Dock, 1953). Of course, these observations influence nutrition to this day, but none has stood the test of time. For example, Osler's observations did not discern whether "the better classes" manifested their afflictions at greater ages than those of "the working classes." Even now, despite the leveling effects of national health services, the life expectancy of professional people in both the UK and Finland is 7.2 years longer than that of unskilled laborers (Cohen, 2003).

The Diet-Heart Theory (DHT) was said to have begun in earnest in 1950 when Gofman et al. (Mann, 1977) described a method for isolating the lipoproteins of blood by ultracentrifugation, and made 2 proposals: (1) that the concentration of

serum low-density lipoprotein (LDL) was a useful index of atherogenesis, and (2) that the concentration could be influenced meaningfully by diet (Mann, 1977). Even though low-carb diets for diabetes were found beneficial at least since 1800 (Allan & Lutz, 2000: 7), the ADbA abandoned them around 1950 without a single trial (Atkins, 2002: 282). By 1977 it was already known that the DHT was false, that total cholesterol (TC) was no better a predictor than LDL, and that high-density lipoprotein (HDL) concentration was inversely related to the incidence of coronary heart disease (CHD) (Mann, 1977).

The DHT was inflated by Prof. Ancel Keys in 1953, who eventually published a graph showing that CHD was correlated with available food fat in 6 (later 7) countries, while ignoring data then available from the WHO on 16 other countries that did not fall on his graph. Moreover, the variation in CHD rates of 5-fold by location within 2 of the countries was not mentioned or resolved (Allen & Lutz, 2000: 76–82; Groves, 1999: 60–61; Mann, 1977; Ravnskov, 2000: 15–19).

Sparked by Keys' earlier reports, in the mid-1950s, the AHA contracted with a team of investigators to evaluate evidence that the fat content of the North American or European diets caused CVD. The report was submitted in 1957 with the conclusions that there was nothing to support the DHT. The AHA could have changed its direction or clung to the DHT with greater fervor; it did the latter after 4 years, beginning in 1961, recommending that *all* Americans reduce their consumption of fat and cholesterol (Smith & Pinckney, 1991: 8).

By 1970 the Framingham Study, begun in 1948, had examined about 1000 people by dietary recall; it was reported that there was no correlation between dietary saturated fat or cholesterol intake and measured serum cholesterol (Groves, 1999: 61–62; Mann, 1977). After 30 years of follow-up, the AHA and NHLBI reported in 1990 in *The Cholesterol Facts* that "The results of the Framingham Study indicate that a 1% reduction . . . of cholesterol [corresponds to a] 2% reduction in CHD risk." But in their *JAMA* paper based on the same data, the authors wrote that those [subjects] whose cholesterol had *decreased* by itself during those 30 years ran a greater risk of dying than those whose cholesterol had *increased*. "For each 1 mg/dL *drop* of cholesterol there was an 11% *increase* in coronary and total mortality" (Anderson et al., 1987; Ravnskov, 2000: 57–58).

Beginning in 1972 the Multiple Risk Factor Intervention Trial (MRFIT), sponsored by the NHLBI, was a randomized clinical trial (RCT), potentially the most scientific sort of comparative study. Of 12,866 men aged 35–57 said to be at high risk of CVD, of the approximately 361,662 who were screened, men were excluded who were at "low risk." Those at very high risk, namely men who had a previous heart attack, angina, diabetes, TC >350 mg/dL, diastolic BP >115 mm Hg, high body mass index (BMI), or treatment with most common drugs, were actually excluded as well! Subjects in the intervention group were asked to eat much less animal fat (high-carb diet) and to smoke less, and they were reported to have a non-significantly lower death rate from CVD after

a mean follow-up of 7 years (MRFIT Research Group, 1982). However, the overall mortality rate in the intervention group was slightly higher due to more cancer. Since smoking does increase cancer rates, one must conclude that eating animal fat protected against cancer (Allan & Lutz, 2000: 84–85). The Lipid Research Clinics trial, reported in 1983, involved similar low-fat diets in both control and intervention groups (Ravnskov, 2000: 171–172), thus the outcome of this huge study is not relevant to this review.

These and other trials moved the NHLBI/AHA to hold a Consensus Development Conference in December, 1984. A 14-member panel and 600 attendees showed considerable disagreement on the effect of diet on TC levels and the frequency of CVD supposedly associated with high levels of TC or LDL. Written before the meeting (Enig, 2003) a statement, quickly incorporated into a press release, and claimed to be a result of consensus, proclaimed that "the blood cholesterol of most Americans is undesirably high [>200 mg/dL], in a large part because of our high intake of calories, saturated fat, and cholesterol." This became the genesis of the NCEP, which was quickly supported by the AMA, Kellogg, Merck, and American Home Products (Moore, 1989: 56–81).

The New Cholesterol Guidelines, approved by the NCEP, and released to the public on May 15, 2001, ignore the adverse effects of excess dietary carbs as well as the imbalance of essential fatty acids created by their recommendation of vegetable oils instead of animal fat. Despite the acknowledgement that the low-fat diets recommended and used by many for 40 years exacerbated obesity and NIDDM, the new recommendation was to intensify the use of the failed diets! (Ottoboni & Ottoboni, 2003: 177–182). In a meta-analysis of 9 RCTs on low-fat (high-carb) diets, both CVD and total mortality were unchanged in 7 trials, up in 1 and down in another. Another meta-analysis supported "... a central role of dietary fat intake in the causation of CVD ...," but to reach this conclusion the authors included a trial with more heavy smokers in the control group, and excluded the trial with the most contradictive result (Ravnskov, 2002a).

Evidence *against* the value of low-fat diets has been attacked at least twice by W. Scott Grundy, MD. As a spokesman for the AHA, he cited 10 studies in support of the AHA's diet recommendations in 1982. On close examination, 6 of the 10 studies used inappropriate liquid formula diets, 1 failed to report the effect of its diet on TC, 2 by Ancel Keys reported almost no effect of dietary cholesterol on serum cholesterol levels, and the final study indicated a *decrease* in serum cholesterol with the *addition* of dietary cholesterol (Smith & Pinckney, 1991: 188–189). More recently, an article by Gary Taubes in *Science,* critical of the NCEP's low-fat recommendation, was attacked by Grundy, who wrote that many studies showed that saturated fatty acids as a class, compared with unsaturated fatty acids and *carbohydrate,* raise serum LDL and thus cause CVD. Grundy cited 2 reviews in support. In one there are no references. In the other, of which Grundy is a co-author, there are 12 references used as support. On close

examination, none of the 12 were really supportive, and some did not even directly address his issue (Ravnskov et al., 2002).

In a study partially funded by the AMA, a prodigious literature review on articles in English on low-carb diets was performed by use of MEDLINE and other searches for those published between January 1, 1966, and February 15, 2003. All 2609 potentially relevant articles were perused, excluding all but 107 articles on 94 studies on 3268 subjects receiving 0–901 g/day of carbs for 4–365 days, but the reasons for exclusion of so many of the trials were obscure. Only 5 studies (non-randomized and no control groups) lasted more than 90 days. ''These [low-carb] diets have not been adequately evaluated for use longer than 90 days, or for individuals aged 53 years or older, or for use by participants with hyperlipidemia, hypertension, or diabetes'' (Bravata et al., 2003). Only books by Atkins, Sears, and Heller were cited, and incorrectly said to recommend more protein and less carb, where, in fact, more fat was also recommended. The abstract's conclusions verbatim are ''There is insufficient evidence to make recommendations for or against the use of low-carbohydrate diets, particularly among participants older than age 50 years, for use longer than 90 days, or for diets of 20 g/d or less of carbohydrates. Among the published studies, participant weight loss while using low-carbohydrate diets was principally associated with decreased caloric intake and increased diet duration but not with reduced carbohydrate content'' (Bravata et al., 2003). Of the 2 main low-carb groups into which the trials were divided, the $\leq$60 g/day group's mean intake of carb was 29 g/day, and the total energy intake all foods was 1446 kcal/day (fuel values). In the $>$60 g/day group, the mean intake of carb was 236 g/day, and total intake of all foods was 1913 kcal/day (their table 3). In the books reviewed below, any intake over about 150–200 g/day of total carb would not be considered low-carb. In the true low-carb group, the mean weight loss in trials was 17 kg, while in the higher-carb group it was 2 kg (their table 5). Bravata et al. (2003) do not consider this significant and attribute the result to the lower total caloric intake. This view has been falsified in several studies. For example, controlled trials in a hospital have shown that a diet of just 1000 kcal/day which is 90% carb led to weight gain, and intakes of 1000–2600 kcal/day with a very low carb content led to weight loss (Groves, 1999: 21–22). Low-carb diets had no adverse effects on serum lipid, fasting serum glucose, fasting insulin levels, or blood pressure, according to Bravata et al. (2003).

Recommendations and Evidence *Favoring* Low-Carb Diets

Individual Observations

Is there evidence for the safety and efficacy of low-carb diets? The examples following are all of multi-year observations that support the thesis that low-carb diets are safe and effective for long periods and promote quality-of-life. These are followed by the results of short-term RCTs. While the RCT is supposed to be

the ultimate for making medical decisions, it is not the only source of acceptable data. The differences in outcomes observed among study centers in a number of multi-center drug RCTs is so extreme as to question their lack of bias (O'Shea & DeMets, 2001). It is true that RCTs of diet of durations longer than a few months are scarce. It is true that clinical experience as in some of the examples given may be biased, but there is support for the possibility that clinical data may be less biased than some RCTs (O'Brien, 2003). It is true that RCTs of low-carb diets have not been carried beyond 1 year.

Undertaker to the rich and famous, William Banting became obese in his 30s. An eminent surgeon and friend recommended extreme exercise, which Banting carried out, only to increase his appetite, so he stopped it. Baths, cathartics, and low-calorie starvation diets did no better. At the age of 64, in 1862, Banting weighed 91 kg and was 1.65 m tall, and had failing eyesight and deafness. The famous William Harvey, MD, advised Banting to give up bread, butter, milk, sugar, beer, and potatoes. After 38 weeks on a fairly low-carb diet, Banting had lost 21 kg, felt better, and no longer had any other ailments. Delighted, he published the details at his own expense and gave away 1000 copies of his now famous *Letter on Corpulence*. He remained a normal weight until his death at age 81 after 17 years on his low-carb diet (Allan & Lutz, 2000: 12–13; Groves, 1999: 15–19).

Vilhjalmur Stefansson, MD and anthropology instructor at Harvard University (Braly & Hoggan, 2002: 204), beginning at age 27 in 1906, spent a total of 15 years with the Canadian Eskimos, including eating their obviously healthful high-fat no-carb diet, which he grew to prefer. In 1928 Stefansson and a former companion, Karsten Anderson, MD, entered Bellevue Hospital in New York for a 1-year trial of Eskimo diets. Anderson did well with an all-meat diet, but Stefansson required considerable fat to feel his best, finally settling on 80% fat and 20% protein, with a total caloric intake of 2000–3100 kcal/day. His TC went *down* about 10 mg/dL. All kinds of other tests were done, but no adverse effects of his diet were seen (Lieb, 1929; McClellan & Du Bois, 1930). In the last 6 years of his life, Stefansson returned to his Arctic diet until his death at age 83, spending a total of 22 years on such a diet (Allan & Lutz, 2000: 14–17; Groves, 1999: 20–21).

Jan Kwasniewski, MD, who ran a clinic in Poland (see book review, pg. 114, this article), has used a low-carb diet for 30 years, and is still practicing medicine at age 66. His colleague, Marek Chylinski, has used one for about 14 years, according to their book translator, Bogdan Sikorski, who has used one for 6 years and looks like a football player at age 45.

Michael R. Eades, MD and Mary Dan Eades, MD, have consumed a low-carb diet themselves for about 19 years, as well as recommending such for thousands of their patients.

Richard K. Bernstein was diagnosed with insulin-dependent diabetes mellitus (IDDM) at the age of 12 in 1946. Following the advice of the AHA and ADbA to eat a high-carb (40%, then 60%) diet, his condition worsened and most of the complications of IDDM began to appear. He found that he could not normalize

his blood sugars with any insulin regimen, and that exercise in his condition did not help. By doing a literature search himself, he realized the potential benefits of normal blood sugars. By using himself as the test animal, he found that ≈30 g/day of slow-acting carb (no simple sugars or high GI starches at all) was necessary to normalize his blood glucose levels. He obtained an MD at about age 45, partly to have his observations published in medical journals, and has continued the diet for 33 years so far, which includes 3 eggs for breakfast on many days and no fruit. His TC dropped from 300 to 179 mg/dL, of which LDL = 63 and HDL = 116. His triglycerides (TG) dropped from 250 to 45. His lipoprotein(a) became undetectable. In 1983 he began his own medical practice for diabetics. At age 70 he still works 12–14-hour days (Bernstein, 2003: xii–xx, 127).

Robert C. Atkins, MD, as a young cardiologist in 1963, gained weight rapidly and needed a hunger-free way to deal with it. Finding that a low-carb diet worked, he remained on it until his death from trauma 39 years later at age 71 (Atkins, 2002: 306).

Barry Groves, PhD, e-mailed that he has eaten a low-carb diet for 41 years and enjoys excellent health at age 67, exemplified by his status as a British and World Champion Archer.

Wolfgang Lutz, MD, who has been operating a clinic for internal medicine in Vienna, has used a low-carb diet (72 g/day) for 50 years and is still active at age 90. His colleague Christian Allan, PhD, age 42, and Christian Allan's wife, Jenny, have each used low-carb diets for 7 years and are healthy (from a recent e-mail from Allan).

Clinical Observations

Lutz followed many of his patients on low-carb diets for many years. Mean baseline fasting serum glucose levels for 15 patients with slight NIDDM was 167 mg/dL; a low-carb diet reduced the level to 125 after 6 months. (These are almost identical to the glucose level changes in your reviewer.) The glucose tolerance curves of 7 patients before and after they adopted a low-carb diet for several months were presented to show how much hyperinsulinemia was brought down (Allan & Lutz, 2000: 37–49). Both too little or too much hemoglobin in the blood is unhealthful; the latter is called polycythemia, with which the blood of the patient is more likely to clot, leading to heart attacks and strokes. Before beginning a low-carb diet, the mean hemoglobin level of 130 patients was 17.3 g/dL. After 10 months on the diet, the 40 patients still available were down to 15.8, and after 30 months the 12 patients still available were down to 15.6. Before beginning a low-carb diet, the mean systolic blood pressure in 38 patients was 194 mm Hg. After 1 week and up to 5 years on a low-carb diet, this varied between 160 and 172 for the 2–27 patients available. One of the longest-lasting fears expressed about low-carb diets is that too much protein will cause gout or kidney stones due to increased levels of uric acid in

the blood. Before beginning a low-carb diet, the mean level of uric acid in 193 patients was 7.3 mg/dL; this plunged to 5.8 after 4 months on a low-carb diet for the 38 patients available, and did not go above 6.9 during 46 months. In patients >60 years old, mean TC was 307 mg/dL before the low-carb diet and 310 25 months after; among those 40–60 years old, TC was 297 before and 280 25 months after; among those <40 years old, mean TC was 285 before and 230 8 months after. Electrocardiograms (EKGs) from 7 patients were presented and said to be improved after a low-carb diet was adopted (Allan & Lutz, 2000: 94–108). Of course, one must trust that these were not selective data.

Epidemiological Studies

According to data from the Nurses Health Study on 65,173 women aged 40–65 years and free from CVD, cancer, and diabetes at the start, who completed a detailed dietary questionnaire and were followed for 6 years, and after adjustment for age, BMI, smoking, and many other factors, those eating the highest quintile GI diet (high-carb) had a relative risk (RR) of 1.37 for 915 cases of diabetes observed. Using glycemic load, those in the highest quintile had RR of 1.47. Combined with low cereal fiber intake, the RR became 2.5; all were highly significant (Bernstein, 2003: 444; Salmeron et al., 1997). Another study on 75,521 nurses followed for 10 years found that high GL predicted CVD better ($RR = 1.57$ for highest quintile vs. lowest, adjusted for age and smoking only, $P < 0.0001$) than did high GI, which was still a better predictor than whether the carb content of the diet was simple or complex (Liu et al., 2000).

A prospective cohort study on 2286 men and 2030 women aged 40–69 and initially free of NIDDM, with a 10-year follow-up, was reported to show an inverse relation of whole-grain consumption ($RR = 0.65$) and much more so with cereal fiber intake ($RR = 0.39$) with the incidence of diabetes (Montonen et al., 2003). Many adjustments to other variables were made. The authors did not remark on the notable inverse relation of magnesium intake with diabetes that they found.

Controlled Trials

More evidence is now provided to show that the common contentions of health authorities—that all calories are alike, and that only caloric restriction will suffice for weight loss—is wrong. For this purpose, studies of shorter duration will have to serve. Water loss of 2–4 kg is generally completed within a week (Atkins, 2002: 84–85) and is not an explanation for the success of low-carb diets.

