The Food Guide Pyramid: Will the Defects Be Corrected?

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ABSTRACT

The USDA-sponsored Dietary Guidelines for Americans (DGA) and its Food Guide Pyramid are nutritionally and biochemically unsound. The DGA was nevertheless accepted wholeheartedly by nutrition authorities, who took Ancel Keys as their guiding spirit and his lipid hypothesis their mantra. They radically changed the food habits of tens of millions of Americans in a massive human experiment that has gone awry. Much evidence suggests that the current epidemics of cardiovascular diseases, type-2 diabetes, and obesity, even in young children, may be the result. The 2005 changes in the DGA and Food Guide Pyramid will add complexity but will not correct the errors.

Background

The Food Guide Pyramid is a graphical representation of the government-sponsored dietary plan that had its origins in the mid-1950s, when nutrition pioneer Ancel Keys published a paper linking coronary heart disease to dietary fat.¹ This marked the beginning of an era of medical interest in the association between diet and cardiovascular disease. Previously, dietary recommendations by the U.S. Department of Agriculture (USDA) and other health organizations were directed toward informally advising the public on nutritional requirements for the basic food groups, vitamins, and minerals. After the diet-heart link was proposed, the government's dietary advice shifted from how to obtain adequate nutrients to how to avoid excessive intakes of food components, such as dietary fats and cholesterol, that were linked to chronic diseases.²

The research of Ancel Keys and colleagues became widely publicized and popular. The nutrition community enthusiastically adopted the lipid hypothesis, the label for the diet-heart connection. The public was encouraged to eliminate the use of butter and other animal fats, whole milk products, and eggs. Vegetable fats (margarines and salad and cooking oils) were listed as the only acceptable dietary fats. Red meats were declared unhealthful because their fats contain cholesterol; they were stigmatized with the label "artery clogging." The whole nation was urged to follow these new guidelines.

By the early 1970s, the lipid hypothesis was fully accepted as fact by the nutrition community. However, because the public was receiving varying and inconsistent information on how to implement these guidelines, it was decided that authoritative and consistent guidance on diet and health was required. The USDA responded with publication of *Nutrition and Your Health: Dietary Guidelines for Americans* (DGA) in 1980.² The dietary plan set



Figure 1. Current Food Guide Pyramid

forth in the DGA was designed to slow or stop the increasing rates of heart disease and stroke. These diseases were essentially unknown in the late 1800s.³ The implicit message of the DGA, which is still the cornerstone of federal nutrition policy,⁴ was that its dietary recommendations were heart healthy and would benefit all who followed them.

Within a few years, it was decided that a simple diagram was required to promote the DGA more intensively. Despite decadeslong promotion and implementation of the heart-healthy diet, the incidence of heart disease had not slowed but continued to climb. Rather than consider that the DGA diet might be to blame, it was assumed that the public was not following the DGA diet with sufficient zeal. Thus, the Food Guide Pyramid was developed and presented to the public in 1992.²

The Pyramid is a graphic subdivided into sections, each of which represents a specific food group (Figure 1). Each section gives the number of portions of its food group that should be consumed each day in order to provide 16 to 20 percent protein, less than 30 percent fat, and the balance (approximately 50 to 60 percent) carbohydrate, all as a percent of total daily calorie intake.⁵ Although the Pyramid is widely recognized and used, it must be remembered in the discussion that follows that the Pyramid does not stand alone but is merely a simplified representation of the much more detailed official DGA.

There is considerable concern today that the diet the Pyramid illustrates is responsible for the current epidemic of cardiovascular disease. The concurrent epidemics of obesity and type-2 diabetes are unintended consequences that can also be attributed to this diet. Evidence for this concern is that the rates of these diseases have increased through the years as this diet became more widely used. By 1998 heart disease was a leading cause of death, accounting for about 31 percent of total U.S. deaths.⁶

Proof that these increases are related to the DGA can be found in the increasing volume of scientific papers in epidemiologic, biochemical, and nutrition journals that confirm the existence of a cause/effect relationship, and explain its basis.⁷ In an artful *mea culpa*, some prominent members of the nutrition community even acknowledge that the Pyramid has failed:⁸

By promoting consumption of complex carbohydrates and eschewing all fats and oils, the Pyramid provides misleading guidance. In short, not all fats are unhealthful, and by no means are all complex carbohydrates healthful.

If revisions to the DGA due in 2005[°] are to correct these past errors and provide nutritional guidance that will prevent chronic nutritional diseases in the future, objective examination of the evidence is essential.