In 1956 Prof. Alan Kekwick and Gaston Pawan, MD, at Middlesex Hospital, London, England, conducted tests of 4 1000 kcal/day diets: 90% fat (by fuel values), 90% protein, 90% carb, and a normal mixed diet. Subjects on the high-fat diet lost much more weight than any of the others. Several subjects on the high-carb diet actually gained weight, even at only 1000 kcal/day! Even at 2600 kcal/day of very low-carb diet, subjects lost weight (Groves, 1999: 21–22).

Reported in 1988, a small study (n = 7) measured serum TC, HDL, TG, and fasting serum glucose after subjects with high baseline TC levels ate a diet of fatty beef, fruits, and vegetables (and no sucrose, milk, or grains) for 3–18 months (mean 8 months). Mean TC dropped from 263 to 189 mg/dL, HDL rose from 57 to 63 mg/dL (n = 6), TG dropped from 113 to 73 mg/dL, and fasting glucose dropped from 167 to 96 mg/dL (n = 2) (Newbold, 1988). Patients became ill if they ate beef without fat.

Numerous other studies confirm their findings (Atkins, 2002: 282–284; Groves, 1999: 21–22; Kauffman, 2002a; Mann, 1977), so just 5 very recent trials will be cited to demonstrate that not all calories are equal in effects on obesity, and further falsify the notion that a ''balanced diet'' is optimal.

A recent 1-year diet trial supposedly designed to evaluate the Atkins (low-carb) diet examined 63 subjects, of whom the 33 assigned to the Atkins diet were given a copy of Atkins' book as reviewed on page 103 of this article and instructed to follow it, including no restriction on the amount of fat and protein. The 30 assigned to the low-fat diet (60% carb, 25% fat, 15% protein by fuel values) were restricted to 1200–1500 kcal/day for women and 1500–1800 kcal/day for men, definitely slimming diets. All subjects met with a registered dietitian 4 times. Since registered dietitians are indoctrinated by the ADtA to promote high-carb diets, this variable was not properly controlled, since the controls would have had reinforcement (placebo effect) and low-carb subjects would not (nocebo effect, O'Mathúna, 2003). In addition, subjects were excluded if they were ill, had NIDDM, were taking lipid-lowering medications or medications that affect body weight (see Atkins book review, p. 103, this article), or were pregnant or lactating. In other words, most subjects who would have benefited the most from the Atkins diet were excluded. Nevertheless, there was more weight loss among subjects on the Atkins diet, highly significant at 3 and 6 months, but claimed not to be significant at 12 months using all participants, including those who did not complete the study but whose values were extrapolated. The absolute difference in weight loss as percentage of body weight was actually 3% between groups, favoring the low-carb group, at 12 months *among those actually completing the study,* and was shown as significant in their figure 1B. Low-carb dieters had increased HDL and decreased TG. Adherence was poor and attrition high in both groups, but adherence was better in the low-carb group. More trials were recommended, and all the usual unfounded dreads about low-carb diets were resurrected (see Atkins book review, p. 103, this article) (Foster et al., 2003).

The subjects of a 12-week RCT of adolescents were 13–17 years old and had an initial mean weight of 92.1 kg in the low-carb group and 99.5 kg in the low-fat group. The low-carb group used an Atkins diet (see book review, p. 103, this article), with very appropriate increases of carb over time, and consumed 1830 kcal/day. The low-fat group at <40 g/day of fat and 75 g of whole-grain carb, with other foods unlimited, consumed 1100 kcal/day. The low-carb group lost 9.9 kg vs 4.1 kg ($P < 0.05$) for the low-fat group (Sondike et al., 2003), despite both the

lower initial weight of the low-carb group and the disparity in calories in favor of the high-carb group.

A 10-week RCT of obese women (ages 45–56 years) had a high-carb arm, following the USDA Food Guide Pyramid, with carb/protein ratio of >3.5; and a low-carb arm in which carb/protein was 1.4, both with 1700 kcal/day and both with 30% energy as fat and equal fiber. Several weeks involved all food being served in the laboratory, and the diets otherwise were strictly controlled. Slightly more weight was lost in the high-protein group, which had a significantly higher fat loss (as in Sears, 1995: 40–53), significantly lower TG and TG/HDL, and these subjects were less hungry (Layman et al., 2003).

Another assault on the Atkins diet claims that it restricts carb to <10% of daily caloric intake, causes ketosis, and promises fat loss, weight loss, and satiety, which have not [sic] been established as outcomes. The authors aired the usual shibboleths that the increased fat, especially saturated fat, will lead to CVD, and used selective citations ''linking'' high fat to increased TG, insulin resistance, glucose intolerance, and obesity (Brehm et al., 2003). So, an RCT of 6-months duration was performed on 53 healthy obese women, BMI 33.6, who were randomized to either an Atkins low-carb diet or a 55% carb diet said to be recommended by the AHA (which actually recommends 75%). Exclusion criteria included CVD, hypertension, and diabetes; again, subjects who might have benefited the most were absent. Both groups were subjected to registered dietitians, so the caveats above apply. Both groups were free-living *and had reduced calorie consumption by similar amounts* at 3 and 6 months. Adherence was good and there were only 3 dropouts in each group. The low-carb group lost 8.5 kg vs. 3.9 kg ($P < 0.001$), including more body fat: 4.8 kg vs. 2.0 kg ($P < 0.01$). Both groups had improved blood lipid concentrations and no changes in EKGs. The authors were so honest as to write, ''This study provides a surprising challenge to prevailing dietary practice.'' Their figure 3 also indicated that the weight loss in the low-fat group was leveling out, while the loss in the low-carb group was continuing down.

The target of an RCT, also of 6-months duration, at the Philadelphia Veterans Affairs Medical Center was *The Protein Power LifePlan* (see book review of Eades & Eades, p. 111, this article). Stratified randomization of 132 severely obese subjects (mean BMI = 43) ensured that the low-carb and low-fat groups would have equal numbers of diabetics and women. The exclusion criteria were thus reasonable, including inability to monitor own glucose; participation in another weight-loss program; use of weight-loss drugs; or severe, life-limiting conditions. The low-fat group was to follow the NHLBI guidelines (55% carb, 30% fat, 15% protein) with calorie restriction to create an energy deficit of 500 kcal/day. The low-carb group was instructed to use 30 g of carb/day, which was incorrectly imputed to Eades, who actually recommended <40 g of total carb/day, even to start, and by the 6-month phase, in Maintenance mode, <120 g digestible carb/day (Eades, & Eades, 2000: 335), which could be 200–240 g of total carb. Both free-living groups were given an exceptional number of contacts

with ''experts in nutritional counseling''; so the placebo and nocebo effects postulated by this reviewer for the study of Foster et al. would be even more intense here. No wonder, then, that adherence and dropout rates were high. But the dropout rate at the 3-month mark was 47% in the low-fat group and 33% in the low-carb group ($P = 0.03$). The difference in consumption of energy from carb was quite narrow: 51% in the low-fat group, and 37% in the low-carb group. Total energy intake at the 6-month mark was 1576 kcal/day in the low-fat group and 1630 kcal/day in the low-carb group. Nevertheless, the low-carb group lost 5.8 kg (and was still losing weight at 6 months) vs. 1.9 kg (and leveled off) in the low-fat group ($P = 0.002$). TG dropped 20% in the low-carb group vs. 4% in the low-fat group ($P = 0.001$). The low-carb group became more insulin-sensitive ($P = 0.01$). Yet the authors want these results to be interpreted with caution ''given the known benefits of fat restriction'' (Samaha et al., 2003).

All these recent studies confirm what had already been known: calorie content is not as predictive of weight loss as is reduced carb consumption. Low-carb diets have other demonstrable benefits besides better adherence and no deleterious effects whatsoever in people of the appropriate metabolic type.

Book Recommendations

Individual detailed book reviews are located in the next-to-last section of this article.

The 2 books in this group which do not contain menus or recipes, those by Braly and Hoggan, and Ottoboni and Ottoboni, are both excellent and complementary. The portions of McCully and McCully on homocysteine and oxycholesterol are excellent and complementary to both of the others. These are the ones to read for general information.

For diagnosed diabetics, Bernstein's book is in a class by itself—outstanding.

For people who are not sure which diet-based affliction they might have, Smith's book has the best tests for self-diagnosis, and the newest, least invasive, most accurate blood tests to confirm the diagnosis, as well as the best suggestions on how to find medical providers who do not merely repeat the diet dogma of the agencies and foundations named above.

For overweight people who want minimal reading and a simple plan to follow, Allan and Lutz's, and Groves' books are the best.

For overweight people who want more information and more diet plans, the book by Eades and Eades is superior, followed closely by Atkins'.

Because conditions in Eastern Europe are so different from those in the USA and UK, people there might do best following Kwasniewski and Chylinski.

For any coach, trainer, or athlete who thinks ''carb-loading'' is a great benefit, Sears' book will nullify that notion.

Unresolved Issues

Whether persons adopting a low-carb diet should plunge in at once to the very low carb levels or approach them gradually is not yet clear.

Whether the amounts of protein and fat should be limited is disputed. Since protein does have a glycemic response, it seems as though the amount should be limited to what is needed to maintain lean body mass (LBM). Sears fixes the calories from fat as equal to the calories from protein. Bernstein and Eades and Eades advise that, if weight loss is still desired after a period of very low-carb diet, the fat intake will have to be cut, whatever it was. These authors show how to work out how much protein is ideal.

Since an effective low-carb diet must be both high protein and high fat, the nature of the fat is important, and the most healthful mix of fats is still disputed. One of the main disagreements among the authors is on the healthful level of saturated fat in the diet, ranging from none to unlimited, with some authors espousing ⅓ of all fat, as recommended by the FDA and NHLBI. Based on a review of diet studies most trusted by this reviewer, saturated fat does not increase the risk of CVD (Ravnskov, 1998), and animal fat may well be protective against cancer (Allan & Lutz, 2000: 84–85). The medium-chain-length saturated fats in butter and coconut and palm kernel oils have antimicrobial effects (Enig, 2000: 87ff). The benefits of canola oil are greatly disputed among our authors based on its perceived content of desirable omega-3 fats and undesirable *trans* fat. Of the non-hydrogenated types, deodorized or otherwise finished samples of canola oil from Belgium, France, Germany, and the UK retained most of their omega-3 linolenic acid (8–10%) and contained only 1–3% *trans* fatty acids, and had only 2–3 times as much omega-6 as omega-3, all as assayed by gas chromatography of their methyl esters (Hénon et al., 1999; Wolff, 1992, 1993). A sample of commercial deodorized canola oil obtained from the Cereol refinery in France contained 0.4% total *trans* 18:2 and 1.26% total *trans* 18:3 (Hénon et al., 1999). The latest type of deodorizers, *SoftColumn*™, can be operated to give canola oils with as little as 0.02% total *trans* 18:2 and 0.08% total *trans* 18:3 (Ahrens, 1999). But according to Fred and Alice Ottoboni, processed canola oil in the USA contains some *trans* fat and very little omega-3.

Fructose is another conundrum, because its low GI yet high GL (Foster-Powell et al., 2002) do not account for the delayed hyperglycemia it causes, observed by Bernstein (2003: 124–125) and Mercola (2003: 106), but not by Smith (2002: 27). It does raise TG levels, and one of the best indicators of ill health is the TG/HDL ratio (Eades & Eades, 2000: 95–96). Eating fruit, in which the main sugar is fructose, is encouraged by some authors, limited by most, and banned by 2 of them. Recent studies show that fructose, besides raising TG levels, causes insulin resistance (Thresher et al., 2000), and that men are more sensitive to fructose than are women (Bantle et al., 2000). Most fructose is converted slowly to glucose in the human liver; it is first catabolized to pyruvate (Berg et al., 2002).

There is no agreement on which, or any, low- to non-caloric sweeteners are most healthful. Trials have shown mixed results, but a recent trial pitting sucrose against aspartame, acesulfame-K, cyclamate, and saccharin showed that the non-caloric sweeteners led to weight and fat loss compared with gains in the sucrose group, as well as lower blood pressures (Raben et al., 2002).

Blanket recommendations for unrefined grains over the corresponding refined ones, as is supported by at least 1 study (Liu et al., 2003), must be tempered by the fact that their sometimes lower GI is not accompanied by a lower GL (Foster-Powell, 2002), and more gluten and other allergens are found in the unrefined grains (Braly & Hoggan, 2002: 101). Rice, oats, and quinoa are ''allowed'' by Mercola (2003: 82) in the Sustain phase of his diets, while Bernstein (2003: 141, 147) wrote that these very grains are hyperglycemic.

One study found benefits of eating fiber for prevention of NIDDM (Montonen et al., 2003) and another study found no benefit in cancer prevention (Fuchs et al., 1999). Since some ethnic groups and some long-term followers of low-carb diets ate no fiber at all and remained in good health, the benefits of fiber are probably moderate, and possibly might be due to an associated micronutrient(s) such as magnesium.

Restrictions on consumption of nuts (except walnuts) (Mercola & Levy, 2003: 111) is at odds with the findings of epidemiological studies (Fraser & Shavlik, 1997), in which the benefits are usually attributed to the high omega-3 content of nuts (Lee & Lip, 2003); but there is an equally good argument for the benefits being due to the copper content (Klevay, 1993).

Soy protein may be acceptable for vegetarians (Bernstein, 2003: 132). Soy flour is both promoted and disparaged. According to Kilmer S. McCully in his e-mail from June 24, 2003, ''. . . soy products are undesirable in the diet in any quantity. The exception would be fermented soy foods in small quantities, as eaten by Asian populations. Soy contains multiple toxic proteins that are anti-thyroid and phytoestrogens that have antifertility and other toxic effects.'' The usual cautions about soy products relates to their phytate content (http://www.westonaprice.org/soy/soy_alert.html), which inhibits absorption of many minerals. However, actual assays of flours show the phytate content of soy to be the same as that of rye and refined wheat, with whole wheat being 2.5 times higher (Lentner, 1981: 265).

Recommendations for alcohol consumption vary from nearly none to several glasses daily of red wine only, to 1.5 drinks daily of any variety (20 mL of ethanol daily), all based on the long-known protection from CVD. In fact, there is no *significant* difference in all-cause death rates between non- and moderate drinkers (Malyutina et al., 2002; Theobald et al., 2001). None is proper for those with the ''leaky gut'' syndrome (Braly & Hoggan, 2002: 150; Smith, 2002: 148–149). For the carb-sensitive only, the carb content matters, so ales and stouts with up to 18 g/355 mL should be avoided in favor of diet beers with $\leq$5 g/355 mL. Low-sugar red wine such as pinot noir at $\leq$150 mL/day is equivalent, but those interested in the antioxidants in wine can obtain them by eating grapes and berries (Smith, 2002: 149).

Recommendations for water consumption range from *ad libitum* to 12 glasses/day!

Recommendations for supplements range from a multivitamin only, to a half dozen known 50 years ago to be taken only when needed, to a dozen recent ones, to a dozen or so with good clinical studies. This reviewer believes that the definitive work on the actions of supplements, along with the symptoms they relieve (Atkins, 1998), does not really indicate who should take which; but like other books on the topic, his may lead one to believe that dozens should be taken by almost everyone. It is true that there are almost universal deficiencies in the intake levels of vitamin C and magnesium (for example, Eades & Eades, 2000: 129–130, 205–226) that cannot be reached by food choices alone, especially when high-GL foods are limited.

As nearly all the authors provide total lifeplans including exercise, not just diet advice on the contents of meals, it is hard to determine what fraction of the benefits of their interventions are due to each factor.

There is no consistent method in general use at present to account for the fact that the NMEs for carb and fat are not 4 and 9 kcal/g. Insoluble fiber has no NME, soluble fiber provides 0–2 kcal/g (Livesy, 2001), and the mean of common fats is nearer 8 kcal/g than 9 (Table 1). Counting a mean NME of 2.5 kcal/g for the low-GI, low-GL carbs usually recommended, the 40:30:30 diet of Sears and others is really nearer to 22:37:41 carb:fat:protein in energy content.

Conclusions

Each of the books individually reviewed in this article describes aspects of low-carb diets that are both unique and useful. All of the authors who are also practicing physicians must be considered courageous for using treatments in their practices that are not recommended by mainstream authorities.

Based on the long-term effects of low-carb diets in a number of individuals—mostly MDs, the long-term results in their patients, the favorable results in all controlled trials—and by using the actual data and not necessarily the trial author(s)' conclusions, the safety of such diets is established, and their efficacy for weight-loss and prevention of the complications of diabetes is indisputable. Controlled trials in a hospital have shown that a diet of just 1000 kcal/day which is 90% carb led to weight gain, and intakes of 2600 kcal/day with a very low carb content led to weight loss (Groves, 1999: 21–22). A patient of Eades and Eades (2000: 50–51) lost weight on a diet of 5000 kcal/day, which contained only 30–40 g (2.4–3.2%) of digestible carb. The dogma that a ''balanced'' diet is best for almost everyone has been falsified.

Sears showed that young athletes, who were supposedly not insulin resistant, at the peak of their training on high-carb diets improved their performance on a medium-carb diet. His and other estimates are that ¾ of all people of European descent are insulin resistant to some degree, so at least this fraction would benefit from eating a low-carb diet. A study from the Centers for

Disease Control and Prevention, using indirect methods, estimated that ¼ of all Americans over age 20 have the metabolic syndrome compared with >42% of those over 60 (Ford et al., 2002); this latter group would obtain the greatest advantages from a low-carb diet. Overlapping any of these populations are those who suffer from gluten or grain sensitivity. They should use low-grain diets as their form of low-carb diet and may have to limit all carb intakes as well.

Obesity and diabetes are strongly associated with CVD (Liu et al., 2000). Even though the absolute values of TC and LDL in serum are not predictive for identifying specific individuals in a given age group who will suffer from CVD soonest (Stehbens, 2001), several authors of the books reviewed have noted how TG, and more so TG/HDL (a strong predictor of CVD [Gaziano et al., 1997]), drop in people using low-carb diets, as do homocysteine levels. Insulin activates HMG-CoA reductase, one of the catalysts for cholesterol synthesis.

One particular isomer of conjugated linoleic acid (CLA) (18:2,n-6,t-10,c-12) formed by exposure to hydrogenation of the natural linoleic acid (18:2,n-6,c-9,c-12) in vegetable oils, soybean in particular, has been found to increase insulin resistance (Risérus et al., 2002a) and oxidative stress (Risérus et al., 2002b) in humans. Thus there is evidence that not just a high-carb intake can cause insulin resistance, justfying the recommendations to avoid *trans* fats. RCTs have shown that there is no benefit to using olive oil or corn oil (Rose et al., 1965) or polyunsaturated oils in general (Morris et al., 1968; Pearce & Dayton, 1971), despite reductions in serum TC with the polyunsaturated oils.

For diabetics, reversal of complications has been noted when blood glucose levels are normalized, and adopting a low-carb diet is an essential factor. A surprising number of other afflictions are stabilized or reversed with a low-carb diet.

A problem with all the books in this review is that one is not told how much longer one might live when following the program, or what the cause of death might become. For example, the lower heart deaths in France and Japan are accompanied by more cancer deaths.

In contrast to this picture, it has been shown that much of the evidence for high-carb (low-fat) diets is a result of poorly designed studies, misinterpretation, exaggeration, and outright fraud. Remember, the original reason for the recommendations for low-fat (high-carb) diets was to limit the intake of cholesterol and saturated fats, especially animal and tropical fats and eggs, and to substitute omega-6 and *trans* fats. ''An almost endless number of observations and experiments have effectively falsified the hypothesis that dietary cholesterol and [saturated] fats, and a high [serum] cholesterol level play a role in the causation of atherosclerosis and CVD'' (Ravnskov, 2002b).

The extent of the evidence for the benefits of low-carb diets in both time and volume is so great as to invite questioning of the motives among all the government agencies and private foundations still recommending high-carb diets and presently coordinating a world-wide attack on high-carb diets, despite

the obvious result of weight loss among at least 40,000,000 Americans alone (based on book sales) who use low-carb diets. Could the vast difference in interpretation be an honest scientific controversy? More likely it is simply resistance in defense of an entrenched organizational position that should not have been adopted originally, an attitude all too familiar to SSE members. Could it also be seen as negligence (or worse) that has led to lower quality of life and premature death for millions of dieters, for which compensation in the courts might be sought?

Individual Reviews

All of the authors of the books reviewed individually below recommend low-carb diets for all diabetics, for most people who are obese, and for people suffering from certain other conditions including hypoglycemia, hyperlipidemia, NIDDM, polycythemia, hypertension, stroke, CVD, cancers, GI problems such as Crohn's disease, as well as multiple sclerosis, rheumatoid arthritis, osteoporosis, and others. The length of this list may strain the empathy of the reader, but for each condition evidence is given, often from the author's own clinical experience. Most authors advise on exercise, water, alcohol, caffeine, and supplement consumption as well, so these topics are addressed. Drafts of all reviews except Atkins' were sent to the authors or associates for criticism; responses were received from all but Eades and McCully.

Dr. Atkins' ***New*** **Diet Revolution** (3rd ed.) by Robert C. Atkins, MD. New York: Evans, 2002. 442 pp. $24.95 (hardcover). ISBN 1-59077-002-1.

Brash, immodest by admission, self-promoting, Atkins' tome is, nevertheless, solidly backed scientifically by individual citations to about 400 peer-reviewed papers. Explanations are given as to why all calories in the diet are not equal, and why people will lose weight on a low-carb diet even if it contains more calories than a high-carb diet. The relation of hyperinsulinemia to a high diet load of carbs is clearly explained, as is the reality that insulin is the primary fat-building enzyme. A large number of diet trials and epidemiological data are quoted (see above) in support. Atkins also has the advantage of about 30 years of clinical experience in his own practice, which comprised "tens of thousands" of patients. However, one of the greatest gaps in Atkins' work is his failure to use clinical assay data from his own patients before and after they tried his low-carb diet, and other aspects of his overall weight-loss plan, to show what fraction really were improved, or not, or had dropped out. His excuse (p. 65) is that his responsibility to his patients would not allow him to use any of them as controls; but that does not prevent Lutz (see above) from citing assay data from his patients, anonymously.

Atkins shows the progression from high-carb diets to higher insulin levels to insulin resistance, to NIDDM (Type 2) with high insulin to later Type 2 with low insulin levels. He also shows the relation of obesity with NIDDM, and of

NIDDM with heart disease, amputations, blindness, kidney failure, and collagen problems, many due to glycation of proteins (p. 277).

Atkins actual program demands not only a low-carb diet, but also at least moderate exercise (distinguishing aerobic from anaerobic, pp. 252–253), use of supplements including fiber, and some standard blood work to follow progress. He reports an over 90% success rate with people who began the program at his Center, but did not define success or provide a dropout rate. The Atkins program diet begins with a few days to weeks eating a low-carb diet with only 20 g of NME from carbs per day (Induction), and to help with the count, there is an extensive table of ''digestible carbohydrate'' per serving in the book, as well as advice on how to determine a rough NME from an FDA food label. Since the carbs recommended in all phases are mostly of low GI, this is not as restrictive as it seems, since 40–50 g of carbs can be used according to Atkins' interpretation of FDA food labels. The objective, from the beginning, is to stop glycolysis as the prime source of energy and replace it partially by lipolysis, burning one's own fat for energy, so the weight loss is mostly to be from one's own fat. This phase is carried to ketosis, monitored with test strips for urine, and the patient is to remain near ketosis until most of the desired weight loss occurs. Atkins clearly distinguishes the non-threatening ketosis of low serum glucose from the potentially lethal ketoacidosis due to the high serum glucose of Type I diabetics (p. 82ff). Ongoing Weight Loss is the next phase, with 20–30 g of NME from carb daily, and the amount is individualized based on testing, symptoms, and weighing. Pre-Maintenance is next, with 25–60 g, then finally Lifetime Maintenance, with 25–100 g. Use of certain low-caloric sweeteners is included, but not aspartame (p. 164). Atkins' favorite is sucralose, but stevia is not mentioned. Eating at least every 6 hours when awake is highlighted. Atkins emphasizes that people with the metabolic problem with carbs will never be cured, so an appetizing, varied, and nutritious diet must be followed for life. Food allergies are addressed briefly (pp. 295–299).

Warnings against ''twisted'' (p. 308) *trans* fats are copious. There are 87 pages of recipes for which preparation times are given (many very short). A daily 100 mL of wine is permitted when it does not hinder weight loss. Coffee is forbidden (p. 164); however, in a recent clinical trial, coffee consumption was inversely and strongly associated with lower risk of NIDDM (van Dam et al., 2002).

Almost every objection to the program has been countered. The weight lost includes 2–4 kg of water at the beginning, but is then truly fat loss (pp. 84–85). Higher protein consumption does not cause kidney damage (p. 86). Higher fat consumption does not cause gallstone formation (p. 87) or heart disease (p. 88). A great variety of related conditions are considered: excessive insulin from use of oral diabetic drugs or injected insulin, interference of many types of prescription drugs on weight loss, thyroid problems, food allergies, and candida infections. Much criticism has been leveled at the unlimited amounts of fat and protein supposedly allowed, and Atkins indeed ballyhoos ''no calorie counting'' (pp. 1, 14, 83, 107, 124). However, his actual instructions are to eat fat and protein until

one is satisfied, not stuffed; to pause when unsure to see whether one can stop eating. One of his major points is that food cravings disappear when carb intakes go down. The low-carb diet usually *lowers* LDL, and especially TG, and may raise HDL. He also notes that one can lose fat and gain LBM (all but fat), so body measurements for the very active can be more important than weight loss.

Among the few minor deviations from best nutritional science are the doubtful recommendations for liberal use of oils containing large amounts of omega-6 fatty acids (Ottoboni & Ottoboni, 2003: 37, 123–126, 193), too much concern about saturated fats (Ottoboni & Ottoboni, 2003: 54, 122, 129; Ravnskov, 2000: 15–46), a recommendation for use of soy flour (p. 207) despite its phytate content that interferes with mineral absorption (Lentner, 1981: 265), and a recommendation to eat 10–20 olives to alleviate hunger, despite their salt content of 8.6–17.3 g! Atkins appears to accept the conventional (but incorrect) view that high TC or LDL levels predict cardiovascular problems. This is mitigated by his preference for the use of high serum HDL levels and also a low ratio of TG/HDL as desirable (p. 304). He admits that some people will not lower their TC levels on his program, while not quite understanding that it does not matter (Ottoboni & Ottoboni, 2003: 119ff; Ravnskov, 2000: 47–93).

Life Without Bread by Christian B. Allan, PhD and Wolfgang Lutz, MD. Los Angeles: Keats, 2000. xiv + 240 pp. $16.95 (paperback). ISBN 0-658-00170-1.

Making it easy to read and very clear, Allan and Lutz state their main message at the beginning and repeat it many times: Without exception, a low-carb diet will improve health by reversing many common conditions from insulin resistance to Crohn's disease to cancer. ''The true fad diet of today is, in fact, eating too many carbohydrates'' (p. 49). The distinction between good and bad carbs is made by christening the bad ones ''utilizable'' (instead of Atkins' ''digestible''). Rather than burden the reader with GI and GL, the old unit of utilizable carb, the bread unit (BU) is adopted. This is 12 g of digestible carb per BU. A short table of foods with their BU per serving is given at the beginning and a long one is given at the end of the book. The main message is simple: Restrict carbs to 6 BU (72 g) per day. Eat any desired non-carbs without restriction. There is no individualization in contrast with Atkins, Eades, and others. The specific benefits of the low-carb diet, oddly, are not given early, but much later.

Another unique feature of this book is that results of clinical assays on Lutz's patients before and after initiation of the low-carb diet are presented (see previous section above for examples).

The history of low-carb nutrition is given, beginning with a possibly apocryphal story from antiquity and leading to modern trials. There are citations in the text to about 100 peer-reviewed papers.

The metabolic balance between insulin and glucagon is given with diagrams showing how hyperinsulinemia can affect growth, sex drive, alertness, and irritability. The special folly of feeding children high-carb diets is presented. It is

noted that there is no requirement for any carb in the human diet, but that bacteria prefer glucose for energy. The DHT is demolished with evidence, including the preference of the heart for fat over carb for energy. A history of the demonization of cholesterol and the discovery of the effects of the undesirable homocysteine (see McCully book review, p. 117, this article) in CVD are presented. The need for fat in the diet is emphasized. The lack of evidence for kidney damage on a low-carb diet is noted (pp. 100–101). Quite a number of GI conditions are alleviated with a low-carb diet. The authors note that the use of oral diabetes drugs, insulin, and the use of other drugs may be diminished or eliminated by means of a low-carb diet. Finally, obesity is addressed (pp. 131–146) with the evidence that low-fat diets have caused the present epidemics of obesity, insulin resistance, or full-blown NIDDM. Then the adequacy of micronutrients in a low-carb diet is presented. An entire chapter on diet and cancer gives examples and evidence that low-carb (high-fat) diets do not increase cancer rates and may reduce them (pp. 172–177). The non-dangers of saturated fat are noted. Then, the authors go back to the history of diets and make several main and unusual points: (1) the reduction of carb intake to nearly zero is not necessary to obtain the benefits of low-carb diets; (2) the 40% carb diet commonly promoted for athletes is not low enough (8–12% carb is what 12 BU/day represents); and (3) too rapid reduction in carb percentage can cause severe problems in the elderly or ill, and the reduction should be slow (just the opposite of Atkins and Eades).

Some final helpful hints are to limit fruit and especially fruit-juice consumption because of the high carb content and fast absorption: ''The USDA Food Pyramid that advocates eating 5 to 9 servings of fruits and vegetables per day is dangerously imprudent.'' This is because many fruits have high carb content and/or high GI. Oddly, 1–2 bottles of beer or ''several glasses of wine'' daily are said not to be harmful. A typical 350-mL bottle of beer contains over 1 BU of carb and stout 1.5 BU. Several 120-mL glasses of a typical low-sugar (''dry'') wine (merlot) would contain 1 BU. The alcohol content would not be a problem (Theobald et al., 2001) except for those with IDDM, in whom these amounts would cause hypoglycemia (Bernstein, 2003: 118–120).

The program is not at all punishing, and this book is recommended for its clarity, lack of errors, and unique clinical findings.

Dr. Bernstein's Diabetes Solution (revised ed.) by Richard K. Bernstein, MD. Boston: Little Brown, 2003. xxiii + 391 pp. $27.95 (hardcover) ISBN 0-316-09906-6.

Based on 20 years of clinical experience and 57 years of suffering from IDDM himself (see above for biographical data), Dr. Bernstein brings a unique background to this book, which is aimed primarily at persons with IDDM or NIDDM. He estimates that ¼ of all adult Americans have impaired glucose tolerance, and that 80% of those persons are overweight (pp. 65–74). The non-diet aspects of

treatment will be given less emphasis in this review than they would deserve in a more general one.

"The evidence is now simply overwhelming that elevated blood sugar is the major cause of the high serum lipid levels among diabetics and, more significantly, [is] the major factor in the high rates of various heart and vascular diseases associated with diabetes . . . My personal experience with diabetic patients is very simple. When we reduce dietary carbohydrate, blood sugars improve dramatically . . . [and in a few months lipid profiles do also]" (pp. 434–435). He notes that excess insulin, produced in response to excess glucose, causes atherosclerosis (p. 40), and that normalizing glucose levels can reverse neuropathy, retinopathy, kidney damage, erectile dysfunction, hypertension, CVD, vision impairments, and mental problems (p. 41). High TG and low HDL, rather than high TC, are associated with insulin resistance and CVD.

Focusing on diet at this point, it may be seen that Bernstein recommends 3–5 meals per day (or 3 meals and 2–3 snacks), eaten 3.5–4 hours apart for those not taking insulin, and 5 hours apart for those who are (pp. 168–169, 360). The main meals may contain just 6, 12, and 12 g of low-GI carb, a total of 30 g/day, about the same as in the initial diets of Atkins or of Eades and Eades. Since fat has no glycemic response, the amount eaten is not controlled. Diet contents (but not the variety of foods) are to be constant from day to day to maintain even blood glucose levels, because most of Bernstein's patients are taking oral hypoglycemic drugs and/or insulin, which must be balanced with food intake, and the results are to be checked frequently by self-determination of blood glucose levels. Individualization is highly encouraged within these parameters (pp. 187–189). Eating just enough to feel satisfied but not stuffed is a goal.

Bernstein is mindful that cooking raises GI (p. 144). Low-GI caloric sweeteners such as sorbitol and fructose, so prominent in so-called "sugar-free" foods for diabetics, are shown to raise blood glucose too much over time, thus they are not suitable (pp. 139–140). Of the non-caloric sweeteners, aspartame is acceptable if not cooked, along with saccharin and cyclamate, all in tablet, not powdered form with added sugars (p. 124). Fruit and honey are not recommended by Bernstein because of their fructose content, which raises blood glucose levels faster than any treatment can bring it down (p. 140). Alcohol in any form does not alter blood glucose, except lowering it at mealtimes for those with IDDM because it prevents the liver from converting protein to glucose. Bernstein is the only author to specify *dry* wine, limited to 1 glass (3 oz, 90 mL) (pp. 132–133, 159, 173). (The "driest," or least sugary, of the common red wines, by the way, is pinot noir.) Some brands of soybean milk and flour are acceptable, as are bran-only crispbreads and toasted nori (pp. 152–154).