The Origin in Flawed Science

Ancel Keys is credited with authorship of the lipid hypothesis that introduced the era of fat phobia. In 1953, Keys published an analysis of data from six countries that showed a direct, almost straight-line correlation between mortality from coronary heart disease (CHD) and percentage of calories from dietary fat. These data showed Japan as the lowest point, with less than 10 percent fat calories and less than 1 in 1,000 CHD mortality, and the U.S. as the highest point, with 40 percent fat calories and CHD mortality of 7 in 1,000.¹

Keys, however, did not include all the data available to him at the time in constructing his nearly straight-line correlation. Ravnskov shows that data for CHD and percentage of calories from dietary fat were available for 22 countries at the time Keys published his analysis. If data from all 22 countries had been used, there would have been no straight line, and no reasonable correlation would have been possible.¹⁰ Keys also ignored a wellestablished epidemiologic principle in claiming that his straightline association between CHD and dietary fat proved a causal relationship. The fact is that statistical associations, no matter how strong, do not prove cause and effect. To be proven, such associations require verification, as by a carefully controlled human feeding study.¹¹

Despite the many exceptions that existed to his hypothesis, and the criticism of his methodology by contemporaries, Keys persevered and made validating the lipid hypothesis his life's work. Other investigators followed, with the result that many epidemiologic studies that supported the lipid hypothesis appeared in the nutrition literature. Animal studies, such as those of Kritchevsky,¹² which showed formation of fatty deposits in the arteries of rabbits that were fed saturated fat and cholesterol, bolstered the lipid hypothesis–even though it was well known that rabbits, being obligate vegetarians, are extremely sensitive to dietary cholesterol.

In 1977, Krehl published a summary of a number of epidemiologic studies, including those of Keys, that demonstrated a statistical association between dietary fats and heart disease.¹³ More than a few of the investigators failed to collect data on dietary

carbohydrate content,⁷ apparently assuming at the outset that sugars and starch could not possibly be implicated. Krehl's conclusion that saturated fat and cholesterol were significant contributors to cardiovascular diseases also ignored conflicting evidence from papers discussed in the body of his article.⁷ For example, Kannel stated that it was difficult to pinpoint what, if any, dietary factor was responsible for high cholesterol levels found in the Framingham Study.¹⁴ Krehl apparently discounted the few studies that did find a link with carbohydrates in the diet. One such report was that of Yudkin, who found a strong association between sucrose consumption and cardiovascular disease.¹⁵

Many of the studies that followed Keys also ignored critical variables–such as carbohydrate consumption–and disregarded the metabolic pathways of macronutrients.⁷

The Errors in the DGA

A Poverty of Protein

Unarguably, protein and its component amino acids are required for the formation and constant maintenance, replacement, and repair of essentially all structural elements, transport systems, and control mechanisms of the body. It's unclear why the DGA considers protein the least important of the macronutrients, recommending only 17 percent of calories from protein as opposed to 30 and 55 percent, respectively, for fat and carbohydrate.⁵ Although many studies have implicated dietary protein in a variety of ills such as heart disease, cancer, osteoporosis, kidney disease, and allergies, at least as many studies have shown that protein does not have these effects. Also, the anthropological and historical research of early nutritionists demonstrates that very high protein diets are not harmful. Ethnic populations such as the Greenland Eskimos¹⁶ existed in good health on diets high in protein and fat for generations before extensive contact with the outside world. Arctic explorers Stefansson and Anderson lived for many years on diets composed almost solely of animal fat and protein, without any evidence of harm.17

Willett and Stampfer⁸ give the most plausible, and probably least defensible reason why the macronutrient that is the most important biochemically is considered least important nutritionally: "Nutritionists did not want to suggest eating more protein, because many of the sources of protein (red meat, for example) are also heavy in saturated fat."

The matter of protein intake requires serious reevaluation. A review of the published literature suggests that a healthy protein requirement is 30 percent of total calories.⁷

Animal proteins are more efficient biologically than are vegetable proteins, because food animals have selected from their vegetable diets and incorporated into their own meat and milk the proper number and ratio of essential amino acids that humans require. Yet the DGA favors vegetable proteins.

<u>Fat Phobia</u>

The pervasive fat/saturated fat phobia in the DGA prevents a reasonable and balanced intake of dietary lipids and leads to a critical deficiency of saturated fats, an unnecessary limitation on dietary cholesterol, and an unhealthful ratio of omega-6 to omega-3 essential fatty acids.

Limitations on dietary fats and cholesterol are based on the claim that these lipid components increase blood cholesterol, and thus are risk factors for cardiovascular disease. Even if the fallacious cholesterol-heart disease connection is accepted, dietary cholesterol is not a significant factor in raising blood cholesterol.¹⁰ This is because human biochemistry is remarkably frugal. If nutrients are provided in the diet, the body will use them rather than expend the energy to make them. Thus, the quantity of cholesterol biosynthesized is decreased by the amount provided by the diet. In an extensive review of the literature, Stehbins reported that animals vary widely in their susceptibility to dietary cholesterol. Rabbits show increases in serum cholesterol of up to 3,000 percent, other species lesser amounts, and humans very little increase at all, with excess dietary intake.¹⁸

The Harvard group reports that the data "do not support the strong association between intake of saturated fat and risk of coronary heart disease,"¹⁹ and "do not support associations between intake of total fat, cholesterol, or specific types of fat and risk of stroke in men."²⁰

Despite the stigma conferred on them by the DGA and the Food Pyramid, saturated fats have beneficial biochemical, anatomical, and physiological effects.^{7, 21} The many functions of and requirements for saturated fats are included in an excellent review by Enig and Fallon.²² Restrictions on fat intake have physiologic consequences, including gall bladder stasis, which fosters development of gallstones, and poor absorption of fat-soluble nutrients.

Contrary to popular belief, dietary fat cannot make body fat in the absence of excess insulin stimulated by dietary high-glycemic carbohydrates.²³ With glucagon rather than insulin in control, the metabolic pathways to body fat deposition and synthesis are reversed to those that mobilize body fat and convert it to ketone bodies. Ketone formation is a normal human metabolic process that is used regularly to provide energy and preserve glucose when supplies of glucose are low, such as during hunger or periods of fasting. Ketone bodies produced in excess of energy need are excreted in breath or urine, along with their calories. This is a simplified explanation of the scientific basis for the weight-loss diet of the late Robert C. Atkins, M.D.

It is important to note that dietary ketosis and diabetic ketosis are two distinct physiologic conditions. Both are the result of excessive ketone formation in response to low blood insulin levels, but the causes for the low insulin levels are quite different. In dietary ketosis, insulin is low because a deficit of dietary glucose prevents the blood glucose level from rising sufficiently to stimulate the pancreas to produce insulin. In diabetic ketosis, insulin is low because no matter how high the blood glucose rises, the pancreas is incapable of producing insulin. Thus, dietary ketosis, rare except in extreme starvation, may be thought of as the body's signal that it needs more glucose, whereas diabetic ketosis may be thought of as the body's signal that it needs more insulin.²⁴

Ignoring the Essential Fatty Acids

The DGA does not provide the public with information and guidance on these critical fatty acid nutrients. It recommends dietary polyunsaturated fats, but it gives no further explanation of what polyunsaturated fats are, why they are so important in a nutritionally sound diet plan, or what foods provide them. This is inexcusable in that the volume of data in the nutrition and medical literature explaining the functions and requirements for these fats, better known as the essential fatty acids, is neither new nor difficult to find.

Briefly, there are two essential fatty acid families: the omega-3 family, with its terminal double bond between the third and fourth carbons from the noncarboxyl end of the chain, and the omega-6 family, with is terminal double bond between the sixth and seventh carbons. The parents of these two families are alpha-linolenic acid (ALA) and linoleic acid (LA), respectively. Both parent compounds are metabolized by the same enzyme systems through a series of elongations and desaturations that eventually yield the omega-3 and the omega-6 eicosanoids.²⁵

Eicosanoids are short-lived, hormone-like messenger biochemicals working at the cellular level that direct a wide variety of biochemical activities. The eicosanoids may be likened to a physiologic yin and yang. They balance each other with opposing effects, such as coagulation/anticoagulation, constriction/dilation, and so forth. When the omega-3 and omega-6 essential fatty acids are in proper dietary ratio, metabolic processes lead to eicosanoid balance. Lack of the proper eicosanoid balance is a critical factor in the etiology of heart disease and stroke plus a host of other medical ills.