Unlike many authors in this group, Bernstein is not excited by many supplements, recommending vitamins C at 500 mg/day and E at 400–1200 IU/day as gamma or mixed tocopherols, as well as B-complex and calcium only when needed, and the latter with vitamin D, magnesium, and manganese (pp. 161, 175–176). As an insulin-mimetic agent, slow-release alpha-lipoic acid is

recommended with evening primrose oil; both reduce insulin requirements (pp. 227–228).

The book begins with some biographical data, followed by 14 testimonials from patients before and after treatment, which demonstrate the appalling failure of conventional treatment of diabetes with concomitant complications. Next, descriptions of IDDM and NIDDM are given (with the little-known observation that no amount of injected insulin of the most rapid-acting type can control blood glucose from a high-GL meal), followed by medical tests to determine the condition of individual patients, a compendium of the supplies needed, and a description of measuring and recording one's own blood-sugar levels (with the revelation that only a few models of glucometer available at publication time were acceptably accurate, and that one should telephone Bernstein to find which ones). Bernstein then explains the virtue of small inputs of food, insulin or oral hypoglycemic drugs as leading to no or small mistakes, and the establishment of an overall treatment plan—all this is given in great detail. As noted above, a low-carb diet is a key part of the plan, so the basic food groups are described, 3 days of initial meal plans are given, and, in a later chapter, recipes are provided for low-carb meals, with grams of carb and protein for each food and individual serving. Some history of the safety of low-carb diets, and conversely, the often devastating effects of high-carb diets (in Pima Indians, etc.), are given. A method of self-hypnosis is described for control of appetite, as is the use of small doses of naltrexone (pp. 191–199). A whole chapter on exercise has the best distinction between aerobic and anaerobic exercise in this whole group of books, with the desirability of regressive anaerobic to build muscle mass in order to reduce insulin resistance. Consideration is given to those who must begin exercise very slowly, and many hints on how to avoid exercise damage are presented.

Oral hypoglycemic drugs are discussed (pp. 224–226). The flaw in the concept behind the old sulfonylurea types is exposed. Among insulin-sensitizing agents in the glitazone class, it was noted that troglitzone (Rezulin™) was withdrawn from the market in May 2002 because of adverse effects. Metformin (Glucophage™), rosiglitazone (Avandia™), and pioglitazone (Actos™) are favored. The former now has an FDA ''black box'' warning for similar adverse effects and is known to increase homocysteine levels (Pelton & LaValle, 2000: 125, 136, 204). Avandia has new labeling, since it also has occasionally fatal adverse effects (Wolfe, 2001a,b).

Next is a chapter on where and how to inject insulin and on the characteristics of slow- and fast-acting insulins, followed by great detail on how to compensate for skipping meals, eating out, crossing time zones, and dealing with hypoglycemia (and not confusing it with alcohol intoxication or autonomic neuropathy or postural hypotension, which may be caused by diuretics and antihypertensive drugs). Then comes a chapter on how to deal with erratic blood sugars caused by gastroparesis (delayed stomach emptying) and another chapter on how to deal with vomiting and diarrhea, in which an excellent discussion of ketoacidosis is

given (pp. 334–335). Advice on consideration for relatives, and tagging oneself as a diabetic, is provided.

Appendix A is more technical. It gives the scientific basis for ignoring the nonsense on dietary cholesterol, fat, protein, carb, salt, and the current high-fiber fad. Appendix B shows how to inform a hospital that food will be chosen by the patient, since ''diabetic'' diets in hospitals (as well as on airliners) are hyperglycemic, that the blood-sugar measurement kit and the insulin supplies should not be confiscated, and that dextrose will not be used for re-hydration. Appendix C gives a long list of drugs that may affect blood-sugar levels. Appendix D is about the all-important (to avoid amputation) foot care for diabetics. A glossary defines many terms for diabetics. In it, polyunsaturated fats and, to a lesser degree, monounsaturated fats are given lukewarm mentions for virtues whose existence is doubtful (Ravnskov, 2000: 15–97). There is a recipe index and an excellent general index.

There are a few footnotes to original medical papers with citations in the text; more would have been welcome. The tone of the book is simultaneously learned, determined, relentless, caring, and considerate, except for utter contempt for the ADbA. Limitations of time, money, and insurance for patients are given consideration. Compared with several other books for diabetics your reviewer has seen, all of which recommended high-carb diets (for example, see Whitaker, 1987), Bernstein's book is in a class by itself—the highest.

Dangerous Grains: Why Gluten Cereal Grains May be Hazardous to Your Health by James Braly, MD and Ron Hoggan, MA. New York: Avery/Penguin Putnam, 2002. xx + 490 pp. $28 (hardcover). ISBN 1-58333-129-8.

Almost alone of the books in this group, *Dangerous Grains* recommends low-gluten diets, rather than generally low-carb diets, for the many sufferers from the ''leaky gut'' syndrome. Grossly under-diagnosed, this is a serious condition affecting 15–42% of humans who use wheat products as a large portion of their diet (p. 30). Gluten is a protein fraction of wheat and other grains, and the toxic sub-fractions are called gliadin and glutenin. Perhaps initiated by preliminary damage to the lining of the GI tract by infection, gluten consumption in people sensitive to it leads to damage of the lining, allowing leaks of partially digested materials into the blood stream. These materials, often peptides, either create havoc or the antibodies generated against them are damaging. Addictions to gluten-containing foods develop because of the presence of ''feel-good'' exorphins in them.

What is actually a sensitivity to gluten may be manifested initially as autism, attention deficit hyperactivity disorder (but see Ottoboni & Ottoboni, 2003), depression, constipation, bloating, or diarrhea, often treated only symptomatically, and may lead to irritable bowel syndrome, colitis, or enteritis. There may be progression to celiac disease in people genetically disposed to it, leading to an almost unbelievable set of dismal conditions, from IDDM, osteomyelitis,

periodontal disease, and Crohn's disease, to gastrointestinal (GI) cancer (evidence on p. 109), schizophrenia, multiple sclerosis, autism, and even epilepsy; more conditions are given in Appendix C. Victims may be under- or overweight; more are overweight. There are 187 symptoms of gluten sensitivity listed in Appendices A and D. Diagnosis is difficult and often delayed for decades of suffering until one of the terminal stages becomes obvious. The former best method of diagnosis, an intestinal biopsy, even the recently improved version, is deemed not as valuable or as safe as a number of new tests on blood (pp. 61–76, 177). Broad population screening beginning in childhood is strongly recommended (pp. 177–179). Cancer treatment by radiation or chemotherapy, both considered worthwhile in most cases by these authors, causes intestinal damage that often leads to gluten sensitivity (pp. 107–109).

The obvious treatment, often reversing serious symptoms, is avoiding gluten in the diet for the remainder of life. This is the basis for the recommendation of low-carb diets, which are actually almost no-grain diets. A 1-day sample menu is given (pp. 89–90) in which high-GI treats are kept separate from the other non-gluten-containing foods. Organic foods are recommended on theoretical grounds. Avoidance of *trans* and omega-6 fats and obtaining enough omega-3 fats are stressed (pp. 86–87). Sources of gluten to avoid are given in Appendix B. As part of the diet, advice is given to supplement with quite a number of well-chosen vitamins and minerals (pp. 92–99). New evidence for supplementation by L-glutamine is given (pp. 176–177). Cautions are given on iron and calcium supplements, with a well-written explanation that calcium supplements are the last choice for prevention of osteoporosis, which is better prevented by magnesium, boron, zinc, and vitamins D and K. Supplements to lower homocysteine levels are also emphasized (pp. 53–54, 97–98). Alcohol is said to cause or worsen a ''leaky gut.'' Water is recommended at 8–12 glasses/day (p. 89). Drugs may contain gluten in formulations, which should be avoided (p. 84).

Of course, the USDA Food Pyramid receives well-deserved ridicule for making no exceptions for people who are gluten sensitive (pp. 6, 135, 169). So, also, does the DHT, and the dangers of *low* cholesterol levels are laid out (p. 87). Widespread recommendations for whole grains are shown to be a disaster for the gluten-sensitive. Appendix E gives biographical data on prescient scientists who identified these sensitivities.

NIDDM is clearly described as a result of excessive carb (not necessarily containing gluten) consumption, which leads to excessive insulin production, leading to obesity, inflammation, cancer, allergy, CVD, and the undesirable prostaglandin E2 series (p. 126). This agrees with all the other authors. However, people with NIDDM and its earlier manifestations get short shrift, unlike in Smith's book (see p. 126, this article). A great deal of fruit and other high-GI foods are recommended (p. 86). It is not clear how increased insulin levels, which are supposed to lower glucose levels, accelerate tumor growth (p. 115). Also, probably an oversight in writing, it does not make sense that removal of L-glutamine from the diet may reverse damage from a ''leaky gut'' (p. 177). A 200-

item bibliography does not have citations in the text, a lack that is particularly annoying in this book, in which so many non-mainstream claims are made. One of the contact websites, www.cerealkillers.com, does not work; it is now info@drbralyallergyrelief.com.

This book is extremely valuable, nevertheless. One must ponder how much of the benefit of low-carb diet is from lower insulin levels, and how much is from avoiding gluten or other grain sensitivities.

The Protein Power LifePlan by Michael R. Eades, MD and Mary Dan Eades, MD. New York: Warner Books, 2000. xxviii + 434 pp. $10 (paperback). ISBN 0-446-52576-6.

Perhaps the title *The Protein Power LifePlan* and the authors' website, www.eatprotein.com, are the least fortunate aspects of this encyclopedic work, since the Eadeses are realistic about the amount and type of fats to be eaten as part of a low-carb diet. The actual diets are near the end of the book, which focuses on obesity and insulin resistance, leading to all the modern nutritional diseases, with attention to gluten and other food sensitivities. The authors have a clinical practice in which "thousands" of patients are using their low-carb diet, and about 3,000,000 adopted the diet based on this and other of the Eadeses' books.

There are 3 phases of diet: Intervention, Transition, and Maintenance, in the manner of Atkins and Mercola. There are 3 levels of restriction, called Purist, Dilettante, and Hedonist, in the manner of Mercola. Individualization is strongly encouraged. GI and GL and their limitations are discussed earlier, and the non-fiber carbs in foods, called effective carbohydrate content (ECC) and shown in a table (pp. 326–331), are used for the guidelines (p. 355), in which Intervention begins at a level of <40 ECC/day, Transition utilizes <60 ECC/day, and Maintenance allows levels of <80, <100, and <120 ECC/day depending on what each individual can tolerate. The minimum amount of protein to maintain one's LBM is readily determined from a table; one must know only one's height and weight (pp. 312–313). The amount of fat to eat is said to take care of itself and not to matter if weight loss is not a goal or the goal has been reached; otherwise, if one is consuming the correct ECC/day and not losing weight, the fat quantity must come down. There are moderate numbers of menus and recipes and advice for buying, cooking foods, eating out, and packing lunches, including those for children. Organic food is recommended for the more restrictive plans, with the usual theoretical reasons; but neither this nor any of the food choices are fanatical or impractical in any way. Since more fat may be eaten than in the LifePlan diet than before, the type of fat is considered important, meaning, for the Eadeses, no *trans* fats, very little omega-6 fats, and at least the minimum few grams of omega-3 to obtain or make DHA and EPA, no oxidized fats, and no restrictions on saturated fat or cholesterol intake. Wasa™ crispbread is recommended as a bread substitute. Nuts and canola oil are seen as healthful (pp. 119–20). Among non-caloric sweeteners, aspartame is banned

(pp. 165–167, 206, 332, 341) because of its effects on the brains of some people. Sucralose and stevia are favored (pp. 341, 356–357). Sorbitol is considered OK, contrary to Bernstein (2003: 124–126). Two liters of liquids are to be drunk daily. Water is preferred and coffee is acceptable, as is alcohol (liquor, beer, or wine containing 15 mL of alcohol/day) (pp. 342–343). A drink called Paleolithic Punch made from blended fruits is suggested (p. 366), and some recipes for drinks made with protein powders and fruit are given also.

The Eadeses give the USDA Food Pyramid a really good ridicule (pp. xv–xxiii, 321–323), and the ADbA gets an equally well-deserved drubbing (pp. 35–36). It is made clear that people are a wide range of metabolic types of whom ¼ will not benefit much from the LifePlan diet, ½ will derive a significant benefit, and ¼ will have their lives resurrected by it. There is a fine discussion of pre-farming diets; of insulin resistance and all the conditions it leads to and the best description of how to measure it; of the best types of fats; and the best review of all the books in this group on the sub-fractions of cholesterol, leading to the conclusion that one of the best indicators of ill-health is the TG/HDL ratio (pp. 95–96). Low cholesterol levels are linked to higher rates of accidents and violent death (p. 263). Very few supplements are recommended, but those that are were very well chosen, including ones to lower homocysteine levels (p. 98), and this selection, too, is to be individualized. After Braly and Hoggan and Smith, the discussion of the leaky-gut syndrome and grain sensitivities is the best. Sweeteners of all kinds rate a chapter of their own with GI discussed. The Eadeses recognize that the degree of cooking, as well as subdivision influence the GI. Iron overload receives an entire chapter with advice to have one's ferritin level measured, and, if it is too high, to reduce iron load by giving blood. (It is odd that ethlenediaminetetraacetic acid [EDTA] chelation for this purpose was not mentioned [Cranton, 2001: 34–35].) Magnesium is considered the single most valuable supplement and is the subject of an entire chapter. Sunning to make one's own vitamin D and the false claims on sun-block preparations are discussed at length. Psychological aids to health also rate a chapter, as does exercise, which is presented in a realistic manner—to be approached for real benefits without self-damage. The distinction between aerobic and anaerobic exercise is given clearly.

This book has few problems beyond misspelling linol*e*nic (pp. 69, 70, 77) and tocopher*o*ls (p. 351) and abusing some transitive verbs. The dangers of toxins in large fish were not quantified (p. 74), so one cannot be certain of how much is safe to eat, and polychlorinated biphenyls (PCBs) are actually biodegradable and not very toxic to humans (Ray & Guzzo, 1990: 86–88). As good as the discussion on cholesterol is, the Eadeses recommend levels of TC of 160–220 mg/dL (pp. 100–101), not realizing that the levels go up naturally with age, that women and men over 60 with the highest levels live the longest, and that high levels are strongly protective against cancer, infections, and other conditions (Ravnskov, 2002a). A cup (235 mL) of almonds (no shells) does not contain 167 g of fat (p. 216) but only 73 g. The fiber on FDA food labels is crude fiber and

omits soluble fiber, which is 2–16 times greater (de Man, 1999: 203–206), so the ECCs are a bit high since soluble fiber has an NME of 0–2 kcal/g (Livesy, 2001). Their figure 10.2 (p. 230) has incorrect wavelengths for ultraviolet and several colors of light, and their figure 10.3 (p. 251) has mislabeled path lengths for summer sunlight.

Overall, this book is very accurate and helpful. At first it seemed that a mere 2 dozen references (as footnotes) with citations in the text was inadequate referencing; but the Eadeses make available on their website their complete bibliography, or will mail copies of it for a fee of $3. Outside sources of information are carefully chosen and cited. Between clear writing, the use of a plethora of American colloquialisms, and many doses of humor, this book comes closest to being literature in this group, as well as one of the most scientifically solid and useful.

Eat Fat Get Thin! by Barry Groves. London: Vermilion, 1999. 220 pp. £6.99 (paperback). ISBN 0-09-182593-8.

With beautifully written reasoning in a compact form, Groves uses his own low-carb diet experience of decades (see above) and an excellent literature review (pp. 15–26), with about 170 references, but no citations in the text, to promote a low-carb diet. Obesity is his main focus, with cancer, CVD, and diabetes receiving attention as well. The diet industry receives a well-deserved drubbing; for example: "... the many commercial interests that rely on overweight people to make a living compound today's weight epidemic" (p. 5). "Low calorie dietary regimes inevitably fail. More importantly they damage your health, engender a feeling of disillusion and failure, lower morale and increase mental as well as physical stress" (p. 54). An entire chapter, "Are You Really Overweight?" shows how to determine your BMI and points out what is not obvious to many young women—dieting when fat is not dangerous.

The Eat Fat, Get Thin diet is simply a matter of reducing the digestible carb content of meals to 60–67 g/day until an acceptable weight is achieved, then the carb weight may be increased, or "... indulge yourself in chocolate once a week," but only with high cocoa butter chocolate (pp. 118–120). There is a BMI table (pp. 96–97). The "Rules" are set out with the wonderful clarity characteristic of this book: "Reduce your intake of refined (but see p. 36) carbohydrates (pp. 112–113); exercise only if you want to; don't try to lose more than 1 kg per week, leave the fat on meat; eat a high-protein breakfast; if you are not overweight, don't diet."

However, other authors (Bernstein, Mercola, Smith, etc.) note that unrefined grains have about the same glycemic response and most of the carb content of refined grains. While other authors wrote that exercise is mandatory, Groves gives very persuasive evidence that it does not aid weight loss and could be dangerous for the obese (pp. 85–90). The other advice is fine, except that a few people who are not overweight may still have IDDM or grain allergies. There are no limits to protein or

fat intake. The 60 g/day limit for carbs may not be low enough for some people, and there is no individualization except noting that the time to reach the desired weight will differ. Groves writes that the 20 g/day (of Atkins) is too low and will cause ketosis, which is not clearly differentiated from ketoacidosis (pp. 110–111).

There are tables of *digestible* carb content per 100 g of many foods, a glossary of nutritional terms with unusual detail, including a table of the fatty acid content of various foods, but lacking *trans* fat and the omega types. There are 54 pages of recipes with carb content, but not preparation times, and an unusual ''extra'': a recipe index. There is nothing punishing about Groves' recommendations. Small amounts of sugar, bread, and other treats are allowed.

''Fats are essential for health. Cut them out and you will shorten your life'' (pp. 9–10). Since high fat intake is justifiably promoted, it is interesting that an old RCT on heart-attack survivors is noted in which those given corn oil (polyunsaturated) had the poorest survival rates, those given olive oil (monounsaturated) did a little better, and those given animal fats (about half of which were saturated) did much better (Rose et al., 1965). The link between low cholesterol levels and cancer incidence was made very clear, and the cholesterol mania was exposed unmercifully. The anti-cancer properties of CLA (18:2,c9,t11) found only in the fat and milk of ruminant animals (Enig, 2000: 46) was noted (pp. 56–84) (not to be confused with the dangerous 18:2,t10,c12 linoleic acid found in partially hydrogenated vegetable oils [Risérus et al. 2000a,b]). ''The low-fat, high-carbohydrate slimming diets of today will inevitably be deficient in protein'' (p. 45) and may lead to gallstones (p. 102). Polyunsaturated and *trans* fats are revealed as the true fatty villains.

The fallback position of so many diet ''authorities,'' that one must cut out sugar and that complex carbs are safe, is demolished by pointing out that all carbs are converted to glucose on digestion (p. 36). This is oversimplified, since fiber is not in humans. The use of non-caloric sweeteners is discouraged on the grounds that one's craving for sweets is exacerbated (p. 39). Water (not juice or sweet soda) intake is to be *ad libitum,* not forced at 6–8 glasses per day. Alcohol, called a carb, without sugar is considered acceptable at the level of 1 drink at lunch (p. 140), contrary to other authors. Many helpful hints are provided for bag lunches at work, dining out, and at dinner parties. It was odd that crispbreads such as Ryvita™ were not recommended, nor were sensitivities to wheat covered. A low-carb breakfast was said to be the most important meal of the day, and this was well supported by research (pp. 123–131).

Where he differs from other authors, Groves may well be correct. This book is easy to read and follow, being ¼ the size of Atkins', Eadeses', or Mercola's books, and, if followed, would probably cut the UK's National Health Service costs in half for a while.

Homo Optimus by Jan Kwasniewski, MD and Marek Chylinski with Engl. transl. by Bogdan Sikorski, PhD. Warszawa, Poland: Wydawnictwo WGP, 2000. 374 pp. $28 (paperback). ISBN 83-87534-16-1.

Jan Kwasniewski, MD, lives and worked in Ciechocinek, a health resort in central Poland. He started his medical career in the 1960s as a specialist in helping people recuperate. After accumulating years of clinical experience, he was able to conclude that the wide variety of ailments in his patients was not the product of a great many pathogenic factors, as other doctors would claim, but the result of one underlying cause—bad nutrition via specific effects on the sympathetic and parasympathetic nervous systems. He also observed that different diseases are caused by different forms of malnutrition. After discovering this, Dr. Kwasniewski embarked on a search for a nutritional model that would not give rise to any detrimental effects and, moreover, would ensure a body's health and proper functioning. The fruit of this search is a dietary model he calls "optimal nutrition" by means of the Optimal Diet, developed around 1965–1967, because he found that it represents the "best" possible way for a human to feed himself. Its basic premise is that an eater should take care to keep proper proportions among the 3 fundamental nutrients in food—carbs, fat, and protein. He found that the ideal proportions by mass are anywhere from 0.3-0.5:2.5-3.5:1, meaning that every gram of protein consumed should be accompanied with between 2.5 and 3.5 g of fat and about half a gram of carbs (p. 51). In short, optimal nutrition is a high-fat, very low-carb diet, not to exceed 50 g of protein and 2000 kcal/day for an average adult. Using the fuel values for food groups, the lower-fat regimen corresponds to 11% energy from carb, 76% from fat, and 13% from protein (p. 235). The Optimal Diet not only is the highest in fat and saturated fat content of all the books in this review, it also recommends substantial amounts of collagen, well cooked for palatability, and organ meats for certain conditions. Eating apples (and fruits in general) is discouraged, contrary to the Ottobonis, who specifically recommend them several times. Potato is recommended as fries on page 234 and not recommended on page 235. Water is to be *ad libitum*. Recipes list many foods high in GI, but since they are allowed in such small amounts, and accompanied by huge amounts of fats, the effects on insulin response are low. Nutrition of infants, children, and pregnant women are all addressed.

This book was included in this review to obtain both diversity and the findings of another unorthodox clinician with long experience. Written in 17th-century style, it has no bibliography, no index, and is replete with testimonials and single-case reports in order to appeal to semi-educated general readers. A dozen or so published findings of other researchers were quoted, but never with enough data to allow a citation to be found easily. The first 135 pages are an interview of Kwasniewski with himself, and this section is a mixture of philosophy with many supposed quotations from biblical, ancient, and medieval sources, and some modern ones, with occasional dabs of diet wisdom. Uninhibited, the authors describe 16-year-old, physically well-developed Polish girls as having "... heads full of garbage soaked in Coca Cola" (p. 128). In contrast, the next section (pp. 137–238) gives actual clinical findings with a much smaller proportion of philosophy, which includes the advice not to vote for the obese for political office because their brains are adversely affected (p. 204). This is

followed by recipes with calorie content (pp. 207–307), menus for 2 weeks, tables with the nutritional value of food products (also using fuel values), and more testimonials. GI, GL, and NME are not addressed. Exercise is addressed realistically: those who are not in condition for it should not damage themselves until and unless the Optimal Diet improves their condition and allows safe exercise (p. 104).

The specific conditions that were stabilized or cured with the Optimal Diet include Bürgers disease, Bechterew's disease, multiple sclerosis (in agreement with Braly & Hoggan, 2002: 157–158), rheumatoid arthritis (in agreement with Ottoboni & Ottobnoi, 2002: 152–153), liver cirrhosis, neurasthenia, IDDM (said to be caused by sugar [not in agreement with Bernstein, 2003: 36] and in which not all pancreatic beta cells are destroyed [in agreement with Bernstein, 2003: 36]), NIDDM (in agreement with all the other authors), atherosclerosis (in which the DHT is demolished, pp. 72–75, 102ff), hypertension, CVD, obesity (in which LBM improved, even if weight did not go down, in agreement with Atkins and Sears), colitis, stomach ulcers, bronchial asthma, migraines, and certain infectious diseases. Clinical findings with results of blood work are given in a believable manner, with the numbers and fractions of patients who recovered. But complicating these findings were simultaneous treatments with electric currents of patients who had a number of the afflictions! Other than noting that the current was limited to 20 mA for safety, no voltage, frequency, or duration was given, let alone a description of the apparatus. So, naturally, a modern scientific view is that one cannot be certain whether the diet or the currents were responsible for healing when both were used. Moreover, there is no recognition that some cures could have been for gluten or grain allergies, not excess carbs. This, however, would clearly be irrelevant to the sufferer.

Many of the theoretical examples in support of actual clinical findings are not good science. For example, ''By increasing the weight of the unsaturated fat molecule by 1% through hydrogenation we can expect an increase in the caloric value by as much as 18%'' (pp. 31–33). In fact, the NME of fats made from saturated long-chain fatty acids is lower than that of their unsaturated analogues because of indigestibility (see Table 1), and the saturates certainly do not yield ''10 cal/g'' when metabolized. Formation of *trans* fats during hydrogenation is totally misunderstood, as is the relative metabolic need for oxygen, for which hydrogen is said to require much less than carbon (p. 35). Margarine is said to be better than the oil from which it is made because margarine contains more hydrogen (pp. 35, 234), contrary to all the other authors in this group. The facts are that 1 g of hydrogen requires 8 g of oxygen, and 1 g of carbon requires 2.7 g of oxygen. ''Carboxylic'' (actually carbonic) acid will *not* react with salt to give hydrochloric acid and sodium carbonate (p. 67); the reverse reaction is the correct one. Apes were said to be plant eaters (p. 162), contrary to the findings of Jane Goodall, who found that animal and insect prey were avidly sought after and eaten.

While this book meets most of the present criteria for junk science, this

reviewer is convinced that the clinical findings are genuine, nevertheless, and that these support the use of low-carb diets for treating many afflictions. Subjective findings were often confirmed by clinical assays of blood as done elsewhere in the world. It is revealing that the negative attitude of the Medical Science Therapy Committee of the Polish Academy of Science towards Kwasniewski's evidence (pp. 9–15) exactly parallels those of the US government agencies towards Atkins, Eades, etc.

The Heart Revolution: The Extraordinary Discovery that Finally Laid the Cholesterol Myth to Rest by Kilmer S. McCully, PhD, MD and Martha McCully. New York: Perennial/HarperCollins, 2000. xxii + 257 pp. $13 (paperback). ISBN 0-06-092973-1.

The USDA Food Pyramid, in perpetuating the anti-cholesterol mania, "... wildly exaggerates the importance of carbohydrates, which erroneously have become known as health foods" (p. 33). "Obesity is another consequence of the [USDA Food] pyramid's push toward carbohydrates ... But fat is not the demon ... *a high fat intake does not lead to obesity* if the diet contains unprocessed whole foods ... when the diet includes a high proportion of calories from refined carbohydrates—as does the American diet—the population develops diabetes, hypertension, tooth decay, obesity and heart disease" (pp. 39–41). The progression from carb in the diet to serum glucose to insulin resistance, obesity, and NIDDM is presented. However, this book is unusual for this group in that its focus is on heart disease.

The McCullys recommend a diet with a typical energy contribution of 45% from carb (all unrefined), 30% from fat, and 25% from protein; this is a medium-carb diet. A detailed program for the first 6 weeks of the Heart Revolution Diet is given (pp. 191–232) with all kinds of hints and many recipes, but without carb or calorie content. Red wine is recommended at 1–2 glasses per day as the only permitted alcohol. Chocolate of 70% or more cocoa butter is recommended (p. 91), as is fruit juice (p. 92). Rye crisp bread is recommended, as are meat, fish, eggs, butter, and other dairy foods with fat; and coffee in moderation but with tea preferred, as well as many vegetables and fruits. There is a half-hearted recommendation for organic food (p. 125). Eating canned and frozen food is discouraged because of vitamin loss on processing, for which actual percent losses are given (pp. 55–70). Irradiated foods are said to have the same fraction of vitamins destroyed as in canning or sterilization and to contain free radicals (pp. 67, 157). (The free radicals seem unlikely to persist, given the high reactivity of free radicals.) Use of powdered egg and powdered milk is strongly discouraged because the processing forms oxycholesterol, said to be a bad actor, unlike cholesterol (p. 167), which is also one reason to avoid fried foods in restaurants. Both an excess of omega-6 and any *trans* fats are banned (pp. 85–87, 163), while omega-3 fats (with a brave recommendation of canola oil) are promoted. Fake fat, such as olestra, prevents the absorption of the fat-soluble

vitamins A, E, and D, as well as coenzyme Q10 and essential fatty acids (p. 123). There is very little individualization and no special emphasis on diabetics as the ones most likely to benefit from this diet; indeed, diabetics are counseled to seek medical care. A week of sample menus is given (pp. 97–102) in addition to a whole section with helpful hints and recipes (no GI, GL, or NME of carbs) (pp. 191–232). Drinking 8 glasses of water/day is recommended (p. 196).

Kilmer S. McCully received his PhD in Biochemistry from Harvard University under the nobelist Konrad Bloch and his MD from Harvard Medical School, where he also did his research. He discovered, beginning in 1968, that one of the main causes of atherosclerosis and the resulting CVD is high levels of homocysteine in the blood. Further tests on animals confirmed that homocysteine, not cholesterol, is a true cause of arterial plaque formation. He worked out that low levels of either vitamins B6 or B12 or folic acid led to high levels of homocysteine, and that supplementation with them brought homocysteine levels down. Current work from other researchers (some cited on pp. 21–27) has confirmed that higher homocysteine levels are associated with both CVD and all-cause mortality (Vollset et al., 2001), congestive heart failure (Vasan, et al., 2003), dementia, and Alzheimer's Disease (Seshadri et al., 2002). Supplementation with folic acid (Stanger et al., 2002) and with vitamin B6 as well (Mark et al., 2002) lowered homocysteine levels and reversed arterial stenosis. Far from receiving a well-deserved Nobel Prize for his work, McCully was eased out of Harvard Medical School and the Massachusetts General Hospital in 1979, prevented from receiving grant funding, and ostracized for 20 years by the Cholesterol Mafia. Only a threatened lawsuit against the pair of ''prestigious'' institutions allowed McCully to obtain employment anywhere. Happily, beginning around 1998, with the confirmation of his work being so overwhelming, McCully is now back in good graces.

So, understandably, much of the book is devoted to exposing all the activities that can raise homocysteine levels and what to eat to lower them (pp. 81–102), and there is a table of what to supplement at each measured serum level of homocysteine (p. 117)—if diet alone does not suffice—and other vitamin advice (pp. 161–163). Each of the following activities are discouraged because they raise homocysteine levels: eating processed foods (p. 10), smoking, or taking the drugs methotrexate, azaribine, Dilantin, phenobarbital, primidone, carbamazepine, valproic acid, and some diuretics (thiazides). Statin drugs come in for well-deserved criticism in several places (see Ravnskov, 2000: 198–211).

Exercise at a moderate level, at least, is considered mandatory. Tables are given of the calories expended in common activities (pp. 146–150); none of the other books have this.

There are a few inconsistencies. The proscription on beer (pp. 140, 204) because it ''is filled with carbohydrates'' is not reasonable, since diet beer contains only 2.5–9 g of carbs per 366 mL bottle, far less than the recommended apple and less than the recommended 1–2 glasses/day of many types of red wine (pp. 131–

133). Saturated fat is to be limited (p. 44) but is acceptable if eaten in meat or fish (pp. 83, 187) and is perfectly fine on page 86. Whole eggs are recommended (pp. 79, 102), but then avoiding yolks is suggested (pp. 91, 199). Low-fat yogurt is unhealthful on page 96 but recommended on pages 102 and 203. Both Häagen-Dazs and Ben & Jerry's ice creams (p. 96) *now* contain corn syrup.

McCully has made an extremely valuable discovery and a great effort to make it accessible to the non-specialist. A revised edition with a simpler arrangement and more consistency would be appreciated.

The No-Grain Diet: Conquer Carbohydrate Addiction and Stay Slim for Life by Joseph Mercola, DO with Alison Rose Levy. New York: Dutton, 2003. vii + 312 pp. $25 (hardcover). ISBN 0-525-94733-7.

The sub-title gives the emphasis *Conquer Carbohydrate Addiction and Stay Slim for Life*. Obesity is partially attributed to the effects of government recommendations, especially the USDA Food Pyramid, which is called a possible product of lobbying and special interests, not science (pp. 22, 34). Recent US Supreme Court rulings were said to reveal conflicts of interest in 6 of the 11 members of the Pyramid committee (p. 10).