In addition to serving as precursors of eicosanoids, ALA, LA, and their metabolites serve other important biochemical functions. For example, eicosapentaenoic acid (EPA), an omega-3 metabolite, plays a key role in balancing eicosanoid production. Another omega-3 metabolite, docosahexaenoic acid (DHA), is the most common lipid involved in the anatomy and physiology of the brain and other nervous system tissues. Arachidonic acid (AA), an omega-6 metabolite of LA, is also a key component of brain and nerve tissue. There is good evidence that a deficiency of DHA, and sometimes of AA, can have profound effects on brain development in the newborn that may lead to such neurologic problems as attention-deficit hyperactivity disorder (ADHD).²⁶

How much omega-6 and omega-3 should be provided by a healthful dietary plan? The DGA recommendation of 14 to 25 grams daily of polyunsaturated fats (8 percent of calories) is adequate; however, no advice is given on how to apportion the total.⁵ Suggestions for a healthful ratio of LA to ALA vary from 4:1 down to 1:1 or less.^{25,27-29} The importance of the ratio was underscored by Yam; Israelis have the highest incidence of cardiovascular disease, obesity, type-2 diabetes, and cancer in the world and the highest omega-6 to omega-3 ratio. The ratio in Israel is between 20:1 and 32:1, whereas the ratio ranges from 12:1 to 20:1 in America and Europe.³⁰

The consensus is that a safe and adequate amount of ALA is from 2 to 4 grams.²⁹ Thus, the LA requirement, assuming a 4:1 ratio, would be 8 to 16 grams. Further, Simopoulos recommends that people who seldom or never eat fish take at least 10 grams of ALA a day because, in the absence of fish in the diet, that amount of ALA is required for the body to make the amount of DHA it needs.²⁸

Food sources of LA are plentiful. It occurs in almost all foods, animal and vegetable, with greatest amounts in vegetable seed oils. AA, an omega-6 essential fatty acid, occurs in abundance in meat and eggs. By contrast, ALA is difficult to obtain in adequate amounts. Small amounts, in a roughly 4:1 ratio of LA to ALA, are present in animal fats and green vegetables, but, with the exception of virgin olive oil, the vegetable oils available commercially have had most or all or their ALA component removed to prevent rancidity and preserve shelf life.²¹ A rich vegetable source of ALA is flax oil.

The lack of guidance in the DGA on specific essential fatty acid requirements is regrettable, but even more so is the lack of information about the importance of EPA and DHA and their dietary sources. DHA is provided primarily by coldwater fish and their oils, yet fish plays only a minor role in the Pyramid. For example, Figure 1 shows fish as only one of six protein foods in the meat group.

Carbohydrates-An Unhealthy Excess

Despite the serious implications of the restrictions on dietary fat in the DGA, probably even more egregious is the fact that it requires 55-60 percent of calories from carbohydrate foods, with more than half supplied by breads and flour products, cereals, and other refined grain foods. To add further insult to this carbohydrate injury, an additional 8 percent of calories from sugar may be added to supply any deficit in calories from the other food groups.⁵ These are high-glycemic carbohydrates.

It is a biochemical fact that no carbohydrate is essential for human nutrition. The body's metabolism can make all of the glucose it needs from proteins, and it can obtain all of the energy normally supplied by glucose from fats. Although not essential, small amounts of carbohydrate are of benefit to conserve protein by eliminating the need for the body to use protein to make glucose.

The importance of carbohydrate foods is not their carbohydrate content, but that they are vehicles of a wide variety of other nutrients that are essential—the vitamins, mineral, and other trace nutrients. They are also valuable sources of fiber. Thus, the recommendations in the guidelines for inclusion of abundant fruits and vegetables, the best carbohydrate sources of trace nutrients, is highly appropriate in a healthful dietary program. Nearly all of these are low-glycemic carbohydrates.

The recommended 6 to 11 servings a day of bread, rice, cereal, and pasta (Figure 1) are not only nutritionally unnecessary, but unwise. Unlike fruits and vegetables, these foods are rapidly converted to glucose. The unfortunate result of this recommendation is a carbohydrate burden that encourages stimulation of excess insulin, which in turn directs excess calories to synthesis of body fat and cholesterol. Excess insulin also interferes in essential fatty acid metabolism.²⁷

Synthesis of body fat and cholesterol is not the only damage from excess insulin. For example, Schwarzbein, in writing about the fallacy of exercising strenuously to justify the overeating of carbohydrates, states:³¹

[I]f you eat a bowl of pasta to "carbo-load" before exercising, you can burn off the excess sugar as energy but you cannot burn off the excess insulin that has been secreted to match the high sugar. Once insulin levels increase to higher than normal levels, damage begins to occur in your metabolism.