The Mercola program is summarized on a single page (vii). Like Atkins and the Eadeses, there are 3 phases: Start-Up (first 3 days), Stabilize (next 50+ days), and Sustain (lifelong). Like the Eadeses, there are 3 levels of diet severity: Booster, Core, and Advanced. So, there are 9 variations in all, the other extreme from Allen and Lutz, Groves, or McCully, each of whom have a simple single plan, as does Smith for each of the 3 food sensitivities in her book. There is a quiz to determine which level of severity to adopt (pp. 90–95). Mercola does not address GI, GL, carb grams, or NME directly, or have tables of them, but he achieves his results by the choices of foods recommended for each phase of each diet. The permitted foods, a wide variety of non-starchy vegetables, fruits, dairy (resumed after a few weeks of abstinence to detect sensitivity), and meat may be eaten *ad libitum*. Many fish, all shellfish, and most pork products are to be avoided in most versions of the diets. There are sample menus for all phases and levels, including a 2-week menu, because Dr. Mercola recognizes that most people have only about 10 types of meals in their diets. There are 48 pages of recipes (pp. 199–247), including Dr. Mercola's Green Juice Drink (celery, cucumber, and fennel subjected to a juicer [p. 200]), but with no preparation times. Of course, nearly all sweets and sweeteners, even stevia (contrary to Bernstein and Smith) and sucralose (contrary to Atkins and Smith), are out. Soy products are out. The main drink is to be purified (not distilled) water, and red wine is permitted without limit (except for alcoholics) once one's target weight level is reached (pp. 120–121). Omega-3 fats are in (albeit mainly as supplements, because so much sea food is out), as are monounsaturated and saturated fats and small amounts of omega-6 fats. Great individualization is encouraged with advice to try eating more or less of this or that food group to

see what happens, and to increase carb consumption only until weight gain reappears, with daily weighing (opposite to Atkins, who suggests it be done once a week or less). Another check is waist size, using a tape or tight-fitting slacks. The claimed success rate is 85% of people who tried this plan.

Digestible carbs, usually called grains, are identified by Mercola and Levy as the main culprits in weight gain because of the insulin response (leading to heart disease) and suppression of glucagon and human growth hormone, as well as leading to CVD, hypertension, NIDDM, osteoporosis, and a number of autoimmune diseases. Stopping grains prevents celiac disease and irritable bowel syndrome (pp. 25–27), but there are no methods given to determine who has these specific conditions. The mainstream myths surrounding cholesterol and saturated fat are demolished (pp. 18–32). For those with profound insulin resistance, a diet with 75–90% of calories from fat at 1000 kcal/day is recommended. A number of standard tests on blood are recommended (pp. 69–72) with a special Appendix (pp. 264–270) that emphasizes thyroid testing, but homocysteine, an important risk factor for heart disease, is conspicuously missing. Mercola has years of clinical experience with about 15,000 patients and feedback from many others via his websites (www.drmercola.com and www.nograindiet.com). About a dozen each of other websites and books are given as sources, but there are no dates, editions, or publishers for the latter. About 120 articles are listed in a bibliography, but with no citations in the text.

To distinguish these diets from those of the other authors, especially Atkins', which is said to have many weaknesses and can fail (p. 6), organically grown food is strongly recommended for the usual reasons (pp. 6, 102, 249). Very high vegetable consumption, with as much raw as possible, is recommended (p. 6). A better transition from the Stabilize to Sustain phases than in the corresponding Weight-Watchers or Atkins plans is claimed, but no evidence is given (pp. 80–81). Metabolic typing to find who should eat what is promoted (pp. 125, 292), but one must go to outside sources to do this. The one really unusual feature of this book (of the books reviewed in this article) is that 20% of the text is on psychological approaches to hunger and food addictions. The scheme is called the Emotional Freedom Technique (EFT), which is a combination of acupressure according to traditional Chinese medicine and use of mantras, which the client may individualize, but the final part of the mantra is to say "I deeply and completely accept myself" (pp. 8–9, 13, 45–46, 80, 85–86, 145–147, 165–195, 258–263). So far it has worked more than half the times tried by this reviewer, but there is no evidence for its effectiveness in this book by comparing results from clients who do or do not use it.

There are a few inconsistencies and omissions. Some of the time "grains" include simple sugars. The most serious is the definition of simple and complex carbs. The former are said to be present in potatoes and grains, as well as in sugars and sweeteners, and the latter in fruits and vegetables (pp. 11, 28, 39, 42, 103). This is at odds with the definitions used by others and is bound to lead to confusion for many readers. Macro- and micronutrients are confused (p. 74), as

are ketosis and ketoacidosis (p. 29). Fructose is among the sweeteners to avoid, yet raw honey is recommended, which contains 39% fructose (p. 118). After the fine endorsement of dietary cholesterol and fat (pp. 18–32), bison and ostrich were recommended over beef, pork, and chicken because the former contain less fat and cholesterol (p. 255). While no limits on protein and fat quantities eaten is the story in the earlier parts of the book, there is an admonition not to eat too much protein, without explaining how much is too much, because so doing is said to damage kidneys and bones, contrary to the findings of Atkins and Bernstein. Coffee is to be eliminated because it ''worsens your insulin levels'' (pp. 120, 131); however, in a recent clinical trial, coffee consumption was inversely and strongly associated with lower risk of NIDDM (van Dam & Feskens, 2002). Figures of 60 lbs, 30 lbs, and $1.5 trillion are given with no time frame (p. 4). Temperatures are given without the C or F (pp. 118–119). Salt is said to change in composition at 1200° (no C or F), but actually it can be distilled at 1413°C with no change. In the calculation of HDL/TC, the decimal point is omitted, making the values 100 times too high (p. 69). Unrefined sea salt is recommended without evidence; see the review of Smith for the likely fallacy. The ''chemical additives'' to be avoided in common salt are calcium silicate to prevent caking and sodium or potassium iodide to prevent goiter; no actual evidence is given against their reasonable use (p. 119). Fish are claimed to be highly contaminated with PCBs, dichlorodiphenyltrichloroethane (DDT), and mercury (pp. 110–111). Actually, PCBs are biodegradable and not very toxic to humans (Ray, 1990: 86–88). The usual metabolite of DDT found in humans is dichlorodiphenyldichloroethane, which has never been shown to have had toxic effects. Listed among fish to avoid because of mercury content are lobster, shrimp, and tuna; however, common tables list these as among the lowest in mercury content. Using the FDA's limit of 0.3 μg/kg body weight daily, one could eat 100 g of tuna or lobster daily without exceeding this limit, which is ⅒ that of the WHO. The Chicago Western Electric Study followed the effects of fish consumption on 2107 men 40–55 years old for 30 years; those who ate the most fish lived the longest (Daviglus et al., 1997). The Nurses' Health Study that followed 84,688 women aged 34–59 years for 16 years showed that those who ate the most fish lived the longest (Hu et al., 2002). If fish and pork (p. 114) were toxic, some of the longest-lived populations in the world (Crete, Japan, Okinawa) would not be so long lived. Eating fiber is strongly recommended (p. 19), but the use of juicers, which separate fiber for disposal, contradicts this.

There are a number of issues addressed in this book that are either not deemed important by the authors of the other books or on which a different position is taken. Most nuts are not recommended due to high omega-6 content (pp. 111–112); this is greatly at odds with the highly significantly lower risks of CHD and all-cause deaths in the oldest old persons in The Adventist Health Study (Fraser & Shavlik, 1997). No consideration is given to extent of cooking on GI for carrots or pasta (p. 105). Microwaves are said to cause the greatest nutrient losses of all cooking methods (pp. 161–162). There is no support given for this,

and it is hard to see how baking, broiling, and grilling could be any gentler. Eggs, said to be high in omega-6 fats, are to be eaten raw, or, if cooked, not scrambled (pp. 107–108, 251–253); there is no evidence whatsoever given for the benefits of doing this. Canola oil was lumped in with safflower and other oils with high omega-6 content despite the fact that the USDA assays show the –6:–3 ratio as 2:1, by far the best of any vegetable oil, and the omega-3 content is the highest (in non-hydrogenated form). Advice not to drink or swim in chlorinated water (pp. 150–151) is not backed by any evidence. Storing water in poly(ethylene) containers is recommended over poly(vinyl chloride) ones to avoid the ''dangerous chemicals'' leached from the latter; there is no evidence given for this. Using reverse osmosis to purify drinking water rather using than distillation, where both methods remove valuable minerals, has no epidemiological support, either. Exercise is recommended for everyone because it ''guarantee[s] ... weight loss'' (contrary to Banting, see above, and Groves, 1999: 88–90) and prevents heart disease; this latter is contradicted by a clinical trial (Dorn et al., 1999).

Except for the criticisms of the Atkins diet, the main messages in this book are the excellent ones, with the same excellent reasons for eating low-carb diets as are used by the other authors for treatment of obesity. There is almost no nonsense about eating cholesterol and saturated fat. The diets are good, if unnecessarily limited in fish, pork, and nuts; and the support of the EFT scheme may be worthwhile. The devil is in the details, as exposed above.

The Modern Nutritional Diseases: Heart Disease, Stroke, Type-2 Diabetes, Obesity, Cancer, and How to Prevent Them by Alice Ottoboni, PhD and Fred Ottoboni, MPH, PhD. Sparks, NV: Vincente Books, 2002, 2nd printing, revised 2003. vi + 218 pp. $30 (paperback). ISBN 0-915241-03-X.

''The American diet over the last hundred years seems to be a story of the triumph of junk science over real science. This is the situation today despite the fact the biochemical pathways that nutrients follow in the body are reasonably well known and competent studies relating diet to human health have scientifically validated the detrimental effects of high-glycemic diets and essential fatty acid imbalances'' (p. 199).

Determined to have non-biochemists follow the scientific evidence for the effect of diet, supplements, and drugs on health, the Ottobonis provide informative word diagrams of metabolic pathways on digestion and beyond, along with clear descriptions. All of this is accompanied by citations in the text to references at the end of each chapter to books, medical journals, and other sources. And all of this is done without using a single chemical structure, certainly a welcome feature to most lay readers. The Ottobonis show how common supplements and drugs influence these metabolic pathways, and they expose the biggest fraud in the history of medicine and diet without restraint—the DHT. Since their interpretation of the science differs so much from that of the desperate

defense of the dictatorial directives by the deceptive defenders of diet dogma, the Ottobonis call their recommendations ''alternative nutrition.'' The authors' advice, which encompasses lifestyle, diet, supplements, and drugs, is given with solid citations and great clarity.

For most people, their recommendation is for a low GI diet with energy from carb:fat:protein of 40:30:30, with homage to Barry Sears, and noting that the human body has no requirement for any carb at all (pp. 85, 90). Not worrying about saturated fats, minimizing omega-6 fats, maximizing omega-3 fats, and eliminating *trans* fats are other important parts of the message, based on published studies and backed by biochemical diagrams showing how the best food leads to low serum insulin levels and the most beneficial eicosanoids. The biochemical path from high-GI carbs to high insulin to undesirable eicosanoids (prostaglandins, etc.) is presented in the clearest manner I have yet seen. For those who cannot accept experimental evidence without a plausible theory, the Ottoboni's provide both. One of the most hard-headed and accurate sections on nutritional supplements is given, with recommendations. This book is informational, and the Ottobonis recommend other books for menus and recipes and give general guidelines for evaluating sources of medical advice (pp. 184–186). Further recommendations include an *ad libitum* intake of water based on thirst (p. 138), 1–2 drinks/day of alcoholic beverages (p. 189), special recognition of homocysteine control (p. 146), the unproven benefits of organic food (p. 185), and the doubtful benefits of non-caloric sweeteners (p. 92). The effect of cooking time on GI is given special attention (p. 85). There is no specific recommendation for individualization or to titrate carb consumption to weight loss or to incipient ketosis, but there is an excellent distinction drawn between ketosis and ketoacidosis (pp. 114–115).

Dozens of their paragraphs are gems of clear, truthful, and practical correlations or conclusions, which should be prize quotations verbatim for many years to come. For example, here are just a few of their accurate observations, all which are presented with ample evidence:

- Acetaminophen is not innocuous.
- Fats made from the 12-carbon lauric acid (12:0) are very beneficial.
- Children on low-fat diets can be harmed seriously.
- Certain vitamins and supplements, (but relatively few herbs) can be very beneficial.
- The vegetarian diet can be perilous.
- Anticholesterol drugs are dangerous.
- A good cholesterol supply in the human body is a vital necessity.

The Ottobonis correctly expose the NCEP Guidelines on diet as an attempt by those with ulterior motives to use the US government umbrella of the NHLBI of the NIH to make it appear that the guidelines have government sanction, and that the process of generating them was the open and complete process we have a right to expect, which would have included public hearings and publication

in the Federal Register and not just in *JAMA*. ''Although the New Cholesterol Guidelines will be a windfall for drug companies and some agricultural interests, they will adversely impact both the practice of medicine and the long-term health of Americans'' (pp. 179–182).

Flaws in dietary studies connecting consumption of saturated fats with the chronic Western diseases are explained. Many examples of better studies are provided, and the contradiction with the Unified Dietary Guidelines of the AHA and other groups still in the Dark Ages regarding GI is exposed fearlessly.

Perhaps the Ottoboni's homage to Barry Sears, author of *Enter The Zone,* should have been qualified. The first edition of the book (September 2002) had less-than-perfect proof-reading and some minor errors in chemistry, nearly all of which were eliminated in the second printing (February 2003), an exception being the meaning of l- and d- prefixes in naming amino acids (p. 95). The recommendation for drinking 1 qt of milk/day (p. 194) is surprising in view of the 44 g of high-GI lactose contained therein (Lentner, 1981: 255). However, these minor complaints do not alter the validity of the conclusions and advice in this marvelous book.

Enter the Zone by Barry Sears, PhD and Bill Lawren. New York: Regan/HarperCollins, 1995. xviii + 328 pp. $25 (hardcover). ISBN 0-06-039150-2.

Brilliant and fearless in debunking the desperate dons of diet dogma—''No cholesterol-lowering diet study has ever decreased total mortality'' (pp. 141–143)—Barry Sears provides a number of insights in the oldest of the books under review (1995), and he reports on a number of small RCTs he initiated without outside funding. The ''Zone'' refers to periods of the highest exhilaration and performance experienced by athletes. Sears' theme is to show how ¾ of us can reach our Zone by eating a low-carb diet. (The other 1/4 are either in their Zone or not harmed by their present diets.)

One of Sears' main themes is that essential fatty acids are converted to good eicosanoids (such as prostaglandin E1, a vasodilator) in the presence of glucagon, the fat-metabolizing hormone and to bad eicosanoids (such as thromboxane A2, a vasoconstrictor) in the presence of insulin, the fat-building hormone (p. 35ff). This is elaborated by noting that thromboxane A2 stimulates the growth of cells in arterial walls to form the lesions of atherosclerosis (p. 149). An interesting offshoot is that insulin activates HMG-CoA reductase, one of the catalysts for cholesterol synthesis. Another is that the poor results in RCTs of antihypertensive drugs may be due to the tendency of the drugs to raise insulin levels (pp. 140–141).

Before and after some history and literature examples supporting his theses, Sears points out that many chronic conditions in aging, obesity, chronic fatigue, and many others can be alleviated by entering the Zone by means of a low-carb diet, because this is the only way to minimize insulin levels. He calls hyperinsulinemia the single most important risk factor for CVD (pp. 136–138).

He is careful to note that about ¼ of people will not obtain a benefit from a low-carb diet; that ¼, including diabetics and the obese, will respond very well; and that ½ will respond well enough to justify maintaining the low-carb diet (pp. 30, 102–103).

The Zone diet—40% carb calories, 30% fat, 30% protein, using the fuel values—is individualized by determining one's LBM, setting the amount of protein high enough to prevent muscle loss (80 g/day for a 70-kg human), matching the protein calories with fat calories (34 g/day), and fixing the calories from carbs at ⅔ those of protein (therefore, 107 g/day of digestible carbs) (pp. 69–76). Since low-GI and high-fiber carbs are strongly recommended, the total carb intake could be 200 g/day. Major supplementation is not needed (pp.105–112). One glass of red wine per day is recommended. A reasonable number of recipes are given (without preparation times), as well as tables for determining LBM, equi-caloric servings of food, rough GI values, and ideal body weights.

In a 1-patient anecdote modestly referred to as an Aunt Millie tale, a CVD patient on a high-carb diet had TG = 650 mg/dL, TC = 229, and HDL = 34. The patient started simvastatin and the Zone diet, which made TG = 108, TC = 152, and HDL = 49. Dropping the drug, but not the diet, made TG = 101, TC = 175, and HDL = 52. ''This ... is a dramatic indication that cholesterol levels are ultimately determined by eicosanoid balance, which is controlled by the food you eat'' (pp. 144–145).

A trial of the Zone diet on 6 college football and 3 professional basketball players, in which all meals in a camp setting were checked by the coach (who was already converted to a Zone diet), was checked against the high-carb diet to which many nutritionists commit athletes. After just 6 weeks, the low-carb diet increased weight 5%, decreased fat 20%, increased LBM 8%, and improved several performance parameters 2–30% (all $P < 0.005$) (pp. 41–44).

An RCT of 15 NIDDM patients was done to compare 8 weeks of ADbA diet of 60% high-GI carb or the Zone diet (40% low-GI carb) with typical American diets to give the following results: (1) ADbA diet = No significant changes occurred in weight loss, fat loss, fasting serum glucose, or the levels of serum TG (up 20%, but not sgnificant), fasting insulin, or blood pressure. Of significance, glycosylated hemoglobin went down 4% and TC up 8%. (2) Zone diet = No significant change in fasting glucose, fasting insulin, or diastolic blood pressure. Significant drops occurred in weight, fat, glycosylated hemoglobin (down 14%), TG, and systolic blood pressure. Another 8 weeks brought diastolic blood pressure down significantly as well. This showed that the ADbA diet was no better than an average American diet, but the Zone diet was far better.