Although long suspected, the relationship of the obesity epidemic to excess carbohydrate was confirmed in a recent study published by the Centers for Disease Control and Prevention.³² The CDC reported that, during the period from 1971 to 2000, the prevalence of obesity in Americans increased from 15 to 31 percent, as average carbohydrate consumption increased by

approximately 300 calories per day. As a percentage of total dietary consumption, this represented an increase from 42 to 49 percent for men, and 45 to 52 percent for women. Protein calories remained fairly constant at about 15 percent, and fat calories decreased from about 36 to 32 percent.

Changes in the 2005 DGA and Food Pyramid

In the current proposals prepared by the Center for Nutrition Policy and Promotion (CNPP), the nutrition policy-setting arm of the USDA,⁹ the low-fat, high-carbohydrate philosophy will continue unmodified. Proposed changes are primarily related to the process of menu planning.³³

The process detailed in five tables is extremely complex and will require a professional nutritionist of exceptional skill to navigate a maze of minutiae. For example, the current DGA is based on three energy (calorie) levels.⁵ Each of the three levels is further divided into four age/gender subgroups, a total of 12 groups. The proposed 2005 DGA has 12 energy levels, ten of which have two subgroups. Ignoring the subgroups and considering only the 12 energy levels, each is then applied to 48 age/activity/gender subgroups (eight age groups X three activity levels X two gender groups), a total of 576 groups.

There probably will be some cosmetic changes in the new Pyramid, but because of size limitations, it will not be amenable to conveying the complexities that underlie the changes proposed in the DGA.

It seems apparent that the CNPP attributes epidemics of obesity and related chronic disease to poor implementation of the DGA, owing to the simplicity of the instructions, rather than to any fundamental flaws in concept. Thus, the uncontrolled national dietary experiment will continue virtually unchanged, and a different result is not to be expected.

A Serious Omission in the 2005 DGA

The Office of Management and Budget requested that the 2005 DGA emphasize the benefits of reducing foods high in trans fats.³⁴ CNPP summarily rejected OMB's request as follows, stating that: "An intake goal for trans fats was not set because no quantified standard is provided in the Dietary Reference Intakes or the Dietary Guidelines. In addition, data on the current amount of trans fats in many food items are not available." The CNPP statement further indicates that it intends only to inform the public that consideration should be given to limiting intake of trans fats.⁹

This answer to an important and valid public health request is irresponsible. The consumer deserves more. There are considerable data in the scientific literature describing adverse health effects of trans fatty acids. Further, there are many studies, including those of Enig,²¹ that belie the contention that data on trans fats in foods are insufficient. Even if specific information about trans fat content is not available for all products, enough is known about the kinds of products that contain trans fats to provide consumers with simple but valuable guidance to help them protect their health.

The major source of trans fats in foods is the hydrogenation process, which is used primarily to extend shelf life of fats and oils derived from vegetable seeds, by eliminating the highly unsaturated fatty acids (omega-3) responsible for rancidity. Thus, consumers can and should be given the very simple advice for avoiding trans fats, which is to read all labels and not buy any product whose list of contents include such words as "partially hydrogenated" or "vegetable shortening."

Conclusion

It is no secret that the lipid hypothesis, now dogma, is facing a serious challenge. America's long dietary experiment with the lowfat, high-carbohydrate diet has failed. Today, there is little doubt that there is a clear temporal association between the "hearthealthy" diet and the current, growing epidemics of cardiovascular disease, obesity, and type-2 diabetes. Many scientific papers and books support this association and explain exactly how and why the low-fat, high-carbohydrate diet causes these diseases.

Long-held beliefs that animal fat is the cause of cardiovascular disease and that grain products are the staff of life will not be relinquished without a struggle. The articles and comments widely circulated in the public press, exemplified by the denigration of the "low carb" diet and its author, the late Dr. Atkins, are evidence of this struggle.

Hope for a solution may well lie with physicians and nutritionists schooled in the biochemistry of nutrient metabolism and open to revisiting past dogmas. As Dr. Sylvan Lee Weinberg, past president of the American College of Cardiology, states in his insightful and courageous critique of the validity of the dietheart hypothesis:³⁵

Defense of the LF-Hcarb[low-fat, high-carbohydrate] diet, because it conforms to current traditional dietary recommendations by appealing to the authority of its prestigious medical and institutional sponsors, or by ignoring an increasingly critical medical literature, is no longer tenable.

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