Sears notes the benefits of the Zone diet in obesity, diabetes, cancer, AIDS, arthritis, MS PMS, lupus, chronic fatigue, depression, and other conditions. Fair to excellent evidence is given for every claim of benefit of the Zone diet.

However, Sears does some things that are at odds with other authors and the medical literature. He understands that aspirin has its adverse effects, and that the male Physician Health Group (PHG) study, after 4 years, found no change in all-cause death rates. He thinks a longer period would change the outcome, but he is ignorant of the 7-year results that had been reported in 1989 (Kauffman, 2000) and, of course, of a newer, 3.1-year study (reported in 2001) that found that more women died on aspirin than on placebo (Kauffman, 2002b). Sears actually calculates the expected death rates based on non-fatal heart attacks in the PHG study rather than using the actual ones! He sends mixed messages on aspirin, the ''miracle drug'' of eicosanoid control, yet notes some of its long-term problems (pp. 113–118, 138, 151–152, 161).

He recommends avoiding saturated fats and egg yolks, accentuating this for cancer patients, for whom he emphasizes sea food and sea vegetables. There is good evidence that saturated fat and meat do *not* cause cancer or CVD (Allen & Lutz, 2000: 172–177; Malhotra, 1967; Ottoboni & Ottonobi 2003: 192), nor do whole eggs (see Atkins, 2002: 96; Hu et al., 1999). Your reviewer cannot help noting that the Japanese eat sea food and sea vegetables and not much red meat in the past and have had a high cancer rate.

In one place, Sears writes that omega-6 fatty acids in the diet lead to good eicosanoids and are more important than omega-3s (p. 120), then he later writes that he had tried to grow a source of a ''good'' omega-6 fatty acid (gamma-linolenic acid), borage oil, in Canada but eventually realized that most people obtained enough from their normal diets (pp. 129–131). It is puzzling that he does not recommend canola oil, an excellent source of alpha-linolenic acid, an omega-3 that the body converts to good eicosanoids (Enig, 2000: 29; Lemaitre et al., 2003). In his newer book, *The Omega Rx Zone,* 2002, the omega-3 and -6 fatty acid message is greatly improved, but the message on saturated fats is not.

Sears writes that all mammals have the same responses to food (p. 12) and that rabbits, which are both mammals and vegetarians, develop atherosclerosis when fed saturated fat. That this is true or is relevant to humans is strongly disputed (Ravnskov, 2000: 137–138).

While very conscious of the benefits of the Zone diet for diabetics, Sears does not address those who cannot tolerate diets as high as 40% carbs (pp. 152–160).

There is a bibliography with about 400 entries, but there are no citations in the text. Despite the many errors and rigidities in this book, the important insights based on biochemistry and the striking results of Sears' trials have made this book a benefit to over 4 million readers.

Going Against the Grain: How Reducing and Avoiding Grains Can Revitalize Your Health by Melissa Diane Smith. Chicago: Contemporary Books, 2002. xv + 304 pp. $15 (paperback). ISBN 0-658-01722-5.

In *Going Against the Grain,* Smith tackles 3 serious diet-related problems: (1) diabetes-related carb sensitivity, which is also addressed by most of the other books in this review, and about which she has written a book (Challem et al., 2000); (2) gluten sensitivity; and (3) wheat sensitivity. No indication is given of the fraction of people afflicted with carb sensitivity, but gluten sensitivity is put at 10–50% and other food allergies at 10–60%. A small US study is described in which 221 adults in a shopping mall who did not admit to having a GI disease were tested for an antibody that indicates gluten sensitivity: 10% had the antibody; 20 of these 22 were further tested; and 1 had celiac disease (p. 77), which is in line with other studies on prevalence (p. 69). The appalling number of health conditions that can result from persisting with a poor and even addictive high-grain diet are given, from cancer to arthritis to osteoporosis and many more. A good historical review is provided.

The USDA Food Pyramid comes in for a well-deserved condemnation (p. 5ff). A history of long-term human diet changes is given. ''Grains aren't the holier-than-thou health foods that people think they are'' (p. 277). ''Grains may seem the staff of life, but they're really scythes that whittle away most people's health . . . To stay fit and free of disease, all of us should eat fewer grains. Some of us should eat no grains at all'' (p. ix). Smith finds, with difficulty, that she herself is gluten sensitive (pp. xi–xiii).

Quizzes are given to diagnose each of the 3 conditions, with points for certain answers, so the reader may diagnose herself. Other medical tests, with emphasis on accurate and non-invasive ones, are recommended for confirmation. The concepts of GI and GL are covered, but the effect of cooking on the GI of carrot or pasta is not given. The presence of undesirable antinutrients (phytate, etc.) in whole grains is cited (pp. 47–65). Diets are given for each condition with great emphasis on observing one's own reactions to food, and thus to individualize each diet. A week of sample menus is provided, as are a number of recipes, which clearly indicate whether they are for the carb, gluten, or wheat sensitive. These do not have the NME or digestible carb content or GI, GL, or preparation times. The major problem with this otherwise very useful and enlightening book is not that the recommended diets are either low carb, low gluten, or low wheat, or all 3, which are all fine, but that the diets also appear to be low in fat, certainly in land animal fat, with a heavy emphasis on the use of olive oil as shown by a week of sample menus (pp. 209–212, 219–222, 228–233). In fact, an RCT of 2-years duration showed that olive oil was inferior to animal fat in protection from cardiac events (Rose et al., 1965). Lean cuts of meat are recommended, as well as coconut oil; the latter is called ''a source of saturated fat that does not contribute to heart disease'' (p. 174). Coconut oil contains 30% of the same longer-chain (14–18 carbon atoms) saturated fats as animal fat; none of these fats is a cause of CVD (Ravnskov, 2000). A ''high'' TC (pp. xiv, 27, 42, 180, 207–208, 263) is not a cause of CVD, either (Ravnskov, 2000). Oddly, the 18-carbon stearic acid, the main saturated one

in animal fat, is called nonatherogenic on page 11 and is said not to be as prevalent in the fat of today's grain-fed animals (but see p. 22), yet fat is to be removed from meat when cooking it (pp. 239–240)! Surviving eating out, unsympathetic associates and feeding children are nicely addressed except for the fat issue. Asthma symptoms in children who consumed milk fat were much rarer than in children who did not (see Wijga et al., 2003). Smith addresses the GI of protein for the very carb sensitive by recommending replacement of some protein by fat, but it is all plant fat (p. 213) except for omega-3 fish oil supplements, despite recent evidence that neither saturated nor total fat intake increases the risk for NIDDM (van Dam et al., 2002). Coffee, tea, and nuts are considered acceptable, with an unusual warning against coffee substitutes that contain gluten (p. 164). Genetically engineered foods are disparaged (p. 101) and organic foods are promoted, both on theoretical grounds with no support.

Other chemistry-related issues are disappointing. Unrefined Real Salt™ is recommended (pp. 174, 200) on theoretical grounds. In fact, this salt contains 98.3% sodium chloride and negligible amounts of anything beneficial (http://waltonfeed.com/self/labels/saltreal.html). In a generally good chapter on supplements, homocysteine is not even mentioned in connection with the B vitamins (p. 261) and is marginalized elsewhere. Vitamin C made from beets or sago palm is recommended rather than vitamin C from the usual source, corn, for people very sensitive to corn (p. 261), as though allergens (proteins in this case) would survive a multi-step synthesis with crystalline intermediates, because some of Smith's corn-sensitive clients told her they reacted badly (e-mail, Smith, July 29, 2003). Egg powder and other refined powders are mentioned as alternate protein sources (p. 139), but with caveats (see McCully, p. 117, this article). Distilled vinegar and alcohol are condemned as though the gliadin fraction of gluten of minimum molecular weight 11,000 daltons (de Man, 1999: 153–156) of such a protein could vaporize during distillation, but Smith also notes that alcohol increases intestinal permeability, which increases allergic reactions, which she urges individuals to watch for (pp. 100, 149, 166, 173, 178). Use of DDT was damned (p. 101) despite lack of evidence for harm (Lagomasini, 2002: 149–177). Unrefined grains are noted to be higher in fiber than are refined grains, but no mention is made of their protein and fat content (pp. 32, 47ff), which is significant (11% protein in 1 whole-wheat pasta). Cramps are said to be due to a calcium deficiency (p. 58), but other sources indicate that low magnesium may be another or additional culprit (Atkins, 1998: 121–131). It is true that magnesium is recommended for other conditions (pp. 261, 266). Exorphins are said to have similar amino acid sequences to those in morphine (p. 95); morphine actually contains no amino acids.

There are about 150 references with citations in the text; more would have been welcomed to back many of the unsupported statements. Other books, magazines, websites, and connections to enlightened nutritionists or nutrition-

oriented physicians are cited; this is an especially valuable feature of this book. The lack of knowledge about food allergies and nutrition of conventionally educated MDs Smith notes is all too true. In this reviewer's opinion, such victims of food allergies would be much better off consulting with Smith than with typical MDs, who typically recommend low-fat diets but rarely low-carb or low-grain diets. The main messages of this book are extremely valuable and apply to the broadest variety of food problems. A rewrite with more reasonable diets and the help of a biochemist would make it even more valuable.

Books on Low-Carb Diets *Not* Reviewed

The authors of the following books, which will not be reviewed, also favor low-carb diets. By no means is this list an exhaustive one:

Cleave, T. L. (1975). *The Saccharine Disease: The Master Disease of Our Time*. New Canaan, CT: Keats.

Cordain, L. (2001). *The Paleo Diet: Lose Weight and Get Healthy by Eating the Food You Were Designed to Eat*. New York: John Wiley & Sons.

Ezrin, C., Kowalski, R. E., & Kowalski, R. E. (1997). *Your Fat Can Make You Thin* (2nd ed.). New York: McGraw-Hill/Contemporary Books.

Gittleman, A. L. (1999). *Eat Fat, Lose Weight*. Los Angeles: Keats.

Price, W. A. (2002). *Nutrition and Physical Degeneration* (6th ed.). New York: McGraw Hill-NTC.

Steward, H. L., Bethea, M., Andres, S., & Balart, L. (1995). *Sugar Busters*. New York: Ballantine Books.

Tarnower, H., & Baker, S. (1995). *The Complete Scarsdale Medical Diet*. New York: Bantam Books.

"Men occasionally stumble over the truth, but most pick themselves up and hurry off as if nothing had happened."

Winston Churchill (McGee, 2001: 82).

Acknowledgments

Expert online searches and editorial aid were provided by Leslie Ann Bowman, AMLS, and other faculty at the University of the Sciences in Philadelphia, including a complete critical reading of the manuscript by William Reinsmith, DA. The searching skills of Wendy H. Kramer, MLS, at the Eastern Regional Research Center of the USDA, Wyndmoor, PA, were extremely valuable. Many members of The International Cholesterol Skeptics Group provided information and/or editorial aid (www.THINCS.org). Thanks to Henry H. Bauer for suggesting this format.

References

Ahrens, D. (1999). Industrial thin-film deodorization of seed oils with SoftColumn™ technology. *Fett/Lipid, 101,* 230–234.

Anderson, K. M., Castelli, W. P., & Levy, D. (1987). Cholesterol and mortality. 30 years of follow-up from the Framingham Study. *Journal of the American Medical Association, 257,* 2176–2180.

Apgar, J. L., Shively, C. A., & Tarka, S. M. (1987). Digestibility of cocoa butter and corn oil and their influence on fatty acid distribution in rats. *Journal of Nutrition, 117,* 660–665.

Atkins, R. C. (1998). *Dr Atkins Vita-Nutrient Solution*. London: Pocket Books, Simon & Schuster.

Atwater, W. O., & Snell, J. F. (1903). Description of a bomb calorimeter and method of its use. *Journal of the American Chemical Society, 25*(7), 659–699.

Bantle, J. P., Raatz, S. K., Thomas, W., & Georgopoulos, A. (2000). Effects of dietary fructose on plasma lipids in healthy subjects. *American Journal of Clinical Nutrition, 72,* 1128–1134.

Berg, J. M., Tymoczko, J. L., & Stryer, L. (2002). *Biochemistry* (5th ed.). New York: W. H. Freeman. pp. 440–441.

Bravata, D. M., Sanders, L., Huang, J., Krumholz, H. M., Olkin, I., Gardner, C. D., & Bravata, D. M. (2003). Efficacy and safety of low-carbohydrate diets: A systematic review. *Journal of the American Medical Association, 289*(14), 1837–1850.

Brehm, B. J., Seeley, R. J., Daniels, S. R., & D'Alessio, D. A. (2003). A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. *Journal of Clinical Endocrinology & Metabolism, 88*(4), 1617–1623.

Carlson, W. E., & Bayley, H. S. (1968). Utilization of fat by young pigs: Fatty acid composition of ingesta in different regions of the digestive tract and apparent and corrected digestibilities of corn oil, lard and tallow. *Canadian Journal of Animal Science, 48,* 315–322.

Challem, J., Berkson, B., & Smith, M. D. (2000). *Syndrome X: The Complete Nutrtional Program to Prevent and Reverse Insulin Resistance*. New York: John Wiley & Sons.

Chen, I. S., Subramanian, S., Vahouny, G. V., Cassidy, M. M., Ikeda, I., & Kritchevsky, D. (1989). A comparison of the digestion and absorption of cocoa butter and palm kernel oil and their effects on cholesterol absorption in rats. *Journal of Nutrition, 119,* 1569–1573.

Cohen, B. L. (2003). Risks in perspective. *Journal of American Physicians and Surgeons, 8*(2), 50–53.

Cranton, E. M. (2001). *A Textbook on EDTA Chelation Therapy* (2nd ed.). Charlottesville, VA: Hampton Roads.

Crick, D. C., Nwokolo, E. N., & Sims, J. S. (1988). Effect of blending dietary oils on growth performance, total and individual fatty acid absorption by the growing chick. *Nutrition Research, 8,* 643–651.

de Man, J. M. (1999). *Princples of Food Chemistry* (3rd ed.). Gaithersburg, MD: Aspen.

De Schrijver, R., Vermeulen, D., & Viaene, E. (1991). Lipid metabolism responses in rats fed beef tallow, native or randomized fish oil and native or randomized peanut oil. *Journal of Nutrition, 121,* 948–955.

Daviglus, M. L., Stamler, J., Orencia, A. J., Dyer, A. R., Liu, K., Greenland, P., Walsh, M. K., Morris, D., & Shekelle, R. B. (1997). Fish consumption and the 30-year risk of fatal myocardial infarction. *New England Journal of Medicine, 336,* 1046–1053.

Deckelbaum, R. J., Fisher, E. A., Winston, M., Kumanyika, S., Lauer, R. M., Pi-Sunyer, F. X., St. Jeor, S., Schaefer, E. J., & Weinstein, I. B. (1999). Summary of a scientific conference on preventive nutrition: Pediatrics to geriatrics. *Circulation, 100,* 450–456.

Dock, W. (1953). The reluctance of physicians to admit that chronic disease may be due to faulty diet. *Journal of Clinical Nutrition, 5,* 1345–1347.

Dorn, J., Naughton, J., Imamura, D., & Trevisan, M. (1999). Results of a multicenter randomized clinical trial of exercise and long-term survival in myocardial infarction patients. *Circulation, 100,* 1764–1769.

Enig, M. G. (2000). *Know Your Fats.* Silver Spring, MD: Bethesda Press.

Enig, M. G. (2003). NHLB Consensus. Personal e-mail of August 9.

Finley, J. W., Klemann, L. P., Leveille, G. A., Otterburn, M. S., & Walchak, C. G. (1994a). Caloric availability of SALATRIM in rats and humans. *Journal of Agriculture and Food Chemistry, 42,* 495–499.

Finley, J. W., Leveille, G. A., Klemann, L. P., Sourby, J. C., Ayres, P. H., & Apppleton, S. (1994b).

Growth method for estimating the caloric availability of fats and oils. *Journal of Agriculture and Food Chemistry, 42,* 489–494.

Ford, E. S., Giles, W. H., & Dietz, W. H. (2002). Prevalence of the metabolic syndrome among US adults. *Journal of the American Medical Association, 287*(3), 356–359.

Foster, G. D., Wyatt, H. R., Hill, J. O., McGuckin, B. G., Brill, C., Mohammed, B. S., Szapary, P. O., Rader, D. J., Edman, J. S., & Klein, S. (2003). A randomized trial of a low-carbohydrate diet for obesity. *New England Journal of Medicine, 348*(21), 2082–2090.

Foster-Powell, K., Holt, S. H. A., & Brand-Miller, J. C. (2002). International table of glycemic index and glycemic load values: 2002. *American Journal of Clinical Nutrition, 76,* 5–56.

Fraser, G. E., & Shavlik, D. J. (1997). Risk factors for all-cause and coronary heart disease mortality in the oldest-old. The Adventist Health Study. *Archives of Internal Medicine, 157,* 2249–2258.

Fuchs, C. S., Giovannucci, E. L., Colditz, G. A., Hunter, D. J., Stampfer, M. J., Rosner, B., Speizer, F. E., & Willett, W. C. (1999). Dietary fiber and the risk of colorectal cancer and adenoma in women. *New England Journal of Medicine, 348*(3), 169–176.

Gaziano, J. M., Hennekens, C. H., O'Donnell, C. J., Breslow, J. L., & Buring, J. E. (1997). Fasting triglycerides, high-density lipoprotein, and risk of myocardial infarction. *Circulation, 96,* 2520–2525.

Hénon, G., Kemény, Z., Recseg, K., Zwobada, F., & Kovari, K. (1999). Deodorization of vegetable oils. Part I.: Modeling the geometrical isomerization of polyunsaturated fatty acids. *Journal of the American Oil Chemists Society, 76,* 73–81.

Hoagland, R, & Snider, G. C. (1942). Digestibility of some animal and vegetable fats. *Journal of Nutrition, 25,* 295–302.

Hu, F. B., Bronner, L., Willett, W. C., Stampfer, M. J., Rexrode, K. M., Albert, C. N., Hunter, D., & Manson, J.-A. E. (2002). Fish and omega-3 fatty acid intake and risk of coronary heart disease in women. *Journal of the American Medical Association, 287*(14), 1815–1821.

Hu, F. B., Stampfer, M. J., Rimm, E. B., Manson, J. E., Ascherio, A., Colditz, G. A., Rosner, B. A., Spiegelman, D., Speizer, F. E., Sacks, F. M., Hennekens, C. H., & Willett, W. C. (1999). A prospective study of egg consumption and risk of cardiovascular disease in men and women. *Journal of the American Medical Association, 281*(15), 1387–1394.

Kauffman, J. M. (2000). Should You Take Aspirin to Prevent Heart Attack? *Journal of Scientific Exploration, 14*(4), 623–641.

Kauffman, J. M. (2001). Book review of *The Cholesterol Myths* by Uffe Ravnskov (2000). *Journal of Scientific Exploration, 15*(4), 531–540.

Kauffman, J. M. (2002a). Alternative medicine: Watching the watchdogs at Quackwatch, website review. *Journal of Scientific Exploration, 16*(2), 312–337.

Kauffman, J. M. (2002b). Aspirin study flawed [Letter to the editor]. *Journal of Scientific Exploration, 16*(2), 247–249.

Kekwick, A., & Pawan, G. L. S. (1969). Body-weight, food, and energy. *Lancet,* 822–825.

Klevay, L. M. (1993). Copper in nuts may lower heart disease risk. *Archives of Internal Medicine, 153,* 401–402.

Lagomasini, A. (2002). Chemical warfare: Ideological enviromentalism's quixotic campaign against synthetic chemicals. In Bailey, R. (Ed.), *Global Warming and Other Eco-Myths.* New York: Forum Random House Prima.

Layman, D. K., Boileau, R. A., Erickson, D. J., Painter, J. E., Shiue, H., Sather, C., & Christou, D. D. (2003). A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles during weight loss in adult women. *Journal of Nutrition, 133,* 411–417.

Lee, K. W., & Lip, G. Y. H. (2003). The role of omega-3 fatty acids in the secondary prevention of cardiovascular disease. *Quarterly Journal of Medicine, 96,* 465–480.

Lemaitre, R. N., King, I. B., Mozzaffarian, D., Luller, L. H., Tracy, R. P., & Siscovick, D. S. (2003). n-3 Polyunsaturated fatty acids, fatal ischemic heart disease, and nonfatal myocardial infarction in older adults: The Cardiovascular Health Study. *American Journal of Clinical Nutrition, 77,* 319–325.

Lentner, C., Ed. (1981). *Geigy Scientific Tables* (Excerpts, 8th ed.). West Caldwell, NJ: CIBA-Geigy.

Lieb, C. W. (1929). The effects on human beings of a twelve months' exclusive meat diet. *Journal of the American Medical Association, 93*(1), 20–22.

Liu S., Sesso, H. D., Manson, J. E., Willett, W. C., & Bering, J. E. (2003). Is intake of breakfast

cereals related to total and cause-specific mortality in men? *American Journal of Clinical Nutrition, 77,* 594–599.

Liu, S., Willett, W. C., Stampfer, M. J., Hu, F. B., Franz, M., Sampson, L., Hennekens, C. H., & Manson, J. E. (2000). A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *American Journal of Clinical Nutrition, 71,* 1455–1461.

Livesy, G. (2001). A perspective on food energy standards for nutrition labeling. *British Journal of Nutrition, 85,* 271–287.

Malhotra, S. L. (1967). Serum lipids, dietary factors and ischemic heart disease. *American Journal of Clinical Nutrition, 20,* 462–474.

Malyutina, S., Bobak, M., Kurilovitch, S., Gafarov, V., Simonova, G., Nikitin, Y., & Marmot, M. (2002). Relation between heavy and binge drinking and all-cause and cardiovascular mortality in Novosibirsk, Russia: A prospective cohort study. *Lancet, 360,* 1448–1454.

Mann, G. V. (1977). Diet-heart: End of an era. *New England Journal of Medicine, 297*(12), 644–650.

Mark, L., Erdei, F., Markizay, J., Kondacs, A., & Katona, A. (2002). Effect of treatment with folic acid and vitamin B6 on lipid and homocysteine concentrations in patients with coronary artery disease. *Nutrition, 18*(5), 428–429.

McClellan, W. S., & Du Bois, E. F. (1930). Prolonged meat diets with a study of kidney function and ketosis. *Journal of Biological Chemistry, 87,* 651–668.

McGee, C. T. (2001). *Heart Frauds: Uncovering the Biggest Health Scam in History.* Colorado Springs, CO: HealthWise.

Montonen, J., Knekt, P., Järvinen, R., Aromaa, A., & Reunanen, A. (2003). Whole-grain and fiber intake and the incidence of type 2 diabetes. *American Journal of Clinical Nutrition, 77,* 622–629.

Moore, T. J. (1989). *Prescription for Disaster. The Hidden Dangers in Your Medicine Cabinet.* New York: Simon & Schuster.

Morris, J. N., et al. for the MRC Social Medicine Research Unit. (1968). Controlled trial of soya-bean oil in myocardial infarction. *Lancet,* 693–699.

MRFIT Research Group (1982). Multiple Risk Factor Intervention Trial. Risk factor changes and mortality results. *Journal of the American Medical Association, 248*(12), 1465–1477.

Newbold, H. L. (1988). Reducing the serum cholesterol level with a diet high in animal fat. *Southern Medical Journal, 81*(1), 61–63.

O'Brien, L. J. (2003). Misplaced notions of simplicity, denial of complexity (i. e., value of randomized controlled clinical trials). *British Medical Journal Electronic,* 11 July.

O'Mathúna, D. P. (2003). The placebo effect and alternative therapies. *Alternative Medicine Alert, 6*(6), 61–69.

O'Shea, J. C., & DeMets, D. L. (2001). Statistical issues relating to international difference in clinical trials. *American Heart Journal, 142,* 21–28.

Ottoboni, F., & Ottoboni, A. (2003). Can attention deficit-hyperactivity disorder result from nutritional deficiency? *Journal of American Physicians and Surgeons, 8*(2), 58–60.

Pearce, M. L., & Dayton, S. (1971). Incidence of cancer in men on a diet high in polyunsaturated fat. *Lancet,* 464–467.

Pelton, R., & LaValle, J. B. (2000). *The Nutritional Cost of Prescription Drugs.* Englewood, CO: Perspective/Morton.

Peterson, C. B., & Vik-Mo, L. (1968). Determination of digestibility and metabolizable energy in pure fats and discussion of analytical methods employed in experiments with growing chickens. *Acta Agricultura Scandinavia, 18,* 42–48.

Raben, A., Vasilaras, T. H., Møller, A. C., & Astrup, A. (2002). Sucrose compared with artificial sweeteners: Different effects on *ad libitum* food intake and body weight after 10 wk of supplementation in overweight subjects. *American Journal of Clinical Nutrition, 76,* 721–729.

Ravnskov, U. (1998). The questionable role of saturated and polyunsaturated fatty acids in cardiovascular disease. *Journal of Clinical Epidemiology, 51,* 443–460.

Ravnskov, U. (2000). *The Cholesterol Myths: Exposing the Fallacy that Saturated Fat and Cholesterol Cause Heart Disease.* Washington, DC: New Trends.

Ravnskov, U. (2002a). Is atherosclerosis caused by high cholesterol? *Quarterly Journal of Medicine, 95,* 397–403.

Ravnskov, U. (2002b). A hypothesis out-of-date: The diet-heart idea. *Journal of Clinical Epidemiology, 55,* 1057–1063.

Ravnskov, U., Allen, C., Atrens, D., Enig, M. G., Groves, B. M., Kauffman, J. M., Kronfeld, R., Rosch, P. J., Rosenman, R., Werkö, L., Vesti-Nielsen, J., Wilske, J., & Worm, N. (2002). Studies of dietary fat and heart disease. *Science, 292,* 1464–1465.

Ray, D. L., & Guzzo, L. (1990). *Trashing the Planet.* Washington, DC: Regnery Gateway.

Risérus, U., Abner, P., Brismar, K., & Vessby, B. (2002a). Treatment with dietary *trans*10*cis*12 conjugated linoleic acid causes isomer-specific insulin resistance in obese men with the metabolic syndrome. *Diabetes Care, 25*(9), 1516–1521.

Risérus, U., Basu, S., Jovinge, S., Fredrikson, G. N., Årnlov, J., & Vessby, B. (2002b). Supplementation with conjugated linoleic acid causes isomer-dependent oxidative stress and elevated C-reactive protein. A potential link to fatty acid-induced insulin resistance. *Circulation, 106,* 1825–1929.

Rose, G. A., Thomson, W. B., & Williams, R. T. (1965). Corn oil in treatment of ischaemic heart disease. *British Medical Journal,* 1531–1533.

Salmeron, J., Manson, J. E., Stampfer, M. J., Colditz, G. A., Wing, A. L., & Willett, W. C. (1997). Dietary fiber, glycemic load and risk of non-insulin-dependent diabetes mellitus in women. *Journal of the American Medical Association, 277*(6), 472–477.

Samaha, F. F., Iqbal, N., Seshadri, P., Chicano, K. L., Daily, D. A., McGrory, J., Williams, T., Williams, M., Gracely, E. J., & Stern, L. (2003). A low-carbohydrate as compared with a low-fat diet in severe obesity. *New England Journal of Medicine, 346,* 476–483.

Seshadri, S., Beiser, A., Selhub, J., Jacques, P. F., Rosenberg, I. H., D'Agostino, R. B., Wilson, P. W. F., & Wolf, P. A. (2002). Plasma homocysteine as a risk factor for dementia and Alzheimer's Disease. *New England Journal of Medicine, 348,* 2074–2081.

Smith, R. E., & Pinckney, E. R. (1991). *The Cholesterol Conspiracy.* St. Louis, MO: Warren H. Green.

Sondike, S. B., Copperman, N., & Jacobson, M. S. (2003). Effects of a low-carbohydrate diet on weight loss and cardiovascular risk factors in overweight adolescents. *Journal of Pediatrics, 142,* 253–258.

Stanger, O., Semmelrock, H.-G., Wonisch, W., Bös, U., Pabst, E., & Wascher, T. C. (2002). Effects of folate treatment and homocysteine lowering on resistance vessel reactivity in atherosclerotic subjects. *Journal of Pharmacology and Experimental Therapeutics, 303,* 158–162.

Stehbens, W. E. (2001). Coronary heart disease, hypercholsterolemia, and atherosclerosis II. Misrepresented data. *Experimental and Molecular Pathology, 70,* 120–139.

Theobald, H., Johansson, S., Bygren, L., & Engfeldt, P. (2001). The effects of alcohol consumption on mortality and morbidity: A 26-year follow-up study. *Journal of Studies on Alcohol, 62*(6), 783–789.

Thresher, J. S., Podolin, D. A., Wei, Y., Mazzeo, R. S., & Pagliasotti, M. J. (2000). Comparison of the effects of sucrose and fructose on insulin action and glucose tolerance. *American Journal of Physiology, Regulatory Integrative and Comparative Physiology, 279,* R1334–R1340.

van Dam, R. M., & Feskens, J. M. (2002). Coffee consumption and risk of type 2 diabetes. *Lancet, 360,* 1477–1478.

van Dam, R. M., Rimm, E. B., Willett, W. C., Stampfer, M. J., & Hu, F. B. (2002). Dietary patterns and risk for type 2 diabetes mellitus in U. S. men. *Annals of Internal Medicine, 136,* 201–209.

Vasan, R. S., Reiser, A., D'Agostino, R. B., Levy, D., Selhub, J., Jacques, P. F., Rosenberg, I. H., & Wilson, P. W. F. (2003). Plasma homocysteine and risk for congestive heart failure in adults without prior mydocardial infarction. *Journal of the American Medical Association, 289*(10), 1251–1257.

Vollset, S. E., Refsum, H., Tverdal, A., Nygård, O., Nordrehaug, J. E., Tell, G. S., & Ueland, P. (2001). Plasma total homocysteine and cardiovascular and noncardiovascular mortality: The Hordaland Homocysteine Study. *American Journal of Clinical Nutrition, 74,* 130–136.

Whitaker, J. M. (1987). *Reversing Diabetes.* New York: Warner Books.

Whitehead, C. C., & Fisher, C. (1975). The utilization of various fats by turkeys of different ages. *British Journal of Poultry Science, 16,* 481–485.

Wijga, A. H., Smit, H. A., Kerkhof, M., de Jongste, J. C., Gerritsen, J., Neijens, H. J., Boshuizen, H. C., & Brunekreef, B. (2003). Association of consumption of products containing milk fat

with reduced asthma risk in pre-school children: The PIAMA birth cohort study. *Thorax, 58,* 567–572.

Wiley, H. W., & Bigelow, W. D. (1898). Calories of combustion in oxygen of cereals and cereal products, calculated from analytical data. *Journal of the American Chemical Society, 20,* 304–316.

Williams, K. I. (1907). The chemical composition of cooked vegetable spreads. *Journal of the American Chemical Society, 29,* 574–582.

Wolfe, S. M. (2001a). The diabetes drug metformin (glucophage) and lactic acidosis. A reminder about a potentially fatal adverse drug reaction. *Worst Pills Best Pills News, 7*(7), 49–51.

Wolfe, S. M. (2001b). Warning! GlaxoSmithKline's disregard for the safety of diabetics taking rosiglitazone (Avandia). *Worst Pills Best Pills News, 7*(9), 68–69.

Wolff, R. L. (1992). *trans*-Polyunsaturated fatty acids in French edible rapeseed and soybean oils. *Journal of the American Oil Chemists Society, 69*(2), 106–110.

Wolff, R. L. (1993). Further studies on artificial geometrical isomers of a-linolenic acid in edible linolenic acid-containing oils. *Journal of the American Oil Chemists Society, 70*(3), 219–224.

APPENDIX

Abbreviations

ADbA	American Diabetes Association
ADta	American Diatetic Association
AHA	American Heart Association
BMI	Body mass index
BU	Bread unit (12 g of digestible carb)
carb	Carbohydrate
CLA	Conjugated linoleic acid
CVD	Cardiovascular disease (same as CHD)
DDT	Dichlorodiphenyl-trichloromethane
DHT	Diet-Heart Theory
ECC	Effective carbohydrate content
EDTA	Ethylenediaminetetraacetic acid
EFT	Emotional Freedom Technique
FDA	Food & Drug Administration
GI	Glycemic index
GL	Glycemic load
HDL	High-density lipoprotein
IDDM	Insulin-dependent diabetes mellitus (Type 1, juvenile onset)
JAMA	*Journal of the American Medical Association*
LBM	Lean body mass
LDL	Low-density lipoprotein
MRFIT	Multiple Risk Factor Intervention Trial
NHLBI	National Heart, Lung and Blood Institute
NIDDM	Non-insulin-dependent diabetes mellitus (Type 2, adult onset)
NIH	National Institutes of Health (USA)
NME	Net metabolizable energy
PCBs	Polychlorinated biphenyls
PHG	Physicians Health Group (also PHS)
PVC	Poly(vinyl chloride)
RCT	Randomized controlled trial
TC	Total cholesterol
TG	Triglycerides
USDA	United States Department of Agriculture
WHO	World Health Organization